Volume 3 Datasheets – Chemical and physical determinands

Part 2.2: Organic chemicals

2019

Part 2.2:  
Organic determinands

### Notes

1. Part 2.2 includes datasheets for those organic determinands with a MAV, and those without a MAV but which may have health concerns. The *Drinking-water Standards for New Zealand* (DWSNZ) define a MAV as the concentration of a determinand, below which the presence of the determinand does not result in any significant risk to a consumer over a lifetime of consumption. For carcinogenic chemicals, the MAVs set in the DWSNZ generally represent a risk of one additional incidence of cancer per 100,000 people ingesting the water at the concentration of the MAV for a lifetime of 70 years.

2. The DWSNZ define a Guideline Value (GV) as the value for an aesthetic determinand that, if exceeded, may render the water unattractive to consumers. This usually involves taste and/or odour. Datasheets for determinands with a GV but no MAV appear in Part 2.4. However, organic determinands that have been reported to have given rise to aesthetic problems, but do not have a GV, appear in this Part.

3. The World Health Organization (WHO) states that their guideline values normally represent the concentration of a constituent that does not result in any significant risk to health over a lifetime of consumption.

4. Some datasheets include the minimal risk levels (MRLs) developed by the US Department of Health and Human Services, Agency for Toxic Substances & Disease Registry (ATSDR). The latest values can be found at <http://www.atsdr.cdc.gov/mrls/mrls_list.html>, or find them via <http://www.atsdr.cdc.gov/toxprofiles/index.asp>. The MRLs in these Guidelines are up-to-date to July 2013, which is the latest version as at November 2014. For a definition of MRLs, see Section 10.2.5.1 in Chapter 10 of the Guidelines. Other expressions explained are ADI, TDI, RfD, NOAEL, LOAEL and UF.

5. Some datasheets include the USEPA’s MCL or Maximum Contaminant Level which is the maximum permissible level of a contaminant in water which is delivered to any user of a public water system in the US. The US has secondary standards (non-enforceable), based on cosmetic or aesthetic effects.

Some datasheets also include the reference dose or RfD, which the USEPA defines as “an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime”. This is a chronic RfD or sometimes written cRfD, as opposed to acute RfDs.

The US also use the concept of Drinking Water Equivalent Level or DWEL, which is defined as “a lifetime exposure concentration protective of adverse, non-cancer health effects, that assumes all of the exposure to a contaminant is from drinking water.

The USEPA use health advisories too, which are an estimate of acceptable drinking water levels for a chemical substance based on health effects’ information; a Health Advisory is not a legally enforceable Federal standard, but serves as technical guidance to assist Federal, State, and local officials. Health Advisories contain a margin of safety to protect sensitive members of the population.

**One-Day HA:** The concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for up to one day of exposure. The One-Day HA is normally designed to protect a 10‑kg child consuming one litre of water per day.

**Ten-Day HA:** The concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for up to ten days of exposure. The Ten-Day HA is also normally designed to protect a 10‑kg child consuming one litre of water per day.

**Lifetime HA:** The concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure. The Lifetime HA is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The USEPA Health Advisory (HA) evaluation of carcinogenic potential includes their classification for the weight of evidence of the likelihood that the agent is a human carcinogen and the conditions under which the carcinogenic effects may be expressed, as well as quantitative estimates of cancer potency (slope factor) where available. The cancer slope factor is the result of the application of a low-dose extrapolation procedure and is presented as the risk per mg/kg/day. The HA includes the drinking water concentration equivalent to cancer risks of 1 in 10,000 (10-4), 1 in 100,000 (10-5), to 1 in 1,000,000 (10-6). See: <http://www.epa.gov/sites/production/files/2014-09/documents/drinking_water_health_advisory_for_24_and_26_dinitrotoluene.pdf>.

The USEPA review their MCLs, DWELs and RfDs regularly. The 2012 values can be found at: <http://water.epa.gov/action/advisories/drinking/upload/dwstandards2012.pdf>.

6. Some chemicals that are not listed in the index are discussed in this Part. For example, the polyaromatic hydrocarbons (PAH), PCBs, halohydrins and perfluoro compounds datasheets refer to a large number of individual chemicals. So if information about an organic chemical that is not listed in the index is required, go to Edit/Find and use the name of the chemical, or its CAS Number. Also check that it is not covered in the pesticides or Aesthetic Determinands Parts.

7. The 2008 ARC publication *Literature Review of Organic Chemicals of Emerging Environmental Concern in Use in Auckland* includes some good introductory discussion on the tools used for assessing health aspects of substances in use in New Zealand. Auckland Regional Council Technical Report No. 028. 193 pp. Available at: <http://www.aucklandcity.govt.nz/council/documents/technicalpublications/TR2008-028%20-%20Literature%20Review%20of%20Organic%20Chemicals%20of%20Emerging%20Environmental%20Concern%20in%20Use%20in%20Auckland.pdf>.

8. Drinking water standards in England and Wales are now set out in European and UK legislation. They are called Prescribed Concentrations or Values (PCVs) and many are different from WHO’s Guideline Values. See: DWI. 2010. The Water Supply (Water Quality) Regulations 2010. Water, England and Wales. No. 994 (W.99). 42 pp. <http://dwi.defra.gov.uk/stakeholders/legislation/wsr2010wales.pdf>.

9. Some generic properties describe the likely environmental fate of various compounds. For example:

* low log Kow (-ve to 3, unlikely to sorb to organic matter and likely to remain in water column)
* moderate log Kow (3 to 5, likely to moderately sorb to organic matter)
* high log Kow (>5, very likely to sorb to organic matter.

Non-volatile (Henry’s Law constant of <1 x 10-7). Henry’s Law constant or coefficient (sometimes called the air-water partition coefficient) is an equilibrium partitioning coefficient that describes the extent to which a chemical partitions between the aqueous and gaseous phases.

* likely to volatilise slowly (Henry’s Law constant of 1 x10-7 to 1 x10-6)
* volatile (Henry’s Law constant of >1 x10-6).

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# Acenaphthene

Acenaphthene, CAS No. 83-32-9, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. Also called 1,2-dihydroacenaphthylene, 1,8‑dihydroacenaphthylene and ethylenenaphthalene.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

Acenaphthene is a component of crude oil and a product of combustion which may be produced and released to the environment during natural fires. Emissions from petroleum refining, coal tar distillation, coal combustion and diesel fuelled engines are major contributors of acenaphthene to the environment. Acenaphthene is used as a chemical intermediate and may be released to the environment via manufacturing effluents and the disposal of manufacturing waste by‑products. Because of the widespread use of acenaphthene in a variety of products, acenaphthene may also be released to the environment through landfills, municipal wastewater treatment facilities and waste incinerators.

Acenaphthene is used on a large scale to prepare naphthalic anhydride, in the manufacture of dyes, pharmaceuticals and plastics, and as an insecticide and fungicide.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

Acenaphthene should biodegrade rapidly in the environment. The reported biodegradation half-lifes for acenaphthene in aerobic soil and surface waters range from 10 to 60 days and 1 to 25 days, respectively. However, acenaphthene may persist under anaerobic conditions or at high concentration due to toxicity to micro-organisms. Acenaphthene is not expected to hydrolyse or bioconcentrate in the environment; yet, it should undergo direct photolysis in sunlit environmental media. A calculated Koc range of 2065 to 3230 indicates acenaphthene will be slightly mobile in soil. In aquatic systems, acenaphthene can partition from the water column to organic matter contained in sediments and suspended solids. A Henry’s Law constant of 1.55 x 10-4 atm‑cu m/mole at 25°C suggests volatilisation of acenaphthene from environmental waters may be important. The volatilisation half-lifes from a model river and a model pond, the latter considers the effect of adsorption, have been estimated to be 11 hours and 39 days, respectively (EAWAG accessed February 2015).

Water solubility is about 4 mg/L.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 and 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. Acenaphthene was not detected. A number of PAHs have been assessed in Phase 1 of the P2 Programme. With the exception of fluoranthene, none have been detected. The limits of detection range from 0.0001 to 0.0002 mg/L.

Five water utilities in the US reported detecting acenaphthene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0037 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation (ATSDR 1995).

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

IARC (2010) classified acenaphthene in Group 3 (not classifiable as to carcinogenicity). The US Environmental Protection Agency has determined that acenaphthene is classifiable as to human carcinogenicity based on no human data and inadequate data from animal bioassays.

The USEPA has a reference dose or RfD of 0.06 mg/L and a Drinking Water Equivalent Level or DWEL of 2 mg/L for acenaphthene.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limits (exposure greater than 10 percent of a lifetime) for acenaphthene is 0.4 mg/L.

ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) has developed oral minimal risk levels (MRLs) for some PAHs:

|  |  |  |
| --- | --- | --- |
| **PAH** | **mg/kg/day** | **duration** |
| acenaphthene | 0.6 | intermediate (15–364 days) |

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA established an organoleptic effect criterion of 0.02 mg/L for acenaphthene. Source: [*Quality Criteria for Water*, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

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USEPA. *National Primary Drinking Water Regulations, Technical Factsheet on: Polynuclear aromatic hydrocarbons (PAHs)*. <http://www.epa.gov/osw/hazard/wastemin/minimize/factshts/acnphthe.pdf>.

WHO. 2011. *Guidelines for Drinking-water Quality* 2011 (4th edition). Geneva: World Health Organization. Available at: [http://www.who.int/water\_sanitation\_health/publications/drinking-water-quality-guidelines-4-including-1st-addendum/en/index.html](http://www.who.int/water_sanitation_health/publications/2011/dwq_guidelines/en/index.html).

# Acenaphthylene

Acenaphthylene, CAS No. 208-96-8, is one of the 17 polynuclear aromatic hydrocarbons (polyaromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the datasheet for polynuclear aromatic hydrocarbons. Also called cyclopenta(de)naphthalene.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

Acenaphthylene comprises about 2 percent of coal tar. Acenaphthylene is a component of crude oil, coal tar and a product of combustion which may be produced and released to the environment during natural fires. Emissions from petroleum refining and coal tar distillation are major contributors of acenaphthylene to the environment. Acenaphthylene is contained in a variety of coal tar products and may be released to the environment via manufacturing effluents and the disposal of manufacturing waste by‑products.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

The reported biodegradation half-lifes for acenaphthylene in aerobic soil range from 12 to 121 days. Acenaphthylene is not expected to hydrolyse or bioconcentrate in the environment; yet, may undergo direct photolysis in sunlit environmental media. A calculated Koc range of 950 to 3315 indicates acenaphthylene will have a low to slight mobility class in soil. In aquatic systems, acenaphthylene may partition from the water column to organic matter contained in sediments and suspended solids. A Henry’s Law constant of 1.13 x 10-5 atm‑cu m/mole at 25°C suggests volatilisation of acenaphthylene from environmental waters may be important. The volatilisation half-lifes from a model river and a model pond, the later considers the effect of adsorption, have been estimated to be 4 and 184 days, respectively (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 and 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. Acenaphthylene was not detected. A number of PAHs have been assessed in Phase 1 of the P2 Programme. With the exception of fluoranthene, none have been detected. The limits of detection range from 0.0001 to 0.0002 mg/L.

Four water utilities in the US reported detecting acenaphthylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00076 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation (ATSDR 1995).

### Analytical methods

Refer to the datasheet for polynuclear aromatic hydrocarbons.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons. Classification – D; not classifiable as to human carcinogenicity (USEPA 1991).

### Derivation of Maximum Acceptable Value

No MAV.

### References

ATSDR. 1995. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs)*. Atlanta, GA: Agency for Toxic Substances & Disease Registry, Department of Health and Human Services. See: <http://www.atsdr.cdc.gov/toxprofiles/index.asp>.

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# Acetaldehyde

CAS No.75-07-0. Also called ethanal, acetic aldehyde or ethylaldehyde.

### Maximum Acceptable Value

Acetaldehyde is not discussed in the WHO Guidelines, and does not have a MAV in the DWSNZ.

The USEPA concluded on 22 September 2009 that acetaldehyde is known or anticipated to occur in PWSs and may require regulation. Therefore they added acetaldehyde to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

Acetaldehyde occurs naturally in ripe [fruit](http://en.wikipedia.org/wiki/Fruit), [coffee](http://en.wikipedia.org/wiki/Coffee), and fresh [bread](http://en.wikipedia.org/wiki/Bread), and is produced by [plants](http://en.wikipedia.org/wiki/Plant) as part of their normal [metabolism](http://en.wikipedia.org/wiki/Metabolism).

The largest use of acetaldehyde is as a chemical intermediate for the production of acetic acid. The production of esters, principally ethyl acetate and isobutyl acetate, is the second most significant use. It is also used in flavourings, foods, beverages, perfumes, plastics, aniline dyes, synthetic rubber manufacturing, the silvering of mirrors, gelatin fibre hardening, and in the laboratory.

Acetaldehyde is a component of cigarette smoke, and is also released to the environment from the combustion and photo-oxidation of hydrocarbons. It has been detected at low levels in drinking-water, surface water, rainwater, effluents, and ambient and indoor air samples. Trucks burning diesel have been reported to produce 0.5 to 15 mg of acetaldehyde per km (Environment Australia 2003). It is also photochemically produced in surface water. Acetaldehyde is the breakdown product of the pesticide metaldehyde (qv).

#### 2. From treatment processes

Low molecular weight aldehydes such as formaldehyde and acetaldehyde have been detected as by‑products of ozonation.

### Forms and fate in the environment

If released to water, acetaldehyde will rapidly biodegrade or volatilise (for a typical river, the half-life is 9.3 hours). Reported half-lifes of acetaldehyde in water and air are 1.9 hours.

If released to soil, acetaldehyde is expected to have very high mobility based upon an estimated Koc of 16. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 6.67 x 10-5 atm‑cu m/mole. Acetaldehyde may volatilise from dry soil surfaces based upon its vapour pressure. If released into water, acetaldehyde is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is expected to be an important fate process based upon its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 11 hours and 5.3 days, respectively. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

Fully miscible with water.

### Typical concentrations in drinking-water

In water, concentrations are generally less than 0.0001 mg/L; therefore, contribution from drinking water is considered negligible. 18 water utilities in the US reported detecting acetaldhyde in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0047 mg/L.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Analysis of a wide range of foodstuffs in the Netherlands showed that concentrations were generally less than 1 mg/kg (1 ppm), but, occasionally, they ranged up to several 100 mg/kg, particularly in some fruit juices and vinegar.

The principal source of exposure to acetaldehyde for the majority of the general population is through the metabolism of alcohol. Cigarette smoke is also a significant source of exposure. With respect to other media, the general population is exposed to acetaldehyde mainly from food and beverages and, to a lesser extent, from air. The amounts contributed to total intake from drinking-water are negligible.

The main source for humans is by inhalation (USEPA 1988). Acetaldehyde is the major metabolite of ethanol. Many of the adverse effects of ethanol (eg, hangover) are attributed to acetaldehyde. Direct administration of acetaldehyde to rats has established alcohol dependency.

A variety of aldehydes have been tested for carcinogenic activity. By the inhalation route formaldehyde and, to a much lesser extent, acetaldehyde are carcinogenic, whereas isobutyraldehyde is not carcinogenic even at doses that cause irritation to the respiratory tract. Numerous aldehydes have been tested for mutagenic activity. In general, only short-chain aldehydes (eg, formaldehyde, acetaldehyde) have been clearly shown to be mutagenic.

A NOEL of 125 mg/kg bw per day was reported for acetaldehyde added to the drinking-water of male and female rats for four weeks at level of 0, 25, 125 or 625 mg/kg bw per day; the only treatment-related effect was hyperkeratosis of the forestomach at 625 mg/kg bw per day. No adverse effects were seen when acetaldehyde in drinking-water at a daily intake level of 0.5 mg/kg bw was given to rats (from IPCS 1998).

Based on the carcinogenicity of acetaldehyde in animals, the USEPA concluded that acetaldehyde is a probable human carcinogen (B2). IARC has also concluded that there is sufficient evidence for the carcinogenicity of acetaldehyde to experimental animals and inadequate evidence for its carcinogenicity to humans, resulting in an classification of 2B, ie, “possibly carcinogenic to humans.” This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Acetamide

CAS No. 60-35-5. Also called ethanamide, acetic acid amide and occasionally methanecarboxamide.

### Maximum Acceptable Value

Acetamide is not discussed in the WHO Guidelines, and does not have a MAV in the DWSNZ.

The USEPA concluded on 22 September 2009 that acetamide is known or anticipated to occur in PWSs and may require regulation. Therefore they added acetamide to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

Acetamide is used as a solvent, [plasticiser](http://en.wikipedia.org/wiki/Plasticizer) and in the synthesis of many other organic compounds. Acetamide can be used intravenously as an antidote for dogs suspected of being poisoned by 1080. If present in wine, acetamide will cause an unpleasant off-flavour usually referred to as “mousey”. It is produced by some of the spoilage bacteria.

#### 2. From the distribution system

Chlorination of amino acids yields haloacetonitriles, which hydrolyse to form haloacetamides (qv).

### Forms and fate in the environment

If released to soil, acetamide is expected to have very high mobility based upon an estimated Koc of 5. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 1.1 x 10-8 atm‑cu m/mole. If released into water, acetamide is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. In water, acetamide reached 1.2 percent, 3.3 percent, and 12.0 percent of its theoretical BOD in 6 hours, 12 hours and 24 hours, respectively, using an activated sludge inoculum, suggesting that this compound may biodegrade in the environment. Volatilisation from water surfaces is not expected to be an important fate process based upon its estimated Henry’s Law constant. Chemical hydrolysis studies demonstrate that acetamide will not hydrolyse under environmental conditions. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low (EAWAG accessed February 2015).

The degradation of the acetamide herbicides acetochlor, alachlor, dimethenamid, flufenacet, and metolachlor do not appear to produce significant quantities of acetamide, their more commonly found degradation products include ethanesulfonic acid, oxanilic acid, and sulfinyl acetic acid.

Highly soluble in water.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Acetamide has been found to cause [cancer](http://en.wikipedia.org/wiki/Cancer) in laboratory animals. Acetamide produced benign and malignant liver tumours in rats following its oral administration, and in male mice, an increased incidence of malignant lymphomas was also observed, therefore IARC classified acetamide in Group 2B “possibly carcinogenic to humans”. The USEPA has not established an RfD for acetamide.

Acetamide, a suspected oncogen, is a minor metabolite of methomyl. Acetamide appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Acetonitrile

CAS No. 75-05-8. Also called methyl cyanide, cyanomethane, ethanenitrile, methanecarbonitrile and ethylnitrile.

### Maximum Acceptable Value

Acetonitrile is not discussed in the WHO Guidelines, and does not have a MAV in the DWSNZ.

### Sources to drinking-water

#### 1. To source waters

Acetonitrile is used primarily as an extraction solvent for butadiene; as a chemical intermediate in pesticide manufacturing; and as a solvent for both inorganic and organic compounds. Also used as a starting material for the production of acetophenone (1-phenylethanone), alpha-naphthalenacetic acid, thiamine, and acetamidine; to remove tars, phenols, and colouring matter from petroleum hydrocarbons not soluble in acetonitrile; in the production of acrylic fibres; and in pharmaceuticals (major use), perfumes, nitrile rubber, and ABS (acrylonitrile-butadiene-styrene) resins.

EU (2002) reports that another important source in some areas results from biomass burning, and that exhaust gases from gasoline-powered engines contain 1.32 percent by mass acetonitrile (pre catalytic converters).

Acetonitrile has been used in formulations for [nail polish remover](http://en.wikipedia.org/wiki/Nail_polish_remover) has been banned in cosmetic products in the [European Economic Area](http://en.wikipedia.org/wiki/European_Economic_Area) since March 2000. It is also a fairly common reagent in analytical laboratories.

Acetonitrile occurs naturally in coal tar and cigarette smoke.

### Forms and fate in the environment

Acetonitrile is expected to adsorb weakly to soils as predicted by its KOC value; removal occurs primarily by volatilisation and leaching into groundwater.

If released to soil, acetonitrile is expected to have high mobility based upon a Koc of 120. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 3.45 x 10-5 atm‑cu m/mole. Acetonitrile may volatilise from dry soil surfaces based upon its vapour pressure of 88.8 mm Hg. Biodegradation studies of acetonitrile with mixed cultures of microorganisms from activated sludge and sewage show that degradation proceeds slowly without acclimatisation of microorganisms. If released into water, acetonitrile is not expected to adsorb to suspended solids and sediment based upon its Koc value. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 18 hours and 7 days, respectively. The biodegradability of acetonitrile was also observed with river water; the 12-day ThOD (theoretical oxygen demand) with river water was 40 percent. An estimated BCF suggests that the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions; the hydrolysis half-life in water is thousands of years (EAWAG accessed February 2015).

EU (2002) reports a vapour pressure of 98.64 hPa at 25°C, a Henry’s law constant of 2.91 Pa m3/mol, and a partition coefficient = logPow = -0.34. Acetonitrile is 100 percent biodegraded in acclimated river water in four days.

The water solubility (miscible) of acetonitrile suggests that dissolution into clouds and raindrops may occur, leading to possible removal in rainfall.

Acetonitrile is removed from soil by microbial degradation; evaporation and leaching are also important.

If released into water, acetonitrile is not expected to adsorb to suspended solids and sediment based upon its Koc value. Acetonitrile is removed from water by biodegradation, with decomposition occurring about five times faster following acclimation of the microorganisms. Decomposition of the chemical in the Ohio River (0.1 to 25 mg/L) was 20 percent in five days and 40 percent in 12 days. Although slow, loss by volatilisation may become more important in shallow waters.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Acetonitrile is metabolised in the body to hydrogen cyanide and thiocyanate; these chemicals are thought to be responsible for the adverse effects of acetonitrile. Generally the onset of toxic effects is delayed, due to the time required for the body to metabolise acetonitrile to cyanide (generally about 2 to 12 hours). It is subsequently conjugated with thiosulphate to form thiocyanate, which is eliminated in urine.

Based on inhalation data obtained in 1983 the USEPA calculated an oral reference dose for acetonitrile of 0.006 mg/kg/day. This was cancelled in 1999.

USEPA has assigned carcinogen as class D, not classifiable as to human carcinogenicity.

Acetonitrile was negative for mutations in Chinese hamster ovary cells and Salmonella strains TA98, TA100, TA1535, and TA1537 when assayed with or without metabolic activation.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Acrolein

CAS No. 107-02-8. Also called acrylaldehyde, acrylic aldehyde, allyl aldehyde, ethylene aldehyde, 2-propenal and prop-2-en-1-al.

### Maximum Acceptable Value

Acrolein is not discussed in the WHO Guidelines, and does not have a MAV in the DWSNZ.

The USEPA concluded on 22 September 2009 that acrolein is known or anticipated to occur in PWSs and may require regulation. Therefore they added acrolein to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

Acrolein is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Acrolein is used to make other chemicals (mainly acrylic acid) and pesticides, and is found in some livestock feeds (with DL-methionine, an essential amino acid), and pesticides. It has previously been used as a pesticide, including weed control in waterways. Acrolein is also used to make glutaric dialdehyde which is used as a biocide and leather tanning agent. An antimicrobial product described as “2-propenoic acid polymer with 2-propenal” appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2012 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). It is used as a slimicide.

Acrolein can be formed from the breakdown of certain pollutants found in outdoor air, from burning tobacco, or from forest fires (incomplete combustion), and burning gasoline. Acrolein has been identified among the decomposition products of cellophane and polyvinyl chloride. Small amounts of acrolein may be found in some foods, such as fried foods, cooking oils, and roasted coffee. It has a pungent acrid odour.

It has been found in US domestic wastewater effluent at 0.02 to 0.2 mg/L. EU (2001) reports concentrations of acrolein in Japanese rainwater of up to 0.011 mg/L and groundwater <0.005 mg/L.

The commercial product may contain up to 0.5 percent as acetaldehyde, and 0.1 to 0.25 percent hydroquinone as an additive (EU 2001).

### Forms and fate in the environment

Acrolein is very soluble in water (at least 20 percent), and evaporates rapidly from soil and water. Acrolein is unlikely to be transported over long distances because of its high reactivity and estimated short half-lifes in air and water. It is also unlikely to partition from these compartments to soil or sediments. The overall reactivity-based half-life of acrolein in surface water is estimated to be between 30 and 100 hours. In groundwater, half-lifes of 11 days and 336 to 1344 h (14–56 days) are estimated based on aerobic and anaerobic degradation, respectively (WHO 2002).

EU (2001) states that acrolein does not contain any hydrolysable groups, but it reacts reversibly with water to form 3-hydroxypropanal. The aerobic soil half-life was measured at 29 days, main degradation products being acrylic acid and 3‑hydroxypropionic acid thence CO2, and anaerobic at 11 days, producing 1,3‑propandiol and 3-hydroxypropionic acid thence CO2. 3-Hydroxypropanol and allyl alcohol are also reported to form.

NPIC (1994) quotes for acrolein a soil half-life of 14 days, water solubility of 20.8 percent and a sorption coefficient (soil Koc) of 0.5. This resulted in a pesticide movement to groundwater rating of very high.

EU (2001) states: vapour pressure = 293 hPa at 20°C; partition coefficient = logPow = about -0.7 to +1.0. Henry’s law constant = 7.46 Pa.m3/mol at 25°C.

### Typical concentrations in drinking-water

Acrolein has not been detected in drinking-water, and is not commonly found in surface water. However, traces have been detected in rain and fog.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Indoor air is an important source of exposure, although the relative contribution of various sources therein is unknown. Considerably higher concentrations of acrolein have been reported in tobacco smoke. For the general population, the relative contribution of ambient air to overall exposure to inhaled acrolein is expected to be small, compared with exposure from indoor air. However, for populations residing in the vicinity of locations heavily impacted by vehicular exhaust, ambient air may be an important source of exposure via inhalation (WHO 2002).

The USEPA (2003) has calculated a provisional oral RfD of 0.0005 mg/kg/d for acrolein, based on a NOAEL of 0.05 mg/kg-day and an uncertainty factor of 100. The RfD is an estimate (including uncertainty) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.004 mg/kg/day for intermediate-duration oral exposure (15–364 days) to acrolein.

No information is available on the carcinogenic effects of acrolein in humans. The USEPA has classified acrolein as a Group C, possible human carcinogen, based on limited evidence of carcinogenicity in animals, the structural similarity of acrolein to substances possibly carcinogenic to humans, the carcinogenic potential of one of its metabolites, and the lack of human data. Later (in 2003) the USEPA stated data were inadequate for an assessment of human carcinogenic potential for either the oral or inhalation route of exposure. There are no adequate human studies of the carcinogenic potential of acrolein. Collectively, experimental studies provide inadequate evidence that acrolein causes cancer in laboratory animals.

EC (2009) quotes a NOAELshort-term of 4.6 mg/kg/d based on a 14-day mouse study (local irritation and mortality at higher dose levels), a NOAELmedium-term of 0.1 mg/kg/d based on a one-year dog study (local irritation at higher dose levels), and a NOAELlong‑term of 0.05 mg/kg bw/d based on a two-year rat study (mortality at higher dose levels).

The Acceptable Daily Intake (ADI) adopted in Australia is 0.0005 mg/kg body weight, with a NOEL of 0.05 mg/kg bw.

The International Agency for Research on Cancer (IARC) has determined that acrolein is not classifiable as to carcinogenicity in humans (Group 3).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Acrylamide

CAS No. 79-06-1. The IUPAC name is prop-2-enamide. Also called 2-propenamide, acrylic amide, acrylic acid amide, ethylene carboxamide, vinyl amide, propenoic acid amide, and sometimes just propenamide.

### Maximum Acceptable Value

Based on health considerations, the concentration of acrylamide in drinking-water should not exceed 0.0005 mg/L (0.5 g/L).

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of acrylamide in drinking water should not exceed 0.0002 mg/L.

USEPA (2006) states “when acrylamide is used in drinking water systems the combination (or product) of dose and monomer level shall not exceed that equivalent to a polyacrylamide polymer containing 0.05 percent monomer dosed at 1 mg/L.”

The Prescribed Concentration or Value (PCV) for acrylamide in England and Wales is 0.0001 mg/L. See Notes.

NSF Standard 60 states that the allowable concentration of acrylamide in drinking water is 0.0005 mg/L.

### Sources to drinking-water

#### 1. To source waters

Acrylamide may enter the aquatic environment as an industrial contaminant. It is used as a chemical intermediate or as a monomer in the production of a variety of high molecular weight polyacrylamides, some of which are used in water and oil treatment. Large quantities of polyacrylamide gel are produced on site for use as a grouting agent (although this has ceased in the US and EU)\*, particularly for the sealing of mineshafts in the mining industry. Polyacrylamides are used as thickeners and binders in paints and coatings, in toiletries and cosmetics, as moisture-retaining additives to concrete, and as binding agents in foundry sand. They play various roles in textile processing and in the production of adhesives, tapes and gels, including gels used for electrophoresis.

\* EU (2002) reports that N-methylolacrylamide (NMA) has replaced acrylamide grouts in some countries.

Acrylamide, a vinyl monomer, is formed catalytically from the hydration of acrylonitrile by sulphuric acid at 90 to 100°C. From the resulting sulphate solution, acrylamide is extracted by neutralisation with ammonia and subsequent cooling to isolate the crystalline monomer. It is sometimes stabilised with copper salts.

#### 2. From treatment processes

The most important source of drinking-water contamination by acrylamide is the use of polyacrylamide flocculants which are used for potable water treatment in New Zealand. Acrylamide occurs as a minor impurity in polyacrylamide polymers. A typical dose level of 1 mg/L of non-ionic or anionic polyacrylamide results in an estimated maximum acrylamide concentration of 0.0005 mg/L (0.5 g/L), with practical concentrations two to three times lower (WHO 2011). Residual levels of acrylamide from the use of cationic polyacrylamides may be higher.

The Scottish Government’s Drinking Water Quality Division List of Approved Products and Processes, December 2003 states (as do BS EN 1407:1998 and 1410:1998):

i) no batch (of polyelectrolyte) must contain more than 0.020% of free acrylamide monomer based on the active ingredient content;

(ii) the dose must average no more than 0.25 mg/L and never exceed 0.50 mg/L of the active ingredient;

(iii) an upper limit for the content of free acrylamide monomer must be stated by the supplier for every batch;

(iv) the method used for the analysis for free acrylamide monomer is entitled ‘Determination of Acrylamide’ published in the series ‘*Methods for the Examination of Waters and Associated Materials*’ by the Environment Agency.

The UK Drinking Water Inspectorate stated in Regulation 31 Letter 02/2004 “Water Supply (Water Quality) Regulations 2000, as amended, set a prescribed concentration or value (PCV) for acrylamide of 0.0001 mg/L. Compliance with the standard is to be achieved by specification of a maximum monomer concentration in the polyacrylamide product and by control of the polyacrylamide dosing concentration.

#### 3. From the distribution system

Acrylamide may enter drinking-water through the use of polyacrylamides as grouting agents in the construction of drinking-water reservoirs and wells.

### Forms and fate in the environment

Acrylamide is highly mobile in aqueous environments based on an estimated Koc of 10, and readily leachable in soil, thereby possibly entering groundwater. Its behaviour in subsurface soil, where most grout application takes place, has not been studied. Acrylamide is susceptible to biodegradation (main fate process) in soil and surface water; common end products being acrylic acid and ammonia. Non-biological hydrolysis may be important in natural water. Based on its vapour pressure volatilisation is not an important removal process. Bioconcentration is not expected to be significant. It is highly soluble in water, variously reported at about 22–40 percent solubility.

At an initial concentration of 10 mg/L, acrylamide was completely degraded in about 12 days using river water; acrylamide, at an initial concentration of 8 μg/L, was rapidly degraded following a lag period of approximately 9 days in well-aerated, sunlit river water obtained from the Thames River (ATSDR 2009).

If released to soil, acrylamide is expected to have very high mobility based upon an estimated Koc of 10. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 1.8 x 10-9 atm‑cu m/mole. Volatilisation from dry soil surfaces is not expected based on acrylamide’s vapour pressure. The nitrogen in acrylamide was recovered as inorganic nitrogen with recoveries after 3 and 14 days at 30°C ranging from 11–71 percent in Clarion soil and 74–95 percent in Canisteo soil, respectively. Results from these studies suggested that acrylamide is hydrolysed in soil under aerobic conditions to produce ammonium ion, which is then oxidised to nitrite ion and nitrate ion. If released into water, acrylamide is not expected to adsorb to suspended solids and sediment based upon the estimated Koc, which EU (2002) quotes as logPow = -1.0). In a river die-away test, 90 percent of acrylamide disappeared in approximately 150 hours. Volatilisation from water surfaces is not expected to be an important fate process based on its Henry’s Law constant. A BCF of 1 for fingerling trout suggests the potential for bioconcentration in aquatic organisms is low. The hydrolysis half-life of acrylamide has been reported as >38 years (EAWAG accessed February 2015).

USEPA (2010) states that if released to water, acrylamide is not expected to adsorb to suspended solids or sediment, based on the Koc. In a river die-away test, 90 percent of acrylamide disappeared in approximately 150 hours. The hydrolysis half-life of acrylamide has been reported as >38 years. Volatilisation of acrylamide from water surfaces is not expected, based on the compound’s Henry’s law constant. Microbial degradation of acrylamide can occur under light or dark, aerobic or anaerobic conditions.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 114 zones, found acrylamide at a concentration of 0.00012 mg/L in one zone (24 percent of the MAV), with the median concentration being “nd” (limit of detection = 0.0001 mg/L) (ESR 2001).

Public drinking water supply wells in West Virginia contained acrylamide concentrations of 0.024–041 μg/L (ATSDR 2009).

NSF (2010) reports levels from <0.00001 to 0.0004 mg/L acrylamide in drinking water that has been treated with polyacrylamide coagulant aids. The median concentration was 0.00006 mg/L.

### Removal methods

The most important source of drinking-water contamination by acrylamide is the use of polyacrylamide flocculants that contain residual levels of acrylamide monomer. Concentrations in drinking-water can be controlled by product specification.

If acrylamide is found to be present in raw water, the concentration can be decreased through ozonation or by treating the water with potassium permanganate. Acrylamide can also be removed by granular activated carbon, but is not removed effectively by powdered activated carbon. If the granular activated carbon is put at the end of the treatment process, acrylamide originating either in the source water or through the use of polyacrylamide flocculants for water treatment will be removed.

Little is known about the rate at which acrylamide leaches from grouting agents in the reticulation.

### Analytical methods

#### Referee method

HPLC/UVD. Determination of acrylamide monomer in waters and polymers, 1987 (HMSO 1988). Subsequently the DWI (2004) has stated “The new method is based on derivatisation of acrylamide monomer to form a dibromo-derivative, which is extracted from water samples using solvent extraction with ethyl acetate. Following concentration of the solvent extract, an aliquot is analysed using gas chromatography-mass spectrometry (GCMS). The method of analysis has a limit of detection of 0.000027 mg/L and it has been shown that the performance of the method is satisfactory over the range 0.00–0.50 mg/L.”

#### Some alternative methods

No alternative methods have been recommended for acrylamide because no methods meet the required criteria.

### Health considerations

Acrylamide is absorbed readily through the skin and following ingestion or inhalation. Absorbed acrylamide is distributed in body fluids and crosses the placental barrier. Acrylamide and its metabolites are accumulated in both nervous system tissues and blood as well as in liver, kidney and the male reproductive system. It is largely excreted as metabolites in urine and bile.

Acrylamide is well-established as a cumulative neurotoxin.

Humans exposed for a short time to groundwater contaminated with up to 400 mg/L of acrylamide showed effects including confusion, disorientation, memory disturbances and hallucinations. They recovered fully within 4 months.

Many other cases of human exposure to acrylamide have been reported, generally dealing with dermal or inhalation exposure of workers in grouting operations or factories manufacturing acrylamide-based flocculants. Typical clinical symptoms were skin irritation, generalised fatigue, foot weakness and sensory changes, which reflect dysfunction of either the central or peripheral nervous system.

[Browning](http://en.wikipedia.org/wiki/Maillard_reaction) during [baking](http://en.wikipedia.org/wiki/Baking), [frying](http://en.wikipedia.org/wiki/Frying) or [deep-frying](http://en.wikipedia.org/wiki/Deep-frying) (especially potatoes) will produce acrylamide, and over-cooking foods may produce large amounts of acrylamide. Carbohydrate-rich foods typically contain the highest levels of acrylamide. It has also been found in coffee. Acrylamides can also be created during [microwaving](http://en.wikipedia.org/wiki/Microwaving) (USFDA 2006).

USEPA (2010) reports that acrylamide is a component of cigarette smoke, and the acrylamide content in mainstream cigarette smoke has been estimated at 1.1–2.34 μg per cigarette. The acrylamide and acrylamide metabolites have been measured in human urine, finding median levels in smokers about four times higher than in non-smokers, indicating that cigarette smoke is clearly an important source of acrylamide exposure.

It is estimated that 40 percent of the acrylamide in the New Zealand diet is contributed by potato crisps and chips (NZFSA 2006). The major foods contributing to dietary acrylamide exposure in New Zealand were potato products, bread, breakfast cereals and beverages. This is largely consistent with international findings. The contribution of potato crisps to acrylamide exposure appears to have decreased between 2006 and 2011 and the contribution from potato, hot chips and oven baked/roasted potatoes appears to have increased. Mean estimates of dietary acrylamide exposure (0.72–1.04 μg/kg bw/day for adults) were very similar to estimates made in 2006 (0.74–0.99 μg/kg bw/day for adults) (MAF 2012).

The reference dose or RfD (USEPA 2009) for acrylamide is 0.0002 mg/kg/d. USEPA (2010) derived a RfD of 0.002 mg/kg/d; the new RfD being based on a more recent chronic exposure studies, as well as current methodology for characterising the dose-response curve. The Drinking Water Equivalent Level or DWEL (USEPA 2009) of 0.007 mg/L was changed to 0.07 mg/L (USEPA (2011).

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 MRLs for acrylamide are:

minimal risk level

0.01 mg/kg/day for acute-duration oral exposure (1–14 days)

0.001 mg/kg/day for intermediate-duration oral exposure (15–364 days)

0.001 mg/kg/d for chronic-duration oral exposure (>364 days).

Based on the fact that the BMDL10 value of 0.43 mg/kg b.w. per day obtained for neurotoxicity is lower than the NOAEL of approximately 2.0 mg/kg b.w. per day for adverse effects on male reproductive parameters and of 1.0 mg/kg b.w. per day for developmental toxicity, the CONTAM Panel concluded that using the BMDL10 for neurotoxicity as the reference point is conservative when considering possible non-neoplastic effects of AA (EFSA 2015).

In mutagenicity assays, acrylamide does not cause mutations in bacterial test systems, but does cause chromosome damage to mammalian cells *in vitro* and *in vivo*. In a long-term carcinogenicity study in rats exposed via drinking-water, acrylamide induced scrotal mesotheliomas. The International Agency for Research on Cancer placed acrylamide in Group 2B (possibly carcinogenic to humans), and in 1994 reclassified it in Group 2A (probably carcinogenic to humans).

The USEPA assigned B2 (probable human carcinogen) to acrylamide based on “inadequate human data and sufficient evidence of carcinogenicity in animals; significantly increased incidences of benign and/or malignant tumours at multiple sites in both sexes of rats, and carcinogenic effects in a series of one-year limited bioassays in mice by several routes of exposures.” The USEPA noted that positive genotoxicity data, adduct formation activity, and structure-activity relationships to vinyl carbamate and acrylonitrile provide support to the classification (ATSDR 2009). USEPA (2009) states that a concentration of 0.0008 mg/L acrylamide represents a 10-4 cancer risk.

Acrylamide appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The short-term, chronic and subchronic health risk limits are 0.007 mg/L, and a limit of 0.0002 mg/L was set for cancer.

Only the acrylamide monomer is toxic; acrylamide polymers are non-toxic.

WHO (2011a) includes a 152-page report on health effects of acrylamide in food.

### Derivation of Maximum Acceptable Value

Based on the available information, it has been concluded that acrylamide is a genotoxic carcinogen. Therefore the risk evaluation was carried out using a non-threshold approach.

On the basis of combined mammary, thyroid, and uterine tumours observed in female rats in a drinking-water study, and using the linearised multistage model, the MAV associated with an excess lifetime cancer risk of one per 100,000 (10-5) is estimated to be 0.0005 mg/L (0.5 g/L).

Drinking-water levels which are considered “safe” for short-term exposures:

for a 10‑kg (22 lb) child consuming one litre of water per day: a one-day exposure of 1.5 mg/L; a 10-day exposure to 0.3 mg/L; up to a seven-year exposure to 0.002 mg/L. USEPA (29 November 2006).

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# Acrylic acid

CAS No. 79-10-7. Also called 2-propenoic acid, ethylenecarboxylic acid, vinylformic acid, propene acid, or acroleic acid. Sometimes called AA.

### Maximum Acceptable Value

There is no MAV in the DWSNZ. Acrylic acid is not mentioned in the WHO Guidelines.

NSF Standard 60 states that the allowable concentration of acrylic acid in drinking water is 0.12 mg/L.

### Sources to drinking-water

#### 1. To source waters

The primary use of acrylic acid, accounting for approximately 67 percent of all use (in the US), is in the production of acrylic esters and resins, which are used primarily in coatings and adhesives. The fastest growing use of acrylic acid is in the production of superabsorbent polymers. It is also used in oil treatment chemicals, detergent intermediates, water treatment chemicals (eg, polyelectrolytes), polishes, paints and water absorbent polyacrylic acid polymers (USEPA 1994).

Acrylic acid polymerises easily when exposed to heat, light or metals, and so a polymerisation inhibitor is added to commercial acrylic acid to prevent the strong exothermic polymerisation. The inhibitors that are usually used in acrylic acid preparations are the monomethyl ether of hydroquinone (methoxyphenol) at 200 ± 20 ppm, phenothiazine at 0.1 percent and hydroquinone at 0.1 percent. Methylene blue at 0.5 to 1.0 percent and N,N’-diphenyl-p-phenylenediamine at 0.05 percent can also be used (IPCS 1997).

An antimicrobial product described as “2-propenoic acid polymer with 2-propenal” appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2012 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)).

2-Propenoic acid polymer with 2-propenal, sodium salt has a CAS No. 52286-11-0. 2‑Propenoic acid polymer with 2-propenal, CAS 28349-72-6, is used as an anti-microbial for the management of intestinal health in broiler chickens and swine, and is administered orally with drinking water (NZFSA 2007).

Acrylic acid can be produced by some marine algae.

### Forms and fate in the environment

Most of the acrylic acid released to the environment is expected to end up in water. Based on its water solubility and vapour pressure, it is not expected to volatilise from water. It is completely stable in sterile water, pH range 3 to 11. Acrylic acid is removed from water by microbial degradation and chemical and photochemical reactions. The chemical can be removed from the atmosphere in rain. If released to soil acrylic acid leaches into groundwater or surface waters (ex USEPA 1994).

Acrylic acid may be formed by hydrolysis of acrylamide monomer from industrial waste in soil, especially under aerobic conditions (IPCS 1997).

If released to soil, acrylic acid is expected to have very high to high mobility based upon Koc values of 6 to 137. The pKa of acrylic acid is 4.26, indicating that this compound will exist almost entirely in the anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilisation from moist soil is not expected because the acid exists as an anion and anions do not volatilise. Acrylic acid may volatilise from dry soil surfaces based upon its vapour pressure. [EU 2002 quotes a soil half-life of <3 days.] Utilising the Japanese MITI test, 68 percent of the theoretical BOD was reached in two weeks indicating that biodegradation may be an important environmental fate process. In a 42–day anaerobic screening study using a sewage seed inoculum, 71 percent of acrylic acid was mineralised. Photodegradation rates in distilled water, river water, artificial seawater, and seawater of 2.1 x 10-4, 5.7 x 10-4, 4.2 x 10-4, and 3.9 x 10-4/sec, corresponding to half-lives of 55, 20, 28, and 30 minutes, respectively, suggest that acrylic acid may be susceptible to direct photolysis by sunlight. If released into water, acrylic acid is not expected to adsorb to suspended solids and sediment based upon the Koc values (EU 2002 quotes logPow = 0.46 at 25°C). The pKa indicates acrylic acid will exist almost entirely in the anion form at pH values of 5 to 9 and therefore volatilisation from water surfaces is not expected to be an important fate process. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Acrylic acid was determined to be stable to hydrolysis at pH 3, 7, and 11 (EAWAG accessed February 2015).

Water solubility is described as miscible.

### Typical concentrations in drinking-water

NSF (2010) reports levels from <0.00002 to 0.01 mg/L acrylic acid in drinking water that has been treated with polyacrylamide coagulant aids. The median concentration was 0.0015 mg/L.

### Analytical methods

#### Referee method

No MAV, so not needed.

### Health considerations

After oral exposure in the rat, the highest concentrations of the chemical were found in the liver, fat, small intestine, brain, and kidneys. Nearly all of the acrylic acid is absorbed and metabolised to carbon dioxide, with very little found in the urine or the faeces (USEPA 1994).

The RfD was calculated at 0.0025 mg/kg/d (USEPA (1994a).

Rats were exposed to acrylic acid in drinking water at 83, 250, or 750 mg/kg/day for 90 days (see USEPA 1994). At the high dose (in both males and females) a reduction in body and organ weights and decreased water intake were observed; the NOAEL for changes in body weight and in organ weight was 250 mg/kg/day. There were no treatment-related histopathologies observed. In other drinking-water studies on rats the no-observed-adverse-effect level (NOAEL) was 140 mg/kg body weight per day for decreased body weight gain in a 12-month study (IPCS 1997).

It is recommended that exposure of the general public to acrylic acid in drinking-water does not exceed the guidance values given in the *Environmental Health Criteria* No. 19 Acrylic Acid (IPCS 1997a), which is: oral exposure via drinking-water: 9.9 mg/L.

Acrylic acid is embryotoxic and teratogenic (IARC 1979).

IARC classified acrylic acid in 1999 as Group 3, not classifiable as to its carcinogenicity to humans.

### Derivation of Maximum Acceptable Value

No MAV.

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# Acrylonitrile

CAS No. 107-13-1. Also called 2-propenenitrile, [vinyl](http://en.wikipedia.org/wiki/Vinyl) [cyanide](http://en.wikipedia.org/wiki/Cyanide), 2-propenenitrile, acrylic acid nitrile, carbacryl, propenoic acid nitrile, or cyanoethylene.

This datasheet also includes some information about methyl acrylonitrile (CAS No. 126‑98-7, also called methacrylonitrile, 2-cyanopropene, isoprene cyanide and 2‑methyl-2-propenenitrile).

### Maximum Acceptable Value

There is no MAV in the DWSNZ. Acrylonitrile is not mentioned in the WHO Guidelines.

Acrylonitrile is one of the “priority pollutants” under the US Clean Water Act.

NSF Standard 60 states that the allowable concentration of acrylonitrile in drinking water is 0.00006 mg/L.

### Sources to drinking-water

#### 1. To source waters

Acrylonitrile is used principally as a [monomer](http://en.wikipedia.org/wiki/Monomer) in the manufacture of synthetic [polymers](http://en.wikipedia.org/wiki/Polymer), especially [polyacrylonitrile](http://en.wikipedia.org/wiki/Polyacrylonitrile) which comprises [acrylic fibres](http://en.wikipedia.org/wiki/Acrylic_fiber). Acrylic fibres are, among other uses, [precursor](http://en.wiktionary.org/wiki/Precursor)s for well-known [carbon-fibr](http://en.wikipedia.org/wiki/Carbon-fiber)e. Acrylic fibres are also used for the manufacture of apparel, including sweaters, fleece wear and sportswear, and home furnishings, including carpets, upholstery and draperies. Acrylonitrile is also a component of [synthetic rubber](http://en.wikipedia.org/wiki/Rubber), elastomers and styrene–acrylonitrile (SAN) resins. Dimerisation of acrylonitrile affords adiponitrile, used in the synthesis of certain [nylons](http://en.wikipedia.org/wiki/Nylon). Small amounts are also used as a [fumigant](http://en.wikipedia.org/wiki/Fumigant). Acrylonitrile is also a precursor in the industrial manufacture of [acrylamide](http://en.wikipedia.org/wiki/Acrylamide) and [acrylic acid](http://en.wikipedia.org/wiki/Acrylic_acid). Technical acrylonitrile is sometimes stabilised with 35–50 mg/kg hydroquinone monomethyl ether inhibitor to prevent polymerisation during storage and transport. Acrylonitrile-based plastic articles or fibres and fabrics contain from 0.0001–0.005 percent residual acrylonitrile (NICNAS 2000).

Releases of acrylonitrile may also occur as a result of combustion of hydrocarbon fuels and cigarette smoking. The incomplete combustion during incineration of municipal wastewater sludge has been identified as a minor source of release of acrylonitrile to the environment (EU 2004).

Acrylonitrile was detected in 46 of 914 samples of surface water and groundwater taken across the United States, generally at levels less than 0.01 mg/L. EU (2004) reports most natural water samples contain <0.002 mg/L, ie, the usual limit of detection.

The USEPA recommends that levels in lakes and streams should be limited to 0.000058 mg/L to prevent possible health effects from drinking water or eating fish contaminated with acrylonitrile.

Methyl acrylonitrile is also used in industry, as an intermediate in the preparation of methacrylic plastics and polymers (known as acrylic glass or plexiglass).

#### 2. From the distribution system

Acrylonitrile is a component in the manufacture of ABS pipes (acrylonitrile-butadiene-styrene), specifications covered AS 3518: 1997 Acrylonitrile butadiene styrene (ABS) pipes and fittings for pressure applications. See also BS 5391.

The Summary Report of NSF International Extraction Results (1991 to 1998) ANSI/NSF Standard 61: Drinking Water System Components – Health Effects stated: “In ABS materials, the extraction of acrylonitrile appears to be more of a function of the surface area of exposure and not necessarily the specific manufacturing process.”

ABS pipes have been used for water supply increasingly since the 1960s. There does not appear to be any reference in the international literature to acrylonitrile leaching from ABS pipes into drinking-water, although it has been found to leach from nitrile-butadiene rubber gaskets and O-rings.

### Forms and fate in the environment

Acrylonitrile is very soluble in water (about 7 to 8 percent) and is not strongly adsorbed to soil or sediment so can enter groundwater by filtering through the soil. However it is not commonly found in groundwater. Acrylonitrile is readily to fairly degradable in water and soil, and is broken down by bacteria in surface water. The characteristics of acrylonitrile indicate that although the volatilisation from aquatic systems is not rapid, it may be a significant removal process in the environment. Methyl acrylonitrile is readily biodegradable and its bioaccumulation potential seems to be low. Methyl acrylonitrile water solubility is about 29,000 mg/L (2.9 percent).

If released to soil, acrylonitrile is expected to have very high mobility based upon estimated Koc values of 9 and 29. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 1.38 x 10-4 atm‑cu m/mole. Acrylonitrile is expected to volatilise from dry soil surfaces based upon its vapour pressure. At concentrations up to 100 ppm, complete degradation of acrylonitrile occurred in <2 days in soil. If released into water, acrylonitrile is not expected to adsorb to suspended solids and sediment based upon the estimated Koc values. Acrylonitrile completely degraded in river water in six days and degraded completely in 20 days in another study, requiring less time for degradation with acclimation. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are seven hours and four days, respectively. A BCF of 48 for bluegills suggests the potential for bioconcentration in aquatic organisms is moderate. Acrylonitrile was stable at pH 4 to 10 for 23 days indicating that hydrolysis is negligible under these conditions (EAWAG accessed February 2015).

EU (2004) quotes: acrylonitrile vapour pressure = 115 hPa at 20°C; water solubility = 7.35 percent; partition coefficient = logPow = 0.25. Acrylonitrile is relatively hydrolytically stable, with no hydrolysis reported to occur in distilled water over the pH range. Decomposition of acrylonitrile has been demonstrated over a period of 23 days at a concentration of 10 mg/L in Mississippi River water at different pHs. Concentrations of acrylonitrile in river water at unadjusted pH fell linearly to undetectable levels by day 6, decomposition was slower at pH 4.0 and pH 10.0, although levels at pH 10 were also below the limits of detection by day 23. The degradation seen in this study may be due to a combination of biodegradation and volatilisation of acrylonitrile from the test medium rather than abiotic degradation. The half-life in water was reported to be 150 days, and soil 300 days.

### Typical concentrations in drinking-water

In a state-wide survey of 1,700 wells in Wisconsin, United States, acrylonitrile was not detected in any sample. Two water utilities in the US reported detecting acrylonitrile in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.005 mg/L.

One water utility in the US reported detecting methyl acrylonitrile in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0025 mg/L.

NSF (2010) reports levels from <0.00001 to 0.00005 mg/L acrylonitrile in drinking water that has been treated with polyacrylamide coagulant aids. The median concentration was 0.00002 mg/L.

### Removal methods

Acrylonitrile is oxidised by strong oxidants such as chlorine and ozone.

### Analytical methods

#### Referee method

No MAV, so not needed.

### Health considerations

Acrylonitrile is rapidly absorbed via all routes of exposure and distributed throughout examined tissues. There is little potential for significant accumulation in any organ, with most of the compound being excreted primarily as metabolites in the urine within the first 24–48 hours following administration.

Acrylonitrile has been detected in the smoke of cigarettes at levels of 3 to 15 mg per cigarette.

IARC (1999) classified acrylonitrile as possibly carcinogenic to humans (Group 2B). Acrylonitrile is possibly carcinogenic to humans (USEPA Group 2B). Studies of people are inconclusive, while animal studies have shown cancers of the brain and mammary glands. USEPA (2009/2011) quotes a health advisory of 0.006 mg/L, representing a 10-4 cancer risk.

Acrylonitrile appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 MRLs for acrylonitrile are:

minimal risk level

0.1 mg/kg/day for acute-duration oral exposure (1–14 days)

0.01 mg/kg/day for intermediate-duration oral exposure (15–364 days)

0.04 mg/kg/day for chronic-duration oral exposure (>364 days).

#### Methyl acrylonitrile

From a two-year carcinogenicity study [NTP], the NOAEL for oral repeated dose toxicity was considered to be 7.14 mg/kg b.w./day for rats and 4.29 mg/kg b.w./day for mice. The NOAEL for developmental toxicity was 3 mg/kg b.w. in rabbits by oral administration. This chemical is not a teratogen and is considered not to be carcinogenic in rodents (INCHEM 2002).

Six oral and four inhalation repeated dose toxicity studies for methyl acrylonitrile including two oral carcinogenic studies are available; the NOAEL for oral repeated dose toxicity was considered to be 7.14 mg/kg b.w./day for rats and 4.29 mg/kg b.w./day for mice. Methyl acrylonitrile was not mutagenic (OECD 2005). The oral RfD for methacrylonitrile was calculated at 0.0001 mg/kg/d (USEPA 1996).

### Derivation of Maximum Acceptable Value

No MAV.

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# Allyl chloride

CAS No. 107-05-1. Also called allylchloride, 3-chloroproprene, 3-chloropropylene, 3‑chloro-1-propene, 1-chloro-2-propene, α-chloropropylene and 2-propenyl chloride.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for allyl chloride. The WHO Guidelines do not mention allyl chloride.

### Sources to drinking-water

#### 1. To source waters

Allyl chloride is used in the preparation of epichlorhydrin (chiefly), and glycerol, resins and plastics.

#### 2. From treatment processes

Allyl chloride and dimethylamine are reacted to form polyDADMAC polyelectrolytes. Allyl chloride has been reported to be a contaminant in the final product, along with diallylether and 5-hexanal (Letterman and Pero 1990 – quoted by Majam and Thompson). The AWWA Standard requires the monomer to be less than 5 percent and the polymer dose to be less than 10 mg/L.

Anticipated products from the reaction of allyl chloride with ozone include formaldehyde, chloracetaldehyde and chloracetic acid.

### Forms and fate in the environment

The estimated hydrolysis half-life of allyl chloride in water at 25°C and pH 7 is 69 days (IARC). However, OECD states that if allylchloride is emitted into water it will rapidly volatilise to the air, model calculations for a river indicate a half life of approximately three hours.

Very soluble in water: 3,600 mg/L at 20°C.

### Analytical methods

#### Referee method

Not needed.

#### Some alternative methods

None needed.

### Health considerations

IARC (1985) stated that “Allyl chloride caused DNA damage in bacteria, and was mutagenic to bacteria and fungi. No case report or epidemiological study of the carcinogenicity of allyl chloride to humans was available to the Working Group.” IARC (1999) considered allyl chloride is not classifiable as to its carcinogenicity to humans (Group 3).

This chemical was delisted (October 1999) from the State of California EPA list of chemicals known to cause cancer or reproductive toxicity.

USEPA (1995) classified allyl chloride as Class C: possible human carcinogen; they do not have an oral RfD.

### Derivation of Maximum Acceptable Value

No MAV.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for 120 contaminants that have been found in Minnesota groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for allyl chloride of 0.03 mg/L was established because of effects on the nervous system.

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# Aniline

CAS No. 62-53-3. Also called phenylamine and aminobenzene.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for aniline. WHO Guidelines do not mention aniline.

The USEPA concluded on 22 September 2009 that aniline is known or anticipated to occur in PWSs and may require regulation. Therefore they added aniline to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

Aniline is used to make a wide variety of products such as polyurethane foam (the main use), agricultural chemicals, synthetic dyes, antioxidants, herbicides, varnishes, perfumes and explosives; it is also used as a stabiliser in the rubber industry. Aniline can be formed from the breakdown of certain pollutants found in outdoor air, from the burning of plastics, from burning tobacco or degradation of rubber and some pesticides (eg, carboxin, fenuron and propham). Some pesticides break down in the soil releasing aniline. Small amounts of aniline may be found in some foods, such as corn, grains, rhubarb, apples, beans, and rapeseed cake (animal feed). Aniline has also been found as a volatile component of black tea, and in coal tar. Aniline is not approved for use in the European Union as a pesticide.

Concentrations of aniline in drinking water range from 0 to 0.18 μg/L, in groundwater from 0 to 720 μg/L, in fresh surface water from 0 to 12 μg/L and in effluents is identified but not quantifiable (DWI 2014).

### Forms and fate in the environment

Aniline in water can adhere to sediment and particulate matter, or evaporate to the air. Aniline will partially stick to the soil. Small amounts may evaporate into the air or pass through the soil to groundwater. Most of the aniline in soil will be broken down by bacteria and other micro-organisms; 80 to 100 percent degradation in aerobic conditions within 28 days, and it is not biodegradable in anaerobic conditions (EU 2004). Aniline is very soluble in water, about 3.6 percent. A half-life for aniline of 2.3 days has been reported in an industrial river.

If released to soil, aniline is expected to have very high to moderate mobility based upon Koc values of 8–497. The pKa of aniline is 4.6, indicating that it will exist partially in the cation form in the environment and cations generally adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Aniline-type compounds’ sorption to soil has been observed to be a rapid initial sorption (a reversible cation-exchange) followed by a slower non-reversible covalent-binding. Volatilisation from moist soil surfaces may occur based upon a Henry’s Law constant of 2.02 x 10-6 atm‑cu m/mole. Aniline is degraded by many common species of bacteria and fungi found in soil with acetanilide, 2-hydroxyacetanilide, 4-hydroxyaniline and catechol as reported metabolites. If released into water, aniline may adsorb to suspended solids and sediment in water based upon the Koc data. Sorption in water has been observed to be a rapid initial loss of aniline from the aqueous phase (a reversible cation-exchange) followed by a much slower rate of disappearance through non-reversible covalent-binding. Complete aniline biodegradation in seven days by industrially-polluted pond water near a factory and no degradation in water from a non-polluted botanical pond and rice paddy, suggests that biodegradation may be an important environmental fate process in water, provided the system is acclimated. Volatilisation from water surfaces is expected to occur based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 12 and 131 days, respectively. Measured BCF values of less than 10 in fish suggest that bioconcentration in aquatic organisms is low. EAWAG. Accessed February 2015. DWI (2014) quote a log Kow of 0.9.

EU (2004) states that 20 percent of the aniline in soil is rapidly mineralised and the other 80 percent is covalently bound to the organic fraction. For this bound aniline a derived half-life of 350 days is used for soil and sediments.

In 1994 aniline was measured in the Rhine and tributaries. Concentrations ranged from <0.0001 to 0.0016 mg/L (EU 2004).

### Typical concentrations in drinking-water

47,000 Americans in 17 communities were served tap water contaminated with aniline between 1998 and 2003, the highest concentration being 0.01 mg/L, most being less than 0.001 mg/L.

### Removal methods

DWI (2014) reports many case studies:

An initial concentration of 100 mg/L was reported to undergo 55 percent adsorption on to Norit Granular Activated Carbon (GAC) at a dose of 500 mg/L.

Using an aniline concentration of 10 mg/L with 1 mg chlorine/L at pH 7, approximately 90 percent of the chlorine dose was consumed within one hour. Aniline initially forms N-chloroaniline and chloroaniline isomers, which may undergo oxidation or polymerisation processes in the presence of excess chlorine.

An aniline concentration of 9 mg/L was decomposed by 10 mg ozone/L, however, in this study it was also noted that the reaction products that were produced were of greater toxicological concern than aniline. The products of a reaction between ozone and aniline that may have potential concern with regards to potential mutagenicity included azobenzene, azoxybenzene, benzidine, nitrosobenzene, nitrobenzene, 4-aminodiphenylamine and phenazine. In another study, an initial aniline concentration of 100 mg/L was completely removed following application of an ozone dose of 10 mg/L/minute for 20 minutes at pH 7. However, the presence of fulvic acid appears to allow the removal of aniline without the formation of these breakdown products.

A concentration of 100 mg/L was 63 percent removed by an ESPA1 reverse osmosis, but only 20 percent removed by an NF 200 nanofiltration membrane.

### Analytical methods

#### Referee method

Not needed.

### Health considerations

The USEPA considers aniline to be a probable human carcinogen (cancer-causing agent) and has ranked it in Group B2, based on induction of tumours of the spleen and the body cavity in two strains of rat, and some supporting genetic toxicological evidence. Aniline appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity. The USEPA estimates that drinking-water containing 0.06 mg/L would result in not greater than a one-in-a-hundred thousand (10-5) increased chance of developing cancer. The International Agency for Research on Cancer (IARC 1982, confirmed 1987) had determined that on the basis of all the available data, no evaluation could be made of the carcinogenicity of aniline to humans (Group 3).

DWI (2014) reports: Tests for the genotoxicity of aniline in bacterial assays are generally negative, although the *majority of other in vitro and in vivo tests indicate a weak genotoxicity of aniline. The EU has classified aniline as a Category 3 mutagen (positive in vivo in somatic cells). For Repeat* Dose Toxicity and Carcinogenicity a LOAEL of 7 mg/kg bw/day was identified based on haematological and splenic effects, from which was derived a Tolerable Daily Intake (TDI) of 0.007 mg/kg bw/day.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Anthracene

Anthracene, CAS No. 120-12-7, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. May also be called paranaphthalene and p‑naphthalene.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

Anthracene occurs in fossil fuels and is also released to the environment as a ubiquitous product of incomplete combustion, occurring in exhaust from coke ovens, motor vehicles, emissions from cigarette smoke, coal-, oil-, and wood-burning stoves and furnaces. On average it comprises 1.5 percent of coal tar, and 1.5 percent of creosote. Anthracene is converted to anthraquinone and used as an intermediate in dye production, in the manufacture of synthetic fibres, and as a diluent for wood preservatives – this use may have ceased. It is also used in smoke screens, pyrotechnics, as scintillation counter crystals, and in organic semiconductor research. Anthracene is used to synthesise the chemotherapeutic agent, Amsacrine.

The commercial product is usually no more than 97 percent pure; main impurities being the following: phenanthrene (1.0 percent); carbazole (1.0 percent); naphthothiophene (0.4 percent); dibenzo[b,c]thiophene (0.3 percent); acridine (0.2 percent); acetophenone (0.4 percent) (EU 2008).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

If released to soil, measured Koc values ranging from 2,600 to 8600 indicate anthracene is expected to have slight to no mobility. Anthracene is expected to volatilise from moist soil surfaces based upon its measured Henry’s Law constant of 4.88 x 10-5 atm‑cu m/mole. However, adsorption to organic matter may attenuate this process. Volatilisation of anthracene from dry soil surfaces is not expected to be an important fate process based on its extrapolated vapour pressure. Biodegradation in soil is expected to be an important fate process based upon half-lifes in unacclimated soils ranging from 50 to 134 days. More rapid biodegradation rates were observed in soils contaminated with coal tar or oil. If released into water, anthracene is expected to adsorb to suspended solids and sediment in the water column based upon the Koc values. Volatilisation from water surfaces is expected to occur given its Henry’s Law constant. However, volatilisation is expected to be attenuated by adsorption to suspended solids and sediment in the water. Estimated volatilisation half-lifes for a model river and model lake are 1.2 and 13 days, respectively when adsorption is not considered. The estimated volatilisation half-life from a model pond is about 11 months when adsorption is considered (EAWAG accessed February 2015).

EU (2008) quotes: vapour pressure = 9.4 x 10-4 Pa at 25°C; water solubility = 0.047 mg/L; partition coefficient = logPow = 4.68; Henry’s law constant = 4.3 Pa.m3/mol. Hydrolysis of anthracene is not expected. The fate of anthracene, a representative polycyclic aromatic hydrocarbon, was followed in a large outdoor stream microcosm. The major non-advective route for the removal of anthracene was photolytic degradation to anthraquinone (half-life 43 minutes). The anthraquinone also photolysed rapidly in this shallow stream system. Excluding the plastic channel liner, the sediment acts as the major sink for anthracene, absorbing 0.2 percent of the 14‑day input dose. The periphyton community was the second most important sink, absorbing 0.04 percent of the input dose. All other compartments were of significantly less importance on a mass basis. Photolysis of anthracene (350 nm) in aerated water yields endoperoxide and 9,10-anthraquinone as the major primary products. Photolysis of anthracene in oxygen-deficient aqueous solutions yields the three isomers of 10,10’‑dihydroxy-9, 9’-10, 10’-tetrahydro-9, 9’-bianthryl as the primary product. In summary, the half-life for photolysis in water lies in the range 20 minutes and 124.8 hours depending on the experimental conditions used. The highest value in this range corresponds to photolysis in winter solar conditions.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 and 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. Anthracene was not detected. A number of PAHs have been assessed in Phase 1 of the P2 Programme. With the exception of fluoranthene, none have been detected. The limits of detection range from 0.0001 to 0.0002 mg/L.

Six water utilities in the US reported detecting anthracene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0002 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation (ATSDR 1995).

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

The US Environmental Protection Agency has determined that anthracene is not classifiable as to human carcinogenicity based on no human data and inadequate data from animal bioassays.

IARC (2010) classified anthracene in Group 3 (not classifiable as to carcinogenicity).

The USEPA has a reference dose or RfD of 0.3 mg/L for anthracene, and a Drinking Water Equivalent Level or DWEL of 10 mg/L.

As at July 2013, ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) has developed oral minimal risk levels (MRLs) for some PAHs:

|  |  |  |
| --- | --- | --- |
| **PAH** | **mg/kg/day** | **duration** |
| anthracene | 10 | intermediate (15–364 days) |

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limits (exposure greater than 10 percent of a lifetime) for anthracene is 2 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

### References

ATSDR. 1995. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs)*. Atlanta, GA: Agency for Toxic Substances & Disease Registry, Department of Health and Human Services. See: <http://www.atsdr.cdc.gov/toxprofiles/index.asp>.

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MDH. 2009/2016. *Groundwater Values Table*. Minnesota Department of Health (MDH). See: <http://www.health.state.mn.us/divs/eh/risk/guidance/gw/table.html>.

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USEPA. 2011. *2011 Edition of the Drinking Water Standards and Health Advisories*. Washington, DC: US Environmental Protection Agency. Available at: [http://water.epa.gov/action/advisories/health\_index.cfm](http://water.epa.gov/action/advisories/drinking/drinking_index.cfm%20) or [www.epa.gov/waterscience/](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.epa.gov\waterscience\).

USEPA. *National Primary Drinking Water Regulations, Technical Factsheet on: Polynuclear aromatic hydrocarbons (PAHs)*. http://www.epa.gov/osw/hazard/wastemin/minimize/factshts/anthrace.pdf.

WHO. 2011. *Guidelines for Drinking-water Quality* 2011 (4th edition). Geneva: World Health Organization. Available at: [http://www.who.int/water\_sanitation\_health/publications/drinking-water-quality-guidelines-4-including-1st-addendum/en/index.html](http://www.who.int/water_sanitation_health/publications/2011/dwq_guidelines/en/index.html).

# Anthraquinone

CAS No. 84-65-1. Also called 9,10-anthraquinone, 9,10-dihydro-anthracene, 9,10‑dihydro-9,10-dioxoanthracene, 9,10-dioxoanthracene and anthracenedione. Refer to the polynuclear aromatic hydrocarbons datasheet.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

Anthraquinone compounds can occur naturally, in products such as rhubarb, aloe and senna.

Anthraquinone is an oxygenated PAH that can be formed from direct combustion processes. Anthraquinone is formed from anthracene through photolytic and biodegradation processes.

Anthraquinone is an important and widely used raw material for the manufacture of vat dyes. Anthraquinone is also used as a seed dressing or in seed treatments. Other major uses are as a pesticide, as a bird repellent (especially for geese), and as an additive in chemical alkaline pulp processes in the paper and pulp industry as a catalyst in the pulp industry to improve delignification of wood and increase pulp yield. Anthraquinone is being trialled as a repellent on 1080 baits in New Zealand at about 0.1 percent w/w (Clapperton et al 2013).

Estimated emission rates of anthraquinone in diesel emission particles were reported to be 24.9 μg/mile [15.5 μg/km]. Another study reported emission rates from exhaust pipes of various vehicles ranging from 4.4 μg/km for cars with a catalyst and 24.3 μg/km for cars without a catalyst to 23.5 μg/km for heavy-duty diesel trucks.

Coal may be another source of exposure to anthraquinone, which was detected (0.7 μg/L) in an extract of model coal piles (Texas lignite) leached with distilled water under simulated rainfall conditions.

Anthraquinone derivatives have been used a laxative. It is also used in the manufacture of hydrogen peroxide in Morrinsville (NZIC).

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

Anthraquinone that is released into water is expected to adsorb on to suspended solids and sediment based upon the Koc. Experimental studies have shown that the majority of the anthraquinone added was degraded within three days in both surface water (82 percent) and groundwater (91 percent). Natural bacterial populations in groundwater and activated sludge were also shown to degrade anthraquinone (range, 50–100 percent) in experiments that lasted between five days and three weeks. Anthraquinone may also be removed through photolysis by sunlight, and its direct photolysis half-life is about nine minutes in aqueous solution. It is not sensitive to aqueous environmental hydrolysis, and volatilisation is not expected to be an important factor in its removal. An estimated BCF of 12 suggests the potential for bioconcentration in aquatic organisms is low.

In soil, anthraquinone is predicted to be slightly mobile or immobile based on its estimated soil absorption coefficients of 2,755–17,416 that were determined using reference European soils. Volatilisation from moist soil surfaces is not expected to be an important fate process based on a Henry’s law constant: 2.35 × 10-8 atm.m3/mol at 25°C (estimated). Anthraquinone is not expected to volatilise from dry soil surfaces based upon its vapour pressure of 1.16 x 10-7 mm Hg.

Biodegradation also appears to be the most important factor that influences the removal of anthraquinone from soil; 67 percent of the anthraquinone added was biodegraded in a mixed soil population within 12 weeks. Other studies have reported half-lifes in different soils of 3–10 days, and a study that used a mixed bacterial population found that 6.5 percent of the initial concentration of anthraquinone remained in the soil after three days.

The ratio of anthracene to anthraquinone in marine sediments was less than 1  
(0.32–0.77) at urban sites, suggesting that the source of the exposure was predominantly discharge, whereas the ratio at remote sites was greater than 1  
(2.45–2.81), suggesting that the source was primarily atmospheric deposition.

Water solubility: 1.35 mg/L. Octanol/water partition coefficient: log Kow, 3.39.

### Typical concentrations in drinking-water

Anthraquinone has been detected in groundwater from industrial sites (see Table 1.3), surface water and drinking-water (at concentrations up to 100 ng/L) in Japan, the USA and Canada (Table 1.5), and also in precipitations in the USA and Norway (IARC 2012).

### Removal methods

PAHs in water can be oxidised by chlorination and ozonation (ATSDR 1995).

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet, and IARC (2012).

### Health considerations

A no observed adverse effect level (NOAEL) of 31.3 mg/kg/d was selected based on the absence of liver histopathology in a feeding study in rats up to 13 weeks (Dodd et al 2013).

IARC (2012) classified anthraquinone in Group 2B (possibly carcinogenic to humans).

EFSA (2012) states that the toxicological properties of anthraquinone remain unknown and a potential carcinogenic effect cannot be excluded.

### Derivation of Maximum Acceptable Value

No MAV.

### References

ATSDR. 1995. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs)*. Atlanta, GA: Agency for Toxic Substances & Disease Registry, Department of Health and Human Services. See: <http://www.atsdr.cdc.gov/toxprofiles/index.asp>.

Clapperton et al. 2014. Efficacy of bird repellents at deterring North Island robins (*Petroica australis longipes*) and tomtits (*P. macrocephala toitoi*) from baits. *New Zealand Journal of Ecology* 38(1). <http://www.newzealandecology.org/nzje/>.

Dodd DE, et al. 2013. Subchronic toxicity evaluation of anthraquinone in Fischer 344 rats. *International Journal of Toxicology* 32(5): 358–67. Abstract: <http://ijt.sagepub.com/content/32/5/358.abstract>.

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# Artificial sweeteners

Artificial sweeteners include acesulfame K, [aspartame](http://www.nhs.uk/Livewell/Goodfood/Pages/the-truth-about-aspartame.aspx), [saccharin](http://www.nhs.uk/Livewell/Goodfood/Pages/the-truth-about-saccharin.aspx), [sorbitol](http://www.nhs.uk/Livewell/Goodfood/Pages/the-truth-about-sorbitol.aspx), [steviol glycosides (stevia plant extracts)](http://www.nhs.uk/Livewell/Goodfood/Pages/are-stevia-plant-extracts-safe.aspx), sucralose and [xylitol](http://www.nhs.uk/Livewell/Goodfood/Pages/the-truth-about-xylitol.aspx).

### Maximum Acceptable Value

The DWSNZ do not have a MAV for any artificial sweeteners. WHO Guidelines do not mention them.

### Sources to drinking-water

#### 1. To source waters

Valued for having no bitter aftertaste, sucralose-based products are found in a broad range of lower-calorie foods, including table top sweeteners, fizzy drinks, chewing gum, baking mixes, breakfast cereals and salad dressings.

The main interest in relation to water supplies is that the concentrations of some artificial sweeteners are tested in raw waters to indicate the presence of sewage, eg, acesulfame K and sucralose. Sucralose concentrations in US WWTP effluents were in the range of 20 to 30 μg/L (median = 27 μg/L). However, other studies have suggested a wider range of levels (WRF 2015).

Environment Canada tested the water from the Grand River at 23 sites between its headwaters and where it enters Lake Erie. The results suggest the artificial sweetener acesulfame is the best at evading wastewater treatment, and it appears in far higher concentrations than saccharin or sucralose at the various test sites. Researchers at the University of Waterloo and Environment Canada are currently using sucralose, cyclamate, saccharin and acesulfame as tracers to identify the source of these elevated artificial sweeteners in groundwater. Artificial sweeteners may be entering groundwater through leaking sewers or septic tanks (*Waterloo News*, 13 December 2013). The maximum concentrations measured were sucralose (21 µg/L), cyclamate (0.88 µg/L), and saccharin (7.2 µg/L) (Spoelstra et al 2013). Acesulfame persists at concentrations that are up to several orders of magnitude above the detection limit over a distance of 300 km. Sucralose had the highest concentration of any of the artificial sweeteners (max. value of 21 µg/L). However, only two samples in the synoptic survey had sucralose concentrations above the quantifiable limit, both at a relatively short distance (23.8 to 35.8 km) below the largest WWTP. Sucralose has previously been measured at concentrations ranging from <mdl to 3.56 µg/L in rivers in European countries. Acesulfame was the most consistently detected artificial sweeteners and present at 21 of the 23 sites.

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

Cyclamate and saccharin are more easily degraded during WWTP processes whereas removal rates for acesulfame and sucralose are very low to not detectable. Acesulfame has been shown to behave conservatively in groundwater indicating that biogeochemical activity in the subsurface does not significantly affect acesulfame. In contrast, sucralose is attenuated under aerobic and sub-oxic to anoxic conditions (Spoelstra et al 2013).

### Typical concentrations in drinking-water

Given the high concentrations of artificial sweeteners in the Grand River, especially downstream of the City of Waterloo, it is not surprising that artificial sweeteners were also detected in tap water in these large cities. Brantford had the highest concentrations of artificial sweeteners of the cities sampled, including high concentrations of more easily degraded saccharin. Acesulfame K was found up to 1.59 µg/L, saccharin up to 0.35 µg/L and cyclamate up to 0.24 µg/L. Brantford’s water treatment system consists of screening, coagulation, sand ballasted flocculation, sedimentation, ozonation, biological filtration, UV disinfection, chlorination and chloramination (Spoelstra et al 2013).

### Health considerations

Acesulfame K is not broken down when digested, nor is it stored in the body. After being consumed, it is quickly absorbed by the body and then rapidly excreted, unchanged. The ADI is 9 mg/kg/d (NHS 2016).

Aspartame is quickly and completely broken down into by-products – including phenylalanine, aspartic acid and methanol – which then enter our system through normal routes. Hardly any aspartame enters the bloodstream. The ADI is 40 mg/kg/d (NHS 2016).

Saccharin is not broken down when digested. It is slowly absorbed into the system and rapidly excreted, unchanged, by the kidneys. The ADI is 5 mg/kg/d (NHS 2016).

Sorbitol is slowly and only partially absorbed in the intestine and [converted into fructose in the liver](http://www.diva-portal.org/smash/get/diva2:145297/FULLTEXT01.pdf). Too much sorbitol in the intestine can cause water retention, resulting in diarrhoea. If consumed in large amounts, it can cause side effects such as bloating and gas. [Unabsorbed sorbitol is broken down into carbon dioxide](http://www.inchem.org/documents/jecfa/jecmono/v05je91.htm) and then eliminated. There is no ADI (NHS 2016).

Steviol glycosides (plant extracts) are broken down into steviol, which is absorbed by the body. The body does not store steviol glycosides and they are rapidly eliminated in faeces and urine. The ADI is 5 mg/kg/d (NHS 2016).

Sucralose is not absorbed by the body and is eliminated by excretion. Between 8 percent and 20 percent enters the blood and is removed through urine, essentially unchanged. The ADI is 15 mg/kg/d (NHS 2016).

Xylitol is slowly and only partially absorbed in the intestine, and is converted into glucose in the liver. Too much xylitol in the intestine can cause water retention, which can result in diarrhoea. If consumed in large amounts, side effects can include bloating and gas. [Unabsorbed xylitol is broken down into carbon dioxide](http://www.inchem.org/documents/jecfa/jecmono/v12je22.htm) and eliminated. There is no ADI (NHS 2016).

### Derivation of Maximum Acceptable Value

No MAV.

### References

NHS. Accessed April 2016. *NHS Choices: How safe is sucralose?* <http://www.nhs.uk/Livewell/Goodfood/Pages/the-truth-about-sucralose.aspx>.

Spoelstra J, Schiff SL, Brown SJ. 2013. *Artificial Sweeteners in a Large Canadian River Reflect Human Consumption in the Watershed*. <http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0082706> <http://dx.doi.org/10.1371/journal.pone.0082706>.

*Waterloo News*. 13 December 2013. Major Canadian river contains artificial sweeteners. University of Waterloo. <https://uwaterloo.ca/news/news/major-canadian-river-contains-artificial-sweeteners> and <http://gizmodo.com/how-diet-soda-can-be-used-to-track-sewage-1482917373>.

WRF. 2015. *Controlling the Formation of Nitrosamines During Water Treatment*. Water Research Foundation in conjunction with USEPA. 163 pp. <http://www.waterrf.org/PublicReportLibrary/4370.pdf>.

# Azobenzene

CAS No. 103-33-3. May be called 1,2-diphenyldiazene, azobenzide, diphenyldiimide or diazobenzene.

Diphenylhydrazine is a related chemical, CAS No. 122-66-7, azobenzene is a degradation product.

Benzidine (CAS No. 92-87-5, and CAS name [1,1’-biphenyl]-4,4’-diamine) is also a degradation product. A benzidine compound 3,3′-dimethylbenzidine (CAS No. 119‑93‑7) also called orthotolidine) used to be used for measuring the chlorine concentration in drinking water and swimming pool water, and as a reagent for detecting manganese.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for azobenzene. WHO Guidelines do not mention azobenzene.

### Sources to drinking-water

#### 1. To source waters

Azobenzene is not found directly in the environment, but usually as a degradation product. It has been used as an insecticide (acaricide) in the past.

1,2-Diphenylhydrazine (hydroazobenzene or hydroazodibenzene) was previously used to make fabric dyes, and is used to make certain medicines. It is used as an antisludging additive to motor oil. Several dyes are based on, or are degraded to, benzidine too.

If released into water, the pesticide diuron will adsorb to the sediment where it will slowly biodegrade after acclimation. The major product of the 6 to 7 degradation compounds that were isolated was 3,4-dichloroaniline and this metabolite may be further metabolised to an azobenzene derivative.

### Forms and fate in the environment

Azobenzene, when incubated in air with four anaerobic lake sediments containing about 2 to 4 percent organic matter, was reduced to aniline with a reaction half-life of about 2,700 to 5,700 minutes.

1,2-Diphenylhydrazine dissolves only slightly in water where it breaks down rapidly (half-life may be less than 15 to 30 minutes, or 60 minutes in anaerobic conditions) to the toxic chemicals azobenzene and benzidine; azobenzene tends to form only at higher pHs.

### Analytical methods

#### Referee method

Not needed.

### Health considerations

The USEPA considers azobenzene to be a probable human carcinogen (cancer-causing agent) and has ranked it in Group B2, based on induced invasive sarcomas in the spleen and other abdominal organs in male and female F344 rats following dietary administration. It is genotoxic and may be converted to benzidine, a known human carcinogen, under the acidic conditions in the stomach. Azobenzene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity. The USEPA estimates that drinking-water containing 0.003 mg/L would result in not greater than a one-in-a-hundred thousand (10-5) increased chance of developing cancer. The International Agency for Research on Cancer (IARC) determined that azobenzene is not classifiable as to its carcinogenicity to humans: Group 3.

1,2-Diphenylhydrazine was mutagenic in *Salmonella typhimurium*, but not in *Escherichia coli*, and produced chromosome aberrations and sister chromatid exchanges in Chinese hamster cells. Overall, the available evidence suggests that 1,2‑diphenylhydrazine may cause chromosomal damage or other genotoxic effects in humans. USEPA (1987) classified 1,2-diphenylhydrazine as B2: probable human carcinogen.

As a matter of interest, the RfD for benzidine was calculated at 0.003 mg/kg/d (USEPA. 1995. Benzidine is carcinogenic to humans, ie, Group I (IARC 2010). Orthotolidine is classified as Group 2B, possibly carcinogenic to humans.

### Derivation of Maximum Acceptable Value

No MAV.

Washington State has a drinking-water standard for azobenzene of 0.0007 mg/L based on a one-in-a-million (10-6) increased chance of developing cancer.

### References

ATSDR. 1990 and 2009 addendum. *Toxicological Profile for 1,2-Diphenylhydrazine*. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine. See http://www.atsdr.cdc.gov/toxprofiles/index.asp.

IARC. 1975. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 8. Some aromatic azo compounds. See: <http://monographs.iarc.fr/ENG/Monographs/allmonos30.php>.

IARC. 2010. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 99. Some aromatic amines, organic dyes, and related exposures. <http://monographs.iarc.fr/ENG/Monographs/vol99/index.php>.

USEPA. 1987. 1,2-Diphenylhydrazine. *Integrated Risk Information System (IRIS)*. http://www.epa.gov/iris/subst/0049.htm.

USEPA. 1988. [*Integrated Risk Information System (IRIS) on*](http://www.epa.gov/iris/subst/0364.htm) *Azobenzene*. Washington, DC: National Center for Environmental Assessment, Office of Research and Development. See <http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showSubstanceList> see also: http://www.epa.gov/iris/subst/0351.htm.

USEPA. 1995. Benzidine. *Integrated Risk Information System (IRIS)*. http://www.epa.gov/iris/subst/0135.htm.

Washington State. 1992. *Defining Water Quality*. Clean water for Washington, EB1721. http://cru.cahe.wsu.edu/CEPublications/eb1721/eb1721.html.

# Benzene

CAS No. 71-43-2. Also called benzol, pyrobenzol, phenyl hydride, cyclohexatriene and benzyl hydride in the past.

### Maximum Acceptable Value

Based on health considerations, the concentration of benzene in drinking-water should not exceed 0.01 mg/L.

The maximum contaminant level (USEPA 2006/2009/2011) is 0.005 mg/L. The maximum acceptable concentration in Canada is 0.005 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that no safe concentration for benzene in drinking water can be confidently set. However, for practical purposes the concentration should be less than 0.001 mg/L, which is the limit of determination.

The Prescribed Concentration or Value (PCV) for benzene in England and Wales is 0.001 mg/L. See Notes.

Benzene is one of the “priority pollutants” under the US Clean Water Act.

EPA established an environmental exposure limit of 2 mg/L for benzene in fresh water (<http://www.epa.govt.nz/search-databases/Pages/substance-exposure-limit-register.aspx>).

### Sources to drinking-water

#### 1. To source waters

Benzene may be present as a natural component of hydrocarbons, hence to petrol and motor vehicle emissions. It can also be produced in the course of natural processes and human activities that involve the combustion of organic matter such as wood and coal. Natural sources of benzene include volcanoes, crude oil, forest fires, and plant volatiles. Since benzene is very water soluble, it may be removed from the atmosphere by rain.

Trucks burning diesel have been reported to produce 3 to 9 mg of benzene per km (Environment Australia 2003). Petrol spillages constitute the main source of benzene in the environment. In New Zealand petrol, a limit of 4.2 percent (by volume) was allowable in 1995 (annual petrol consumption in 2001 around 2,900 million litres). In 2005 the Ministry of Economic Development proposed a stage 1 reduction to 3 percent, followed a stage 2 reduction to 1 percent. Benzene is a useful source of octane, and its reduction will require an increase in other high-octane constituents or additives such as toluene and xylene which have lesser health effects.

Benzene may also enter source waters as an industrial contaminant since it is used by the chemical industry in the production of styrene, phenol and cyclohexane. Benzene is also used in the manufacturing of some types of rubbers, lubricants, dyes, detergents, drugs, and pesticides. Because of health risks, use as a solvent should decrease. There is however, a steady demand in New Zealand which will likely result in the continuing occurrence of benzene in the environment.

In overseas studies benzene has been detected in the Rhine River in Germany at approximately 0.0003 mg/L, and occasionally in groundwater supplies in the United States. Concentrations are usually less than 0.001 mg/L, but concentrations up to 0.18 mg/L have been detected in chemical plant effluent.

EU (2008) reports benzene levels in surface water ranging from <0.1 to 32 µg/L, and from 0.005 to 1 µg/L in drinking water.

Data from the USEPA’s STORET database (2003–2005) showed that benzene was positively detected in 38 percent of the surface water samples collected at 571 observation stations ranging from concentrations too low to quantify to 0.10 mg/L.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Volatilisation with subsequent oxidation is considered to be the primary fate of benzene entering the environment. In soil, benzene degrades under aerobic conditions only. In surface water, benzene rapidly volatilises to the air, biodegrades, or reacts with hydroxyl radicals. Gasoline leaks from underground storage tanks release benzene into groundwater and soil. Hydrolysis and photolysis are not important processes.

Based on a reported Henry’s Law constant of 0.0053 atm.m3/mol and a model river 1 metre deep flowing at 1 m/s with a wind velocity of 3 m/s, the half-life of benzene was 2.7 hours at 20°C (NICNAS 2001).

If released to soil, benzene is expected to have high mobility based upon a Koc of 85. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 5.56 x 10-3 atm‑cu m/mole. Benzene may volatilise from dry soil surfaces based upon its vapour pressure. Using a base-rich para-brownish soil incubated for 10 weeks, 20 ppm benzene was 24 percent degraded in one week, 44 percent in five weeks, and 47 percent in 10 weeks, indicating that biodegradation may be an important environmental fate process in soil. If released into water, benzene is not expected to adsorb to sediment and suspended solids in water based upon the Koc. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 2.7 hours and 3.5 days, respectively. Utilising the Japanese MITI test, 40 percent of the Theoretical BOD was reached in two weeks indicating that biodegradation is important environmental fate process in water. In aqueous solution, benzene will react with hydroxyl radical (OH radical average concentration = 1.0 x 10-17 molec/cu cm) at a reaction rate of 7.8 x 10+9 L/mol sec which results in an estimated half-life of 103 days. A BCF ranging from 1.1–20 suggests bioconcentration in aquatic organisms is low. Hydrolysis is not expected to occur due to the lack of hydrolysable functional groups (EAWAG accessed February 2015).

Solubility of benzene in water is about 1,700–1,800 mg/L. Octanol/water partition coefficient: log Kow, 2.13. Vapour pressure = 99.7 hPa at 20°C. The half-life in water is 15 days, 30 days in soil and 300 days in sediments. A half-life of 11.5 days was calculated for volatilisation from aqueous solutions (EU 2008).

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 301 zones, found benzene in 4 zones at concentrations ranging from “not detectable” (nd) to 0.0036 mg/L (36 percent of the MAV), with the median concentration being “nd” (limit of detection = 0.0001 mg/L) (ESR 2001).

225 water utilities reported detecting benzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.013 mg/L.

In Alberta, benzene levels in municipal treated surface water ranged from 0.00001 to 0.005 mg/L (mean 0.00028 mg/L, 30 samples) for 26 locations from 1998 to mid-2005; levels in more than 96 percent of samples were less than 0.001 mg/L. Levels in municipal treated groundwater ranged from 0.00001 to 0.00023 mg/L (mean 0.0001 mg/L, 15 samples) for 11 locations for the same period. These ranges represent 45 detects out of a total of 1,500 samples (Health Canada 2009). One municipal treated groundwater sample taken near a leaky underground storage tank contained 1.7 mg/L.

### Removal methods

Removal of benzene from contaminated source waters can be achieved through adsorption on to granular activated carbon and by air stripping. High doses of ozone can be effective too.

WRF (2014) reports that benzene is characterised with a moderate Henry’s law constant (0.171 dimensionless air/water at 20°C). Low profile air stripping is very effective for benzene removal even at low temperatures and low air to water ratios (below 100). This VOC exhibited 100 percent removal rates at the three temperatures and air to water ratio of about 150. The very high removal efficiency of 99.6 and 99.8 was achieved at the relatively low air to water ratio of about 70 and temperatures of 4°C and 12°C. At the low air to water ratio of 53, the temperature effect on benzene removal became clear with 99.6 percent removal at 20°C, 99.18 percent removal at 12°C, and a lowest removal efficiency of 95.3 percent, achieved at the worst case scenario of 4°C.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

Most benzene enters the body by inhalation, perhaps up to 99 percent of the total intake. About half of the exposure to benzene in the United States results from smoking tobacco or from exposure to tobacco smoke. IARC (2012) states: Amounts of benzene measured per cigarette ranged from 5.9 to 75 μg in mainstream smoke and from 345 to 653 μg in sidestream smoke.

Benzene can be formed at very low levels in beverages that contain both ascorbic acid (vitamin C) and sodium benzoate (see benzyl benzoate datasheet). Sodium benzoate is a permitted food preservative that may be added to many food products to ensure the microbiological safety of the food. Ascorbic acid is also an approved food additive (antioxidant) which may be added to drinks. It also occurs naturally in fruit and fruit juices. Ascorbic acid reacts with metals (copper, iron) found in water to form hydroxyl radicals, which react with benzoic acid to form low levels of benzene. FSANZ (2007) sampled 68 flavoured beverages in March/April 2006. Of the 68 samples tested, 38 beverage products contained trace levels of benzene. The levels detected ranged from 1 to 40 ppb. More than 90 percent of all beverages surveyed had levels of benzene below the WHO guidelines for drinking water (10 ppb).

Following ingestion, almost all of the benzene is absorbed from the gastrointestinal tract. Benzene is also rapidly and efficiently absorbed following inhalation. Less than 1 percent is absorbed through the skin. Following absorption, benzene is widely distributed throughout the body, independent of the route of administration. It is metabolised predominantly into phenol by the liver, and also by bone marrow.

Benzene has a low acute toxicity. Acute exposure of humans to high concentrations of benzene primarily affects the central nervous system. In fatal cases, extensive haemorrhages have been observed. There is considerable evidence that exposure to high benzene concentrations may eventually result in leukaemia (acute myelogenous leukemia or AML).

Chronic benzene toxicity has been attributed to the formation of reactive metabolites that appear to exert their toxic effect in combination, with no one metabolite accounting for all of the observed effects. The principal toxic effect following repeated exposure to low levels of benzene is in the blood and blood-forming tissues including bone marrow.

Although benzene does not induce mutations or DNA damage in standard bacterial assay systems, it has caused chromosomal aberrations in a variety of species *in vivo*.

Benzene is carcinogenic in mice and rats after inhalation and oral exposure, producing malignant tumours at many sites. It is considered as a human carcinogen and is classified by the International Agency for Research (IARC 2002) on Cancer in Group 1 (carcinogenic to humans). USEPA (2009) states that a concentration of 0.1 mg/L benzene represents a 10-4 cancer risk.

Benzene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at July 2013 ATSDR quotes a minimal risk level (MRL) of 0.0005 mg/kg/day for intermediate-duration oral exposure (15–364 days) to benzene.

The reference dose or RfD (USEPA 2002, 2006, 2009 and 2011) is 0.004 mg/kg/d, based on an equivalent oral dose rate of 1.2 mg/kg/day and an uncertainty factor of 300. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.1 mg/L.

### Derivation of Maximum Acceptable Value

Owing to the unequivocal evidence of the carcinogenicity of benzene to humans and animals and its documented chromosomal effects, quantitative risk assessment was used to calculate lifetime cancer risks. Based on a risk estimate using data on leukaemia from epidemiological studies involving inhalation exposure, it was calculated that a drinking-water concentration of 0.01 mg/L was associated with a lifetime excess cancer risk of one per one hundred thousand (10-5).

As data on the carcinogenic risk to humans following ingestion of benzene are not available, risk estimates were also carried out on the basis of a two-year gavage study in rats and mice. The robust linear extrapolation model was used, as there was a statistical lack of fit of some of the data with the linearised multistage model. The estimated range of concentration in drinking-water corresponding to a excess lifetime cancer risk of 10-5, based on leukaemia and lymphomas in female mice and oral cavity squamous cell carcinomas in male rats, is 0.01–0.08 mg/L. The lower end of this estimate corresponds to the estimate derived from epidemiological data, which formed the basis for the previous MAV of 0.01 mg/L associated with a 10-5 lifetime cancer risk. The MAV corresponding to an excess cancer risk of 10-5 is therefore 0.01 mg/L.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic and subchronic health risk limits (exposure greater than 10 percent of a lifetime) for benzene are 0.003 mg/L; the acute and short-term limits (one day exposure) are 0.01 mg/L, and the limit for cancer is 0.002 mg/L.

Most people can begin to taste benzene in water at 0.5–4.5 mg/L.

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# Benzo[a]anthracene

Benzo[a]anthracene, CAS No. 56-55-3, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. Also called benzo(α)anthracene and sometimes benz(a)anthracene.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; benzo[a]pyrene (BaP) levels range from 6 to 35 mg/kg TEQ depending on land use. The equivalent BaP concentration is calculated as the sum of each of the detected concentrations of nine carcinogenic PAHs which are benz(a)anthracene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene and indeno(1,2,3-cd) pyrene, multiplied by their respective potency equivalency factors.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 and 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. Benzo[a]anthracene was detected in four of the New Zealand samples with concentrations ranging from 0.0000012–0.0000031 mg/L (1.2–3.1 ng/L).

Five water utilities in the US reported detecting benzo[a]anthracene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00067 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation. Pyrene was the most rapidly degraded PAH. Benz[a]anthracene, benzo[a]pyrene, and perylene were also highly degraded. Indeno[1,2,3-c,d]pyrene and benzo[g,h,i]pyrene were intermediate with respect to relative degradation. Benzo[k]fluoranthene and fluoranthene were the most slowly degraded of the compounds tested. A variety of complex end-products have been identified (ATSDR 1995).

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess. The relative carcinogenic potencies of various polynuclear aromatic hydrocarbons, based on bioassays by several routes of administration and related toxicological data, have been ranked in decreasing order as follows: dibenz[a,h]anthracene, benzo[a]pyrene, anthanthrene, indeno[1,2,3-cd]pyrene, benz[a]anthracene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, benzo[j]fluoranthene, cyclopentadieno[c,d]pyrene, benzo[g,h,i]perylene, chrysene and benzo[e]pyrene.

IARC (2010) classified benz[*a*]anthracene in Group 2B (possible human carcinogen).

Benzo[a]anthracene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

No MAV.

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# Benzo 3,4-fluoranthene

Benzo 3,4-fluoranthene, CAS No. 205-99-2, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. Also called benzo[b]fluoranthene and 3,4‑benzofluoranthene.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. From treatment processes

No known sources.

#### 2. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

Octanol/water partition coefficient as log Pow: 6.12.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 and 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. Sixteen New Zealand samples contained detectable concentrations of benzo[b]fluoranthene. Detected concentrations ranged from 0.0000001–0.00000099 mg/L (0.1–0.99 ng/L).

Eleven water utilities in the US reported detecting benzo[b]fluoranthene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00063 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess. The relative carcinogenic potencies of various polynuclear aromatic hydrocarbons, based on bioassays by several routes of administration and related toxicological data, have been ranked in decreasing order as follows: dibenz[a,h]anthracene, benzo[a]pyrene, anthanthrene, indeno[1,2,3-cd]pyrene, benz[a]anthracene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, benzo[j]fluoranthene, cyclopentadieno[c,d]pyrene, benzo[g,h,i]perylene, chrysene and benzo[e]pyrene.

IARC (2010) classified benzo[*b*]fluoranthene in Group 2B (possible human carcinogen).

Benzo[b]fluoranthene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

No MAV.

### References

ATSDR. 1995. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs)*. Atlanta, GA: Agency for Toxic Substances & Disease Registry, Department of Health and Human Services. See: <http://www.atsdr.cdc.gov/toxprofiles/index.asp>.

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# Benzo 11,12-fluoranthene

Benzo 1,12-fluoranthene, CAS No. 207-08-9, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. Also called benzo[k]fluoranthene and 11,12-benzofluoranthene.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

The principal route of entry to source water is via atmospheric deposition. Based on the literature reviewed, the presence of chrysene and benzo(k)fluoranthene may be indicators for coal combustion emissions (Environment Australia 2003).

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; benzo[a]pyrene (BaP) levels range from 6 to 35 mg/kg TEQ depending on land use. The equivalent BaP concentration is calculated as the sum of each of the detected concentrations of nine carcinogenic PAHs which are benz(a)anthracene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene and indeno(1,2,3-cd) pyrene, multiplied by their respective potency equivalency factors.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 and 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. Twenty-seven New Zealand samples contained detectable concentrations of benzo[k]fluoranthene. Detected concentrations ranged from 0.00000003–0.00000042 mg/L (0.03–0.42 ng/L).

Eight water utilities in the US reported detecting benzo[k]fluoranthene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00063 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation. Pyrene was the most rapidly degraded PAH. Benz[a]anthracene, benzo[a]pyrene, and perylene were also highly degraded. Indeno[1,2,3-c,d]pyrene and benzo[g,h,i]pyrene were intermediate with respect to relative degradation. Benzo[k]fluoranthene and fluoranthene were the most slowly degraded of the compounds tested. A variety of complex end-products have been identified (ATSDR 1995).

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess. The relative carcinogenic potencies of various polynuclear aromatic hydrocarbons, based on bioassays by several routes of administration and related toxicological data, have been ranked in decreasing order as follows: dibenz[a,h]anthracene, benzo[a]pyrene, anthanthrene, indeno[1,2,3-cd]pyrene, benz[a]anthracene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, benzo[j]fluoranthene, cyclopentadieno[c,d]pyrene, benzo[g,h,i]perylene, chrysene and benzo[e]pyrene.

IARC (2010) classified benzo[*k*]fluoranthene in Group 2B (possible human carcinogen).

Benzo[k]fluoranthene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Benzo 1,12-perylene

Benzo 1,12-perylene, CAS No. 191-24-2, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. Also called (benzo[g,h,i]perylene).

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

Whereas other PAHs are indicators of other combustion process, benzo(g,h,i)perylene is an indicator for motor vehicle emissions (Environment Australia 2003). It has also been used to make bile acids, cholesterol and steroids.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 and 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. Benzo(g,h,i)perylene was not detected. A number of PAHs have been assessed in Phase 1 of the P2 Programme. The concentration of benzo 1,12-perylene was less than 0.00000046 mg/L (0.46 ng/L) in all the New Zealand samples. With the exception of fluoranthene, none have been detected.

Eight water utilities in the US reported detecting benzo[g,h,i]perylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00047 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation. Pyrene was the most rapidly degraded PAH. Benz[a]anthracene, benzo[a]pyrene, and perylene were also highly degraded. Indeno[1,2,3-c,d]pyrene and benzo[g,h,i]pyrene were intermediate with respect to relative degradation. Benzo[k]fluoranthene and fluoranthene were the most slowly degraded of the compounds tested. A variety of complex end-products have been identified (ATSDR 1995).

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess. The relative carcinogenic potencies of various polynuclear aromatic hydrocarbons, based on bioassays by several routes of administration and related toxicological data, have been ranked in decreasing order as follows: dibenz[a,h]anthracene, benzo[a]pyrene, anthanthrene, indeno[1,2,3-cd]pyrene, benz[a]anthracene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, benzo[j]fluoranthene, cyclopentadieno[c,d]pyrene, benzo[g,h,i]perylene, chrysene and benzo[e]pyrene.

IARC (2010) classified benzo[*ghi*]perylene in Group 3 (not classifiable as to carcinogenicity).

### Derivation of Maximum Acceptable Value

No MAV.

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# Benzo[a]pyrene

CAS No. 50-32-8. Also called benzo 3,4-pyrene, 3,4-benzopyrene, 6,7-benzopyrene, benzpyrene, benzo[*def*]chrysene, B[α]P and BaP. Refer also to datasheet for polynuclear aromatic hydrocarbons.

### Maximum Acceptable Value

Based on health considerations, the concentration of benzo[a]pyrene in drinking-water should not exceed 0.0007 mg/L (0.7 g/L).

The maximum contaminant level (USEPA 2004/2009/2011) is 0.0002 mg/L. The maximum acceptable concentration in Canada is 0.00001 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of benzo(a)pyrene in drinking water should not exceed 0.00001 mg/L (10 ng/L, or 0.01 g/L).

The Prescribed Concentration or Value (PCV) for benzo[a]pyrene in England and Wales is 0.00001 mg/L, ie, 0.01 µg/L. See Notes.

Benzo[a]pyrene is one of the “priority pollutants” under the US Clean Water Act.

Benzo[a]pyrene is listed as a “priority contaminant” in the Ministry for the Environment’s *Toxicological Intake Values for Priority Contaminants in Soil* (MfE 2011).

### Sources to drinking-water

#### 1. To source waters

Polynuclear aromatic hydrocarbons comprise a large group of organic compounds formed from the incomplete combustion of organic matter, of which benzo[a]pyrene (BaP) has received the most extensive toxicological study. They have no industrial use but are formed naturally in forest fires, volcanic activity, or from anthropogenic activities such as domestic fires, vehicle emissions (especially from diesel engines), coke ovens, the coal gas industry and related contaminated soils, and aluminium smelters. The principal route of entry to source water is via atmospheric deposition. There is no quantitative relation between measured BaP and concentrations of any other PAH; however, if BaP is found, other PAHs are probably also present.

As both roasted and instant coffee are derived by roasting procedures (at temperatures of the order of 210°C), polycyclic aromatic hydrocarbons (PAHs), and in particular benzo(a)pyrene, can be expected to be present. Determinations in roasted coffee suggest a range of about 0.1 to 1.2 µg/kg, with an average of 0.3 µg/kg (IARC 1991).

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; benzo[a]pyrene levels range from 6 to 35 mg/kg TEQ depending on land use. The equivalent BaP concentration is calculated as the sum of each of the detected concentrations of nine carcinogenic PAHs which are benz(a)anthracene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene and indeno(1,2,3-cd) pyrene, multiplied by their respective potency equivalency factors.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar linings of water distribution systems. The presence of significant concentrations of BaP in drinking-water in the absence of very high concentrations of fluoranthene indicates the presence of coal-tar particles, which may arise from seriously deteriorating coal-tar pipe linings. It is recommended that the use of coal-tar-based and similar materials for pipe linings and coatings on storage tanks be discontinued; its use in New Zealand is minimal.

### Forms and fate in the environment

Polynuclear aromatic hydrocarbons enter the environment through atmospheric deposition. Because of its low water solubility (about 0.002 mg/L at 25°C) benzo[a]pyrene adsorbs to sediments and suspended solids in aquatic systems. It does not hydrolyse. In some natural waters there are organisms capable of metabolising it, but in most waters it does not undergo biodegradation. Volatilisation may be important over periods exceeding 1 month, and if near the surface of waters, it is likely to undergo significant photodegradation. Losses through volatilisation, photodegradation and biodegradation are retarded by adsorption on to solids.

If released to soil, benzo(a)pyrene is expected to have very low to no mobility based upon measured soil Koc values of 930 to 6,300. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon a Henry’s Law constant of 4.57 x 10-7 atm‑cu m/mole. The persistence of benzo(a)pyrene in soil is expected to vary depending upon the nature of compounds accompanying it and the nature and previous history of the soil; biodegradation half-lifes of 309 and 229 days were observed in Kidman and McLaurin sandy loam soils, respectively. If released into water, benzo(a)pyrene is expected to adsorb to suspended solids and sediment based upon the measured Koc values. Biodegradation of benzo(a)pyrene is expected to occur in aquatic systems; the removal of 64 percent of benzo(a)pyrene over 36 days in an activated sludge pilot reactor was attributed to biodegradation. Volatilisation from water surfaces is not expected to be an important fate process based on its Henry’s Law constant. Measured BCF values ranging from 8.7 to 1 x 10+5 suggest bioconcentration in aquatic organisms can be low to very high. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

Log Kow (octanol–water): 6.35. Henry’s Law Constant: 0.034 Pa m3/mol at 20°C (IARC 2012).

Model calculations indicate that B[a]P persists in soil with half-lifes in the range from 420 to 1250 days. The calculated half-lifes of B[a]P in water and sediments are in the range of 42 to 125 days and >1250 days respectively (ECHA 2016).

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 to 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. Ten samples contained detectable concentrations of benzo[a]pyrene. Detected concentrations ranged from 0.00000004–0.00000086 mg/L (0.04–0.086 ng/L).

The P2 Chemical Determinand Identification Programme, sampled from 346 zones, did not find any benzo[a]pyrene concentrations (limit of detection = 0.0001 mg/L) (ESR 2001).

Ninety-five water utilities in the US reported detecting benzo[a]pyrene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest being 0.003 mg/L.

### Removal methods

Benzo[a]pyrene, like the polynuclear aromatic hydrocarbons in general, is very insoluble in water and hence adsorbs readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which it may be adsorbed, and by providing floc surfaces on to which benzo[a]pyrene in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

Chlorination can reduce benzo[a]pyrene concentrations by approximately 60 percent after three hour contact time. Oxidation by ozone results in a much more rapid reduction in benzo[a]pyrene concentration; 100 percent destruction is achieved in less than 15 minutes.

As polynuclear aromatic hydrocarbons can be leached from coal-tar lined pipes, this surface covering should not be used for pipes in the treatment plant or reticulation.

### Analytical methods

#### Referee method

Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry (EPA 525).

#### Some alternative methods

1. Liquid–Liquid Extraction and HPLC with Coupled Ultraviolet and Fluorescence Detection (EPA 550).

2. Liquid-Solid Extraction and HPLC with Coupled Ultraviolet and Fluorescence Detection (EPA 550.1).

3. Liquid–Liquid Extraction with Gas Chromatography/Mass Spectrometry (APHA 6410B).

### Health considerations

Drinking water is estimated to account for only 0.1 to 0.3 percent of the total BaP ingested. Air is estimated to contribute about 0.9 percent of the total exposure, and the greatest source is foods, which contribute 99 percent.

Benzo[a]pyrene is absorbed principally through the gastrointestinal tract and the lungs. Absorbed benzo[a]pyrene is rapidly distributed to the organs and tissues and may be stored in mammary and fatty tissues. It crosses the placenta and is distributed in the developing tissue. Metabolism of benzo[a]pyrene occurs primarily in the liver. Benzo[a]pyrene metabolites are eliminated primarily in the faeces, with minor amounts excreted in urine.

Benzo[a]pyrene has also been detected in tobacco smoke, reported to range from 52 to 95 ng/cigarette, more than three times the concentration in mainstream smoke. Human subjects skin painted with benzo[a]pyrene developed skin lesions. Occupations associated with exposures to polynuclear aromatic hydrocarbons, of which benzo[a]pyrene is a component, have been associated clearly with human cancer.

Benzo[a]pyrene has been shown to be mutagenic in tests with a strain of bacteria, however the diol-epoxide metabolite was considerably more mutagenic than the parent compound.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The acute limit is 0.002 mg/L, short-term, subchronic, chronic values are 0.0003 mg/L, and a limit of 0.00006 mg/L was set for cancer.

The health effect of primary concern is carcinogenicity. Benzo[a]pyrene is one of the most potent carcinogens amongst the PAHs that have been tested to-date. The International Agency for Research on Cancer had previously classified benzo[a]pyrene in Group 2A (probably carcinogenic to humans). Note that in IARC (2005 and 2012), benzo[a]pyrene appears in Group 1 (carcinogenic to humans).

USEPA (2009/2011) has a health advisory of 0.0005 mg/L benzo[a]pyrene, representing a 10-4 cancer risk. USEPA (2017) describes benzo[a]pyrene as “carcinogenic to humans” based on strong and consistent evidence in animals and humans; the RfD was calculated at 0.0003 mg/kg/d.

Benzo[a]pyrene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

MfE (2011) states:

Benzo(a)pyrene is considered to be a genotoxic carcinogen, and therefore is a non-threshold contaminant. An oral-risk-specific dose of 0.0048 µg/kg bw/day (slope factor of 2.08 per mg/kg bw/day) is recommended for use. This value is the geometric mean of 14 BMDL10 estimates from four studies divided by 10,000 and allometric scaling, maximising the use of available data. A dermal absorption of 0.026 (2.6 percent) is recommended for use. BaP is considered representative of a range of carcinogenic polycyclic aromatic hydrocarbons (PAHs), and potency equivalence factors (PEF) are used to estimate the potential carcinogenicity of environmental PAH mixtures. A consistent set of PEFs is recommended to enable assessment of potential carcinogenicity of PAH mixtures through comparison with a BaP-equivalent soil contaminant standard in New Zealand. Further, it is recommended that the range of PAHs routinely analysed is expanded to include additional PAHs considered carcinogenic by FAO/WHO.

Benzo[a]pyrene is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

ECHA (2016) identifies B[a]P as a substance of very high concern owing to its classification in the hazard class carcinogenicity category 1A or 1B, the hazard class germ cell mutagenicity category 1A or 1B, the hazard class reproductive toxicity category 1A or 1B, and is identified as very persistent, very bioaccumulative and toxic.

### Derivation of Maximum Acceptable Value

Benzo[a]pyrene appears to be a local carcinogen in that it induces tumours at the site of administration. Administration of benzo[a]pyrene in the diet of mice resulted in an increased incidence of forestomach tumours. Owing to the unusual protocol followed in this study which involved variable dosing patterns and age of sacrifice, these data could not be extrapolated accurately using the linearised multistage model normally applied to the derivation of the MAVs for carcinogens.

However, an oral carcinogenicity quantitative risk assessment was conducted using the two-stage birth-death mutation model and incorporating variable dosing patterns and time of sacrifice. Using this model the estimated concentration of benzo[a]pyrene in drinking-water corresponding to an excess life-time cancer risk of one per 100,000 (10‑5) is 0.0007 mg/L (0.7 g/L) (WHO 2017). Data on forestomach tumour incidence in mice gives nearly identical results, giving support for the validity of the aforementioned study.

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# Benzophenone

CAS No. 119-61-9. Also called benzoylbenzene; phenyl ketone; diphenylketone; diphenylmethanone; α-oxodiphenylmethane; α-oxoditane.

### Maximum Acceptable Value

The is no MAV for benzophenone in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Benzophenone is used as a flavour ingredient, a fragrance enhancer, a perfume fixative and an additive for plastics, coatings and adhesive formulations; it is also used in the manufacture of insecticides, agricultural chemicals, hypnotic drugs, antihistamines and other pharmaceuticals. Benzophenone is used as an UV-curing agent in sunglasses, and to prevent UV light from damaging scents and colours in products such as perfumes and soaps, and in sunscreen. Moreover, it can be added to plastic packaging as a UV blocker, which allows manufacturers to package their products in clear glass or plastic rather than opaque or dark packaging. It is also used in laundry and household cleaning products. Benzophenone is widely used as a photoinitiator for inks and varnishes that are cured with UV light. In addition to being a drying catalyst, benzophenone is an excellent wetting agent for pigments; it can also be used in printing to improve the rheological properties and increase the flow of inks by acting as a reactive solvent.

According to the USEPA, benzophenone was classified in 2003 as a high volume chemical, with an annual production exceeding 1 million pounds (453,000 kg), in the USA.

Benzophenone was reported to occur naturally in wine grapes (*Vitis vinifera* L.) at concentrations of 0.08–0.13 mg/kg.

Benzophenones have been found in surface waters at more than 0.002 mg/L.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Benzophenones in general have the environmentally critical properties of high lipophilicity and persistence, and are known to have adverse effects on the reproduction and hormonal functions of fish. Because of its high octanol:water partition coefficient and its insolubility in water, benzophenone partitions in soil and sediment, and its adsorption to soil is proportional to the organic content therein.

Water solubility: practically insoluble in water.

### Typical concentrations in drinking-water

The data on benzophenone in drinking-water are limited. Levels of 8.8 ppb (0.009 mg/L) were found in tap-water in Japan, and 0.26 μg/L in finished drinking-water in a water filtration plant in the USA in 2001/02 (DWI 2014).

### Removal methods

Because benzophenone partitions to soil and sediment, treatment processes that remove particulate matter will reduce its concentration.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Based on its concentration in muscat grapes, the Working Group estimated that consumption of 200 g of grapes would result in exposure to approximately 0.02 mg benzophenone, ie, 0.3 μg/kg body weight for a 60‑kg adult. In the USA, the average reported levels of use of benzophenone as an additive range from 0.57 mg/L in non-alcoholic beverages to 1.57 mg/kg in baked goods, and maximum reported levels range from 1.28 mg/L in non-alcoholic beverages to 3.27 mg/kg in frozen dairy products. Other reported uses are in soft candy, gelatins and puddings.

Benzophenone was not mutagenic in *Salmonella* and did not induce micronuclei in mice. Benzophenone and its metabolites induced *umu* gene expression, an indication of DNA damage, in *Salmonella* in the presence of *Escherichia coli* membranes expressing recombinant human cytochrome P450s. The benzophenone metabolite, 4‑hydroxybenzophenone, elicits estrogenic activity and anti-androgenic activity *in vitro*, and the *in vivo* estrogenic activity of benzophenone has been confirmed in multiple uterotrophic assays. Benzophenone may alter endocrine signalling through multiple effects on receptors. The mechanistic evidence for tumour induction by benzophenone is weak, but the relevance of the tumour response in experimental animals to humans cannot be excluded.

IARC (2012) concluded that there is sufficient evidence in experimental animals for the carcinogenicity of benzophenone, so benzophenone is possibly carcinogenic to humans (Group 2B).

EFSA (2009) states: For the current opinion the Panel after having examined the toxicological data on benzophenone considers that the liver hypertrophy seen in rat in the two-generation study is an adaptive response and not an adverse response. Therefore the Panel considered the EFSA approach to derive a LOAEL of 6 mg/kg b.w. per day based on this effect as conservative. Due to the limited amount of time available to EFSA for the release of its statement the Panel considered this conservative approach reasonable. The Panel derives a TDI of 0.03 mg/kg b.w. per day for benzophenone based on the BMDL10 value of 3.1 mg/kg b.w. per day following BMD analyses on the non-neoplastic kidney effects in male rats.

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2014. *Risks to Drinking Water from Personal Care Products and Domestic Cleaning Products*. WRc Ref: DWI9879.03. Report No: DWI9879.03. 121 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-283.pdf>.

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# Benzotriazoles

Three common benzotriazoles are:

CAS No. 95-14-7: 1-H-benzotriazole

CAS No. 29385-43-l: 1-H-benzotriazole, 4 (or 5) methyl

CAS No. 64665-57-2: 1-H-benzotriazole, 4 (or 5) methyl, sodium salt

The sodium salt will dissociate to the methyl benzotriazole in aqueous solution.

### Maximum Acceptable Value

There is no MAV for the benzotriazoles in the DWSNZ, and they are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Benzotriazoles contain a five-membered ring with three nitrogen atoms directly bonded to one another as substituents on a benzene ring. The compounds called methyl (or tolyl) derivatives have a methyl substituent on the benzene ring. Many other derivatives are possible and a number have been used in various applications.

There are three primary uses for benzotriazoles: corrosion inhibitor (typically used as trace additives in industrial chemical mixtures such as coolants, deicers, surface coatings, cutting fluids, and hydraulic fluids), ultraviolet light stabiliser for plastics, and antifoggant in photography. Because benzotriazoles are used in large quantities as a corrosion inhibitor, it is mainly through this type of use that they become an environmental contaminant. As a corrosion inhibitor (mainly for copper, including under water) and fire retardant, they are used in antifreeze in concentrations of  
0.01–2.0 percent and in airplane deicing/anti-icing fluids. Used antifreeze may leak or be poured down drains and thence enters the environment. Also, an estimated 80 percent of aircraft deicing/anti-icing fluids are deposited on the ground due to spray drift, jet blast, and wind shear during taxiing and takeoff. Taken from Wu et al (1998). Benzotriazole is detected in the groundwater below de-icing platforms at several international airports.

There has been a suggestion that benzotriazoles be used as inhibitors to economise N fertilisers by helping prevent leaching and denitrification.

DWI (2014) identified benzotriazoles as a risk to drinking water from personal care products and domestic cleaning products, eg, dishwasher cleaning products. About 1,000 tonnes pa are used in the UK. From 0.005 to 0.01 mg/L have been detected in surface water and sewage.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Due to resistance of benzotriazoles to oxidation under environmental conditions, and the UV stability of the compounds, benzotriazoles may persist in the environment for a very long time. USEPA (2001) states all benzotriazoles have degradation rates ≥70 percent after 28 days.

Breedveld et al (2003) found high concentrations of benzotriazole in the groundwater below the de-icing pad, regeneration plant and snow disposal site one to two years after de-icing activities had ceased at an abandoned airport. Propylene glycol is the de‑icing component (see ethylene glycol datasheet); benzotriazoles are added at about 0.5 percent to assist controlling viscosity, corrosion and flammability.

Water solubility:

1-H-benzotriazole: 1–5 mg/L at 24°C

1-H-benzotriazole, 4 (or 5) methyl: <100 mg/L at 18°C

1-H-benzotriazole, 4 (or 5) methyl, sodium salt: 55 percent at 20°C.

### Typical concentrations in drinking-water

DWI (2014) quotes a reference where 0.008 mg/L benzotriazoles were found in drinking water in the UK.

### Removal methods

No significant removal by chemical coagulation. Poor removal by PAC. Not all benzotriazole is removed during drinking water treatment by ozone (DWI 2014).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Benzotriazoles are of low concern for aquatic and mammalian toxicity. USEPA (2001) quotes a NOAEL for 1-H-benzotriazole, 4 (or 5) methyl of 150 mg/kg/d, based on oral testing on rats for 29 days.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Breedveld GD, et al. 2003. Persistence of the de-icing additive benzotriazole at an abandoned airport. *Water, Air and Soil Pollution: Focus* 3: 91–101. http://link.springer.com/article/10.1023%2FA%3A1023961213839#page-2.

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Wu X, Chou N, Lupher D, et al. 1998. Benzotriazoles: toxicity and degradation. *Proceedings of the 1998 Conference on Hazardous Waste Research* 374–82. https://www.engg.ksu.edu/HSRC/98Proceed/32Wu/32wu.pdf.

# Benzyl benzoate

CAS No. 120-51-4. The IUPAC name is benzyl benzoate. The CAS name is phenylmethyl benzoate. Also called benzyl benzenecarboxylate, benzoic acid (phenylmethyl ester), and benzyl phenylformate. It has many trade names.

This datasheet includes information on the related compounds:

benzoic acid (CAS No. 65-85-0) and its potassium (CAS No 582-25-2) and sodium (CAS No. 532-32-1) salts

benzyl alcohol (CAS No. 100-51-6)

benzaldehyde (CAS No. 100-52-7)

### Maximum Acceptable Value

The is no MAV for any of these compounds in the DWSNZ, and they are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Benzyl benzoate used to flavour foods, in perfumes, as a [plasticiser](http://en.wikipedia.org/wiki/Plasticizer) in [cellulose](http://en.wikipedia.org/wiki/Cellulose) and other [polymers](http://en.wikipedia.org/wiki/Polymer), and is a solvent, a bridged diphenyl acaricide, insecticide and fungicide used on carpets, mattresses, upholstery, and on furniture, and for control of mites on dogs.

Benzyl benzoate appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)).

Benzyl benzoate can be used without a prescription to treat lice and scabies infestations. Benzyl benzoate is believed to be absorbed by the lice and mites and to destroy them by acting on their nervous system.

Benzoates also occur commercially as benzoic acid (CAS No. 65-85-0) and the potassium (CAS No 582-25-2) and sodium (CAS No. 532-32-1) salts. OECD (2004) also covers benzyl alcohol (CAS No. 100-51-6) due to its similarity regarding human health. The major outlet (60 percent) for sodium benzoate is as a preservative in food and beverages. Benzoic acid occurs naturally in free and bound form in many plant and animal species, eg, many berries contain around 0.05 percent; see WHO (2000) for other details.

Benzoic acid, a bactericide, fungicide and virucide, is used as a food preservative and appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)) as a skin/coat conditioner; it can also be used as a fungicide in ointments, eg, for ringworm and tinea. It is also found in vehicle exhaust. Benzoic acid can also be called phenylformic acid and carboxybenzene. EFSA (2016) states that use of benzoic acid at the maximum level of 125 mg/kg complete feed is safe for all animal species, and the use of benzoic acid in animal nutrition will not pose a risk for the environment. EFSA (2017) found benzoic acid is safe at the supplementation level of 5,000 mg/kg complete feed for minor porcine species for fattening and for reproduction, and at 10,000 mg/kg complete feed for pigs for fattening, with a margin of safety of 1.5 (EFSA 2019).

Note that benzaldehyde (CAS No. 100-52-7) is used to impart an almond flavour in foods. It occurs naturally in several foods, particularly almonds. Benzaldehyde can be used as a bee repellent by apiarists.

For Australia, France, New Zealand, the UK and the USA, the food category that contributed the most to dietary exposure to benzoates was carbonated water-based flavoured drinks (GSFA food category 14.1.4.1), typically 100 to 300 mg/L of benzoate. In Finland, 40 percent of the benzoates used in food was in soft drinks. Soya sauce was the main source in China and the second most important source in Japan (WHO 2015).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Benzyl alcohol is a metabolite of benzyl benzoate which in turn is metabolised in experimental animals and humans to benzaldehyde and benzoic acid. Benzyl alcohol can also be called phenylmethanol, benzenemethanol, phenylcarbinol, benzoyl alcohol, alpha-toluenol, benzenecarbinol, hydroxytoluene, and phenylmethyl alcohol.

If released to soil, benzyl alcohol is expected to have very high mobility based upon a Koc range of <5 to 29. An experimentally derived first-order aerobic biodegradation rate constant of 0.05 days, corresponding to a half-life of about 13 days, suggests that biodegradation is an important environmental fate process. Volatilisation from moist soil surfaces is not expected to be an important fate process based on a Henry’s Law constant of 3.37 x 10-7 atm‑cu m/mole. Benzyl alcohol is not expected to volatilise from dry soil surfaces based upon a vapour pressure. If released into water, benzyl alcohol is not expected to adsorb to suspended solids and sediment based upon the Koc. Volatilisation from water surfaces is not expected to be an important fate process based on its Henry’s Law constant. An estimated BCF of 0.3 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

Above pH 6, the benzoic acid molecule dissociates to the benzoate anion. From their use patterns, it can be expected that benzoic acid and sodium benzoate will be released to surface waters and to leaching water (and groundwater). Only minor amounts are expected to be emitted to the atmosphere. Great variability in degradation of benzoic acid or benzoate is seen in tests using environmental matrices. It depends mainly on substance concentration and time for acclimation. Test durations exceeding two days resulted in removal of >40 percent when initial concentrations were below 20 mg/L. Rapid mineralisation occurred in groundwater and subsurface soil samples. In groundwater, a half-life of 41 h has been found for benzoic acid (initial concentration 1–0.1 mg/L; metabolised to CO2) under aerobic conditions. Anaerobic degradation requires a period of acclimation.

If released to soil, benzoic acid is expected to have very high mobility based upon an estimated Koc of 15. The pKa of benzoic acid is 4.20, indicating that it will exist almost entirely in the anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilisation from moist soil is not expected because the compound exists as an anion and anions do not volatilise. Benzoic acid is not expected to volatilise from dry soil surfaces based upon its vapour pressure. Mineralisation half-life in Captina silt loam for benzoic acid in solution was 4.5 hours after a 30-minute lag and complete degradation occurred in one day using a Niagra silt loam inoculum, suggesting that biodegradation may be an important fate process in soil. If released into water, benzoic acid is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Biodegradation half-lifes of 0.85 and 3.6 days using inoculum from a polluted river and a reservoir, respectively, suggest that biodegradation may be an important fate process in water. The pKa indicates benzoic acid will exist almost entirely in the anion form at pH values of 5 to 9 and, therefore, volatilisation from water or moist soil surfaces is not expected to be an important fate process. A BCF range of <10 to 21 suggests bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (pH 5 to 9) (EAWAG accessed February 2015). EC (2013) quotes the vapour pressure as 0.04–0.07 Pa at 20°C; the calculated pKa value as 4.19; and Henry’s law constant is calculated for the water solubility at pH 2.94 to be 0.0016-0.0029 Pa x m3 x mol-1.

If released to soil, benzaldehyde is expected to have very high mobility based upon an estimated Koc of 34. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 2.67 x 10-5 atm‑cu m/mole. Benzaldehyde may volatilise from dry soil surfaces based upon its vapour pressure. A number of biological screening studies have demonstrated that benzaldehyde is readily biodegradable. If released into water, benzaldehyde is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is expected to be an important fate process based upon this compound’s Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 1.5 and 14 days, respectively. An estimated BCF of 2.7 suggests the potential for bioconcentration in aquatic organisms is low. Five-day theoretical BODs of 77.2 percent and 63.5 percent were measured using the standard dilution method and seawater dilution method, respectively. Benzaldehyde had a five-day theoretical BOD of 36 percent using the AFNOR T test and inocula from three polluted surface waters. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015). Easily penetrates the soil to contaminate groundwater and nearby waterways. Water solubility about 7 percent.

Water solubilities:

benzyl benzoate is about 15 mg/L

benzoic acid: 2,900 mg/L

benzyl alcohol about 4 percent

the sodium and potassium salts of benzoic acid, about 50–60 percent

benzaldehyde: 6570 mg/L (ie, about 6.5 percent) (<http://www.rsc.org/learn-chemistry/wiki/Substance:Benzaldehyde>).

None of these is expected to hydrolyse. All are readily biodegradable. None has bioaccumulative potential (OECD 2004).

### Typical concentrations in drinking-water

Two water utilities in the US reported detecting benzoic acid (a degradation product) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.051 mg/L.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

For benzoic acid, see EFSA (2016a).

### Health considerations

Benzyl benzoate rapidly breaks down *in vivo* to benzoic acid and benzyl alcohol.

DEFRA (1995) quotes a NOAEL of 150 mg/kg/d for benzyl benzoate, based decreased urinary pH level in a 13-week repeated oral toxicity rat study.

No safety concern at current levels of intake of benzyl benzoate when used as a flavouring agent. The 1996 group ADI of 0–5 mg/kg bw for benzoic acid, the benzoate salts (calcium, potassium and sodium), benzaldehyde, benzyl acetate, benzyl alcohol and benzyl benzoate, expressed as benzoic acid equivalents, was maintained at the 57th meeting (2001) (IPCS 2003).

The RfD for benzaldehyde was calculated at 0.1 mg/kg/d (USEPA 1988). The RfD for benzoic acid was calculated at 4 mg/kg/d (USEPA 1993).

WHO (2000) states: JECFA (WHO, 1996) has allocated an acceptable daily intake (ADI) for benzoic acid and sodium benzoate of 0–5 mg/kg body weight.

OECD (2004) found benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values >1,000 mg/kg/day are obtained. For benzyl alcohol the long-term studies indicate a NOAEL >400 mg/kg bw/d for rats and >200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur. All chemicals showed no mutagenic activity in in vitro Ames tests. Various results were obtained with other in vitro genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity in vivo. While some mixed and/or equivocal in vitro chromosomal/chromatid responses have been observed, no genotoxicity was observed in the in vivo cytogenetic, micronucleus, or other assays. The weight of the evidence of the in vitro and in vivo genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies.

EC (2013) reports the medium and long term oral NOAELs of benzoic acid to be 500 mg/kg bw/d, with an ADI of 5 mg/kg/d using an uncertainty factor of 100; an ARfD was not necessary. EFSA (2016a and 2019) confirmed these values.

WHO (2015) refers to the upper bound of the ADI as 0.3–4.1 mg/kg bw per day (expressed as benzoic acid) for toddlers and young children, 0.2–2.7 mg/kg bw per day for other children including adolescents, and 0.1–1.7 mg/kg bw per day for adults.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The chronic health risk limit for benzoic acid is 30 mg/L.

USEPA (2007) states that benzyl benzoate was tested for mutagenicity and found negative in inducing unscheduled DNA synthesis in rat hepatocyte cultures, and it was not clastogenic nor did it cause mammalian cell gene mutations *in vitro*. Neither acute nor chronic RfDs are required. Benzyl benzoate is not expected to be carcinogenic. No cancer risk assessment is needed.

### Derivation of Maximum Acceptable Value

No MAV.

### References

DEFRA. 1996. *Evaluation on Benzyl Benzoate: Use as an acaricide*. York, UK: Department for Environment, Food and Rural Affairs. 44 pp. See: www.pesticides.gov.uk/PSD\_PDFs/159\_benzyl\_benzoate\_use\_as\_an\_acaricide.pdf.

EAWAG. Accessed February 2015. *Biocatalysis/Biodegradation Database: Select 1396 compounds*. <http://eawag-bbd.ethz.ch/index.html>.

EC. 2013. Benzoic acid. *Standing Committee on Biocidal Products Assessment Report*. 74 pp. <https://circabc.europa.eu/faces/jsp/extension/wai/navigation/container.jsp>.

EFSA. 2016. Safety and efficacy of benzoic acid as a feed additive for pigs for fattening when used as acidity regulator and all animal species when used as flavouring. *EFSA Journal* 14(1): 4353 [13 pp.]. <http://www.efsa.europa.eu/en/efsajournal/pub/4353>.

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EFSA. 2017. *Safety and Efficacy of VevoVitall® (Benzoic Acid) as Feed Additive for Minor Porcine Species* 15(10). <http://onlinelibrary.wiley.com/doi/10.2903/j.efsa.2017.5026/full>.

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IUCLID. 2000. *Benzyl Benzoate Dataset*. 40 pp. See: http://ecb.jrc.ec.europa.eu/iuclid-datasheet/120514.pdf.

IUPAC. Accessed 2009. *Benzyl Benzoate, Benzoic Acid Phenylmethyl Ester*. See: <http://sitem.herts.ac.uk/aeru/iupac/1473.htm>.

MDH. 2016. *Human Health-Based Water Guidance Table*. Minnesota Department of Health. <http://www.health.state.mn.us/divs/eh/risk/guidance/gw/table.html>.

NIH. Accessed 2017. Benzaldehyde. Compound summary for CID 240. *PubChem Open Chemistry Database*. National Center for Biotechnology Information. [https://pubchem.ncbi.nlm.nih.gov/compound/benzaldehyde#section=Top](https://pubchem.ncbi.nlm.nih.gov/compound/benzaldehyde%23section=Top).

OECD. 2004. *SIDS Initial Assessment Report: Benzoates* 320 pp. See: http://www.inchem.org/documents/sids/sids/BENZOATES.pdf or <http://www.inchem.org/pages/sids.html>.

USEPA. 1988. Benzaldehyde. *Integrated Risk Information System (IRIS)*. http://www.epa.gov/iris/subst/0332.htm.

USEPA. 1993. Benzoic acid. *Integrated Risk Information System (IRIS)*. http://www.epa.gov/iris/subst/0355.htm.

USEPA. 2007. *Reregistration Eligibility Decision (RED) for Benzyl Benzoate (Benzoic Acid case 4013)* [27 pp]. Also *Benzyl Benzoate: Risk assessment – Preliminary (Phase 1) HED Chapter of the Re-registration Eligibility Decision Document (RED)* [33 pp]. <http://www.epa.gov/pesticides/reregistration/benzoic_acid/>.

WHO. 2000. Benzoic acid and sodium benzoate. *Concise International Chemical Assessment Document (CICAD)* 26. International Programme on Chemical Safety (IPCS) Geneva. <http://www.inchem.org/pages/cicads.html>.

WHO. 2015. *Safety evaluation of certain food additives and contaminants*. *WHO Food Additives Series*: 71. Prepared by the 80th meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). 142 pp. <http://www.who.int/foodsafety/publications/monographs/en/>.

# 2-benzyl-4-chlorophenol

CAS No. 120-32-1. Also called chlorophene (not to be confused with chloroprene), clorophene, 4-chloro-2-benzylphenol, o-chloro-p-benzylphenol, o-benzyl p‑chlorophenol, and 5-chloro-2-hydroxydiphenylmethane.

### Maximum Acceptable Value

There are insufficient data to derive a MAV for 2-benzyl-4-chlorophenol in drinking-water. The WHO Guidelines do not refer to 2-benzyl-4-chlorophenol.

### Sources to drinking-water

#### 1. To source waters

2-benzyl-4-chlorophenol is used in cosmetics and deodorants. It has been approved by the EU as a preservative in cosmetics at levels up to 0.3 percent, and ERMA allows up to 0.2 percent (Schedule 7: Preservatives Cosmetic Products May Contain with Restrictions).

2-benzyl-4-chlorophenol is also used in a large range of domestic, agricultural, industrial and commercial disinfectants (eg, lysols) with good, broad spectrum activity, usually when mixed with other phenolic compounds. It is also an active ingredient in pine oil.

2-benzyl-4-chlorophenol appeared in the “List of active ingredients to be removed” in July 2003 under Directive 91/414/EEC.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Disposal of o-benzyl-p-chlorophenol occurs primarily in municipal wastewater treatment plants, where bio-degradation removes 95 percent of the o-benzyl-p‑chlorophenol; it also degrades rapidly in the environment. Measured influent and effluent concentrations from 16 sites in the US averaged 0.015 mg/L and 0.0008 mg/L respectively.

Water solubility is about 70 mg/L (moreso as the pH increases).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Under the conditions of two-year gavage studies, there was [no evidence](http://ntp.niehs.nih.gov/index.cfm?objectid=07027D0E-E5CB-050E-027371D9CC0AAACF#CARCDEF) of carcinogenic activity of o-benzyl-p-chlorophenol in male F344/N rats receiving 30, 60, or 120 mg/kg body weight. There was [equivocal evidence](http://ntp.niehs.nih.gov/index.cfm?objectid=07027D0E-E5CB-050E-027371D9CC0AAACF#CARCDEF) of carcinogenic activity of o-benzyl-p-chlorophenol in female F344/N rats based on the occurrence of two rare renal transitional cell carcinomas. There was [some evidence](http://ntp.niehs.nih.gov/index.cfm?objectid=07027D0E-E5CB-050E-027371D9CC0AAACF#CARCDEF) of carcinogenic activity of o-benzyl-p-chlorophenol in male B6C3F1 mice based on increased incidences of renal tubule adenoma and renal tubule adenoma or carcinoma (combined). There was [no evidence](http://ntp.niehs.nih.gov/index.cfm?objectid=07027D0E-E5CB-050E-027371D9CC0AAACF#CARCDEF) of carcinogenic activity of o-benzyl-p-chlorophenol in female B6C3F1, mice receiving 120, 240, or 480 mg/kg.

o-Benzyl-p-chlorophenol was nephrotoxic for male and female F344/N rats and B6C3F1 mice. The severity of nephropathy was increased in male and female rats and the incidence and severity of nephropathy was increased in male and female mice. The incidence and severity of nephropathy increased with length of treatment. Other lesions considered to be associated with the nephropathy and the secondary hyperparathyroidism in male rats and in male and female mice included fibrous osteodystrophy and soft tissue mineralisation. Increased incidences of squamous cell hyperplasia of the forestomach were observed in mice (NTP 1994). o-Benzyl-p-chlorophenol has been classified by the USEPA as a Group C, possible human carcinogen.

### Derivation of Maximum Acceptable Value

The are insufficient data to derive a MAV for 2-benzyl-4-chlorophenol at this time.

### References

NTP. 1994. *NTP Technical Report on the Toxicology and Carcinogenesis Studies of o‑benzyl-p-chlorophenol (CAS No. 120-32-1) in F344/N Rats and B6C3F1 Mice*. NTP TR 424. US Department Health and Human Services. National Institutes of Health. See: <http://ntp.niehs.nih.gov/index.cfm?objectid=0709C5FC-AE98-D164-0DB806C8F6094853>.

USEPA. 1995. *Reregistration Eligibility Decision ortho-benzyl-p-chlorophenol*. Environmental Protection Agency, Office of Pesticide Programs, Special Review and Reregistration Division. 141 pp. See: <http://www.epa.gov/oppad001/reds_list_ad.htm> or http://www.epa.gov/oppsrrd1/REDs/2045red.pdf.

# 4-benzylphenol

CAS No. 101-53-1, and 7563-63-5 for the sodium salt. Also called p-benzylphenol, p‑benzyl phenol, α-phenyl-p-cresol, 4-hydroxydiphenylmethane, 4-hydroxyditane; the IUPAC name is 4-(phenylmethyl)phenol.

### Maximum Acceptable Value

Neither WHO (2004) nor the DWSNZ refer to 4-benzylphenol.

### Sources to drinking-water

#### 1. To source waters

4-benzylphenol is used as a germicide, antiseptic, preservative; also used in organic syntheses. Human exposure is expected through presence in consumer goods, mainly food packaging, hence high exposure concern (EC 2002).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

EC (2002) described p-benzylphenol as moderately persistent. Release to soil will lead to adsorption, based on the high KOC. Volatilisation from water is expected to be low, based on a low Henry’s law constant. Because bioconcentration is only moderate, p‑benzylphenol will tend to stay in the water, and degrade there. The high KOC predicts that adsorption to suspended matter is an important mechanism for removal from the water compartment (EC 2002).

Water solubility is about 75 mg/L at 25°C (moreso as the temperature increases).

### Removal methods

Treatment processes that remove particulate matter should reduce the concentration of p-benzylphenol.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

4-benzylphenol is considered to increase the uterine glycogen content (EC 2002).

In a UK study a literature review identified 509 articles suggesting 325 potential endocrine disrupting compounds could be present in water bodies of potential relevance. Thirty-five of these chemicals were predicted to have highest (worst case) concentrations. The extent of the risk posed was determined by establishing the margin of safety (MOS). For six chemicals (p-benzylphenol, dibutyl phthalate (qv), 4‑nitrophenol (qv), digoxin, fluticasone and salbutamol), the MOS were ≤10, and hence were considered to warrant a more detailed consideration. See (DWI 2012) and datasheet for endocrine disrupting compounds for further details.

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2012. *A Review of Latest Endocrine Disrupting Chemicals Research Implications for Drinking Water*. Final Report DWI:70/2/266. 210 pp. http://dwi.defra.gov.uk/research/completed-research/reports/DWI70\_2\_266.pdf.

EC. 2002. *Endocrine Disrupters: Study on gathering information on 435 substances with insufficient data*. EU DG Environment B4-3040/2001/325850/MAR/C2. 279 pp. <http://ec.europa.eu/environment/endocrine/documents/bkh_report.pdf>.

# 2,2-bis(bromomethyl) propane-1,3-diol

CAS No. 3296-90-0. Also called 1,3-dibromo-2,2-dihydroxymethylpropane, 1,3‑dibromo-2,2-dimethylolpropane, 2,2-dibromomethyl-1,3-propanediol, dibromoneopentyl glycol, pentaerythritol dibromide, and pentaerythritol dibromohydrin. Trade names include FR-522 and FR-1138.

BBMP (FR-1138) is a technical-grade mixture of approximately 78 percent 2,2‑bis(bromomethyl)-1,3-propanediol, 6 percent 2,2-bis(hydroxymethyl)-1-bromo-3-hydroxypropane, 7 percent 2,2-bis(bromomethyl)-1-bromo-3-hydroxypropane, less than 1 percent pentaerythritol, and 8 percent dimers and structural isomers.

2,2-bis(bromomethyl)propane-1,3-diol is a halohydrin (see datasheet for a list of halohydrins).

### Maximum Acceptable Value

There are insufficient data to derive a MAV for 2,2-bis(bromomethyl)propane-1,3-diol in drinking-water. The WHO Guidelines do not refer to 2,2‑bis(bromomethyl)propane-1,3-diol.

### Sources to drinking-water

#### 1. To source waters

2,2-bis(bromomethyl)propane-1,3-diol can be produced by replacement of the hydroxyl groups of pentaerythritol with bromide. 2,2-bis(bromomethyl)propane-1,3-diol is a reactive flame retardant that is used primarily in unsaturated polyester resins for moulded products and in rigid polyurethane foams. It is increasingly used (alone or in mixtures) in CFC (chlorofluorocarbon)-free foam products designed to meet more stringent standards of flame retardancy. The total world market for flame retardants is estimated at just under 1 million tonnes per year.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

2,2-bis(bromomethyl)propane-1,3-diol may enter the environment as fugitive dust, through wastewater and through disposal of resins and foams which may contain the compound as an additive or impurity. 2,2-bis(bromomethyl)propane-1,3-diol may be persistent in water. 2,2-bis(bromomethyl)propane-1,3-diol is very soluble in water.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

The primary routes of human exposure to BBMP are inhalation and dermal contact.

2,2-bis(bromomethyl)propane-1,3-diol was tested for carcinogenicity as a commercial mixture (FR-1138) containing 80 percent of the parent compound in one experiment in mice and in two experiments in rats by oral administration in the diet. In mice, it increased the incidence of tumours of the Harderian gland, forestomach and lung in both males and females and of subcutaneous sarcomas in females. In one study in male rats, it increased the incidences of tumours of the skin, subcutaneous tissue, mammary gland, Zymbal gland, oral cavity, oesophagus, forestomach, small and large intestine, peritoneum, lung and thyroid. In female rats the incidences of oesophageal, mammary gland and thyroid follicular tumours were increased.

IARC (2000) stated that there is sufficient evidence in experimental animals for the carcinogenicity of 2,2-bis(bromomethyl)propane-1,3-diol so classified it as possibly carcinogenic to humans (Group 2B).

### Derivation of Maximum Acceptable Value

No MAV.

### References

IARC. 2000. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 77. Some industrial chemicals. <http://monographs.iarc.fr/ENG/Monographs/vol77/index.php>.

IPCS. 1997. *Flame Retardants: Environmental Health Criteria* 192. INCHEM. International Programme on Chemical Safety. http://www.inchem.org/documents/ehc/ehc/ehc192.htm.

IPCS. 1998. *Flame Retardants: Environmental Health Criteria* 209. INCHEM. International Programme on Chemical Safety. http://www.inchem.org/documents/ehc/ehc/ehc209.htm.

IPCS. 2000. *Flame Retardants: Environmental Health Criteria* 218. INCHEM. International Programme on Chemical Safety. http://www.inchem.org/documents/ehc/ehc/ehc218.htm.

NTP. 1996. *Toxicology and Carcinogenesis Studies of 2,2-Bis(Bromomethyl)-1,3-Propanediol (FR-1138) (CAS no. 3296-90-0) in F344/N Rats and B6C31F Mice (Feed Studies)*. Technical Report Series No 452. Research Triangle Park, NC: National Toxicology Program. http://www.ncbi.nlm.nih.gov/pubmed/12594523.

NTP. 2002. *2,2-Bis(bromomethyl)-1,3-Propanediol (Technical Grade) CAS No. 3296-90-0*. First listed in the 10th Report on Carcinogens (2002). US Government: National Institute of Environmental Health Sciences, National Institutes of Health. ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s024bbmp.pdf.

# Bis(2-chloroethyl)ether

CAS No. 111-44-4. Also called 1,1’-dichlorodiethyl ether, 1,1’-oxybis(2-chloro)ethane, 2,2’-dichloroethyl ether, or BCEE.

### Maximum Acceptable Value

There are insufficient data to derive a MAV for bis(2-chloroethyl)ether in drinking-water. The WHO Guidelines do not refer to bis(2-chloroethyl)ether.

Bis(2-chloroethyl)ether is one of the “priority pollutants” under the US Clean Water Act. (So is bis(2-chloroisopropyl)ether).

### Sources to drinking-water

#### 1. To source waters

Bis(2-chloroethyl)ether is mainly used as a chemical intermediate to make pesticides, but some of it is used as a solvent and cleaner. Bis(2-chloroethyl)ether has been found at 821 of the 1,518 National Priorities List sites identified by the Environmental Protection Agency (USEPA).

The USEPA recommends that levels in lakes and streams should be limited to 0.00003 mg/L to prevent possible health effects from drinking water or eating fish contaminated with bis(2-chloroethyl) ether.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Bis(2-chloroethyl)ether is very soluble in water (about 1 percent), so when released to soil, some will filter through the soil to groundwater; however some will be broken down by bacteria, and some will evaporate to the air. Most ethers are very resistant to hydrolysis, and the rate of cleavage of the carbon-oxygen bond by abiotic processes is expected to be insignificant. The carbon-chlorine bond is also quite stable to abiotic cleavage. The USEPA calculated a half-time for volatilisation of bis(2-chloroethyl)ether from a river to be 3.4 days.

### Typical concentrations in drinking-water

Low levels, 0.00001 to 0.0005 mg/L, have been detected in the drinking-water supplies of several cities. The median bis(2-chloroethyl)ether concentration in water samples taken across the United States is <0.01 mg/L based on 808 samples.

1 water utility in the US reported detecting bis(2-chloroethyl) ether in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0013 mg/L.

### Analytical methods

#### Referee method

A referee method cannot be selected for bis(2-chloroethyl)ether because a MAV has not been established and therefore the sensitivity required for the referee method is not known.

### Health considerations

There is some evidence that bis(2-chloroethyl)ether causes cancer in mice. The International Agency for Research on Cancer (IARC) has determined that bis(2‑chloroethyl)ether is not classifiable as to its carcinogenicity in humans (Group 3). USEPA (1994) classified it as B2: a probable human carcinogen. Bis(2-chloroethyl)ether appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

The are insufficient data to derive a MAV for bis(2-chloroethyl)ether at this time.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The cancer health risk limit for bis(2-chloroethyl)ether is 0.0003 mg/L.

### References

ATSDR. 1989. *Toxicological Profile for Bis(2-chloroethyl)ether*. Atlanta, GA: Agency for Toxic Substances and Disease Registry. Department of Health and Human Services. <http://www.atsdr.cdc.gov/toxprofiles/index.asp>.

Environmental Working Group (EWG). Accessed 2010. *National Drinking Water Database – Chemical Contaminants*. <http://www.ewg.org/tap-water/chemical-contaminants>.

IARC. 1999. *Bis(2-chloroethyl)ether*. International Agency for Research on Cancer (IARC). http://www.inchem.org/documents/iarc/vol71/060-bis2chleth.html.

IPCS. 1995. Chloroalkyl ethers. *Environmental Health Criteria* 201. INCHEM. International Programme on Chemical Safety. http://www.inchem.org/documents/ehc/ehc/ehc201.htm.

MDH. 2009/2016. *Groundwater Values Table*. Minnesota Department of Health (MDH). See: http://www.health.state.mn.us/divs/eh/risk/guidance/gw/table.html.

USEPA. 1994. Bis(2-chloroethyl)ether. *Integrated Risk Information System (IRIS)*. http://www.epa.gov/iris/subst/013.htm.

# Bis(2-chloromethyl) ether

CAS No. 542-88-1. Also called 1,1’-dichlorodimethyl ether, 1,1’-oxybis(2-chloro)methane, 2,2’-dichloromethyl ether, BCME, and chloromethyl ether – note: this name is sometimes used incorrectly for chloromethyl methyl ether (CAS No. 107-30-2) which can also be known as CMME, chloromethoxymethane or chlorodimethyl ether.

### Maximum Acceptable Value

There are insufficient data to derive a MAV for bis(2-chloromethyl)ether in drinking-water. The WHO Guidelines do not refer to bis(2-chloroethyl)ether.

### Sources to drinking-water

#### 1. To source waters

Bis(2-chloromethyl)ether and chloromethyl ether are mainly used as chemical intermediates and alkylating agents. BCME is used as a laboratory reagent; to manufacture plastics, ion-exchange resins, and polymers. BCME formerly was used for crosslinking of cellulose, surface treatment of vulcanised rubber to increase adhesion, and in the manufacture of flame-retardant fabrics, however its use is now highly restricted. CMME is used as an alkylating agent and industrial solvent to manufacture dodecylbenzyl chloride, water repellants, ion-exchange resins, and polymers, and as a chloromethylating reagent.

BCME has not been detected in ambient air or water (ATSDR 1989).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Bis(2-chloromethyl)ether in water is broken down quickly to formaldehyde and hydrochloric acid. When released to soil, some will evaporate to the air but most of it will be broken down by reacting with soil moisture.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

The primary routes of potential human exposure to BCME is inhalation and dermal contact, however, the potential for exposure is low, because these chemicals are no longer produced or sold in large quantities, and most industrial operations involving them take place in closed process vessels.

The USEPA has classified bis(2-chloromethyl)ether as a Group A: a human carcinogen (USEPA 1991).

The International Agency for Research on Cancer (IARC 1974) determined that because bis(2-chloromethyl)ether exhibits a high incidence of predominantly oat-cell carcinoma in a small population of laboratory workers exposed to BCME, exposure to this compound constitutes a serious human lung cancer hazard, and has classified it in Group 1: carcinogenic to humans. IARC (2012) classified bis(chloromethyl) ether and chloromethyl methyl ether in Group 1: carcinogenic to humans.

The USEPA recommends that levels in lakes and streams should be limited to 0.0000038 parts per billion parts of water (0.0038 ng/L) to prevent possible health effects from drinking water or eating fish contaminated with bis(chloromethyl) ether.

Bis(2-chloromethyl)ether is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

### Derivation of Maximum Acceptable Value

There is little need to derive a MAV for bis(2-chloromethyl)ether because it breaks down rapidly in drinking-water.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The cancer health risk limit for bis(2-chloromethyl)ether is 0.000002 mg/L.

### References

ATSDR. 1989. *Toxicological Profile for Bis(2-chloromethyl)ether*. Atlanta, GA: Agency for Toxic Substances and Disease Registry. Department of Health and Human Services. http://www.atsdr.cdc.gov/toxprofiles/index.asp.

EFSA. 2016. Identification of the substances from the Carcinogenic Potency Database (CPDB) which are of particular concern even if ingested at doses below 0.0025 μg/kg body weight per day. *EFSA Journal* 14(3): 4407. 11 pp. http://www.efsa.europa.eu/sites/default/files/scientific\_output/files/main\_documents/4407.pdf.

IARC. 1974. Bis(2-chloromethyl)ether. Some aromatic amines, hydrazine and related substances, n-nitroso compounds and miscellaneous alkylating agents. International Agency for Research on Cancer. http://monographs.iarc.fr/ENG/Monographs/allmonos30.php.

IARC. 2012. A review of human carcinogens: chemical agents and related occupations. Bis(chloromethyl) ether and chloromethyl methyl ether. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 100F. <http://monographs.iarc.fr/>.

IPCS. 1995. Chloroalkyl ethers, *Environmental Health Criteria* 201. INCHEM. International Programme on Chemical Safety. http://www.inchem.org/documents/ehc/ehc/ehc201.htm.

MDH. 2009/2016. *Groundwater Values Table*. Minnesota Department of Health (MDH). See: http://www.health.state.mn.us/divs/eh/risk/guidance/gw/table.html.

USEPA. 1991. Bis(2-chloromethyl)ether. *Integrated Risk Information System (IRIS)*. See <http://www.epa.gov/iris/subst/0375.htm>.

# Bisphenol A

CAS No. 80-05-7. Also called 4,4-bisphenol A, 2,2-bis(4-hydroxyphenyl)propane, 4,4’‑isopropylidenediphenol and bis(4-hydroxyphenyl)dimethylmethane.

Bisphenol A is one of dozens of bisphenols. Some other bisphenols occasionally mentioned in the literature include: bisphenol A diglycidyl ether (CAS No. 1675-54-3, sometimes called BADGE); bisphenol A dimethylacrylate and bisphenol-A diglycidylether dimethacrylate (both used in the dental industry). Industrial grade bisphenol A will include many of the bisphenols, somewhat analogous to the PCBs.

Technical bisphenol A (higher quality than industrial-grade) is usually >98 percent bisphenol A; the main impurities are phenol, and the ortho and para isomers of bisphenol A.

Up to 15 other contaminants have been identified in commercial grade bisphenol A. One, p-cumylphenol, was found to be 12-times more estrogen-active than bisphenol A. Also, 4,4`-(1,3-dimethylbutylidene)bisphenol and 2-(4`-hydroxyphenol)-2,2,4-trimethylchroman were 9-times more active. Some of the other compounds included 4-hydroxyacetophenone, 4-hydroxyphenyl isobutyl methyl ketone, and 2,4‑bis(4-hydroxycumyl)phenol (Terasaki et al 2005; NTP 2007).

### Maximum Acceptable Value

There are insufficient data to derive a MAV for bisphenol A in drinking-water. The WHO Guidelines do not refer to bisphenol A.

### Sources to drinking-water

#### 1. To source waters

The primary sources of environmental release of bisphenol A are expected to be effluents and emissions from its manufacturing facilities and facilities which manufacture epoxy, polycarbonate, and polysulfone resins. Bisphenol A (BPA) is a chemical produced in large quantities for use primarily in the production of polycarbonate plastics (commonly used in drink bottles) and epoxy resins (a common lining in food cans). Bisphenol A is one of the highest volume chemicals produced worldwide; over six billion pounds are produced each year. The concentration of bisphenol A in found in various waters has been reported in IUPAC. 2003. The use of bisphenol-A as an inhibitor in PVC production ceased voluntarily in the EU in 2003 (EU 2008).

EU (2008) reports concentrations of bisphenol A from 848 freshwater samples. Median = 0.00001 mg/L, mean = 0.00013 mg/L, 95th percentile = 0.00035 mg/L.

Bisphenol A diglycidyl ether is an ingredient in some Araldite products.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Bisphenol A can leach into drinking-water from plastic bottles, chiefly polycarbonate, and some epoxy-lined storage tanks. Polycarbonate plastic bottles and containers are usually identified by the plastic recycling symbol #7.

### Forms and fate in the environment

If released to soil, bisphenol A may have high to slight mobility based upon a Koc range of 115 to 3886 (EU 2008 quotes logKow = 3.4). Most of the measured Koc values suggest that bisphenol A may have moderate to low mobility in soil. The pKa of bisphenol A is 9.6, indicating that this compound will exist partially in anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. The partial dissociation of bisphenol A in environmental media may be one reason for the wide range of observed soil adsorption. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 4.0 x 10-11 atm‑cu m/mole. If released into water, bisphenol A is expected to adsorb to suspended solids and sediment based upon the Koc. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. A BCF range of 5.1 to 73.4 suggests bioconcentration in aquatic organisms is low to moderate. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions. Sensitised photooxidation may be an important as a fate process for bisphenol A in sunlit natural water (EAWAG accessed February 2015).

Removal appears to be rapid once the waters have become acclimatised to bisphenol-A. The reported lag-phases before degradation are between 3–8 days. After the lag phase removal was rapid with 50 percent removal in 1–2 days and 100 percent removal in 2 to 17 days. The half-life for biodegradation of bisphenol-A in soil is calculated to be 30 days (EU 2008).

If released to soil, bisphenol A is expected to have moderate mobility due to its solubility of 120 mg/L (EU 2008 quotes 300 mg/L) and very low vapour pressure. This compound may biodegrade under aerobic conditions following acclimation. If released to acclimated water, biodegradation would be the dominant fate process (half-life less than or equal to four days). In non-acclimated water, bisphenol A may biodegrade after a sufficient adaptation period (1 to 150 days), it may adsorb extensively to suspended solids and sediments, or it may photolyse.

### Typical concentrations in drinking-water

Bisphenol A is found frequently in German drinking-water around the 0.005 µg/L or 0.000005 mg/L concentration.

WHO (2011) reports that the maximum concentration of bisphenol A in drinking water and bottled water was 1 µg/L, or 0.001 mg/L, based on >100 samples.

In 2017 16 bores in Lower Hutt were tested; the highest concentration of BPA found was 280 ng/L, ie, 0.28 µg/L, or 0.00028 mg/L (*New Zealand Herald*, Friday 5 October 2018, <https://www.nzherald.co.nz/nz/news/article.cfm?c_id=1&objectid=12133631>).

### Removal methods

When dosing ozone at 1, 1.5, and 2 mg/L into water containing 11 mg/L of bisphenol A, the removal efficiencies were measured at 70 percent, 82 percent, and 90 percent respectively.

### Analytical methods

#### Referee method

A referee method cannot be selected for bisphenol A because a MAV has not been established and therefore the sensitivity required for the referee method is not known.

#### Some alternative methods

Many methods have been suggested for testing substances for endocrine disruption – see the OECD website: <http://www.oecd.org/env/testguidelines>.

### Health considerations

WHO (2011) reported that on the basis of the most relevant national published estimates, the exposure of adults to BPA was <0.01–0.40 μg/kg body weight (bw) per day at the mean and 0.06–1.5 μg/kg bw per day at the 95th/97.5th percentile. For young children and teenagers, mean exposure was 0.1–0.5 μg/kg bw per day, and exposure at the 95th/97.5th percentile was 0.3–1.1 μg/kg bw per day. Based on the limited data available, exposure to BPA from non-food sources is generally lower than that from food by at least an order of magnitude for most population subgroups.

There is a lot of debate at present about the safety of using polycarbonate bottles due to claims that bisphenol A is an endocrine disruptor, or is “weakly” estrogenic. Counterclaims are that polycarbonate bottles have been used for decades without any reported health problems. Chlordane is on the EC List of 66 Category 1 substances showing evidence of endocrine disrupting activity in at least one species using intact animals (EC 2015).

Bisphenol A belongs to the bis(hydroxyphenyl)methanes, for which several endocrine effects are reported (IUPAC 2003).

The National Toxicology Program (NTP 2008) released a draft report affirming “some concern” for potential adverse brain and behaviour-related developmental effects associated with exposures to BPA. However, NTP acknowledges that the concerns stem from limited evidence based on laboratory animals, which demonstrates the need for additional research, especially on the long-term health effects of current exposure levels. The NTP has negligible concern that exposure of pregnant women to bisphenol A will result in fetal or neonatal mortality, birth defects or reduced birth weight and growth in their offspring. The NTP concludes that there is negligible concern that exposure to bisphenol A causes reproductive effects in non-occupationally exposed adults and minimal concern for workers exposed to higher levels in occupational settings.

Evidence based on animal testing suggests that adverse developmental effects related to BPA exposures occur. Some of the observed effects include neural and behavioural abnormalities in rodents. However, these effects occur at levels several thousand times higher than any child or adult would be expected to consume on a daily basis.

Bisphenol A is rapidly converted to bisphenol-glucuronide or the sulphate (non-estrogenic) in the human body.

The RfD was calculated at 0.05 mg/kg/d (USEPA 1993). In 2008 EFSA set a Tolerable Daily Intake (TDI) for bisphenol A of 0.05 mg/kg body weight/day (50 μg/kg body weight/day). This was based on effects on the liver in animal studies. It was concluded that reproductive effects occurred at doses ten-fold higher than those at which liver effects were seen (ex NZFSA).

IARC considers bisphenol A diglycidyl ether as not classifiable as to its carcinogenicity to humans (Group 3). Glycidaldehyde (CAS No. 765-34-4), a metabolite of bisphenol A diglycidyl ether, is carcinogenic to experimental animals and classified as possibly carcinogenicto humans (Group 2B).

### Derivation of Maximum Acceptable Value

The are insufficient data to derive a MAV for bisphenol A.

Willhite et al (2008) studied the toxic effects of bisphenol A and concluded that, assuming a 70‑kg adult consumes 2 L of water each day and adopting the default 20 percent USEPA drinking-water relative source contribution yields a 0.10 mg/L total allowable concentration.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The short-term level is 0.1 mg/L, chronic and subchronic health risk limits are 0.02 mg/L.

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# Bis(trichloromethyl) sulfone

CAS No. 3064-70-8. The IUPAC name is trichloro(trichloromethylsulfonyl)methane. Also called hexachlorodimethyl sulfone and sulfonyl bis(trichloromethane).

### Maximum Acceptable Value

There are insufficient data to derive a MAV for bis(trichloromethyl) sulfone in drinking-water. The WHO Guidelines do not refer to bis(trichloromethyl) sulfone.

### Sources to drinking-water

#### 1. To source waters

Bis(trichloromethyl) sulfone is primarily used to control microbes, algae, and fungi in cooling water systems, waste disposal systems, pulp and paper mill water systems, oil extraction systems, and other industrial settings.

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

Bis(trichloromethyl) sulfone does not hydrolyse in sterile aqueous buffered solutions at pH 4, 7, or 9. An anaerobic aquatic metabolism study shows that the major route of dissipation of bis(trichloromethyl) sulfone is rapid microbial degradation with an estimated half-life of <0.5 days. Two metabolites are pentachlorodimethylsulfone and hexachloroethane. Photodegradation half-lifes were 16 days in water. Leaching adsorption/desorption studies indicate low mobility (USEPA 1995).

If released to soil, bis(trichloromethyl) sulfone is expected to have very high mobility based upon an estimated Koc of 43. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 1.2 x 10-8 atm‑cu m/mole. If released into water, bis(trichloromethyl) sulfone is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. A half-life of <0.5 days in an anaerobic aquatic biodegradation test indicates that biodegradation may be in important fate process under anoxic aquatic conditions. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. Bis(trichloromethyl) sulfone was found to photodegrade in water when irradiated with a non-specific sunlamp. An estimated BCF of 70 suggests the potential for bioconcentration in aquatic organisms is moderate. Hydrolysis is not expected to be an important environmental fate process since bis(trichloromethyl) sulfone is stable at pH 5, 7, or 9. See [http://pubchem.ncbi.nlm.nih.gov/compound/Chlorosulfona#section=Ecotoxicity-Values](http://pubchem.ncbi.nlm.nih.gov/compound/Chlorosulfona%23section=Ecotoxicity-Values).

Water solubility is 67 mg/L. The Octanol/Water Partition Coefficient: Log P = 3.3.

### Health considerations

From its review of the toxicology data, the USEPA (1995) determined that bis(trichloromethyl) sulfone was slightly toxic to non-toxic in acute oral and dermal toxicity tests. From evidence of changes in the blood and clinical chemistry values, the systemic NOEL is established at 2.0 mg/kg/day for male rabbits and equal to or greater than 5.0 mg/kg/day for female rabbits. The developmental toxicity for albino rats was a NOEL of 10 mg/kg/day based on decreased fetal body weights and an increase in skeletal and external anomalies. The establishment of a reference dose (RfD) for bis(trichloromethyl) sulfone is not warranted, based on the use patterns and exposure profile for this active ingredient.

### Derivation of Maximum Acceptable Value

No MAV.

### References

USEPA. 1995. *Reregistration Eligibility Decision (RED): Bis(trichloromethyl) sulfone*. EPA 738-R-96-028. 126 pp. <http://www.epa.gov/opp00001/reregistration/REDs/2055red.pdf>.

# Brominated phenols

The commoner bromophenols include:

2-bromophenol CAS No. 95-56-7

3-bromophenol CAS No. 591-20-8

4-bromophenol CAS No. 106-41-2

2,4-dibromophenol CAS No. 615-58-7

2,5-dibromophenol CAS No. 626-41-5 and 28165-52-8

2,6-dibromophenol CAS No. 608-33-3

3,5-dibromophenol CAS No. 626-41-5

2,4,6-tribromophenol CAS No. 118-79-6

2,3,4,6-tetrabromophenol CAS No. 14400-94-3

pentabromophenol CAS No. 608-71-9

### Maximum Acceptable Value

There are no MAVs in the DWSNZ for these bromophenols, and they are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

There are very limited data on environmental levels of the brominated phenols. There are also limited data on the toxicity of mono- and dibromophenols and pentabromophenol; 2,4,6-tribromophenol is the most data rich of the brominated phenols. Compounds such as 2,5-dibromophenol, 3,5-dibromophenol, and 2,3,4,6‑tetrabromophenol appear to exist only in the laboratory.

Brominated phenols that have been identified as reactive flame retardants include 2,4‑dibromophenol (2,4-DBP), 2,4,6-tribromophenol (2,4,6-TBP), pentabromophenol (PBP), and tetrabrominated bisphenol S (TBBPS). They are added to epoxy resins, phenolic resins, polyester resins, polyolefins and vinyl-aromatic polymers (EFSA 2012).

No data are available on levels in and possible leaching of unreacted brominated phenols from plastics containing fire retardants derived from 2,4,6-tribromophenol in particular. Some brominated phenols are released to the atmosphere from burning plastics and hydrocarbon fuels.

Several species of marine algae are known to contain simple brominated phenols. For example, 2,4-dibromophenol, 2,6-dibromophenol and 2,4,6-tribromophenol are released by the large perennial brown algae *Eisenia bicyclis* and *Ecklonia kurome*. Dibromophenols have been found in other marine biota as well. Consequently these compounds have been detected in fish flesh, giving rise to phenolic or iodoform taints.

2,4-Dibromophenol and 2,4,6-tribromophenol are marine secondary metabolites, with 2,4,6-tribromophenol playing an important role as industrially produced flame retardant, and as a pesticide/fungicide (but not registered in New Zealand). 2,4‑Dibromophenol is also reported to have been used as a flame retardant.

#### 2. From treatment processes

Bromophenols can be formed during the chlorination of natural water and wastewater containing phenol and bromide ions. For example, the formation of 2,4,6‑tribromophenol has resulted from the chlorination of water containing phenol and bromine at pH 7.4. Direct bromination with hypobromous acid was compared with bromination by hypochlorous acid and bromide ion. Under conditions when hypochlorous acid was not limiting, a higher yield of bromine substitution products could be expected from the bromination by hypochlorous acid plus bromine than from direct bromination by hypobromous acid.

WHO (2005) states: Chlorination of natural water containing bromide ion can result in the production of dibromo-, bromodichloro-, dibromochloro-, and tribromophenols (Bean et al, 1980; Sweetman and Simmons 1980; Rivera and Ventura 1984; Sithole and Williams 1986). In a survey of 40 potable water treatment plants located in 39 cities distributed geographically in proportion to population and covering about 40 percent of Canadian consumers, between October 1984 and June 1985, mean concentrations of 2,4-DBP ranged from 0.6 to 1.2 ng/L for raw water and from 0.4 to 2.5 ng/L for treated water; mean 2,4,6-TBP concentrations ranged from 0.2 to 0.6 ng/L (maximum 10 ng/L) and from 0.2 to 1.3 ng/L (maximum 20 ng/L) for raw and treated water, respectively (Sithole and Williams 1986). Raw water from water treatment plants in six Canadian cities and treated water from water treatment plants in five of six Canadian cities, collected in February 1985, contained 2-BP, 2,6-DBP, and 2,4,6-TBP at concentrations below the quantification limit of 2–4 ng/L; samples of treated water from one city contained 2,4,6-TBP at a mean concentration of 5 ng/L, while samples of treated water from another city contained 2-BP and 2,6-DBP at mean concentrations of 42 and 60 ng/L, respectively.

#### 3. From the distribution system

2,6-Dibromophenol has been found in water that has been in contact with materials usually not intended for use with drinking water.

### Forms and fate in the environment

In water, pentabromophenol would be expected to adsorb to suspended solids and sediment. However, other less brominated phenols would tend to remain in the water phase. Volatilisation of non-dissociated 2,4,6-tribromophenol and pentabromophenol from water surfaces is not expected to be an important fate process. Henry’s law constants for mono- and dibrominated phenols would suggest little volatilisation of these compounds. All of the brominated phenols, if released to soil, essentially stay there and will not be mobile. Brominated phenols are generally not readily biodegradable and will persist in the environment (EAWAG accessed February 2015).

If released to soil, 2,6-dibromophenol is expected to have low mobility based upon an estimated Koc of 1600. The pKa of 2,6-dibromophenol is 6.67, indicating that this compound will partially exist in the anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 8.9 x 10-8 atm‑cu m/mole. If released into water, 2,6-dibromophenol is expected to adsorb to suspended solids and sediment based upon the estimated Koc. 2,6-Dibromophenol degraded in a soil-water system under anaerobic conditions with a half life of 7 days, suggesting that biodegradation may be an important environmental fate. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. An estimated BCF of 31 suggests the potential for bioconcentration in aquatic organisms is moderate. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

Maximum reported concentrations in surface fresh water are:

2,4-dibromophenol 0.04 mg/L

2,6-dibromophenol 0.003 mg/L

2,4,6-tribromophenol 0.0003 mg/L

Generally, concentrations in drinking-water are less than 3 ng/litre (0.000003 mg/L), with levels higher in treated than in raw water. Drinking water samples in Canada have been found to contain about 0.00004 mg/L of 2-bromophenol, 2,6-dibromophenol and 2,4,6-tribromophenol.

### Removal methods

No records available.

### Analytical methods

See WHO (2005); EFSA (2012).

### Health considerations

Toxicity studies are scarce and mostly relates to 2,4,6-TBP. The main targets are liver and kidneys. In a limited repeated dose oral toxicity study a no-observed-adverse-effect level (NOAEL) for 2,4,6-TBP of 100 mg/kg bw per day was identified. 2,4,6-TBP was not genotoxic in bacterial tests *in vitro*, and not *in vivo*, but induced chromosomal aberrations in mammalian cells *in vitro.* No long-term toxicity or carcinogenicity studies with 2,4,6-TBP were identified (EFSA 2012).

### Derivation of Maximum Acceptable Value

No MAV.

The *Australian Drinking Water Guidelines* (2011) state that taste and odour can also arise from impacts on the supplied water within the customer’s property, such as contaminants in direct or indirect contact with water (eg, contaminants from kettles, refrigerators, dishwashers or washing machine hoses). The compound 2,6‑dibromophenol, identified as probably responsible for a “plastic” or “chemical” taste in water after it is boiled, has a taste threshold concentration of 0.0005 mg/L (Whitfield et al 1992, Adams et al 1999, Heltz et al 2002).

Flavour thresholds in water are reported as (RSC 1996):

2-bromophenol 3 x 10-2 ng/g = 0.00003 mg/L

4-bromophenol 23 ng/g = 0.023 mg/L

2,4-dibromophenol 4 ng/g = 0.004 mg/L

2,6-dibromophenol 5 x 10-4 ng/g = 0.0000005 mg/L

2,4,6-tribromophenol 6 x 10-1 ng/g = 0.0006 mg/L

Reported in Dietrich (2006) is an observation that biofilm in the distribution system can cause the biomethylation of chlorophenols and bromophenols to form haloanisoles which have earthy and musty odours at concentrations less than 1 ng/L, ie, <0.000001 mg/L.

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# Bromoacetonitriles

The haloacetonitriles are a subgroup of the nitrogen-containing disinfection by‑products (N-DBPs). This bromoacetonitrile (BAN) datasheet could have been titled haloacetonitriles (HANs). However, datasheets already exist for:

2-chloroacetonitrile CAN

2,2-dichloroacetonitrile DCAN

2-bromo-2-chloroacetonitrile BCAN also called bromochloroacetonitrile

2,2-dibromoacetonitrile DBAN

2,2,2-trichloroacetonitrile TCAN

The haloacetonitriles without an individual datasheet that are discussed collectively here are:

2-bromoacetonitrile CAS No. 590-17-0

2-bromo-2,2-dichloroacetonitrile CAS No. 60523-73-1 or bromodichloroacetonitrile

2,2-dibromo-2-chloroacetonitrile CAS No. 60523-73-1 or dibromochloroacetonitrile

2,2,2-tribromoacetonitrile CAS No. 75519-19-6

3-bromopropanenitrile CAS No. 2417-90-5

There will be some overlap, so refer to the 5 individual datasheets mentioned above as well.

### Maximum Acceptable Value

There are no MAVs in the DWSNZ for the five bromoacetonitriles discussed in this datasheet, and they are not mentioned in the WHO Guidelines.

MAVs exist for two haloacetonitriles (see individual datasheets for details):

2,2-dibromoacetonitrile 0.08 mg/L

2,2-dichloroacetonitrile 0.02 mg/L (provisional MAV)

### Sources to drinking-water

#### 1. From treatment processes

DWI (2010) states (regarding the US Nationwide DBP Occurrence Study): Among the three main N-DBPs classes noted (ie, the haloacetamides, halonitromethanes and haloacetonitriles), the haloacetonitriles (HANs) had the highest formation, with median and maximum values of 3 μg/L and 14 μg/L, respectively. The most prevalent HAN was dichloroacetonitrile (DCAN), with a median concentration of 1 μg/L. Compared with the results from US Nationwide DBP Study in 2000–2002, the median and 75th percentile values for dichloroacetonitrile, bromochloroacetonitrile, dibromoacetonitrile and trichloroacetonitrile were slightly higher in the N-DBP Study. This may be attributed to the high content of nitrogenous precursors in the selected source waters.

The most frequently reported haloacetonitrile species in drinking water are the dihaloacetonitriles. Dichloroacetonitrile and other haloacetonitriles have been shown to be produced from the chlorination of selected free amino acids, heterocyclic nitrogen in nucleic acids, proteinaceous materials, and combined amino acids bound to humic structures.

In the presence of excess chlorine, free amino acids react quickly to form nitriles; for the reactive amino acids, this in turn leads to dihaloacetonitrile formation. Combined amino acids, such as peptide chain structures, react more slowly compared with free amino acids and are believed to be the predominant species in drinking water.

The reaction of chlorine with proteins can be regarded as a two-step process. Firstly, a series of fast reactions involving chlorine and reactive side groups gives rise to the formation of THMs and total organic halides (TOX), but insignificant levels of dihaloacetonitriles. The second step is the slower, base-catalysed, degradation of the polypeptide backbone, which forms haloacetonitriles.

In one study, dichloroacetonitrile was the only haloacetonitrile recorded at significant levels, and its formation was approximately five times higher after chloramination rather than after chlorination. Use of chlorine dioxide and ozone also increase haloacetonitriles levels.

When chlorinating, the molar yields of total dichloroacetonitriles increased with elevated bromide concentrations. When chlorinating or ozonating, increasing bromide concentrations shifted the distribution of dichloroacetonitriles from dichloroacetonitrile to bromochloroacetonitrile and then to dibromoacetonitrile.

3-Bromopropanenitrile has been detected in water sources with high bromide levels and for pre-oxidation with chlorine dioxide. In this particular study, which used Israeli water, bromide levels were far higher than in other investigations, at 2 mg/L. At present, its incidence in waters with lower bromide is uncertain.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Hydrolysis of haloacetonitriles leads to haloacetamides (qv), and then to haloacetic acids (qv). Higher concentrations of haloacetonitriles have been observed under weakly acidic and neutral pH conditions, under which conditions, they are the most stable in chlorinated water. Degradation of the dihaloacetonitriles was accelerated at pH 7 and pH 8 in the presence of free chlorine, but not at pH 6. By studying the hydrolysis and chlorination kinetics of all nine bromine and chlorine-containing haloacetonitriles, it was shown that trihaloacetonitriles had the highest rates of hydrolysis, followed by the dihaloacetonitriles, and finally the monohalogenated forms. Reaction rates with chlorine follow a similar trend, while all degradation rates increased with increasing pH.

|  |  |
| --- | --- |
|  | **Water solubility** |
| 2-bromoacetonitrile | 3.7% |
| 2-bromo-2,2-dichloroacetonitrile | 0.2% |
| 2,2-dibromo-2-chloroacetonitrile | 0.1% |
| 2,2,2-tribromoacetonitrile | 0.05% (490 mg/L) |
| 3-bromopropanenitrile | 1.2% |

WRF (2016) reports half-lifes for three haloacetonitriles in water at pH 8.3 and 25°C:

DCAN 30 hours

BCAN 55 hours

BDAN 85 hours

### Typical concentrations in drinking-water

A Canadian study at 53 sites found dichloroacetonitrile (in 97 percent of samples), 2‑bromo-2-chloroacetonitrile (92 percent), 2,2-dibromoacetonitrile (57 percent) and 2,2,2-trichloroacetonitrile (in 9 percent of samples).

DWI (2010) reports the concentration (mg/L) found in US drinking water:

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | **Minimum** | | **Median** | | **Maximum** | |
| chloroacetonitrile | ND | ND | | 0.0009 | |
| bromoacetonitrile | ND | ND | | 0.0002 | |
| dichloroacetonitrile | ND | 0.001 | | 0.012 | |
| bromochloroacetonitrile | ND | 0.0006 | | 0.003 | |
| dibromoacetonitrile | ND | 0.0002 | | 0.002 | |
| trichloroacetonitrile | ND | ND | | 0.0004 | |
| dibromochloroacetonitrile | ND | ND | | 0.0006 | |

DWI (2012) reported a UK study of 20 water supplies that had suspected risk factors for the formation of N-DBPs; several supply systems with no risk factors were also included. The lowland water sources that were included in the survey formed more N‑DBPs than the upland and groundwater sources. The six treatment works that applied ozone were associated with higher concentrations of HANs and HAcAms than non-ozone treatment works, although this was potentially confounded because all the ozone works were treating lowland source waters which may have had higher N-DBP formation potential. None of the N-DBPs exhibited consistent links with total trihalomethanes (THMs). There were no trends linking HNMs to either THMs or HAA9. The mean HAN concentration was 0.0032 mg/L (3.2 µg/L). The main HANs were dichloroacetonitrile, bromochloroacetonitrile and dibromoacetonitrile. Trichloroacetonitrile was not detected.

The N-DBPs included in the study were:

**Haloacetamides (HAcAms)**

2-Chloroacetamide (CAcAm)

2-Bromoacetamide (BAcAm)

2,2-Dichloroacetamide (2,2-DCAcAm)

2,2-Dibromoacetamide (2,2-DBAcAm)

2,2,2-Trichloroacetamide (2,2,2-TCAcAm)

**Haloacetonitriles (HANs)**

Chloroacetonitrile (CAN)

Bromoacetonitrile (BAN)

Dichloroacetonitrile (DCAN)

Bromochloroacetonitrile (BCAN)

Dibromoacetonitrile (DBAN)

Trichloroacetonitrile (TCAN)

Dibromochloroacetonitrile (DBCAN)

**Halonitromethanes (HNMs)**

Chloronitromethane (CNM)

Bromonitromethane (BNM)

Dichloronitromethane (DCNM)

Bromochloronitromethane (BCNM)

Dibromonitromethane (DBNM)

Trichloronitromethane (TCNM)

Bromodichloronitromethane (BDCNM)

Dibromochloronitromethane (DBCNM)

**Cyanogen halides (CNXs)**

Cyanogen chloride (CNCl)

Cyanogen bromide (CNBr)

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See DWI (2010).

### Health considerations

#### 2-bromoacetonitrile

Limited data are available on the toxicity of 2-bromoacetonitrile. The available studies indicate that 2-bromoacetonitrile is cytotoxic and causes genotoxicity *in vitro*. Results of the NTP (1997) study indicate that there was significant aversion to 2‑bromoacetonitrile in the water and suggest that 2-bromoacetonitrile may be a possible mild renal toxicant at 100 ppm (approximately 7 mg/kg/day), and a potential reproductive toxicant at 100 ppm, as evidenced by increased (p >0.05) post‑implantation loss. There is ‘low to moderate’ concern about carcinogenicity based on SAR analysis.

#### 2-bromo-2,2-dichloroacetonitrile

Very limited data are available on the toxicity of 2‑bromo-2,2-dichloroacetonitrile. There is ‘low to moderate’ concern for carcinogenicity based on SAR analysis.

#### 2,2-dibromo-2-chloroacetonitrile

Very limited data are available on the toxicity of 2,2-dibromo-2-chloroacetonitrile. There is ‘low to moderate’ concern for carcinogenicity based on SAR analysis.

#### 2,2,2-tribromoacetonitrile

Very limited data are available on the toxicity of 2,2,2-tribromoacetonitrile. There is ‘low to moderate’ concern for carcinogenicity based on SAR analysis.

#### 3-bromopropanenitrile

No data are available on the toxicity of 3-bromopropanenitrile.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Bromobenzene

CAS No. 108-86-1. May also be called phenyl bromide or monobromobenzene.

### Maximum Acceptable Value

Bromobenzene does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

Bromobenzene is on the USEPA Drinking Water Contaminant Candidate List.

### Sources to drinking-water

#### 1. To source waters

Bromobenzene is used as an additive to motor oils, and in the production of drugs. It has been found in US river water samples at concentrations up to 0.04 mg/L.

#### 2. From treatment processes

Bromobenzene can be formed in small quantities during chlorination.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

If released to water, bromobenzene is not expected to adsorb to suspended solids or sediment. Based on its Henry’s Law constant, bromobenzene will volatilise from water surfaces. Hydrolysis of bromobenzene should be very slow because halogenated aromatics are generally resistant to hydrolysis. Bromobenzene does not appear to be degraded rapidly by aquatic microorganisms.

Water solubility about 400 mg/L.

### Typical concentrations in drinking-water

In the US, 16 communities were served tap water containing bromobenzene between 1998 and 2003, all at less than 0.0012 mg/L. 15 water utilities in the US reported detecting bromobenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.013 mg/L.

### Removal methods

Treating water containing bromobenzene with ozone will release bromide, which may then form disinfection by‑products.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

The USEPA (2006/2011) derived a children’s health-based limit for 10-day exposure to bromobenzene of 4 mg/L, being the concentration of a chemical in drinking water that is not expected to cause any adverse, non-carcinogenic health effects for up to ten days of exposure. The main concern is considered to be bromobenzene-induced liver necrosis, and perhaps to a lesser extent, nephrotoxicity.

Independent *in vivo* and *in vitro* studies indicate that bromobenzene and monochlorobenzene (see datasheet – aesthetics) have similar toxicokinetic properties and share the same critical target of toxicity (liver).

Under the USEPA’s Guidelines for Carcinogen Risk Assessment, there is inadequate information available for an assessment of the human carcinogenic potential of bromobenzene.

A chronic oral RfD for bromobenzene of 0.008 mg/kg-day was established by the USEPA (2009 and 2011), and drinking water equivalent level (DWEL) of 0.3 mg/L. USEPA (2009a) quotes a subchronic RfD of 0.02 mg/kg/d.

### Derivation of Maximum Acceptable Value

No MAV.

### References

EWG. 2008. *National Tap Water Quality Database. National Contaminant Report: Bromobenzene*. http://www.ewg.org/tapwater/contaminants/contaminant.php?contamcode=2993.

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# Bromochloroacetic acid

CAS No. 5589-96-8. The IUPAC name is bromochloroethanoic acid. Also called chlorobromoacetic acid. Refer also to the haloacetic acids datasheet.

### Maximum Acceptable Value

WHO (2004 and 2011) states that the available data relating to bromochloroacetic acid was considered inadequate to permit recommendation of a health-based guideline value.

### Sources to drinking-water

#### 1. To source waters

There is no reported industrial use of bromochloroacetic acid. Brominated acetic acids are formed during disinfection of water which contains bromide ions and organic matter. Bromide ions occur naturally in surface water and groundwater and exhibit seasonal fluctuations in concentrations. Bromide ion concentrations can increase due to saltwater intrusion resulting from drought conditions, or due to pollution. Bromide is introduced into New Zealand surface waters usually by wind blown seaspray.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

In dilute solutions at pH >6, more than 99.99 percent of the chemical exists as the dissociated carboxylate anion, bromochloroacetate. Bromochloroacetate contains an asymmetric carbon atom and, therefore, can exist in two non-superimposable forms, the (+)- and (–)-bromochloroacetate stereoisomers.

### Typical concentrations in drinking-water

ESR (2001) reported that 488 samples were tested for bromochloroacetic acid and was found in 51 distribution zones above the detection limit of 0.005 mg/L, with a maximum concentration of 0.023 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found <0.0005 mg/L bromochloroacetic acid in the raw water, and 0.0022 to 0.0036 mg/L in the treated water.

Brominated acetates generally are present in surface water and groundwater distribution systems at mean concentrations below 0.005 mg/L. Bromochloroacetic acid was detected in groundwater and surface water distribution systems in the US at mean concentrations of 0.0015 and 0.0036 mg/L respectively.

3,997 water utilities in the US reported detecting bromochloroacetic acid in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.169 mg/L.

### Removal methods

Brominated acetic acids arise in waters as a disinfection by-product, so the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine.

Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps.

### Analytical methods

#### Referee method

A referee method cannot be selected for bromochloroacetic acid because a MAV has not been established and therefore the sensitivity required for the referee method is not known.

#### Some alternative methods

Liquid/liquid extraction, gas chromatography-electron capture detection (APHA 6251B; EPA 552.3). DWI (2011) includes a thorough discussion on the analysis of the nine haloacetic acids.

### Health considerations

Data are limited on the oral toxicity of bromochloroacetic acid. Limited mutagenicity and genotoxicity data give generally positive results for bromochloroacetic acid (WHO 2011).

Data gaps include subchronic or chronic toxicity studies, multigeneration reproductive toxicity studies, standard developmental toxicity studies and carcinogenicity studies. The available data are considered inadequate to establish guideline values for these chemicals.

NTS reports from a recent study that there was [clear evidence](http://ntp.niehs.nih.gov/index.cfm?objectid=07027D0E-E5CB-050E-027371D9CC0AAACF#CARCDEF) of carcinogenic activity of bromochloroacetic acid in male F344/N rats based on increased incidences of malignant mesotheliomas and adenomas of the large intestine.

There was [clear evidence](http://ntp.niehs.nih.gov/index.cfm?objectid=07027D0E-E5CB-050E-027371D9CC0AAACF#CARCDEF) of carcinogenic activity of bromochloroacetic acid in female F344/N rats based on increased incidences of adenomas of the large intestine; increased incidences of multiple fibroadenomas of the mammary gland in female rats were also considered to be exposure related.

Increased incidences of pancreatic islet adenomas in male rats and of hepatocellular adenomas in male and female rats may have been related to bromochloroacetic acid exposure.

There was [clear evidence](http://ntp.niehs.nih.gov/index.cfm?objectid=07027D0E-E5CB-050E-027371D9CC0AAACF#CARCDEF) of carcinogenic activity of bromochloroacetic acid in male and female B6C3F1 mice based on increased incidences of hepatocellular neoplasms and hepatoblastoma (males only).

Exposure to bromochloroacetic acid for two years resulted in increased incidences of non-neoplastic lesions in the liver of male rats, liver and lung of female rats, and liver of male and female mice.

IARC (2012) considers that bromochloroacetic acid is possibly carcinogenic to humans (Group 2B).

The NTP (2009) completed a toxicological and carcinogenic assessment for BCAA subsequent to the Stage 2 Rule as part of their research agenda on water disinfectants and disinfection by‑products. BCAA was administered to F344/N rats and B6C3F1 mice in drinking water at daily doses up to 40 and 50 mg/kg/day in male and female rats, respectively, and 90 and 60 mg/kg/day in male and female mice, respectively in a two-year study. NTP concluded that there is clear evidence of carcinogenic activity in rats based on an increased incidence of malignant mesotheliomas in males, multiple fibroadenomas of the mammary gland in females and adenomas of the large intestine in males and females. There was also clear evidence of carcinogenic activity in mice based on increased incidences of hepatocellular neoplasms in male and female mice and hepatoblastoma in male mice. The lowest dose in rats that demonstrated an increased incidence of malignant mesotheliomas and pancreatic islet adenoma compared to controls was 20 mg/kg/day. The lowest dose in the mouse study with an increase in tumours compared to controls was 15 mg/kg/day for hepatoblastomas. Taken from USEPA (2016).

### Derivation of Maximum Acceptable Value

The are insufficient data to derive a MAV for bromochloroacetic acid at this time.

Limited mutagenicity and genotoxicity data give generally positive results for bromochloroacetic acid. Because bromochloroacetic acid has not been tested in subchronic or chronic toxicity studies, the available data are considered inadequate to establish guideline values for this chemical. Other data gaps include the absence of multigeneration reproductive toxicity studies, standard developmental toxicity studies and carcinogenicity studies (WHO 2004a).

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# Bromochloroacetonitrile

CAS No. 83463-62-1. Can also be called bromochloromethyl cyanide, bromochloroethanenitrile and 2-bromo-2-chloroacetonitrile. Datasheets also exist for 2-chloroacetonitrile, 2,2-dichloroacetonitrile, 2,2-dibromoacetonitrile, 2,2,2-trichloroacetonitrile, and bromoacetonitriles.

### Maximum Acceptable Value

WHO (2004 and 2011) considered that the available data was inadequate to permit recommendation of a health-based guideline value.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for haloacetonitriles in drinking water.

### Sources to drinking-water

#### 1. To source waters

Halogenated acetonitriles are not produced on an industrial scale.

#### 2. From treatment processes

Halogenated acetonitriles arise during water chlorination, chloramination, and to a lesser extent, when dosing with chlorine dioxide, from naturally occurring substances including algae, fulvic acid and proteinaceous material. In general, increasing temperature and/or decreasing pH have been associated with increasing concentrations of halogenated acetonitriles. Ambient bromide levels appear to influence, to some degree, the speciation of halogenated acetonitrile compounds. Dichloroacetonitrile is by far the most predominant halogenated acetonitrile species detected in drinking-water from sources with bromide levels of 0.02 mg/L or less. In chlorinated or chloraminated water from sources with higher bromide levels (0.05 to 0.08 mg/L), bromochloroacetonitrile was the second most prevalent compound. However, none of the treated water from any of these sources had a dibromoacetonitrile concentration exceeding 0.0005 mg/L, including treated water from one source that had a much higher bromide level (DWI 2010).

#### 3. From the distribution system

Halogenated acetonitriles that form at the treatment plant tend to hydrolyse as they pass through the distribution system.

### Form and fate in the environment

Haloacetonitriles are reported to undergo hydrolysis in water, yielding non-volatile products.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 209 zones, found bromochloroacetonitrile concentrations in 1 zone to range from “not detectable” (nd) to 0.005 mg/L, with the median concentration being “nd” (limit of detection = 0.002 mg/L) (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.00014 mg/L bromochloroacetonitrile in the raw water, 0.0006 to 0.0007 mg/L in the treated water, and up to 0.0013 mg/L in the distribution system.

Health Canada reported 0.0006 and 0.0015 mg/L in treated water drawn from the Winnipeg River; the raw water contained <0.0001 mg/L. Bromochloroacetonitrile was detected in US groundwater and surface water distribution systems at mean concentrations of 0.0007 and 0.0011 mg/L respectively.

DWI (2012) reported a UK study. The lowland water sources that were included in the survey formed more N-DBPs than the upland and groundwater sources. The six treatment works that applied ozone were associated with higher concentrations of HANs and HAcAms than non-ozone treatment works, although this was potentially confounded because all the ozone works were treating lowland source waters which may have had higher N-DBP formation potential. None of the N-DBPs exhibited consistent links with total trihalomethanes (THMs). There were no trends linking HNMs to either THMs or HAA9. The mean HAN concentration was 0.0032 mg/L (3.2 µg/L). The main HANs were dichloroacetonitrile, bromochloroacetonitrile and dibromoacetonitrile. Trichloroacetonitrile was not detected.

### Removal methods

As bromochloroacetonitrile arises in waters as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine.

Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

### Analytical methods

#### Referee method

A referee method cannot be selected for bromochloroacetonitrile because a MAV has not been established and therefore the sensitivity required for the referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for bromochloroacetonitrile for the above reason. However, the following information may be useful.

APHA (2005) states that haloacetonitriles in water may be determined by solvent extraction with methyl tert-butyl ether and analysed by capillary column/electron capture/gas chromatography, electron capture (EPA Method 551, 1990). Quantitation limits of 0.0004 mg/L (0.4 g/L) are achievable. Interference may come from contaminated reagents or glassware.

### Health considerations

Several halogenated acetonitriles have been detected in chlorinated drinking-water in a number of countries as a consequence of the reaction of chlorine with natural organic substances and bromide present in untreated water. The only known route of human exposure is through chlorinated drinking-water. Haloacetonitriles tend to be more toxic than the haloacids.

Haloacetonitriles are absorbed rapidly from the gastrointestinal tract and metabolised to single carbon compounds. Insufficient data are available to determine whether haloacetonitriles can accumulate in specific organs.

No data are available on the health effects of haloacetonitriles in humans. IARC considers bromochloroacetonitrile is not classifiable as to its carcinogenicity to humans (Group 3).

Bromochloroacetonitrile was a direct-acting mutagen in tests on bacteria and induced DNA damage (sister chromatid exchange and DNA strand breaks) in mammalian cells.

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. One of their findings was that for cell induced genomic DNA damage, the rank order from the most genotoxic to the least genotoxic of the DBP classes was haloacetonitriles > haloacetamides > halonitromethanes > haloacetaldehydes > haloacetic acids > >2C‑haloacids > halomethanes.

2-Bromo-2-chloroacetonitrile is cytotoxic and causes genotoxicity in vitro. There is ‘moderate’ concern for carcinogenicity based on SAR analysis, and an increase in squamous cell carcinomas was shown, but there is insufficient evidence for carcinogenicity due to the lack of tests. The LOAEL for developmental and teratogenic effects was the lowest dose tested, 5 mg/kg of body weight per day, based on decreased fetal growth and teratogenicity. However, this may be overestimated because of the use of tricaprylin as gavage vehicle. The NOAEL for maternal toxicity was 45 mg/kg of body weight per day, based on adverse maternal effects; the high dose of 65 mg/kg of body weight per day was a LOAEL, based on decreased maternal weight (after adjusting for gravid uterine weight) and increased dam mortality (DWI 2010).

### Derivation of Maximum Acceptable Value

There are insufficient data to determine a MAV for bromochloroacetonitrile in drinking-water.

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# Bromochlorodimethylhydantoin

CAS No. 126-06-7; it appears as though 107846-11-7 and 16079-88-2 have also been allocated (possibly commercial products). The full name is 3-bromo-1-chloro-5,5-dimethylhydantoin. Also called bromochlorodimethyl-2,4-imidazolinedione, BCDMH or bromo chloro dimethyl hydantoin. It is sold under various trade names. At least one publication (mis)spells hydantoin as hydrantoin. The commercial product comprises about 63 percent bromine and 28 percent chlorine.

### Maximum Acceptable Value

Bromochlorodimethylhydantoin does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to water

3-Bromo-1-chloro-5,5-dimethylhydantoin appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)) as a bactericide and fungicide.

Bromochlorodimethylhydantoin (listed under various trade names) appears in the NSF/ANSI Standard 60: Drinking Water Treatment Chemicals – Health Effects, see <http://www.nsf.com/Certified/PwsChemicals/Listings.asp?=&ChemicalName=Bromochlorodimethylhydantoin>), where its function is described as disinfection, oxidation, algicide, bactericide and other. The most common maximum dose is quoted as 3 mg/L with the proviso: “The residual levels of chlorine (hypochlorite ion and hypochlorous acid), chlorine dioxide, chlorate ion, chloramine and disinfection by‑products shall be monitored in the finished drinking water to ensure compliance to all applicable regulations.” Some maximum doses reach 15 mg/L, with the proviso: “These products are to be added to the feed water for evaporation desalination units.”

It is used in industrial water supplies at dosages recommended to achieve exposures to 1 mg/L of ‘active’ residual bromine to control algal, bacterial and fungal slimes in industrial recirculating cooling towers; influent water systems, eg, flow-through filters, and lagoons etc; heat exchange water systems; industrial water scrubbing systems; and industrial air-washing systems equipped with a mist eliminator.

The Australia New Zealand Food Authority (ANZFA 2000) assessed BCDMH for sanitising water used to wash fruit and vegetables, both post harvest and in the production of minimally processed fruit and vegetable products, largely as a substitute for chlorine.

BCDMH produces hypobromous acid (650 g/kg available bromine) and hypochlorous acid (260 g/kg available chlorine) in water.

WHO (2006) states: liquid bromine is not commonly used in pool disinfection. Bromine-based disinfectants for pools are available in two forms, bromochlorodimethylhydantoin (BCDMH) and a two-part system that consists of sodium bromide and an oxidiser (usually hypochlorite).

### Forms and fate in the environment

BCDMH breaks down to produce hypobromous and hypochlorous acids (which would lead to the formation of halides on the treated produce) and dimethylhydantoin (also called 5,5-dimethyl-2,4-imidazolidinedione or DMH).

Water solubility is about 1,500–2,000 mg/L.

### Recommended analytical techniques

#### Referee method

No MAV, so not needed.

### Health considerations

Charles River rats (20/sex/group) received 0, 500, 5,000 or 50,000 ppm DMH in drinking water for 13 weeks. Ten males and three females in the high-dose group died. At high-dose animals showed thinness and emaciation, urogenital staining, hunching, decreased motor activity, ataxia, irritability and reduced bodyweight gains and food and water consumption. Histo-pathological changes in high-dose animals included atrophy of the thymus, spleen and lymph nodes, renal necrosis of the tip of the papilla, pelvic transitional cell hyperplasia, hyperplasia of the epithelial lining of the renal papilla, atrophy of the uterine wall and gastric necrotic inflammation. The NOEL of 500 ppm was determined which corresponded to approximately 50 mg/kg bw/day in the diet. Based on this subchronic study and using a safety factor of 2000, an ADI of 0.025 mg/kg bw/day can be established for DMH. Based on this ADI, dietary intakes calculations show that only 42 percent of the ADI would be reached (ANZFA 2000).

ANZFA also performed a dietary exposure calculation based on residues in fruit and vegetables of DMH and conservative values in other commodities for inorganic bromide (50 mg/kg for cereal grains and 400 mg/kg for spices). A total dietary exposure was calculated at 0.16 mg/kg bw/day (16 percent of ADI for bromide) for average consumers and 0.39 mg/kg bw/day (38 percent of ADI for bromide) for high consumers (95th percentile).

Hydantoins (diphenylhydantoins) are used therapeutically, particularly as antiepileptic agents.

DWI (2011) includes a case report of irritant contact dermatitis to 1-bromo-3-chloro-5,5-dimethylhydrantoin in a hydrotherapy pool in the UK. The case involved a 46-year-old male with no previous history of atopy and no family history of note. The skin reaction involved asteatotic eczema with severe excoriation and lichenification of the upper and lower limbs. The trunk was also affected, but there were no signs of facial involvement. The symptoms developed upon immersion into a hydrotherapy pool disinfected with 1-bromo3-chloro-5,5-dimethylhydrantoin. The method of disinfection had recently been changed from the use of sodium hypochlorite. The patient had routinely used the pool prior to the change in disinfectant, and continued to do so, without skin reaction, following a further change to a chlorine-based disinfectant. The association between bromide-based disinfectants and irritant contact dermatitis is well recognised.

### Derivation of Maximum Acceptable Value

No MAV.

### References

ANZFA. 2000. *Full Assessment Report and Regulatory Impact Statement, Application A393, Bromo-chloro-dimethylhydantoin (BCDMH) as a Processing Aid*. 25 pp. [www.foodstandards.gov.au/standardsdevelopment/applications/applicationa393bromo934.cfm](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.foodstandards.gov.au\standardsdevelopment\applications\applicationa393bromo934.cfm) or http://www.foodstandards.govt.nz/\_srcfiles/A393\_FA.pdf.

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# Bromochloromethane

CAS No. 74-97-5. Also called chlorobromomethane, methylene chlorobromide, Halon 1011, or CBM.

### Maximum Acceptable Value

There are insufficient data to determine a MAV for bromochloromethane in drinking-water.

The USEPA concluded on 22 September 2009 that bromochloromethane is known or anticipated to occur in PWSs and may require regulation. Therefore they added bromochloromethane to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a). The USEPA (2006/2011) established a lifetime health advisory of 0.09 mg/L, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

### Sources to drinking-water

#### 1. To source waters

Bromochloromethane is an intermediate in chemical manufacturing and a fire extinguishing agent. These uses should diminish because bromochloromethane is listed as a controlled substance in the Handbook for the Montreal Protocol on Substances that Deplete the Ozone Layer – 7th edition (2006). It had been found in 15 of 91 samples taken from Lake Ontario in 1981, up to 0.00001 mg/L.

#### 2. From treatment processes

Bromochloromethane is a disinfection by-product, although it is not produced by the classic reactions that produce the trihalomethanes.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Bromochloromethane is very soluble (about 16,000 mg/L) so has the potential to migrate to groundwater where it may be fairly stable. It will volatilise from soil and surface water.

### Typical concentrations in drinking-water

ESR (2001) reported that 173 samples were tested for bromochloromethane and was found in 12 distribution zones above the detection limit of 0.0005 mg/L, with a maximum concentration of 0.0011 mg/L.

An Environmental Working Group (EWG) analysis of bromochloromethane tests reported by 18,379 USA public water suppliers in 33 states shows that between 1998 and 2003, 75 communities had water containing bromochloromethane. Most were below the ‘lifetime exposure concentration’ of 0.05 mg/L that is protective of adverse, non-carcinogenic health effects, that assumes all of the exposure to a contaminant is from drinking water; the highest concentration found was 7 mg/L. 121 water utilities in the US reported detecting bromochloromethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.012 mg/L.

Bromochloromethane has been found in groundwater in the Netherlands up to 0.008 mg/L.

### Removal methods

Bromochloromethane can be removed from water by air stripping. Activated carbon is not likely to be effective.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

The USEPA has stated that bromochloromethane is not classifiable as to human carcinogenicity, ie, Class D, based on the lack of data regarding the carcinogenicity of bromochloromethane in humans or animals. However, there are data indicative of genotoxic effects and structural relationships to halogenated methanes (eg, dichloromethane) classified as B2 probable human carcinogens.

The reference dose or RfD (USEPA 2006/2009/2011) is 0.01 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.5 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

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USEPA. 2009a. *Contaminant Information Sheets for the Final CCL 3 Chemicals*. EPA 815‑R-09-012. 216 pp. <http://water.epa.gov/scitech/drinkingwater/dws/ccl/upload/Final-CCL-3-Contaminant-Information-Sheets.pdf>.

# Bromodichloroacetic acid

CAS No. 71133-14-7. The IUPAC name is 2-bromo-2,2-dichloroacetic acid. May also be called dichlorobromoacetic acid or BDCAA. Refer also to the haloacetic acids datasheet.

### Maximum Acceptable Value

Bromodichloroacetic acid does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Bromodichloroacetic acid is not a natural product, and not manufactured for any particular use.

#### 2. From treatment processes

Bromodichloroacetic acid is not often listed as a disinfection by‑product, however, it is found (Wu 1998). This chemical is formed after disinfection of water with halogenated oxidants, usually chlorine (NTP 2010). The sum of bromodichloroacetic acid, dibromochloroacetic acid, and tribromoacetic acid concentrations is known as HAA3.

#### 3. From the distribution system

No known sources.

### Typical concentrations in drinking-water

351 water utilities in the US reported detecting bromodichloroacetic acid in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.018 mg/L.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

DWI (2011) includes a thorough discussion on the analysis of the nine haloacetic acids.

### Health considerations

In genetic toxicology studies using *Salmonella*, bromodichloroacetic acid was found to be weakly positive (NTP 2010).

Studies are being planned for bromodichloroacetic acid, one of the brominated HAAs, which (based on preliminary data) are suspected of being more toxic than their chlorinated HAA sisters (EHP 2000).

NTP (2015) conducted a two-year bioassay of BDCAA in treated F344/NTac rats and B6C3F1 mice. BDCAA administration in drinking water to rats resulted in increased incidences of malignant mesothelioma and combined incidences of epithelial tumours of the skin in males, increased incidences of fibroadenoma and carcinoma of the mammary gland in females, along with adenoma or carcinoma of the Harderian gland in males and hepatocellular adenoma in females. In mice there was an increased incidence of hepatocellular carcinoma and hepatoblastoma in males and females. The lowest dose to induce tumours at levels above controls in female rats was 13 mg/kg/day for mammary gland fibroadenoma. In male rats, the lowest dose to observe increased incidence of keratoaceanthoma, basal cell ademona or carcinoma, squamous cell carcinoma (SCC) and other carcinogenic endpoints was 43 mg/kg/day. The lowest dose with evidence of carcinogenicity in male and female mice was 23 mg/kg/day based on an increased incidence of hepatocellular adenoma. NTP (2015) concluded that there was clear evidence of carcinogenicity in male and female rats and mice. Taken from USEPA (2016).

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2011a. *Evaluation of Haloacetic Acid Concentrations in Treated Drinking Water*. Report No. WT1236. 111 pp. http://dwi.defra.gov.uk/research/completed-research/reports/DWI70\_2\_242.pdf.

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Wu WW. 1998. Effect of bromide ion on haloacetic acid formation during chlorination of Biscayne aquifer water. [*Journal of Environmental Engineering*](http://cedb.asce.org/cgi/WWWdisplay.cgi?168569) 124(10): 932–8. See abstract http://cedb.asce.org/cgi/WWWdisplay.cgi?9804487.

# Bromodichloromethane

CAS No. 75-27-4. Also called dichlorobromomethane or BDCM.

### Maximum Acceptable Value

Based on health considerations, the concentration of bromodichloromethane in drinking-water should not exceed 0.06 mg/L. Trihalomethanes (BDCM) are included in the [plan of work of the rolling revision](http://www.who.int/entity/water_sanitation_health/dwq/en/index.html) of the WHO Guidelines for Drinking-water Quality.

Bromodichloromethane is one of the four trihalomethanes with a MAV in the DWSNZ. The others are bromoform, chloroform and dibromochloromethane. The sum of the ratio of the concentrations of these four trihalomethanes to each of their respective MAVs must not exceed one. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, section 10.2.5.3).

As at 2006 Health Canada stated the maximum acceptable concentration (MAC) for bromodichloromethane (BDCM) in drinking water was 0.016 mg/L (16 µg/L) monitored at the point in the distribution system with the highest potential THM levels. Effective April 2009, the guideline statement for trihalomethanes in drinking water was modified to remove the separate guideline for BDCM, recognising that the maximum acceptable concentration for THMs is protective of the health effects of all THMs, including BDCM.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) do not include a specific guideline value for bromodichloromethane. They do have one for total trihalomethanes though (qv).

The maximum contaminant level for total trihalomethanes (USEPA 2006/2009/2011) is 0.08 mg/L. The maximum contaminant level goal (MCLG) for bromodichloromethane is zero (USEPA 2006/2009).

Dichlorobromomethane is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Brominated trihalomethanes, such as bromodichloromethane (sometimes abbreviated to BDCM), may occur in raw water as industrial contaminants. They are used as laboratory reagents, chemical intermediates, as fluids for mineral ore separation, as solvents for waxes, fats, and resins and as flame retardants.

#### 2. From treatment processes

Trihalomethanes, including bromodichloromethane, are most likely to be formed as by-products of the chlorination of drinking-water. Naturally-occurring bromide is oxidised by chlorine to form bromine (hypobromous acid and hypobromite ion) which reacts with organic matter, such as humic and fulvic acids, in the water, with the result that the trihalomethanes found in the water show varying degrees of bromine incorporation. When full bromine substitution occurs, bromoform is produced; mixed substitution of chlorine and bromine results in dibromochloromethane and bromodichloromethane. The concentration of trihalomethanes produced depend upon: pH, organic matter concentration, chlorine dose, bromide concentration, contact time and temperature.

Being a disinfection by-product, the USEPA (2007) regulates bromodichloromethane.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

In air, brominated trihalomethanes may be removed through oxidation with atmospheric hydroxyl radicals. Bromodichloromethane is soluble in water (about 4,500 mg/L). Volatilisation to the atmosphere is the major removal mechanism for bromodichloromethane from drinking-water, as it leaves the tap or shower. Biodegradation occurs under anaerobic conditions. Hydrolysis is extremely slow. Bioaccumulation in aquatic organisms may occur. Brominated trihalomethanes are expected to be mobile in soil.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987-1992 contained bromodichloromethane results from 370 samples representing 157 chlorinated supplies. Bromodichloromethane was detected in 304 samples in concentrations ranging from 0.0002 to 0.076 mg/L (0.2–76 g/L).

The P2 Chemical Determinand Identification Programme sampled, from 511 zones, found bromodichloromethane concentrations to range from “not detectable” (nd) to 0.055 mg/L, with the median concentration being 0.0031 mg/L (limit of detection = 0.002 mg/L). The Priority 2 Identification Programme found no distribution zone supplying drinking-water with bromodichloromethane at greater than the MAV, but four distribution zones supplied 8080 people at >50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0005 mg/L bromodichloromethane in the raw water, 0.0041 to 0.0043 mg/L in the treated water, and up to 0.0067 in the distribution system.

A large study in Canada found mean bromodichloromethane levels were typically less than 0.01 mg/L, although some averages were higher, and several locations reported one-time samples in excess of 0.2 mg/L. From those suppliers that reported BDCM data, 87 water systems (8 percent of reporting systems), representing a sampled population of 285,000 (2 percent of population served), reported having mean BDCM levels greater than 0.01 mg/L, while 192 water systems (18 percent), serving a sampled population of 1,165,000 (8 percent), reported at least one instance of BDCM levels being greater than 0.01 mg/L. Generally, the smaller centres with less sophisticated treatment systems had higher THM levels in their drinking-water (Health Canada 2006).

16,971 water utilities in the US reported detecting bromodichloromethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.15 mg/L.

### Removal methods

As bromodichloromethane arises in waters principally as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine.

Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Trihalomethane concentrations in a chlorinated water increase with increasing pH. Concentrations in the finished water therefore can be reduced by ensuring that high pH levels are not present once the water is chlorinated.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Bromodichloromethane can be removed from contaminated source waters by adsorption on to granular activated carbon, or by air stripping. Adsorption efficiency increases, and air stripping efficiency decreases, with increasing bromine substitution in the trihalomethane. BDCM removal using packed tower aeration depends on the air-to-water ratio, water loading rate, and packing depth; 74 percent removal from an inlet concentration of approximately 0.1 mg/L was achieved in pilot-scale studies.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551).

### Health considerations

The major human exposure to bromodichloromethane is via drinking-water. Available studies indicate that gastrointestinal absorption is high for all trihalomethanes. They are fat soluble and accumulation is higher in tissues with high lipid content, including body fat, liver and kidneys.

In a 90-day study in rats administered bromodichloromethane in drinking-water, mild to moderate histological changes in the liver and thyroid and a significant increase in the severity of hepatic lesions were observed at the highest dose.

The International Agency for Research on Cancer has classified bromodichloromethane in Group 2B (possibly carcinogenic to humans).

USEPA (2009) quotes a health advisory of 0.1 mg/L for bromodichloromethane, representing a 10-4 cancer risk.

Bromodichloromethane appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

In the Stage 1 D/DBPR, USEPA established an MCLG of zero for BDCM and classified BDCM as a “*probable human carcinogen*” based on a weight of evidence evaluation of both the cancer and non-cancer effects. They later classified BDCM as “*likely to be carcinogenic by all routes of exposure*” following the new cancer guidelines. The MCLG of zero was assigned based on intestine and kidney tumour data from a chronic animal carcinogenicity study. The low-dose extrapolation approach was used to estimate cancer risk since there was insufficient evidence regarding the mode of action of BDCM. The RfD presented on IRIS at the time of the Stage 1 D/DBPR (and which is still currently on IRIS) is 0.02 mg/kg/day, based on a lowest observed adverse effect level (LOAEL) of 17.9 mg/kg/day for renal cytomegaly in male mice with the application of an uncertainty factor of 1000. In support of the Stage 2 D/DBPR, a criteria document for brominated THMs was used in which USEPA derived an RfD of 0.003 mg/kg/day for BDCM based on degeneration of the liver in a 24-month dietary study in rats. However, for the Stage 2 D/DBPR, USEPA determined that there were no new significant health effects data suggesting the need for a change in the categorization of BDCM as a *likely* human carcinogen nor for a change in the MCLG of zero. Copied from USEPA 2016).

Although BDCM has given mixed results in bacterial assays for genotoxicity, the results have tended to be positive in tests employing closed systems to overcome the problem of the compound’s volatility. Among the four THMs commonly found in drinking-water, BDCM appears to be the most potent rodent carcinogen. BDCM caused tumours at lower doses and at more target sites than for any of the other THMs.

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. One of their findings was that diiodoacetamide was the most cytotoxic agent and bromodichloromethane was the least cytotoxic.

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <https://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>. As at April 2018 draft MRLs for bromodichloromethane are:

minimal risk level

0.1 mg/kg/day for acute-duration oral exposure (1–14 days)

0.008 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD (USEPA 2006/2009/2011) is 0.003 mg/kg/d (it had been 0.02 mg/kg/d in 1993). The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.1 mg/L.

### Derivation of Maximum Acceptable Value

The estimated range of concentrations corresponding to an upper-bound excess lifetime cancer risk of 10-5 is 0.025–0.077 mg/L for the critical tumour types (ie, intestinal adenomatous polyps and adenocarcinomas; renal tubular cell adenomas and adenocarcinomas) in rats, based on a linearised multi-stage model, and 0.021 mg/L for the critical tumour types, ie, renal adenomas and adenocarcinomas (combined) in male mice.

Although a guideline value for BDCM in drinking-water of 0.021 mg/L, which is the most conservative of the values noted above, could be derived, the previous guideline value of 0.06 mg/L is retained for two reasons.

First, and most important, both the previous guideline and the above calculations were based on the same study. The only differences between the new calculation and the previous guideline are the model and model assumptions used to derive the guideline value. Both calculations support the observation that a guideline based on the mouse tumours is more conservative than a guideline based on the rat tumours. There is therefore no scientific basis on which to justify a change in the guideline value.

Second, concentrations of BDCM below 0.05 mg/L may be difficult to achieve using currently available technology without compromising the effectiveness of disinfection.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The cancer health risk limit for bromodichloromethane is 0.006 mg/L.

The WHO guideline value only considers exposure to THMs in water via the oral route. However, WHO accepts THMs are volatile chemicals, and therefore, exposure via the inhalation and dermal routes may be significant sources of exposure, particularly during bathing and showering, as increasing water temperature will increase the rate of volatilisation, and ventilation may be poor. WHO suggested that in colder countries with low rates of ventilation in houses or where the incidences of showering and bathing are high, this guideline value may be lowered (WHO 2005, in DWI 2010).

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# Bromoethane

CAS No. 74-96-4. Also called ethyl bromide.

### Maximum Acceptable Value

There is no MAV for bromoethane in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Bromoethane has limited commercial use, mainly used as an intermediate in the production of other chemicals, primarily in the pharmaceutical industry, but also in the manufacture of pesticides. Small amounts could potentially be released from landfill and incinerator sites from disposal of bromoethane-containing goods. It has also been used as an anti-knocking agent in fuels. Bromoethane was found to be a minor component of bromomethanes and bromochloromethanes released from brown algae collected from the North and South Atlantic to the surrounding air.

#### 2. From treatment processes

The only reference to bromoethane being a disinfection by‑product was in Techneau (2007) where it appears in a list of chlorination by‑products.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

If released to water, bromoethane will be removed by hydrolysis and volatilisation. Aqueous hydrolysis half-lifes range from five days at 35°C to 21–30 days at 25°C. The volatilisation half-lifes from a model river and pond have been estimated to be 3.2 hours and 38.2 hours, respectively. If released to soil, bromoethane will be susceptible to hydrolysis under wet soil conditions.

In groundwater under reducing conditions in the presence of hydrogen sulphide, bromoethane may react with naturally occurring nucleophiles (such as the sulfhydryl ion found near sulphur and sulphide deposits) to form aliphatic sulphur-containing products.

Bromoethane is quite soluble in water, about 900 mg/L. Volatilisation is the major removal mechanism for bromoethane from water.

### Typical concentrations in drinking-water

No information available.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Since bromoethane is an ethylating agent, it would, like other alkylating agents, be predicted to possess genotoxic activity, particularly at sites of initial contact. Such direct-acting genotoxicity is observed in bacteria and in Chinese hamster ovary cells in vitro, but there are no other relevant studies. Thus, it is not possible to assess whether or not such activity would be expressed in vivo.

The main health concerns relating to exposure to bromoethane are the potential for neurotoxicity, haematological and hepatic toxicity, irritation of the respiratory tract, damage to genetic material, and carcinogenicity. The characterisation of the risk of developing these effects is somewhat complicated by the lack of dose–response information. Descriptions of neurotoxicity in humans are essentially qualitative in nature, but studies in animals have indicated that such effects are seen only following exposure to high concentrations. Similarly, haematological and liver damage in animals has been observed only at high exposure concentrations (WHO 2002).

There is limited evidencein experimental animals for the carcinogenicity of bromoethane. Studies assessed by IARC were all inhalation studies, so are not particularly relevant to water supply. The International Agency for Research on Cancer (IARC) has classified bromoethane in Group 3 (not classifiable as to its carcinogenicity in humans).

The Office of Environmental Health Hazard Assessment (OEHHA) of the California Environmental Protection Agency added bromoethane to their list of chemicals known to cause cancer on December 2000, based on increased incidence of malignant and combined malignant and benign tumours in female mice to an unusual degree with regard to site and incidence, concluding that there is clear evidence of the carcinogenic activity of bromoethane in female mice. Note: these were inhalation studies.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# 2-bromoethanol

CAS No. 540-51-2. Also called ethylene bromohydrin, 2-hydroxyethyl bromide, 1‑bromo-2-ethanol, 2-bromoethan-1-ol and EBH. Also see halohydrin datasheet.

This datasheet also covers ethylene chlorohydrin (CAS 107-07-3).

### Maximum Acceptable Value

There is no MAV for 2-bromoethanol or 2-chloroethanol in the DWSNZ, and they are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Several halohydrins are said to have been reported to form in water following disinfection with ozone or chlorine. However, no specific references were found.

Ethylene bromohydrin and ethylene chlorohydrin have been shown to result from fumigation of foods with ethylene oxide due to interaction with natural bromides and chlorides present in the food items. Food presents the main intake for humans, mainly as a result of fumigation, especially with ethylene oxide, with ethylene chlorohydrin and ethylene bromohydrin being the main by-products of health concern; drinking-water is not an important route (IUPAC 1997; INCHEM 1971).

#### 2. From the distribution system

No known sources.

### Form and fate in the environment

If released on soil, 2-chloroethanol would be expected to leach because of its estimated very low adsorptivity to soil and may biodegrade. It should not volatilise from moist soil, but may evaporate from dry soil and other surfaces. 2-chloroethanol’s fate in the aquatic environment is not clear. Based on data from screening tests, it may biodegrade. It would not be expected to volatilise from surface waters, adsorb to sediment or bioconcentrate in aquatic organisms. 2-chloroethanol reacts with photochemically-produced hydroxyl radicals in the atmosphere, as a result of which its half-life in the atmosphere will be approximately 11.5 days. 2-chloroethanol is soluble in water and would be washed out of the air by rain (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

No information available.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Ethylene oxide reacts with available chlorine and bromide to form detectable residues of ethylene chlorohydrin and ethylene bromohydrin in treated food. The residues of ethylene oxide itself decrease rapidly (usually to <20 ppm) but residues of chlorohydrin may persist for much longer periods (and up to 1500 ppm). The available data indicates that ethylene chlorohydrin is significantly less mutagenic than ethylene oxide and, as such, presents a significantly reduced public health and safety risk. There is no mutagenicity data available on ethylene bromohydrin. There are no known significant cancer risks associated with consumption of ethylene chlorohydrin and ethylene bromohydrin at the levels found in spices. It is acknowledged, however, that the toxicological database on these compounds is limited. There are no regulatory limits internationally for ethylene bromohydrin in foods (ANZFA 2000).

The USEPA assessed ethylene oxide and its reaction products (ethylene bromohydrin, ethylene chlorohydrin and ethylene glycol) for dietary (oral) exposure and risk; they determined that the dietary risk was below the Agency’s level of concern after risk mitigation options were adopted.

Some halohydrins are suspected human carcinogens.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Bromoform

CAS No. 75-25-2. Also called methyl tribromide, tribromomethane or methenyltribromide.

### Maximum Acceptable Value

Based on health considerations, the concentration of bromoform in drinking-water should not exceed 0.1 mg/L.

Bromoform is one of the four trihalomethanes with a MAV in the DWSNZ. The others are bromodichloromethane, chloroform and dibromochloromethane. The sum of the ratio of the concentrations of these four trihalomethanes to each of their respective MAVs must not exceed one. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, section 10.2.5.3).

The maximum contaminant level for total trihalomethanes (USEPA 2006/2009/2011) is 0.08 mg/L. The maximum contaminant level goal (MCLG) for bromoform is zero (USEPA 2006/2009/2011).

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) do not include a specific guideline value for bromoform. They do have one for total trihalomethanes though (qv).

### Sources to drinking-water

#### 1. To source waters

Brominated trihalomethanes may occur in raw water as industrial contaminants and from human activity. They have been used as laboratory reagents, chemical intermediates, as fluids for mineral ore separation, as solvents for waxes, fats, resins, and as flame retardants. Bromoform has been used as a sedative and cough depressant. Very little is manufactured today.

#### 2. From treatment processes

Brominated trihalomethanes are most likely to be formed as by-products of the chlorination of drinking-water. Naturally-occurring bromide is oxidised by chlorine to form bromine (hypobromous acid and hypobromite ion), which reacts with organic matter in the water, such as humic and fulvic acids, with the result that the trihalomethanes found in the water show varying degrees of bromine incorporation. When full bromine substitution occurs, bromoform is produced; mixed substitution of chlorine and bromine results in dibromochloromethane and bromodichloromethane. The concentration of trihalomethanes produced depend upon: pH, organic matter concentration, chlorine dose, bromide concentration, contact time and temperature.

Being a disinfection by-product, the USEPA (2007) regulates bromoform.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

In air, brominated trihalomethanes may be removed by oxidation with atmospheric hydroxyl radicals. Bromoform is quite soluble in water (about 1,000–3,000 mg/L). Volatilisation is the major removal mechanism for bromoform from water. Biodegradation occurs under anaerobic conditions. Hydrolysis is extremely slow. Bioaccumulation in aquatic organisms may occur. Brominated trihalomethanes are expected to be mobile in soil.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987–1992, contained bromoform results from 370 samples representing 157 chlorinated supplies. Bromoform was detected in 82 samples in concentrations ranging from 0.00033–0.029 mg/L (0.33–29 g/L).

The P2 Chemical Determinand Identification Programme, sampled from 511 zones, found bromoform concentrations to range from “not detectable” (nd) to 0.049 mg/L, with the median concentration being “nd” (limit of detection = 0.001 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with bromoform at greater than 50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0005 mg/L bromoform in the raw water, 0.0009 to 0.0012 mg/L in the treated water, and up to 0.0017 mg/L in the distribution system.

A large study in Canada found mean bromoform levels were typically less than the detection limit of 0.0005 mg/L and individual values were less than 0.01 mg/L, with occasional values over 0.03 mg/L. Generally, the smaller centres with less sophisticated treatment systems had higher THM levels in their drinking-water (Health Canada 2006).

11,205 water utilities in the US reported detecting bromoform in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.21 mg/L.

### Removal methods

Bromoform present in contaminated source waters may be removed by adsorption on to granular activated carbon, or by air stripping. Adsorption efficiency increases, and air stripping efficiency decreases, with increasing bromine substitution in the trihalomethane.

However, as this compound arises in waters principally as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine.

Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Trihalomethane concentrations in chlorinated water increase with increasing pH. Finished water concentrations can therefore be reduced by ensuring that high pH levels are not present once the water is chlorinated.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products. Bromoform reacts very slowly with ozone.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Bromoform can be removed by adsorption on to granular activated carbon, or by air stripping. Adsorption efficiency increases, and air stripping efficiency decreases, with increasing bromine substitution in the trihalomethane.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

Action to reduce THMs is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than THMs.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551).

### Health considerations

Available studies indicate that gastrointestinal absorption is high for all trihalomethanes. They are fat soluble and accumulation is higher in tissues with high lipid content, including body fat, liver and kidneys.

In a 90 day study in rats administered bromoform in drinking-water, mild to moderate histological changes in the liver and thyroid and a significant increase in the hepatic lesions were observed at the highest dose. In an NTP bioassay, bromoform induced a small increase in relatively rare tumours of the large intestine in rats of both sexes but did not induce tumours in mice. Data from a variety of assays on the genotoxicity of bromoform are equivocal.

In the past, orally administered bromoform was used as a sedative for children with whooping cough. Occasionally, instances of death were reported due to accidental overdose. The clinical signs in fatal cases were central nervous system depression followed by respiratory failure.

The International Agency for Research on Cancer has classified bromoform in Group 3 (not classifiable as to its carcinogenicity in humans). USEPA (1993) classified bromoform as B2: a probable human carcinogen. USEPA (2009) quotes a health advisory of 0.8 mg/L for bromoform, representing a 10-4 cancer risk.

The USEPA established an MCLG of zero for bromoform and classified bromoform as a “*probable human carcinogen*” based on a weight of evidence evaluation of both the cancer and non-cancer effects. Under the 2005 cancer guidelines it was classified as “*likely to be carcinogenic by all routes of exposure*”). The MCLG is based on a chronic animal carcinogenicity study that reported uncommon neoplasms of the large intestines in rats. Insufficient evidence exists regarding the mode of carcinogenic action of bromoform, therefore, the low-dose extrapolation approach was used to be protective of public health. The RfD of 0.02 mg/kg/day is based on a No-Observed-Adverse-Effect-Level (NOAEL) of 25 mg/kg/day from subchronic data for hepatic lesions in male rats with the application of an uncertainty factor of 1,000 (copied from USEPA 2016).

Bromoform appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

There is some evidence to suggest that bromoform may be weakly mutagenic. Bromoform, in common with the other brominated THMs, is largely positive in bacterial assays of mutagenicity conducted in closed systems.

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 and August 2018 MRLs for are:

minimal risk level

0.7 mg/kg/day for acute-duration oral exposure (1–14 days)

0.2 mg/kg/day for intermediate-duration oral exposure (15–364 days)

0.02 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD (USEPA 2006/2009/2011) is 0.03 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 1 mg/L.

### Derivation of Maximum Acceptable Value

A TDI of 17.9 mg/kg of body weight has been used (derived on the basis of a NOAEL of 25 mg/kg of body weight per day), based on the absence of histopathological lesions in the liver in a well-conducted and well-documented 90-day study in rats, using an uncertainty factor of 1,000 (100 for intra- and interspecies variation and 10 for possible carcinogenicity and short duration of exposure). This NOAEL is supported by the results of two long-term studies.

The MAV for bromoform in drinking-water was derived as follows:

17.9 mg/kg body weight per day x 70 kg x 0.2 = 0.125 mg/L (rounded to 0.1 mg/L)

2 L x 1000

where:

* tolerable daily intake (TDI) = 17.9 mg/kg body weight
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for possible carcinogenicity and short duration of the study).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The cancer health risk limit for bromoform is 0.04 mg/L.

The WHO guideline value only considers exposure to THMs in water via the oral route. However, WHO accepts THMs are volatile chemicals, and therefore, exposure via the inhalation and dermal routes may be significant sources of exposure, particularly during bathing and showering, as increasing water temperature will increase the rate of volatilisation, and ventilation may be poor. WHO suggested that in colder countries with low rates of ventilation in houses or where the incidences of showering and bathing are high, this guideline value may be lowered (WHO 2005, in DWI 2010).

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# 2-bromo-4-hydroxyacetophenone

CAS No. 2491-38-5. Also called 1-(4-hydroxyphenyl)-2-bromoethanone, 2-bromo-1-(4-hydroxyphenyl)ethan-1-one and BHAP. Products such as Busan contain 2-bromo-4-hydroxyacetophenone.

### Maximum Acceptable Value

There is no MAV for 2-bromo-4-hydroxyacetophenone in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

2-Bromo-4-hydroxyacetophenone is used as microbiocide/microbiostat for control of slime forming bacteria and fungi in water cooling systems, oil recovery drilling muds and secondary injection water, pulp and paper water systems, adhesives, coatings, latex paints, and wet-end additives, and industrial processing chemicals. It is used at up to 10 to 20 ppm active ingredient in cooling waters; higher dose rates (up to 80 ppm) are required for algal and fungal slimes.

### Form and fate in the environment

Based on submitted supplemental data, BHAP appears to be non-persistent, and the data indicate that photolysis plays a major role in the degradation pathway. BHAP photodegrades in water with a half-life of less than two days. Also, BHAP’s half-life in aerobic aquatic environments is 2.5 days. Its hydrolytic half-life is 272 hours, 250 hours, and 173 hours at pH 5, 7, and 9, respectively. BHAP appears to be immobile to moderately mobile in silty clay loam, sandy loam, and silt loam soils, respectively. However, when applied to sandy soils BHAP appears to be very mobile (USEPA 1995).

The dissociation constant (pKa) = 7.6 ± 0,3 at 24°C. Water solubility is about 2500 mg/L.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

BHAP generally is of moderate acute toxicity. BHAP is moderately toxic by the oral and inhalation routes (Toxicity Category II). The use of bromohydroxyacetophenone will not likely result in human exposure over a significant portion of human life span. Therefore, the chronic toxicity were not required (USEPA 1995).

### Derivation of Maximum Acceptable Value

No MAV.

### References

USEPA. 1995. *Re-registration Eligibility Decision (RED) for Bromohydroxyacetophenone (BHAP)*. EPA 738-R-95-010. 184 pp. <http://www.epa.gov/pesticides/reregistration/REDs/3032red.pdf> or http://www.epa.gov/pesticides/reregistration/status.htm.

# Bromomethane

CAS No. 74-83-9. Also called methyl bromide and monobromomethane.

### Maximum Acceptable Value

There is no MAV for bromomethane in the DWSNZ, and bromomethane is not mentioned in the WHO Guidelines.

The USEPA concluded on 22 September 2009 that bromomethane is known or anticipated to occur in public water supplies and may require regulation. Therefore they added bromomethane (listed as methyl bromide) to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a). Methyl bromide is on the USEPA List of Priority Pollutants.

The USEPA (2006/2009) established a lifetime health advisory of 0.01 mg/L, where the lifetime health advisory is the concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on human health concerns, methyl bromide in drinking water should not exceed 0.001 mg/L. Excursions above this level would need to occur over a reasonably significant period to be a health concern, because the health-based guideline is based on medium-term effects.

Methyl bromide is a Class I ozone depleting substance and many of its uses are required to be phased out under the Montreal Protocol.

### Sources to drinking-water

#### 1. To source waters

Some bromomethane is formed in the ocean (about 1 to 2 ng/L), probably by algae or kelp. However, most is made by humans (overseas) to kill various pests (rats, insects, fungus, etc) that might be present in homes, foods, or soil. Some bromomethane is also used to make other chemicals.

Bromomethane appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). Some commercial products also contain from 2 to 70 percent chloropicrin; ERMA (2010) no longer allows these products to be used in New Zealand.

This datasheet is not included in the pesticides section because being a gas, methyl bromide is not expected to reach drinking-water when used as a fumigant. Methyl bromide fumigation is required of New Zealand by importing countries on a number of products prior to their shipment, and is also used in quarantine applications on imported goods. Commercial methyl bromide may contain odorants – chloropicrin (2 percent), amyl acetate (0.3 percent). Ethylene dibromide or hydrocarbons may be present as inert diluents.

Methyl bromide is no longer used to fumigate soil in conjunction with strawberry growing in New Zealand. Potato wart is a disease that disfigures potatoes and is caused by a persistent soil-borne fungus *Synchitrium endobioticum*. Methyl bromide is regarded as an essential tool in the management and eradication of potato wart in New Zealand and that the use of methyl bromide for controlling potato wart is a legitimate quarantine use (ie, exempted from the Montreal Protocol). Methyl bromide is also used in a limited way for some horticulture products where there is no alternative available such as fumigation to protect honeycomb from wax moth.

The application of methyl bromide for large-scale fumigation of export logs under tarpaulins has attracted considerable public interest. ERMA (2010) has reassessed its use. The decision was to approve the continued use of methyl bromide but impose a new overall management regime which includes strengthening the tolerable exposure limits, requiring air quality monitoring and reporting, and imposing minimum buffer zones. ERMA is also requiring all methyl bromide fumigations to be subject to recapture within a 10-year period. In addition, ERMA recommends more research into alternatives to methyl bromide and recapture technology.

#### 2. From treatment processes

Bromomethane may be generated in drinking-water as the result of chlorination, but this has not yet been quantified (ATSDR 1992). Theoretically, bromine can be reduced by chlorine to form hypobromous acid, and bromomethane can be a breakdown product.

#### 3. From the distribution system

Bromomethane is heavier than air and has the ability to penetrate many substances, including usually impermeable materials, such as concrete, leather, and rubber. Methyl bromide is able to diffuse through certain plastics; permeation through low density polyethylene (LDPE) is eight times higher than through high density polyethylene (HDPE). However, methyl bromide does not permeate through PVC. Metal piping is also impervious.

### Form and fate in the environment

Methyl bromide quickly evaporates at temperatures ordinarily encountered in fumigating, but some may be entrapped in soil micropores following application where it is moderately persistent, with a field half-life of between 30 and 60 days. Any bromomethane that does not evaporate when used to fumigate soil may leach to groundwater where it may persist for several months. In water, a half-life of three hours was calculated for a model river, this half-life relates to loss due to evaporation. Methyl bromide hydrolyses slowly to form methanol and hydrobromic acid in aqueous solution.

The USEPA (2008) has concerns regarding methyl bromide’s potential to leach into groundwater and surface water. While methyl bromide has certain properties and characteristics in common with chemicals that have been detected in groundwater (methyl bromide is highly soluble in water and has low adsorption to soil), volatilisation is this chemical’s most important route of dissipation.

NSW Government (2013) reports that methyl bromide hydrolyses very slowly in water and more rapidly in alkaline media.

Water solubility is about 15,000 mg/L (1.5 percent).

NPIC (1994) quotes for methyl bromide a soil half-life of 55 days, water solubility of 1.34 percent and a sorption coefficient (soil Koc) of 22. This resulted in a pesticide movement to groundwater rating of very high.

### Typical concentrations in drinking-water

Tests reported by 25,443 US public water suppliers in 38 states shows that between 1998 and 2003, people in 113 communities drank water containing bromomethane. The highest concentration was 0.02 mg/L, which is above the 0.01 mg/L level, the concentration in drinking water that the USEPA does not expected to cause any adverse, non-carcinogenic health effects for a lifetime of exposure. The lifetime health-based limit (or Health Advisory) is based on exposure for a 70‑kg adult consuming two litres of water per day (EWG 2008). 141 water utilities in the US reported detecting bromomethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.014 mg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,848 drinking water samples for bromomethane between 2013 and 2015, and found 115 samples exceeded the minimum reporting level (MRL) of 0.2 µg/L.

### Removal methods

Due to its volatility, bromomethane is expected to be removed from water by aeration processes.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Because bromomethane evaporates very quickly, it is usually not found in food, surface water, or soil, thus the main intake route for humans is by respiration. The air in some cities may contain up to about 1 to 2 ppb due to releases from chemical factories and automobile exhausts.

Bromomethane is a powerful fumigant gas which is one of the most toxic of the common organic halides. In massive dosage, it is narcotic like a halogenated hydrocarbon solvent. It has a characteristic delayed neurotoxic action.

Metabolism studies show that methyl bromide is rapidly metabolised and excreted in the body. The primary route of excretion is exhalation as CO2. Studies in animals suggest that bromomethane does not cause birth defects and does not interfere with normal reproduction except at high exposure levels. Animals that breathed bromomethane for two years did not develop cancer. Animals that swallowed bromomethane for 25 weeks had changes in their stomachs that could have been an early sign of cancer, but we do not know if swallowing bromomethane for a longer time would cause cancer. Both the International Agency for Research on Cancer (IARC) and the USEPA have determined that bromomethane is not classifiable as to its carcinogenicity in humans. Methyl bromide has been shown to be genotoxic in a variety of assays but has not been shown to be carcinogenic in animals or human epidemiology studies (OECD 2003).

The reference dose or RfD (USEPA 2006/2009/2011) is 0.001 mg/kg/d based on epithelial hyperplasia of the forestomach in rats. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.05 mg/L. As at May 2014, the USEPA Human Health Benchmarks for Pesticides (<http://water.epa.gov/drink/standards/hascience.cfm>) quotes a cRfD of 0.02 mg/kg/d. The USEPA acute one day HHBP (Human Health Benchmark for Pesticides) in drinking water for methyl bromide is 4.62 mg/L.

The Acceptable Daily Intake (ADI) adopted in Australia for methyl bromide is 0.0004 mg/kg body weight with a NOEL of 0.4 mg/kg bw, from a short-term (90-day) dietary rat study. The NOEL is based on injury to the forestomach. The ADI incorporates a safety factor of 1,000.

EXTOXNET (1996) quotes an ADI of 1.0 mg/kg/d, and a RfD of 0.0014 mg/kg/d. IPCS (1996) also reports an ADI of 1 mg/kg bw, based on the toxicology of the bromide ion.

As at July 2013 – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html> – ATSDR quotes a minimal risk level (MRL) of: 0.003 mg/kg/d for intermediate oral exposure  
(15–364 days).

EFSA (2013) states: The toxicological profile of methyl bromide was evaluated in the framework of Directive 91/414/EEC, which resulted in an ADI and an ARfD being established at 0.001 mg/kg bw per d and 0.003 mg/kg bw, respectively. The toxicological profile of bromide ion, the main metabolite of methyl bromide, was evaluated by JMPR, but EFSA considers that the proposed ADI of 1 mg/kg bw per d is not sufficiently supported by data and that the necessity of an ARfD for bromide ion should be reassessed. Pending a revision of the toxicological profile of bromide ion by JMPR, the values set by JMPR are considered on a tentative basis.

### Derivation of Maximum Acceptable Value

No MAV.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for bromomethane is 0.01 mg/L.

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# 3-bromopropan-1,2-diol

CAS No. 4704-77-2. Also known as 3-bromo-1,2-propanediol, α-bromohydrin, monobromoglycerol and alpha-glycerol bromohydrin. It can appear as the (R)‑enantiomer and (R,S)-enantiomers.

### Maximum Acceptable Value

3-Bromopropan-1,2-diol does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

3-Bromopropan-1,2-diol belongs to the large, rather loosely defined group of chemicals called halohydrins. Chlorohydrins occur as well, see datasheet for 1,3‑dichloropropan-2-ol. Other halohydrins are listed in the halohydrin datasheet.

Like some of the other halohydrins, 3-bromopropan-1,2-diol can appear in processed edible oils.

#### 2. From treatment processes

3-Bromopropan-1,2-diol has been reported as a disinfection by-product when using ozone.

#### 3. From the distribution system

No known sources.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See Matthew and Anastasio (2000).

### Health considerations

(R,S)- and (R)-3-bromopropan-1,2.diol (α-bromohydrin) produced diuresis and glucosuria when administered to male rats but had no effect on the metabolic activity of rat kidney tubules *in vitro*.

α-Bromohydrin may be formed following oral administration of 2,3-dibromopropan-1-ol (qv) to rats (IARC 2000).

### Derivation of Maximum Acceptable Value

No MAV.

### References

IARC. 2000. *3-Bromopropan-1,2-diol*. 15 pp. See: <http://monographs.iarc.fr/ENG/Monographs/vol77/mono77-17.pdf>.

Matthew BM, Anastasio C. 2000. Determination of halogenated mono-alcohols and diols in water by gas chromatography with electron-capture detection. *J Chromatography A* 886(1): 65–77.

# 1-bromopropane

CAS No. 106-94-5. Also known as 1-propyl bromide and n-propyl bromide. Sometimes 1BP or nPB.

### Maximum Acceptable Value

1-Bromopropane does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

In 2001, the United Nations Environment Programme (UNEP) projected that manufacture and use of 1-bromopropane would expand because it was being marketed to replace ozone-depleting solvents (such as trichloroethylene, tetrachloroethylene (perchloroethylene) and methylene chloride) with high production volume for a range of applications.

1-Bromopropane is a solvent for fats, waxes and resins and is primarily used as a chemical intermediate in the production of pesticides, quaternary ammonium compounds, flavours and fragrances, and pharmaceuticals in closed processes. It is used as a cleaning solvent for metals, plastics, optical and electronic components, and for dry cleaning fabrics.

1-Bromopropane has been detected qualitatively in six species of marine algae that produce it naturally.

#### 2. From the distribution system

No known sources.

### Forms and fate in the environment

Octanol/water partition coefficient: log Kow 2.10. Organic carbon:water partition coefficient (log Koc) 1.6. Henry’s law constant 7.3 x 10-3 atm-m3. Water solubility 2.45 mg/L at 20°C. Hydrolysis half life 26 days.

1-Bromopropane is mobile in soils, and volatile. Adsorption to soils is not expected; therefore, 1-BP can migrate through soil to groundwater.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Exposure to 1-bromopropane in humans is most likely to occur by inhalation or dermal contact.

In the 13th Report on Carcinogens, the United States National Toxicology Program (NTP) classified 1-bromopropane as “reasonably anticipated to be a human carcinogen” The state of California has also listed 1-bromopropane as a developmental hazard under proposition 65 of the California Clean Water Act.

In November 2012, in accordance with Article 57 and 59 of the European Committee regulation 1907/2006, the European Chemical Agency identified 1-bromopropane as a substance of very high concern due to the risk of reproductive toxicity.

IARC (2018) concluded that 1-bromopropane is possibly carcinogenic to humans (Group 2B).

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <https://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>. As at August 2017 the MRL for 1-bromopropane are:

minimal risk level

0.2 mg/kg/day for acute-duration oral exposure (1–14 days)

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Bronopol

CAS No. 52-51-7. IUPAC name is 2-bromo-2-nitropropane-1,3-diol. Also called 2‑bromo-2-nitro-1,3-propanediol, BNPD and various trade names.

### Maximum Acceptable Value

Bronopol does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

EPA established an environmental exposure limit of 0.0007 mg/L (0.70 µg/L) for bronopol in fresh water (<http://www.epa.govt.nz/search-databases/Pages/substance-exposure-limit-register.aspx>).

### Sources to drinking-water

#### 1. To source waters

Bronopol is a highly active general purpose [antimicrobial](http://en.wikipedia.org/wiki/Antimicrobial) chemical compound. Its first applications were as a [preservative](http://en.wikipedia.org/wiki/Preservative) for [pharmaceuticals](http://en.wikipedia.org/wiki/Pharmaceutical). Bronopol’s low [mammalian](http://en.wikipedia.org/wiki/Mammal) [toxicity](http://en.wikipedia.org/wiki/Toxicity) (at in-use levels) and exceptional activity against [bacteria](http://en.wikipedia.org/wiki/Bacteria) (especially the troublesome [Gram-negative](http://en.wikipedia.org/wiki/Gram-negative) species) ensured that it became popular as a preservative in many consumer products such as [shampoos](http://en.wikipedia.org/wiki/Shampoo), wet-wipes and [cosmetics](http://en.wikipedia.org/wiki/Cosmetics). The use of bronopol in [personal care products](http://en.wikipedia.org/wiki/Personal_care_products) ([cosmetics](http://en.wikipedia.org/wiki/Cosmetics), [toiletries](http://en.wikipedia.org/wiki/Toiletries)) has declined since the late 1980s due to the recognised potential for [nitrosamine](http://en.wikipedia.org/wiki/Nitrosamine) formation.

Bronopol has been taken up as an effective antimicrobial in many industrial environments (aerobic and anaerobic) such as [paper mills](http://en.wikipedia.org/wiki/Paper_mill), [oil exploration](http://en.wikipedia.org/wiki/Oil_exploration) and production facilities, as well as cooling water disinfection plants, often at as low as 25 mg/L, particularly at pH below 8. It has also been use as an algicide. It is mixed with some pesticides, eg, ametryn (qv).

Bronopol appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2012 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). It is registered as a skin/coat conditioner.

Bronopol with Contact has been trialled for control of Psa on kiwifruit (Plant and Food 2011).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Bronopol has low volatility, so evaporation from products containing it will be minimal. The substance is very soluble in water, and when introduced, will have a tendency to remain in water. It has minimal tendency to bind to soil or sediment. Bronopol is unlikely to persist in the environment. The substance is susceptible to both biodegradation and hydrolysis which suggests the chemical will be removed from water and soil environments, including biological wastewater treatment plants.

The USEPA (1995) does not anticipate groundwater contamination from the uses of bronopol. Although bronopol has high water solubility, high solubility in polar solvents, low solubility in nonpolar solvents, and favourable partitioning into water, the Agency feels that bronopol’s shortlived environmental persistence reduces the potential for groundwater contamination. Bronopol is stable to hydrolysis under normal conditions. However, at warmer temperatures and/or higher pHs, rapid hydrolysis may occur. Under these conditions, hydrolysis products include formaldehyde and lesser amounts of other degradates. Judging from its low octanol/water ratio and high solubility in water, bronopol is not expected to bioaccumulate. In tested mammalian species metabolism is reported to be rapid and complete, and accumulation does not occur.

When mixed with water the half-life of bronopol decomposition to formaldehyde is 18 years at pH 4; 1.5 years at pH 6; and two months at pH 8 at 20°C. Decomposition products detected include bromide ion, nitrite ion, bromonitroethanol, formaldehyde and 2-hydroxymethyl-2-nitropropane-1,3-diol.

Water solubility: about 28 percent.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

No dietary exposure is expected from the pesticide uses of bronopol since no food or feed uses are registered. However, an RfD was established recently at 0.1 mg/kg/day because the data base is available and because of possible long-term exposure to bronopol-containing products (USEPA 1995). Based on chronic oral studies, the systemic NOEL and LOEL for both sexes are 10 mg/kg/day and 40 mg/kg/day, respectively.

The USEPA (1995) classified bronopol as a Group E chemical (one for which there is evidence of non-carcinogenicity for humans), based on a lack of evidence of cancer effects in acceptable studies with two animal species, the rat and mouse.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# 1,3-butadiene

CAS No. 106-99-0. Also known as butadiene, buta-1,3-diene, trans-butadiene, biethylene, diethylene, pyrrolylene, bivinyl, divinyl, vinylethylene, erythrene or pyrrolylene.

### Maximum Acceptable Value

1,3-Butadiene does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

The USEPA concluded on 22 September 2009 that 1,3-butadiene is known or anticipated to occur in PWSs and may require regulation. Therefore they have added [it](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane) to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

At least 60 percent of 1,3-butadiene is used to make man-made rubber, including styrene–butadiene rubber (SBR), polybutadiene rubber, styrene–butadiene latex, chloroprene rubber (eg, neoprene) and nitrile rubbers (NBR), which is then used mostly for car and truck tyres. 1,3-Butadiene is also used to make certain types of plastics such as acrylics (polymers) and thermoplastic resins such as acrylonitrile-butadiene-styrene (ABS). Smaller amounts are constantly released into the air from evaporating motor vehicle fuel and in vehicle exhaust. Total dienes (mainly 1,3-butadiene) are regulated in LPG to <0.5 percent. ATSDR (2012) states that large amounts of 1,3-butadiene are released into the air by industrial sources; releases to water and soil are relatively low.

1,3-Butadiene can be used to manufacture adiponitrile which is subsequently hydrogenated to hexamethylenediamine, an intermediate in the manufacture of nylon 6,6.

Other sources of 1,3-butadiene include cigarette smoke and the smoke of wood fires. Forest fires (usually due to incomplete combustion) are considered to be a natural source of 1,3-butadiene in the air – WHO estimates 770,000 tonnes pa worldwide. Some commercial liquefied petroleum gases (LPG) may contain up to 8 percent 1,3‑butadiene by volume (EU 2002).

1,3-Butadiene was found in 1 of 204 water samples taken in 1975–1976 from surface waters near known industrialised areas across the United States. The single positive sample contained an approximate concentration of 0.002 mg/L. The maximum concentration found in sewage is 0.13 mg/L.

The commercial product contains 0.01–0.02 percent w/w 4-tert-butylpyrocatechol (CAS No. 98-29-3) as an inhibitor, preventing peroxide formation and spontaneous exothermic self-polymerisation.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

1,3-Butadiene does not occur naturally but has been detected in air after fires. 1,3‑Butadiene that is spilled on to water or soil is expected to evaporate quickly into the air based on its physical and chemical properties. Based on estimated soil adsorption coefficient values, 1,3-butadiene is not expected to adsorb significantly to soil or sediment, nor is it expected to bioconcentrate in fish or aquatic organisms based on estimated bioconcentration and bioaccumulation factors.

1,3-Butadiene is quite soluble in water: about 735 mg/L. The Log KOW 1.99; KOC is 288 or log Koc is 1.86 to 2.36; Henry’s law constant is 0.0736 atm.m3/mole at 25°C (DWI 2014). EU (2002) also quotes the vapour pressure = 240 kPa at 20°C.

EU (2002) states that 1,3-butadiene is not expected to hydrolyse appreciably in the environment; direct photolysis is assumed to be an insignificant process; 1,3-butadiene may biodegrade but it is considered that volatilisation and subsequent photodegradation in the atmosphere is likely to be the most important removal mechanism for 1,3-butadiene from soil and surface water.

### Typical concentrations in drinking-water

DWI (2014) reports:

No data were located on the removal of 1,3-butadiene during drinking water treatment, however, some predictions on the fate of this chemical in drinking water treatment can be made based on its physico-chemical properties.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,848 drinking water samples for 1,3-butadiene between 2013 and 2015, and found two samples exceeded the minimum reporting level (MRL) of 0.1 µg/L.

### Removal methods

A log Koc of 1.86–2.36 has been reported for 1,3-butadiene, which would suggest that it has high mobility in the water column and therefore is unlikely to be amenable to removal by GAC.

1,3-Butadiene is highly volatile; a vapour pressure of 2110 mm Hg and a Henry’s Law constant of 0.0736 atm.m³/mole have been reported at 25°C. This may indicate that 1,3-butadiene will be amendable to removal from water by air stripping.

Oxidation technologies such as UV/ozone may also be effective.

WRF (2014) reports that 1,3-butadiene is characterised with high Henry’s law constant (2.23 dimensionless air/water at 20°C) which is the highest among the 13 VOCs trialled. Low profile air stripping is very effective for 1,3-butadiene removal even at low temperatures and low air to water ratios (below 100). 1,3-Butadiene was completely removed in most of the tested scenarios, and showed a very high removal efficiency (99.8 percent) at the lowest temperature (4°C) and lowest air to water ratio (53) which is the worst case examined.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Exposure to 1,3-butadiene through ingestion of food and drinking-water is expected to be very low compared with exposure through breathing contaminated air. Certain cooking oils release butadiene to the air on heating (USEPA 2003). This probably occurs primarily by volatilisation, but could also involve formation due to low levels of combustion. Tobacco smoke may contribute significant amounts of 1,3-butadiene.

Reproductive and developmental effects are the most sensitive non-cancer effects observed in rodents. In the short term, 1,3-butadiene exposure in humans may cause nausea, dry mouth and nose, headache, and decreased blood pressure and pulse rate.

Studies of workers exposed to 1,3-butadiene suggest that workers may have an increased risk for cancers of the stomach, blood, and lymphatic system. Laboratory animals have developed cancer in multiple body tissues after exposure to 1,3‑butadiene for 13 weeks or longer. Animals appear to be most sensitive to blood and lymphatic system cancers. The International Agency for Research on Cancer (IARC – Group 1), National Toxicology Program (NTP), and USEPA all classify 1,3-butadiene as a human carcinogen, and may also be genotoxic in humans. IARC (2012) states that there is strong evidence that the carcinogenicity of 1,3-butadiene in humans operates by a genotoxic mechanism that involves formation of reactive epoxides, interaction of these direct acting mutagenic epoxides with DNA, and resultant mutagenicity.

Positive genotoxicity results have been reported with the three major metabolites of 1,3-butadiene (epoxybutene, diepoxybutane and butanediol) in a variety of *in vitro* test systems, where diepoxybutane is reported to be the most mutagenic (DWI 2014).

The USEPA has not set levels in drinking water for 1,3-butadiene. An oral RfD is not calculated because 1,3-butadiene is a gas and causes hazard by inhalation only. Nearly all health testing is related to inhalation. DWI (2014) states that an oral Tolerable Daily Intake (TDI) can not be derived from oral studies but can be derived by extrapolation from inhalation studies – an oral Tolerable Daily Intake (TDI) of 0.005 mg/kg bw/day (5 μg/kg bw/day; rounded) can be derived. They added: However, 1,3-butadiene is classified as a Group 1 carcinogen by the IARC, therefore its concentration in water should be as low as reasonably practicable.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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WRF. 2014. *Removal of Volatile Organic Contaminants via Low Profile Aeration Technology*. 58 pp. <http://www.waterrf.org/PublicReportLibrary/4439.pdf>.

# Butanol

Butanol (butyl alcohol) includes four possible isomers; normal (n); iso, secondary (sec or s) and tertiary (tert or t).

CAS No. 71-36-3: n-butanol. Also called 1-butanol, butan-1-ol, n-butyl alcohol, butyric alcohol or propyl carbinol.

CAS No. 78-83-1: isobutanol. Also called 2-methylpropan-1-ol, isobutyl alcohol, isopropylcarbinol, 1-hydroxymethylpropane or IBOH.

CAS No. 78-92-2: sec-butanol. Also called butan-2-ol, 2-butanol, sec-butyl alcohol, butylene hydrate, 2-hydroxybutane, methyl ethyl carbinol.

CAS No. 75-65-0: tert-butanol. Also called tert-butyl alcohol, t-butanol, 2-methyl-2-propanol, 1,1-dimethylethanol, trimethylcarbinol or TBA.

### Maximum Acceptable Value

Butanol does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

The USEPA concluded on 22 September 2009 that 1-butanol is known or anticipated to occur in PWSs and may require regulation. Therefore they have added [it](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane) to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

Butanol is being studied for use as a biofuel, one such process using anaerobic conversion of carbohydrates by strains of *Clostridium* into acetone, butanol and ethanol. Most of the simpler processes produce n-butanol, but branched chain butanols have a higher octane rating, so research is being directed towards production of isobutyl alcohol.

n-Butanol is a volatile organic compound (VOC) which is emitted by natural sources such as plants (beans, split peas, lentils, rape, rye and grass), trees (beech, birch and hornbeam), carbohydrates, animal waste, microbes and insects. 1-Butanol is used primarily as a chemical intermediate in the production of butyl acrylate and methacrylate. It is also used in the production of glycol ethers and butyl acetate. n-Butanol is used to make other chemicals, or used as a solvent or an ingredient in formulated products such as cosmetics, and its use as a flavouring agent.

Isobutanol (isobutyl alcohol) is useful in organic synthesis, as a chemical intermediate, as a lube oil additive, and as a solvent in coating applications. Isobutanol is similar in property to n-butyl alcohol and may be used as a supplement or replacement for n-butyl alcohol in many applications. As a relatively slow evaporating latent solvent in lacquers and ambient-cured enamels, isobutanol is effective in reducing the viscosities of many formulations while simultaneously promoting flow and retarding blush. It is also used as a flavouring agent in butter, cola, fruit, liquor, rum, and whisky. Almost all human beings are exposed daily to low concentrations of isobutanol from natural sources, such as in foods, animal wastes and from fermentation of carbohydrates.

sec-Butanol is used to make methyl ethyl ketone (MEK or butanone); small amounts are used as perfumes or in artificial flavours. 2-Butanol occurs naturally as a product of fermentation of carbohydrates. It is used for the extraction of fish meal to produce fish protein concentrate. It is also used for the production of fruit essences, as a flavouring in food, and as a solvent.

tert-Butanol is used as a solvent, as a [denaturant](http://en.wikipedia.org/wiki/Denatured_alcohol) for ethanol, as an ingredient in [paint removers](http://en.wikipedia.org/wiki/Paint_remover), as an [octane](http://en.wikipedia.org/wiki/Octane_rating) booster for [gasoline](http://en.wikipedia.org/wiki/Gasoline), as an [oxygenate](http://en.wikipedia.org/wiki/Oxygenate) [gasoline additive](http://en.wikipedia.org/wiki/Gasoline_additive), and as an intermediate in the synthesis of other chemical commodities such as [MTBE](http://en.wikipedia.org/wiki/MTBE), [ETBE](http://en.wikipedia.org/wiki/ETBE), [TBHP](http://en.wikipedia.org/wiki/TBHP), other flavours and perfumes. tert-Butyl alcohol is a metabolite of MTBE (qv). It has been found to leach from some plastics.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

If released to the soil, the butanols may be expected to leach to groundwater.

In a natural river die-away test, using river water as the only inoculum, about 56 percent of 3 mg/L of 1-butanol degraded after four days. Under anaerobic conditions, 1-butanol (initial concentration of 500 mg/L) was degraded 100 percent after a four-day lag period (USEPA 1994). OECD (2005) quoted a half life for n-butyl alcohol in river water of about 4 hours and in lakes about 126 days.

Isobutanol is readily biodegradable under aerobic conditions. Isobutanol volatilises moderately from moving rivers, but less so from quiescent lakes and other surface water bodies (calculated volatilisation half-lifes of 43 hours from a river and 23 days from a lake). Isobutanol is not persistent in the environment and is not likely to bioaccumulate in food webs (OECD 2005).

If released to soil, t-butyl alcohol is expected to have very high mobility based upon a reported Koc of 37. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 9.05 x 10-6 atm‑cu m/mole. t-Butyl alcohol may volatilise from dry soil surfaces based upon its vapour pressure. The half-life of t-butyl alcohol under anoxic conditions in a non-amended soil was about 200 days, but the half-lifes in the same soil amended with nitrate and sulfate nutrients were 100 and 50 days, respectively. Biodegradation of t‑butyl alcohol in unamended soils collected at different depths had rates of <0.01 to 0.15 mg/L/day/gram dry soil. If released into water, t-butyl alcohol is not expected to adsorb to suspended solids and sediment based upon the Koc. The biodegradation half-life of t-butyl alcohol was reported to range from about 28 to 180 days in aerobic water and 100 to 500 days in anaerobic water. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lives for a model river and model lake are 3.6 and 29 days, respectively. A reported BCF of <5 in carp suggests bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

Water solubility:

n-butanol: about 7 percent

iso-butanol: about 8 percent

sec-butanol: about 12.5 percent

tert-butanol: fully miscible

### Typical concentrations in drinking-water

Thirty-two water utilities in the US reported detecting tert-butyl alcohol (a degradation product of MTBE) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.088 mg/L.

### Removal methods

Aeration possibly offers the best option.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

In man, the most likely acute effect of 2-butanol is alcoholic intoxication. No published data are available concerning other effects on man (IPCS 1987a).

In man, tert-butanol is mildly irritating to the skin. No other effects in man have been reported, and there have been no reports of poisoning (IPCS 1987b).

Isobutanol is rapidly absorbed following inhalation and oral exposures. Isobutanol is rapidly metabolised to isobutyraldehyde and isobutyric acid in rodents and humans (OECD 2005). An oral gavage subchronic study has also been reported for isobutanol. Clinical signs related to treatment with 1,000 mg/kg dose level included hypoactivity, ataxia, and salivation. Clinical signs of hypoactivity and ataxia were resolved by the fourth week of the study. Slight decreases in feed consumption and body weight gains were noted in the first two weeks and were restricted to the 1000 mg/kg/day group. There were no changes in organ weights or gross or histopathology at any exposure level. The NOAEL was 316 mg/kg bw/day (INCHEM 2004; OECD 2005).

1-Butanol causes adverse central nervous system effects in animals by both the oral and inhalation routes of exposure. The USEPA (1991/1994) derived a chronic oral reference dose (RfD) of 0.1 mg/kg/day for 1-butanol, based on the absence of ataxia and hypertension observed in animals exposed by mouth at a 125 mg/kg/day level. IPSC (1998) stated no adverse effects were observed when 6.9 percent butyl alcohol and 25 percent sucrose (about 5.6 mg/kg bw per day butyl alcohol) were added to the drinking-water of male rats for 13 weeks.

Based on no human and no animal cancer data, the USEPA (1994) classified 1-butanol as class D, not classifiable as to human carcinogenicity.

The oral RfD for isobutyl alcohol was calculated at 0.3 mg/kg/d (USEPA (1991), based on hypoactivity and ataxia in an oral subchronic rat study.

### Derivation of Maximum Acceptable Value

No MAV.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for n-butanol is 0.7 mg/L.

And the subchronic limit is 0.7 mg/L; the chronic health risk limit (exposure greater than 10 percent of a lifetime) for isobutanol is 0.3 mg/L.

In 1983 the maximum allowable concentration (MAC) in the USSR for 2-butanol was 0.2 mg/L (IPCS 1987a). In 1983 the maximum allowable concentration (MAC) in the USSR for tert-butanol was 1.0 mg/L (IPCS 1987b).

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# 2-butanone

CAS No. 78-93-3. Also known as butan-2-one, methyl ethyl ketone, ethyl methyl ketone, MEK, and occasionally butanone or methyl acetone.

### Maximum Acceptable Value

2-Butanone does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

The USEPA (2006/2011) established a lifetime health advisory of 4 mg/L for methyl ethyl ketone, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

### Sources to drinking-water

#### 1. To source waters

2-Butanone is manufactured in large amounts for use in paints and other finishes, adhesives and in food processing. 2-Butanone is often found dissolved in water. 2‑Butanone is also a natural product produced by some trees and is found in some fruits and vegetables. It has also been found to be produced by some algae. The exhausts of cars and trucks release 2-butanone into the air. As at 1987, US uses of 2‑butanone were broken down into the following categories: coatings/solvents, 50 percent; adhesives, 13 percent; magnetic tapes, 8 percent; lube oil dewaxing, 4 percent; printing inks, 3 percent; exports, 16 percent; and miscellaneous, 6 percent.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Its use as a component in adhesives used to join PVC pipes is a potential route for entry of 2-butanone into potable water (ATSDR 1992).

### Forms and fate in the environment

In water, 2-butanone is expected to undergo microbial degradation under both aerobic and anaerobic conditions. It will not be deposited in the sediment of rivers or lakes, and it is not expected to concentrate in fish. 2-Butanone will not adhere to soil, and if it is spilled on to soil, it will travel through the soil into underground waters. Chemical oxidation, direct photolysis, and hydrolysis of 2-butanone under environmental conditions are not expected to occur to any significant extent. Some of the 2-butanone found in soil or water will evaporate to the air.

The most important fate process for methyl ethyl ketone in water is volatilisation (estimated half-life of 3 and 12 days, for rivers and lakes, respectively). Complete aerobic biodegradation of methyl ethyl ketone has been reported in about 5 to 10 days following inoculation with sewage or polluted surface water. Anaerobic degradation occurred after an acclimation period of about one week (USEPA 1994).

If released to soil, methyl ethyl ketone is expected to have very high mobility based upon Koc values of 29 and 34 obtained in silt loams. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 5.69 x 10-5 atm‑cu m/mole. Methyl ethyl ketone may volatilise from dry soil surfaces based upon its vapour pressure. The volatilisation half-life of methyl ethyl ketone from silt and sandy loams was measured as 4.9 days. This compound is expected to biodegrade under aerobic and anaerobic conditions in soil. If released into water, methyl ethyl ketone is not expected to adsorb to suspended solids and sediment based upon the Koc values. Methyl ethyl ketone was shown to biodegrade 89 percent in 20 days in fresh water and 69 percent in 20 days in salt water. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 16 hours and 7.3 days, respectively. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

2-Butanone is very soluble in water: 13–14 percent; note that USEPA (1994) states 35.3 percent at 10°C, and USEPA (2003) states 27.5 percent.

### Typical concentrations in drinking-water

Numerous studies have qualitatively detected 2-butanone in drinking-water supplies. 2-Butanone was detected in tap water eight months after the installation of new PVC pipes at a concentration ranging from 0.4 to 4.5 mg/L. It resulted from the glue used to cement the water pipes together. The concentration of 2-butanone in the water increased with the amount of time the water sat in the pipes.

196 water utilities in the US reported detecting methyl ethyl ketone in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.34 mg/L.

### Removal methods

Aeration possibly offers the best option.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

MEK has been detected as a natural component of numerous foods, including raw chicken breast, milk, nuts (roasted filberts), cheese (Beaufort, Gruyere, and cheddar), bread dough and nectarines at concentrations ranging from 0.3 to 19 ppm. MEK is also found in tobacco smoke and volatile releases from building materials and consumer products (ATSDR 1992). WHO (1992) estimated levels of daily MEK intake from different sources as follows: foodstuffs – 1,590 μg/day; drinking water (two litres) – 3.2 μg/day; rural outdoor air – 36 μg/day; urban outdoor air up to 760 μg/day; and tobacco smoke up to 1,620 μg/day.

2-Butanone alone is a relatively safe chemical, widely used as a solvent. For some uses, 2-butanone is combined with other chemicals that have serious neurotoxic and hepatotoxic effects. Clinical reports and animal studies have clearly shown that exposure to 2-butanone alone causes minimal chronic neurological or hepatic deficits, if any. It does potentiate both the neurotoxicity of n-hexane (qv) and methyl-n-butyl ketone (MBK or 2-hexanone, qv) and the hepatotoxicity of carbon tetrachloride and chloroform.

The International Agency for Research on Cancer and the Environmental Protection Agency (USEPA) have not classified 2-butanone as to its human carcinogenicity. USEPA (2003) states that “*data are inadequate for an assessment of human carcinogenic potential*” for MEK, because studies of humans chronically-exposed to MEK are inconclusive, and MEK has not been tested for carcinogenicity in animals by the oral or inhalation routes. The majority of short-term genotoxicity testing of MEK has demonstrated no activity; MEK is unlikely to be carcinogenic.

The reference dose or RfD (USEPA 2003 and 2006/2009/2011) for MEK is 0.6 mg/kg/d, based on a LED05 of 639 mg/kg-day and an uncertainty factor of 1000. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 20 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA Reference Dose ([RfD](http://www.epa.gov/ttn/atw/hlthef/hapglossaryrev.html#rfd)) for methyl ethyl ketone is 0.6 mg/kg/d based on decreased fetal birth weight in rats. Various States in the US have adopted drinking-water quality guideline values for MEK, the lowest being 0.017 mg/L.

The odour threshold in water is about 8 mg/L.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for methyl ethyl ketone is 4 mg/L.

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# 2-butoxyethanol

CAS No. 111-76-2. Its IUPAC name is ethylene glycol butyl ether and it is also known as butyl ethoxol, n-butoxyethanol, 2-butoxyethanol, butyl glycol and ethylene glycol monobutyl ether (EGBE), 3-oxa-1-heptanol, o-butyl ethylene glycol, plus many trade names.

2-Butoxyethanol acetate (CAS No. 112-07-2) has many similar uses and properties so has been included with this datasheet. Also called butyl glycol acetate, 2-butoxyethyl acetate, butoxyethyl acetate, butyl ethoxol acetate, ethylene glycol butyl ether acetate and EGBEA.

### Maximum Acceptable Value

2-Butoxyethanol does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

In the 1970s, it was estimated that more than 740 products marketed in the US contained 2-butoxyethanol, at an average concentration of 2.8 percent; approximately half of these were products for household use (stated in ATSDR 1998). 2-Butoxyethanol is mainly used in paints and surface coatings, followed by detergents, cleaning products with over 400 of them used in Australia, and inks. Cleaning products which may contain 2-butoxyethanol include general surface cleaners, floor strippers, window cleaners, spot cleaners, rust removers and ink and resin removers.

IEH (2014) selected for consideration all those substances reported as being involved in taste and odour incidents in a developed country, excluding those for which there was no evidence of UK production or import, as well as those already regulated to a limit value either lower than or close to the reported taste and odour threshold. Other prioritised substances were then categorised according to amounts used and their reported taste and odour threshold. This process gave a list of compounds from which substances formed during water treatment were excluded leaving 18 priority compounds. This included ethylene glycol butyl ether.

The commercial product contains an additive of 0.008–0.012 percent w/w 2,6-bis(1,1-dimethylethyl)-4-methylphenol (CAS 128-37-0) to prevent the formation of peroxides.

2-Butoxyethanol acetate is used mainly in paints and coatings.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

2-Butoxyethanol is miscible in water. 2-Butoxyethanol is unlikely to hydrolyse as alcohols and ethers are generally resistant to hydrolysis. A Koc of 67 indicates that 2‑butoxyethanol will not partition into organic matter contained in sediments and suspended solids, and should be highly mobile in soil. 2-Butoxyethanol is not expected to volatilise readily from water. 2-Butoxyethanol does not absorb light of wavelength >290 nm so photolysis by absorption of sunlight is not an important process.

For 2-butoxyethanol EU (2006) quotes : vapour pressure = 1.41 hPa at 25°C; partition coefficient n-octanol/water = logPow = 0.8; Henry’s law constant = 0.08 Pa.m3/mol at 25°C. Alcohols and ethers are generally resistant to hydrolysis; according to standard tests on ready biodegradation and further experimental data which confirmed high biodegradation rates, EGBE can be regarded as readily biodegradable; the surface water half-life is 15 days, 30 days in soil and 300 days in sediments.

For 2-butoxyethanol acetate EU (2006a) quotes :water solubility = 1.6 percent; vapour pressure = 0.56 hPa at 25°C; partition coefficient n-octanol/water = logPow = 1.51; Henry’s law constant = 0.55 Pa.m3/mol at 25°C. Hydrolysis is not a major process of ultimate degradation for EGBEA with a value of more than 1,000 days for the abiotic degradation of EGBEA in water. According to standard tests, EGBEA can be regarded as readily biodegradable; the surface water half-life is 15 days, 30 days in soil and 300 days in sediments. EGBEA is not likely to adhere to particulate or organic matter.

The risk to the environment is expected to be low as 2-butoxyethanol is readily biodegradable and is of low toxicity to aquatic organisms. However, it should not be disposed of to landfill as it may leach to groundwater due to its expected high mobility in soil and low adsorption potential. Biodegradation studies indicate that 2‑butoxyethanol will be readily degraded by micro-organisms present at sewage treatment plants. Any 2-butoxyethanol that passes through sewage treatment plants and enter receiving waters is likely to remain in the water column until biodegraded by micro-organisms present in the water. 2-Butoxyethanol half-lifes in surface water range from seven days to four weeks. The half-life in anaerobic water is 4–16 weeks.

### Typical concentrations in drinking-water

IARC states that 2-butoxyethanol was listed as a contaminant in drinking-water samples analysed between September 1974 and January 1980 in a survey of cities in the USA (Lucas, 1984, cited in ATSDR, 1998). 2-Butoxyethanol was detected in 68 percent of 50 drinking-water samples (limit of detection, 0.02 μg/L) collected from Ontario, Nova Scotia and Alberta, Canada, in 1997, with a mean concentration of 0.21 μg/L. 2-Butoxyethanol was detected at a concentration of 23 µg/litre (0.023 mg/L) in one of seven groundwater samples in the US (WHO 1998; 2005).

2-Butoxyethanol and the acetate can be smelt in the air at less than 0.5 ppm. The taste and odour threshold is 0.88 mg/L; IEH (2014).

Ethylene glycol butyl ether (EGBE) was involved in a taste/odour incident in the UK in relation to the so called ‘Worcester/Wem incident’ of 11 April 1994. On this occasion several organic chemicals, including EGBE, were discharged into a sewer of a company specialising in the recovery of solvents used in the production of resins. The released effluent passed through the Wem sewage works and into two small tributaries feeding the River Severn. A few days later, consumers complained that their drinking water supply exhibited an adverse taste and odour. From IEH (2014).

### Removal methods

One of the techniques used for trapping 2-butoxyethanol from air samples is to pass it through charcoal (ATSDR 1998), so activated carbon may reduce its concentration from water. IEH (2014) notes that conventional water treatment and chlorination may remove 55 percent.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

2-Butoxyethanol is well absorbed via the inhalational, dermal and oral routes. It is widely distributed throughout the body and efficiently metabolised to 2-butoxyacetic acid, which is rapidly excreted in urine.

The critical health effect from animal studies is haemolysis of the blood cells. The severity of the effect, caused mainly by 2-butoxyacetic acid, differs markedly between species, with rats and mice the most sensitive, rabbits less sensitive, and then guinea pigs. Humans appear to be the least sensitive from the results of *in vitro* studies and *in vivo* inhalational studies.

NICNAS (1996) reported results of a 1993 National Toxicology Program (NTP) in the USA: under the conditions of the 13-week studies, the NOAEL for haematological effects in male rats was 129 mg/kg/day, but the corresponding NOAEL for females was not reached as slight anaemia was observed at the lowest dose (LOAEL 82 mg/kg/day).

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 MRLs for 2‑butoxyethanol are:

minimal risk level

0.4 mg/kg/day for acute-duration oral exposure (1–14 days)

0.07 mg/kg/day for intermediate-duration oral exposure (15–364 days)

No MRL was derived for chronic-duration oral exposure because no data were found.

USEPA (2010) reports a chronic oral RfD of 0.1 mg/kg/d, revising their 1999 value of 0.5 mg/kg/d. Following the USEPA *Guidelines for Carcinogen Risk Assessment*, a nonlinear approach to dose-response assessment is taken for agents such as EGBE, for which the most plausible mode of action at low doses is consistent with nonlinearity. The RfD of 0.1 mg/kg-day represents the outcome of nonlinear assessment based on hemolytic effects (ie, hemosiderin deposition) associated with oral and exposure to EGBE. Doses (or concentrations) of EGBE below the RfD would not be expected to produce hemolytic effects and is therefore not expected to produce any increase in cancer risk.

##### 2-Butoxyethanol acetate (EGBEA) (from EU 2006a)

Humans may be exposed to EGBEA at workplace, via consumer products and indirectly via the environment. The highest potential exposure is likely to occur during occupational exposure.

The molecule of 2-butoxyethanol acetate is rapidly cleaved, presumably by esterases, into 2-butoxyethanol and acetate. It can therefore be anticipated that systemically available EGBEA will be metabolised into EGBE and acetate. Based on the structural similarities between EGBE and EGBEA and the high likely metabolism of EGBEA to EGBE at least in the systemic circulation, it is reasonable to assume that a read-across from EGBE data to EGBEA could be conducted when no specific or valid data on systemic toxicity are available on EGBEA.

Some studies are available to assess acute oral toxicity using various animals. The toxicological effects were mainly haemolysis and associated lesions. All the studies are old, with some uncertainties about the purity and the experimental procedure, but indicate that rabbits are more sensitive with a LD50 around 940 mg/kg.

A LOAEL of 400 mg/kg bw can be taken into account for acute toxicity by oral route in humans; it should be noted that this is a worst case estimation. A chronic oral LOAEL of 69 and 82 mg/kg bw/d of EGBE (in males and females respectively) can be fixed from a three-month study on cats, and 94 mg/kg bw/d based on male rats.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Butylbenzenes

Butylbenzenes are made up of a benzene ring with a butyl group attached. They comprise:

* n-butylbenzene (CAS No. 104-51-8). Also called 1-butylbenzene or 1-phenylbutane.
* sec-butylbenzene (CAS No.135-98-8). Also called 2-phenylbutane or 1‑methylpropyl)-benzene.
* tert-butylbenzene (CAS No. 98-06-6). Also called pseudobutylbenzene (1,1‑dimethylethyl) benzene, or 2-methyl-2-phenylpropane.

The xylenes, trimethylbenzenes and butylbenzenes are part of a bigger family of related chemicals called the alkyl substituted benzene derivatives.

### Maximum Acceptable Value

Butylbenzenes do not have a MAV in the DWSNZ, and are not mentioned in the WHO Guidelines.

The USEPA concluded on 22 September 2009 that sec-butylbenzene is known or anticipated to occur in PWSs and may require regulation. Therefore they have added [it](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane) to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

n-Butylbenzene is used in the production of other chemicals, and liquid crystals.

Sec-butylbenzene and tert-butylbenzene are flammable liquids used in US commerce for synthetic organic chemistry, as solvents, and tert-butylbenzene is used as a polymer linking agent. Sec-butylbenzene is a component of crude oil, vehicle emissions, ambient air, and is a possible migrant from microwave heating of thermoset polyester plastics. Tert-butylbenzene has been identified in landfill gas (OEHHA 2000).

tert-Butylbenzene is a useful intermediate for manufacturing agrochemicals and pharmaceuticals.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Water solubility of n-butylbenzene is about 12 mg/L. Water solubility of tert-butylbenzene is about 15 mg/L.

n-Butylbenzene does not absorb light with wavelengths >290 nm, and is not expected to be susceptible to direct photolysis by sunlight. If released to soil, n-butylbenzene is expected to have slight mobility based upon Koc values ranging from 2,450–2,510. Volatilisation from moist soil surfaces is expected to be an important fate process based upon an estimated Henry’s Law constant of 0.016 atm‑cu m/mole. A persistence of three months to one year in unadapted soils indicates that biodegradation in soil is may not be an important environmental fate process. If released into water, n-butylbenzene is expected to adsorb to suspended solids and sediment based upon the Koc values. Volatilisation from water surfaces is expected to be an important fate process based upon n-butylbenzene’s estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 3.5 and 110 hours , respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column (NCBI, accessed 2013).

### Typical concentrations in drinking-water

Twenty-three water utilities in the US reported detecting n-butylbenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.024 mg/L.

Nineteen water utilities in the US reported detecting sec-butylbenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0095 mg/L.

Thirteen water utilities in the US reported detecting tert-butylbenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0039 mg/L.

### Removal methods

Aeration appears to offer the best option.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

The US EPA National Center for Environmental Assessment (NCEA) has recommended that a provisional reference dose (RfD) for these compounds be derived from the toxicity data for a closely-related branched-chain alkylbenzene, cumene (isopropylbenzene), qv. Cumene (isopropylbenzene) has a dose-schedule-adjusted NOAEL of 110 mg/kg-day based on 139 gavage doses to female rats in a 194-day period. Other data on cumene and similar alkylbenzenes support this as an approximate NOAEL for the chemicals. OEHHA therefore agrees that this NOAEL is appropriate to be used for sec-butylbenzene and tert-butylbenzene. However, the lack of specific data justifies a large uncertainty factor.

Staff of the Office of Environmental Health Hazard Assessment (OEHHA) have reviewed the Department of Health Services proposed Notification Levels of 0.08 mg/L for sec-butylbenzene and tert-butylbenzene. OEHHA recommends action levels of 0.26 mg/L for sec-butylbenzene and tert-butylbenzene. The toxicological basis is the same, except for the use of an uncertainty factor of three instead of 10 for the subchronic to chronic toxicity study extrapolation.

### Derivation of Maximum Acceptable Value

No MAV.

NCBI quotes State Drinking Water Guidelines of 0.28 mg/L in Florida and 0.26 mg/L in North Hampshire for n-butylbenzene.

### References

Environmental Working Group (EWG). Accessed 2010. *National Drinking Water Database – Chemical Contaminants*. <http://www.ewg.org/tap-water/chemical-contaminants>.

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# N-(n-butyl) thiophosphoric triamide

CAS No. 94317-64-3. Also called butylphosphorothiotriamide NBPT and BTPT. Agrotain (20 percent NBPT) is a trade name for the commercially available product. A urea-based product containing NBPT used in New Zealand is called SustaiN.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for NBPT. The WHO Guidelines do not mention NBPT.

### Sources to drinking-water

#### 1. To source waters

NBPT is a urease inhibitor which can reduce nitrogen loss due to ammonium production (and subsequent nitrification) from urea by the enzyme urease.

Agrotain may contain up to 2 percent tetrahydrofuran (CAS No. 109-99-9), and up to about 15 percent N-methyl pyrrolidone (CAS No. 872-50-4; this is on USEPA’s third CCL, 2009) which is toxic to reproduction. Agrotain is used as a fertiliser additive in agricultural applications, often added to urea at about 0.05 percent.

Dicyandiamide (DCD) is a commonly used nitrification inhibitor; dimethylpyrazole-phosphate (DMPP) is a much superior nitrification inhibitor to DCD and is effective at lower concentrations. Nitrapyrin is used as well – see datasheets.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

NBPT may be absorbed by crops grown on treated soil. Residues of the chemical in food commodities are expected to be negligible. The relatively high water solubility of NBPT gives the potential to leach to groundwater. However, microbial activity which will occur in the subsoil and groundwater would be expected to lower the half-life, and taking into account the fast rate of mineralisation and proven ability of NBPT to bind to soil, this would combine to limit the extent of NBPT leaching to groundwater. Therefore leaching into groundwater is expected to be low.

The worst case predicted environmental concentrations (PEC) are 0.68 mg/L in a rural sewage treatment plant and 0.003 mg/L in fresh water.

The hydrolysis half-life at pH 7 is about 90 days at 25°C.

Water solubility is about 4300 mg/L.

### Health considerations

NBPT is of low acute oral and dermal toxicity in rats.

Oral absorption of NBPT is almost complete. After absorption, NBPT is distributed to various organs in animals, and NBPT concentrations in the blood stream decrease following a biphasic pattern. After a single oral dosing, more than 80 percent of the given dose was excreted in 7 days, mainly via expired air (35 percent), urine (24 percent) and faeces (9 percent). From the urine samples, two major metabolites of NBPT have been identified: N-(n-butyl)-thiophosphoric diamide and the glucuronic acid conjugate of NBPT.

The repeated dose NOAEL for males is 74 mg/kg bw/d based on liver effects and changes in neurobehaviour and haematology and the LOAEL is considered to be 377 mg/kg bw/d. A NOAEL could not be determined in females in the study, but the LOAEL for females is 17 mg/kg bw/d based on effects seen in the uterus.

NBPT showed little evidence of mutagenicity in two Ames tests with and without metabolic activation. An *in vivo* mouse micronucleus study was negative for evidence of clastogenicity.

### Derivation of Maximum Acceptable Value

No MAV.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. For tetrahydrofuran the short-term, chronic and subchronic health risk limits are 0.6 mg/L.

### References

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# Cadin-4-ene-1-ol

### Maximum Acceptable Value

Cadin-4-ene-1-ol does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Cadin-4-ene-1-ol is a metabolite of the actinomycetes which imparts a woody-earthy odour to water (EA 1998). It is known chemically as a sesquiterpenoid, and has been described as an enantiomer of epicubenol from cubeb oil.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Analytical methods

#### Referee method

No MAV.

### Derivation of Maximum Acceptable Value

No MAV.

### References

EA. 1998. *The Assessment of Taste, Odour and Related Aesthetic Problems in Drinking Waters 1998*. *Methods for the Examination of Waters and Associated Materials*. Environment Agency. [www.environment-agency.gov.uk/static/documents/Research/171\_taste\_odour\_in\_water.pdf](http://www.environment-agency.gov.uk/static/documents/Research/171_taste_odour_in_water.pdf) or go to [www.netregs.eu/default.aspx](http://www.netregs.eu/default.aspx) and enter title in search box.

EA. 2004. *The Microbiology of Drinking Water (2004) – Part 11 – Taste, odour and related aesthetic problems. Methods for the Examination of Waters and Associated Materials*. Environment Agency. www.netregs.eu/static/documents/Research/mdwpart112004-859972.pdf or go to [www.netregs.eu/default.aspx](http://www.netregs.eu/default.aspx) and enter title in search box.

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# Caffeine

CAS No. 58-08-2. IUPAC name: 3,7-dihydro-1,3,7-trimethyl-1*H*-purine-2,6-dione-1,3,7-trimethyl-1*H*-purine-2,6(3*H*,7*H*)-dione. Has also been called guaranine and methyltheobromine.

### Maximum Acceptable Value

Caffeine does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Caffeine is a bitter, white [crystalline](http://en.wikipedia.org/wiki/Crystalline) methyl[xanthine](http://en.wikipedia.org/wiki/Xanthine) [alkaloid](http://en.wikipedia.org/wiki/Alkaloid) that acts as a [stimulant](http://en.wikipedia.org/wiki/Stimulant) drug. Caffeine is found in varying quantities in the seeds, leaves, and fruit of some 60 plants, where it acts as a natural pesticide that paralyses and kills certain insects feeding on the plants. It is most commonly consumed by humans in infusions extracted from the seed of the [coffee plant](http://en.wikipedia.org/wiki/Coffea_arabica) and the leaves of the [tea bush](http://en.wikipedia.org/wiki/Camellia_sinensis), as well as from various foods and drinks containing products derived from the [kola nut](http://en.wikipedia.org/wiki/Kola_nut). Guarana is the richest known natural source of caffeine. Global consumption of caffeine has been estimated at 120,000 tonnes per year, making it the world’s most popular psychoactive substance. This amounts to one serving of a caffeinated beverage for every person every day. Although tea contains more caffeine than coffee (by dry weight), a typical serving contains much less, as tea is normally brewed much weaker.

Caffeine appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2012 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). It is registered as an oral nutrient/electrolyte and respiratory tract modifier.

Caffeine citrate appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2012 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). It is registered as an oral nutrient, oral, medicated antibiotic.

Because of the wide use of caffeine it is now present in a wide variety of environments, including wastewater treatment plant effluents, groundwater and even remote mountain lakes; typically in the ng/L range in many freshwater environments. In certain areas levels appear to be sufficiently high to approach threshold toxicity values for aquatic biota, primarily in locations near the discharge of raw or treated wastewater.

Several studies have determined that there is a strong correlation between the levels of caffeine in water and the level of bacteria, and that scientists can therefore use caffeine levels as an indicator of pollution due to sewerage systems. The presence of caffeine in water is a sure indicator of human sewage contamination, as agriculture and industry do not tend to release caffeine into the environment. Finding *E. coli* in natural water in the absence of caffeine has been suggested as a test for animal (non-human) wastes.

An evaluation of 139 stream sites in the US for the occurrence of organic wastewater contaminants found that caffeine was the fourth most frequently detected chemical, and occurred in 70 percent of the samples (Kolpin et al 2002).

Caffeine concentrations ranged between 0.007 to 0.07 mg/L in wastewater influents, and 0.00003 to 0.01 mg/L in effluents of wastewater treatment plants, which amounts to 81–99 percent removal; septic tanks will be a lot less effective. Ambient concentrations of caffeine in lakes and rivers ranged from 0.006 to 0.25 micrograms (µg)/L. Caffeine was detected in groundwater samples at concentrations up to 0.0002 mg/L. Remote mountain lakes contained <0.002 µg/L, ie, <2 ng/L (reported in USEPA 2006).

Many groundwaters in the UK were found to contain more than 0.001 mg/L caffeine (British Geological Survey 2011).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

If released to soil, caffeine will display very high mobility. It will not volatilise from either moist or dry soil, or from water, to the atmosphere. Caffeine degrades within a few weeks to two to three months in the environment.

If released to soil, caffeine is expected to have low to no mobility based upon Koc values of 741 and 7762 determined in silt and sandy loam soils. An approximated Koc of 71 suggests high mobility in sand which contains no clay and very low organic carbon content. Caffeine is both a weak acid and a weak base with pKa values of 14.0 and 0.7. Although partial ionisation to cation and anion forms may occur, electrochemical studies have found that the neutral form of caffeine was predominant in the pH range of 5.5 to 9. Cations generally adsorb more strongly to organic carbon and clay than their neutral counterparts suggesting that the cation form of caffeine may have higher Koc values than the neutral form. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 1.1 x 10-11 atm‑cu m/mole. Caffeine is not expected to volatilise from dry soil surfaces based upon its vapour pressure. Biodegradation studies in three Canadian soils observed mineralising to CO2; mineralisation in sandy loam and loam soils reached about 60 percent in 20 days (50 percent in 3-10 days) with mineralisation in silt loam soil reaching about 25 percent in 34 days. Various biodegradation studies have found caffeine and similar analogs to be readily biodegradable. If released into water, caffeine is expected to adsorb to suspended solids and sediment based upon the Koc. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. The hydrolysis half-life of caffeine in water is reported to be >1 year. Degradation in natural water can occur through photodegradation and biodegradation. Photooxidation via singlet oxygen radicals may be on the order of hours to days based on analogs containing imidazole rings. Caffeine had a mean half-life of 1.5 days in an aquatic microcosm study using sunlight exposure with photodegradation suggested as the primary fate process. The estimated half-life for caffeine in the Rhine River was 0.8 days which was thought to occur as a result of biodegradation (EAWAG accessed February 2015).

Water solubility: 2 percent in cold water; 66 percent in boiling water.

### Typical concentrations in drinking-water

No information; the maximum permissible level of caffeine in soft drinks in New Zealand is 145 mg/L.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

EPA Method 553.

### Health considerations

Caffeine and pharmaceuticals are discharged into the aqueous environment in wastewater, and they serve as source tracking indicators of human sewage pollution. Their presence in groundwater suggests a possibility of septic or sewage contamination. Their concentration in treated drinking water seldom exceeds the detection limit of available analytical methods whereas the concentration in wastewater is typically much higher, thus a wastewater contamination event may result in a significantly higher level, that is detectable over baseline (USEPA 2006).

Absorbed caffeine is readily distributed throughout the entire body. It passes across the blood-brain barrier, through the placenta into amniotic fluid and the foetus, and into breast milk. In humans, caffeine acts as a [central nervous system](http://en.wikipedia.org/wiki/Central_nervous_system) stimulant, temporarily warding off drowsiness and restoring alertness. It is the world’s most widely consumed [psychoactive drug](http://en.wikipedia.org/wiki/Psychoactive_drug), but, unlike many other psychoactive substances, it is both legal and unregulated in nearly all parts of the world. Beverages containing caffeine, such as [coffee](http://en.wikipedia.org/wiki/Coffee), [tea](http://en.wikipedia.org/wiki/Tea), [soft drinks](http://en.wikipedia.org/wiki/Soft_drink), and [energy drinks](http://en.wikipedia.org/wiki/Energy_drink), enjoy great popularity; in North America, 90 percent of adults consume caffeine daily.

Caffeine has a range of adverse effects. Common acute adverse effects associated with stimulation of the central nervous system following caffeine ingestion include dizziness, rapid heartbeat, irritability, anxiety, tremors and insomnia. Irritation of the gastrointestinal tract can result in diarrhoea, nausea and/or vomiting. Single high doses of caffeine can affect the cardiovascular system causing rapid heart beat and high blood pressure. Moderate daily caffeine intake by healthy adults with adequate nutrition, up to 400 mg/day (5.7 mg/kg bw/day for a 70 kg adult) is unlikely to result in adverse effects (MPI 2010).

Although caffeine was reported to induce mutations and inhibit DNA repair in a number of micro-organisms and cell lines, it is considered unlikely that at normal levels of exposure, caffeine would result in mutagenic effects in humans (MPI 2010).

There is currently no recognised reference health standard established for caffeine exposure, such as an Acceptable Daily Intake (ADI). An upper exposure of 2.5 mg/kg bw/day has been suggested as a cautious toxicological limit on which to base risk assessments for children, based on limited evidence. An adverse effect level of 3 mg/kg bw/day for adults is a conservative reference level based on limited evidence of acute anxiety effects. A reference level of 200 mg/day for pregnant women is used in this report and is based on recent evidence of foetal growth restriction (MPI 2010).

Single doses of caffeine up to 200 mg (about 3 mg/kg bw for a 70‑kg adult) from all sources do not give rise to safety concerns for the general healthy adult population. The same amount of caffeine does not give rise to safety concerns when consumed less than two hours prior to intense physical exercise under normal environmental conditions. No studies are available in pregnant women or middle-aged/elderly subjects undertaking intense physical exercise. Single doses of 100 mg (about 1.4 mg/kg bw for a 70‑kg adult) of caffeine may increase sleep latency and reduce sleep duration in some adult individuals, particularly when consumed close to bedtime (EFSA 2015).

### Derivation of Maximum Acceptable Value

No MAV.

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# Carbon tetrachloride

CAS No. 56-23-5. Also known as tetrachloromethane; has also been called perchloromethane.

### Maximum Acceptable Value

Based on health considerations, the concentration of carbon tetrachloride in drinking-water should not exceed 0.005 mg/L (5 g/L).

The maximum contaminant level or MCL (USEPA 2006/2009/2011) is 0.005 mg/L. The maximum acceptable concentration in Canada is 0.002 mg/L; short-term exceedances above the guideline value are unlikely to have an effect on health. However, in the event that monitoring data show elevated levels on a yearly basis, it is suggested that a plan be developed and implemented to address these situations.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of carbon tetrachloride in drinking water should not exceed 0.003 mg/L.

Carbon tetrachloride is one of the “priority pollutants” under the US Clean Water Act.

The odour threshold in water is 0.52 mg/L (ICPS 1998).

### Sources to drinking-water

#### 1. To source waters

Carbon tetrachloride can be released to the aquatic environment as an industrial contaminant. It was used mainly in the production of chlorofluorocarbon refrigerants, foam blowing agents, and solvents. It is also used in the manufacture of paints and plastics, as a solvent in metal cleaning, and in fumigants. Since the mid-1970s, annual use and production has generally declined.

Carbon tetrachloride is listed as a controlled substance in the New Zealand Ozone Layer Protection Act, 1990 that has been phased out so can only be obtained with a permit. Its occurrence in the New Zealand environment should therefore decrease.

#### 2. From treatment processes

Carbon tetrachloride may appear in water as the result of its presence as a trace impurity in the liquid chlorine used to treat the water.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Most carbon tetrachloride released to the environment reaches the atmosphere, where it may be removed by photolysis. Carbon tetrachloride has an estimated half-life of 50 years in the atmosphere.

Carbon tetrachloride is reasonably soluble in water (about 800 mg/L). Carbon tetrachloride dissolved in water does not photodegrade or oxidise in any measurable amounts. It migrates from surface water to the atmosphere in a matter of days or weeks. It is expected to leach to lower soil horizons and groundwater. Carbon tetrachloride is capable of adsorbing to organic matter in soils. Bioaccumulation has not been observed. Carbon tetrachloride may biodegrade in soil or water under anaerobic conditions; however, biodegradation of carbon tetrachloride under aerobic conditions does not occur readily, so levels in anaerobic groundwater may remain elevated for months or years.

If released to soil, carbon tetrachloride is expected to have high mobility based upon a Koc of 71. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 2.76 x 10-2 atm‑cu m/mole. Carbon tetrachloride may volatilise from dry soil surfaces based upon its vapour pressure. Aerobic degradation of carbon tetrachloride measured >87 percent in 7 days. Carbon tetrachloride found in leachate samples was degraded 100 and 90 percent within 10 and 50 days, respectively. Nearly complete degradation of carbon tetrachloride was observed at low concentrations after three weeks of incubation under methanogenic conditions. If released into water, carbon tetrachloride is not expected to adsorb to suspended solids and sediment in water based upon the Koc. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 1.3 hours and five days, respectively. A BCF of 3.2–7.4 suggests bioconcentration in aquatic organisms is low. The hydrolysis half-life in water is 7,000 years at 25°C (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987–1992 contained carbon tetrachloride results from 70 samples. Carbon tetrachloride was detected in three samples in concentrations ranging from  
0.0004–0.0006 mg/L (0.4–0.6 g/L).

The P2 Chemical Determinand Identification Programme, sampled from 332 zones, found no carbon tetrachloride at detectable concentrations (limit of detection = 0.0005 mg/L) (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0007 mg/L carbon tetrachloride in the raw water, the treated water and the distribution system.

Owing to its high volatilisation from water, carbon tetrachloride concentrations are normally low in surface water (<0.001 mg/L). However, in groundwater systems where volatilisation and biodegradation are limited, concentrations may be higher if contamination has occurred in the vicinity and leaching has taken place. Concentrations of carbon tetrachloride have been measured in various water sources at limited locations across Canada. In Québec, carbon tetrachloride was detected in 10 distribution systems at a maximum concentration of 0.001 mg/L between the years 2001 and 2005 (Health Canada 2010).

274 water utilities in the US reported detecting carbon tetrachloride in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.013 mg/L.

### Removal methods

Carbon tetrachloride present in contaminated source waters can be removed by adsorption on to granular activated carbon or by air stripping.

As carbon tetrachloride may appear in water as the result of its presence in gaseous chlorine used to treat the water, reducing the organic loading of the water, and hence the chlorine demand, will minimise the amount of chlorine that has to be added, and therefore the amount of carbon tetrachloride appearing in the water.

WRF (2014) reports that carbon tetrachloride is characterised with a very high Henry’s law constant (0.969 dimensionless air/water at 20°C) which is the second highest among the 13 VOCs trialled. Low profile air stripping is very effective for carbon tetrachloride removal even at low temperatures and low air to water ratios (below 100). Carbon tetrachloride was completely removed in most of the tested scenarios, and showed a very high removal efficiency (99.4 percent) at the lowest temperature (4°C) and lowest air to water ratio (53) which is the worst case examined.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551).

### Health considerations

Carbon tetrachloride is absorbed readily from the gastrointestinal tract, the respiratory tract and the skin. It is distributed to all major organs, with highest concentrations in fatty tissues. Carbon tetrachloride is thought to be metabolised in the liver to chloroform and other products, which binds to macromolecules, initiating lipid peroxidation and destroying cell membranes. USEPA (2010) states that in humans and animals exposed to carbon tetrachloride by any route, the unmetabolised parent compound is excreted in exhaled air. Additionally, animal studies show that volatile metabolites are released in exhaled air, whereas non-volatile metabolites are excreted in feces and to a lesser degree, in urine.

Although available data on concentrations in food are limited, the intake from air is expected to be much greater than that from food or drinking-water.

Workers exposed to 20 to 80 ppm carbon tetrachloride for two to three months experienced nausea, depression, dyspepsia and narcosis. Kidney and liver damage have been reported after short term exposures to 200 ppm. Similar effects have been reported following acute oral exposures. Death may result from ingestion of as little as 1.5 mL for an adult. Alcohol consumption enhances carbon tetrachloride-induced hepatic and renal effects in adults.

Carbon tetrachloride does not exhibit any evidence of mutagenic activity in tests with bacteria or cultured liver cells. Many genotoxicity assays have been conducted with carbon tetrachloride. On the basis of available data, carbon tetrachloride can be considered to be a non-genotoxic compound.

Carbon tetrachloride has been classified by the International Agency for Research on Cancer in Group 2B (possibly carcinogenic to humans). Carbon tetrachloride appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

USEPA (2010) states that carbon tetrachloride is “likely to be carcinogenic to humans” based on: (1) inadequate evidence of carcinogenicity in humans and (2) sufficient evidence in animals by oral and inhalation exposure, ie, hepatic tumours in multiple species (rat, mouse, and hamster) and pheochromocytomas (adrenal gland tumours) in mice. USEPA (2009) quotes a health advisory of 0.03 mg/L for carbon tetrachloride, represents a 10-4 cancer risk; USEPA (2001) changed this to 0.05 mg/L.

Carbon tetrachloride has caused liver and other tumours in rats, mice and hamsters after oral, subcutaneous, and inhalation exposure. The length of time to the development of the first tumour has sometimes been short, within 12–16 weeks in some experiments.

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 MRLs for carbon tetrachloride are:

minimal risk level

0.02 mg/kg/day for acute-duration oral exposure (1–14 days)

0.007 mg/kg/day for intermediate-duration oral exposure (15–364 days)

The reference dose or RfD (USEPA 2006/2009) was 0.0007 mg/kg/d; USEPA (2011) changed this to 0.004 mg/L. USEPA (2010) states that an RfD of 0.004 mg/kg-day for carbon tetrachloride is derived by applying a composite UF of 1,000 to the BMDL2X-ADJ of 3.9 mg/kg-day, where the composite UF of 1,000 includes a factor of 3 (100.5) to extrapolate from a subchronic to chronic duration of exposure, a factor of 10 to protect susceptible individuals, a factor of 10 to extrapolate from rats to humans, and a factor of 3 to account for database deficiencies. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009) is 0.03 mg/L.

### Derivation of Maximum Acceptable Value

A tolerable daily intake (TDI) of 1.4 mg/kg of body weight was used, based on a NOAEL of 1 mg/kg of body weight per day for hepatotoxic effects in a 12-week oral gavage study in rats, incorporating a conversion factor of 5/7 for daily dosing and applying an uncertainty factor of 500 (100 for inter- and intraspecies variation, 10 for the duration of the study and a modifying factor of 0.5 because it was a bolus study).

The MAV for carbon tetrachloride in drinking-water was derived as follows:

1 x (5/7) mg/kg body weight per day x 70 kg x 0.1 = 0.005 mg/L (5 g/L)

2 L x 500

where:

* no-observable-adverse-effect level = 1 mg/kg body weight per day based a 12‑week oral gavage study in rats (normalised for 5 days/week dosing in derivation)
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 500 (100 for inter- and intraspecies variation, 10 for the duration of the study and a modifying factor of 0.5 because it was a bolus study).

This value is lower than the range of values associated with lifetime upperbound excess cancer risks of 10-4, 10-5 and 10-6 calculated by linear extrapolation.

The MAV for carbon tetrachloride in the 1995 DWSNZ had been 0.002 mg/L. The 1995 datasheet showed the derivation as follows:

1 x (5/7) mg/kg body weight per day x 70 kg x 0.1 = 0.002 mg/L (rounded down)

2 L x 100

where:

* no-observable-adverse-effect level = 1 mg/kg body weight per day based a 12‑week oral gavage study in rats (normalised for 5 days/week dosing in derivation)
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for inter- and intraspecies variation and 10 for evidence of possible non-genotoxic carcinogenicity), No additional factor for the short duration of the study was incorporated. It was considered to be unnecessary because the compound was administered in corn oil in the critical study and available data indicate that the toxicity following administration in water may be an order of magnitude less.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The acute limit is 0.1 mg/L, the short-term, chronic and subchronic limits are 0.003 mg/L. The cancer health risk limit for carbon tetrachloride is 0.001 mg/L.

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# Chloramine t

CAS No. 127-65-1. The IUPAC name is N-chloro-4-methylbenzenesulfonamide, sodium salt. Also called N-chloro-para-toluenesulfonylamide, *N*-chloro tosylamide sodium salt, or sodium chloro[(4-methyl phenyl)sulfonyl]azanide. Also spelt with or without the hyphen. CAS No. 7085-50-4 refers to the trihydrate form. Chloramine-T also goes under a host of trade names.

The main reason for preparing this datasheet is to remove any confusion between chloramine T and the chloramines used for or resulting from more conventional water disinfection.

### Maximum Acceptable Value

Chloramine T does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Chloramine-T has used since the early 1900s for disinfection and as an [algicide](http://en.wikipedia.org/wiki/Algicide), [bactericide](http://en.wikipedia.org/wiki/Bactericide), [germicide](http://en.wikipedia.org/wiki/Germicide), for [parasite](http://en.wikipedia.org/wiki/Parasite) control, and has even been said to have been used for drinking water disinfection. It has widespread applications in agriculture, aquaculture, medical and dental facilities, air conditioning, and the food industry. In agricultural practices, chloramine-T has been approved as a broad spectrum biocide for foot-and-mouth disease, swine vesicular disease, diseases of poultry, and tuberculosis in the United Kingdom, and is used in numerous branches of industry such as intensive farming, slaughterhouses, and kitchens.

Chloramine-T is slightly basic (pH typically 8.5). In water, it breaks down to the disinfectant [hypochlorite](http://en.wikipedia.org/wiki/Hypochlorite). The sulfonamide component disrupts bacterial metabolism rather like a sulpha-drug.

Chloramine-T appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). It is registered as an antimicrobial agent.

#### 2. From treatment processes

Nil.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Chloramine-T does not bind to soil or sludge and is readily biodegradable if it is in sufficiently low concentrations in water.

The metabolite *p-*toluenesulfonamide (*p-*TSA), CAS No. 70-55-3, has high mobility if released into soil, and volatilisation should not be important from moist or dry soil surfaces. It is stable in neutral, acidic or alkaline solutions, and is classified as “not readily biodegradable”. p-Toluenesulfonamide can also be called 4-methyl-benzenesulfonamide, toluene-p-sulfonamide, and p-tosylamide.

The molecular structure of toluenesulfonylamide is similar to [para-aminobenzoic acid](http://en.wikipedia.org/wiki/Para-aminobenzoic_acid), a naturally occurring intermediate in bacterial metabolism.

Water solubility of chloramine T is about 15 percent. Water solubility of p-TSA is about 3200 mg/L.

### Health considerations

Chloramine-T has a low degree of cytotoxicity and has been used in direct contact with tissues. As such, it is used in the treatment of burns, in whirlpools for the treatment of wounds, and as an oral mouthwash.

The metabolite *p-*toluenesulfonamide is of importance as the primary residue of chloramine-T in chloramine-T treated fish intended for human consumption. *p-*TSA is used as an intermediate for pesticides and drugs and is used as an additive to outdoor paints in Sweden. Mixtures of *o*-TSAand *p-*TSA can be used as reactive plasticisers in hot-melt adhesives to improve flow properties of thermosetting resins. This mixture also adds flexibility to coatings based on some resins. Both *o*-TSAand *p-*TSA were common, and quantitatively important, contaminants of saccharin. *p-*TSA is used in the formulation of toluenesulfonamide/formaldehyde resin (TSFR), which is used in fingernail polishes and enamels at concentrations up to 10 percent (NEIHS 2002).

p-Toluenesulfonamide showed no genotoxic effects, and the LOAEL for repeated dose toxicity was 120 mg/kg/day and the NOAEL for reproductive toxicity was 300 mg/kg/day. Estimated dose of low concern (EDLC) was calculated as 0.024 mg/kg/day and 0.6 mg/kg/day for repeated dose toxicity and reproductive toxicity, respectively (OECD 1995).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Chlorendic acid

CAS No. 115-28-6. The IUPAC name is 1,4,5,6,7,7-hexachlorobicyclo[2.2.1]-hept-5-ene-2,3-dicarboxylic acid. Also called HET acid, hexachloroentomethylenetetrahydrophthalic acid. The anhydride, CAS No. [115-27-5](http://www.emolecules.com/cgi-bin/search?t=ss&q=115-27-5&c=1&v=), is discussed in INCHEM (2009).

### Maximum Acceptable Value

Chlorendic acid does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Chlorendic acid is used as an intermediate in synthesis of unsaturated flame-retardant [polyester](http://en.wikipedia.org/wiki/Polyester) [resins](http://en.wikipedia.org/wiki/Resin) and [plasticisers](http://en.wikipedia.org/wiki/Plasticizer), and as a finishing flame-retardant treatment for [wool](http://en.wikipedia.org/wiki/Wool). A major use is in production of [fiberglass](http://en.wikipedia.org/wiki/Fiberglass)-reinforced resins for chemical industry equipment. It can be used to make [alkyd resins](http://en.wikipedia.org/wiki/Alkyd_resin) for use in special [inks](http://en.wikipedia.org/wiki/Ink) and [paints](http://en.wikipedia.org/wiki/Paint). It is used as a hardening agent in [epoxy resins](http://en.wikipedia.org/wiki/Epoxy_resin) used in manufacture of [printed circuit boards](http://en.wikipedia.org/wiki/Printed_circuit_board). When reacted with non-halogenated [glycols](http://en.wikipedia.org/wiki/Glycol), it forms halogenated [polyols](http://en.wikipedia.org/wiki/Polyol) used as [flame retardants](http://en.wikipedia.org/wiki/Flame_retardant) in [polyurethane](http://en.wikipedia.org/wiki/Polyurethane) [foams](http://en.wikipedia.org/wiki/Foam). It is also used for production of [dibutyl chlorendate](http://en.wikipedia.org/w/index.php?title=Dibutyl_chlorendate&action=edit&redlink=1) and [dimethyl chlorendate](http://en.wikipedia.org/wiki/Dimethyl_chlorendate), which are used as reactive flame retardants in [plastics](http://en.wikipedia.org/wiki/Plastic). In limited amounts, it is used as an additive in [acrylonitrile butadiene styrene](http://en.wikipedia.org/wiki/Acrylonitrile_butadiene_styrene) (ABS) [copolymer](http://en.wikipedia.org/wiki/Copolymer). Esters and amine salts of chlorendic acid are used as [extreme pressure additives](http://en.wikipedia.org/wiki/Extreme_pressure_additive) in synthetic [lubricants](http://en.wikipedia.org/wiki/Lubricant).

Chlorendic acid may be released via hydrolytic degradation of polyesters and as an oxidation product of chlorinated cyclodiene insecticides, eg, [endosulfan](http://en.wikipedia.org/wiki/Endosulfan), [chlordane](http://en.wikipedia.org/wiki/Chlordane), [heptachlor](http://en.wikipedia.org/wiki/Heptachlor), [aldrin](http://en.wikipedia.org/wiki/Aldrin), [dieldrin](http://en.wikipedia.org/wiki/Dieldrin), [endrin](http://en.wikipedia.org/wiki/Endrin), and [isodrin](http://en.wikipedia.org/wiki/Isodrin).

The combined worldwide production for chlorendic acid and anhydride is at present around 4,000 tonnes per year (IPCS 1996).

#### 2. From treatment processes

Nil.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Ultraviolet light degrades chlorendic acid with a half-life of 16 days in a solid thin layer and 5 days in an aqueous solution. Its half-life in [soil](http://en.wikipedia.org/wiki/Soil) is 140–280 days.

Chlorendic acid is not expected to volatilise from water or soil; it has a low potential for binding to soil and sediment and is expected to have high mobility in soil, so in theory, it could leach to groundwater. Chlorendic acid has been found in the leachate from landfills at concentrations of up to 455 mg/L.

In aqueous solution, chlorendic anhydride is rapidly hydrolysed to chlorendic acid, with a half-life of approximately one hour.

Water solubility of chlorendic acid is about 3500 mg/L.

### Analytical methods

#### Some alternative methods

See IPCS (1996).

### Health considerations

Both chlorendic acid and the anhydride seem to have low acute and subacute oral toxicity, but they are dermal, eye and respiratory irritants. From the results of long-term toxicity/carcinogenicity studies with chlorendic acid on rats and mice, it is concluded that chlorendic acid induces tumours in rats and mice and is, therefore, considered to have a carcinogenic potential. However, a full hazard assessment for humans and the environment cannot be made in view of the lack of data (IPCS 1996).

IARC (1990) states that chlorendic acid is possibly carcinogenic to humans (Group lB).

The primary route of potential human exposure to chlorendic acid is dermal contact, but inhalation exposure also is possible. No epidemiological studies were identified that evaluated the relationship between human cancer and exposure specifically to chlorendic acid, however, chlorendic acid is reasonably anticipated to be a human carcinogen (NIH 2011).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Chloroacetones (haloketones)

Chloroacetone or monochloroacetone: IUPAC name is 1-chloro-2-propanone. Also called acetonyl chloride. CAS No. 78-95-5.

Dichloroacetones, or collectively, dihaloketones, include:

* 1,1-dichloropropanone (also called DCP, 1,1-dichloro-2-propanone or 1,1‑dichloropropan-2-one). CAS No. 513-88-2
* 1,3-dichloropropanone (also called 1,3-dichloro-2-propanone or 1,3‑dichloropropan-2-one). CAS No. 534-07-6.

Trichloroacetones (collectively trihaloketones) include:

* 1,1,1-trichloropropanone (also called 1,1,1-trichloro-2-propanone or 1,1,1‑trichloropropan-2-one). CAS No. 918-00-3.
* 1,1,3-trichloropropanone (1,1,3-trichloro-2-propanone or 1,1,3-trichloropropan-2-one). CAS No. 921-03-9.

Tetra- and penta- chloroacetones exist too.

### Maximum Acceptable Value

There are insufficient data to determine a MAV for any of the chloroacetones in drinking-water.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for chloroketones in drinking water. These are: 1,1‑dichloropropanone (dichloroacetone), 1,3-dichloropropanone, 1,1,1‑trichloropropanone (trichloroacetone) and 1,1,3-trichloropropanone.

### Sources to drinking-water

#### 1. To source waters

Dichloroacetones may be present in source waters as a result of their discharge from industries in which they are used. Chloroacetones are reagents in the synthesis of drugs, perfumes, insecticides and vinyl compounds.

#### 2. From treatment processes

Dichloroacetones may form as a result of chlorination from the reaction between chlorine and large organic molecules. Ozone can react with natural organic matter to form acetone and acetaldehyde, which can then react with chlorine to form trichloroacetones. Chloroacetones do not appear in many lists of disinfection by-products.

IARC (1991) reported concentrations of 1,1-dichloropropanone and 1,1,1‑trichloropropanone that were produced during chlorination. Other chlorinated ketones that have been detected in drinking-water but have not been quantified, include 1,1,3,3-tetrachloropropanone, 3,3-dichloro-2-butanone, 1,1-dichloro-2-butanone, 1,1,1-trichloro-2-butanone and 2,2-dichloro-3-pentanone.

WHO (2011) states: 1,1-Dichloroacetone is formed from the reaction between chlorine and organic precursors and has been detected in chlorinated drinking-water. Concentrations are estimated to be less than 10 μg/l (0.01 mg/L) and usually less than 0.001 mg/L.

In a large US study WRF (2016) found 1,1-dichloropropanone (DCP) in most samples; higher levels of 1,1-dichloropropanone were observed at chloraminated utilities. The highest concentration of 1,1-dichloropropanone (6.55 μg/L) was detected in the water sample collected at the point of entry to a distribution system and points in the distribution system exhibited lower levels. This utility was the only system in the study where chloramines were used without a significant free chlorine contact period. Dichloropropanone concentration was found to decrease very quickly in many systems. For example, it dropped from 6.55 ug/L (water age = 16.7 hours) to 2.20 µg/L(water age = 67.7 hours) within 50 hours in Utility #10. In other systems, 1,1-DCP concentrations also decreased, but usually at a lower rate.

As described by Krasner et al (2006), USEPA selected the following haloketones as priority DBPs for a nationwide occurrence study: chloropropanone, 1,3‑dichloropropanone, 1,1-dibromopropanone, 1,1,3-trichloropropanone, 1-bromo-1,1-dichloropropanone, 1,1,3,3- tetrachloropropanone, 1,1,1,3-tetrachloropropanone, 1,1,1,3,3-pentachloropropanone and hexachloropropanone. In this study, 1,1,3,3‑pentachloropropanone and hexachloropropanone were not analysed because they are not stable in water. While 1,1,3,3-tetrabromopropanone was not initially prioritised, it was identified in drinking water after the initial prioritisation and was included in the monitoring study report due to its similarity to the other priority compounds. Several haloketone species were identified in drinking water, with the priority haloketone 1-bromo-1,1-dichloropropanone reaching a maximum concentration of 3 μg/L in a distribution sample from a plant using ozone-chlorine disinfection. From USEPA (2016).

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

The chlorinated acetones undergo hydrolysis. The rate of this reaction depends on the number of chlorine atoms in the molecule and where they are positioned. 1,1‑Dichloroacetone is likely to undergo hydrolysis more rapidly than 1,3‑dichloroacetone.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme (ESR 2001) sampled from 491 zones, found 1,1-dichloropropan-2-one concentrations in four zones to range from “not detectable” (nd) to 0.0031 mg/L, with the median concentration being “nd” (limit of detection = 0.002 mg/L).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0003 mg/L 1,1‑dichloropropan-2-one in the raw water, the treated water and the distribution system.

In 2013/14 Hamilton’s six-monthly analyses have found <0.0003 mg/L 1,1,1‑trichloropropan-2-one in the raw water, the treated water once (the other sample contained 0.0003 mg/L) and the distribution system.

IARC (1991) reported 1,1-dichloropropanone and 1,1,1-trichloropropanone concentrations to reach a maximum of 0.002 mg/L, with many values around 0.0008 mg/L.

1,1-Dichloroacetone concentrations are estimated to be less than 0.01 mg/L and usually less than 0.001 mg/L (WHO 2004). 3 water utilities in the US reported detecting 1,1-dichloropropanone in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0055 mg/L.

The P2 Chemical Determinand Identification Programme (ESR 2001) sampled from 491 zones, found 1,1,1-trichloropropan-2-one concentrations in 50 zones to range from “not detectable” (nd) to 0.009 mg/L, with the median concentration being “nd” (limit of detection = 0.002 mg/L).

### Removal methods

As chloroacetones arise in waters principally as a disinfection by-product, the preferred method for minimising their formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Trihalomethane concentrations in chlorinated water increase with increasing pH. Concentrations in the finished water can therefore be reduced by ensuring that high pH levels are not present once the water is chlorinated.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Chloroacetones can be removed by adsorption on to granular activated carbon, or by air stripping.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

### Analytical methods

#### Referee method

A referee method cannot be selected for chloroacetones because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for chloroacetones for the above reason. However, the following information may be useful:

1. Chloroacetones in drinking-water may be analysed by purge and trap gas chromatography with mass spectrometry or electron capture detection (Method APHA 6251 or EPA Method 502.1). The limit of quantification is approximately 0.001 mg/L (1 g/L). Interference may occur from impurities in the purge gas and organic compounds outgassing from the trap system.

2. A solvent extraction procedure with methyl tert-butyl ether (MTBE) may be used (EPA Method 551) and analysis by gas chromatography with mass spectrometry or electron capture detection. The detection limit is approximately 0.00002 mg/L (0.02 g/L). Interference may occur from impurities in the reagents or glassware used for extraction.

### Health considerations

Studies with single doses of 1,1-dichloroacetone indicate that it affects the liver.

A number of chlorinated acetones including 1,1- and 1,3- and 1,1,1,- and 1,1,3,3,- and pentachloroacetone were direct-acting mutagens in one or both of *Salmonella typhimurium* strains TA98 and TA 100. Mutagenic activity decreased with increased chlorine substitutions at the C-1 and C-3 positions although 1,1,1-trichloroacetone was 25 times as potent as 1,1-dichloroacetone.

One carcinogenicity study concluded that 1,3-dichloroacetone is a tumour initiator in mouse skin. However, it is not listed as carcinogenic by ACGIH, IARC, NIOSH, NTP, or OSHA. 1,3-Dichloroacetone in the presence of FAC has a pronounced irritant effect on the eyes.

Acute oral toxicity studies in mice using 1,1-dichloropropanone and 1,3‑dichloropropanone found no toxic effects with single doses of 130 mg/kg and 20 mg/kg respectively. No long-term toxicity studies have been reported.

WHO (2003a) investigated the data for chlorinated acetones (propanones) and determined that the data on dose-response were limited. Single doses of 1,1‑dichloroacetone revealed effects on the liver at 325 mg/kg and no toxicity was observed below 130 mg/kg. No liver toxicity was observed for 1,3-dichloracetone at doses up to 20 mg/kg, but it was shown to potentially act as a tumour initiator in mouse skin. No guideline or regulatory value was derived by WHO (2003a). From USEPA (2016).

### Derivation of Maximum Acceptable Value

There are limited and insufficient data on the chloroacetones on which to propose a MAV for any of the chloroacetones.

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WRF. 2016. *Fate of Non-Regulated Disinfection By-products in Distribution Systems*. Web Report #4242. 240 pp. Water Research Foundation (US) and Drinking Water Inspectorate (UK). <http://www.waterrf.org/PublicReportLibrary/4242.pdf>.

# Chloroacetonitrile

CAS No. 107-14-2. Also called chloroethanenitrile, 2-chloroacetonitrile, α‑chloroacetonitrile, chloromethyl cyanide, monochloroacetonitrile, monochloromethyl cyanide, or CAN.

Datasheets have also been prepared for dichloroacetonitrile and trichloroacetonitrile. Also, bromochloroacetonitrile, dibromoacetonitrile, and a general datasheet for the other haloacetonitriles called bromoacetonitiles.

### Maximum Acceptable Value

Chloroacetonitrile is not mentioned in the WHO Guidelines, and does not have a MAV in the DWSNZ.

### Sources to drinking-water

#### 1. To source waters

Halogenated acetonitriles are not produced on an industrial scale. Chloroacetonitrile has been used on a limited basis in the past as a pesticide, or the manufacture of pesticides (eg, fenoxycarb – but this is not used in New Zealand).

#### 2. From treatment processes

Several halogenated acetonitriles have been detected in chlorinated drinking-water in a number of countries as a consequence of the reaction of chlorine with natural organic substances (and bromine in the case of brominated acetonitriles) present in untreated water (DWI 2010).

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

If released to water, chloroacetonitrile can be transported to air through volatilisation. Volatilisation half-lifes of 16 hr and 7.3 days have been estimated for a model river and environmental pond, respectively. If released to soil, chloroacetonitrile may leach readily. Adsorption to sediment and bioconcentration are not important fate processes.

Chloroacetonitrile water solubility is reported to be about 70,000 mg/L (7 percent).

### Typical concentrations in drinking-water

An Environmental Working Group (EGW 2008) analysis of chloroacetonitrile tests reported by 213 public water suppliers in the US shows that between 1998 and 2002, no communities drank water containing chloroacetonitrile (LoD not quoted). One water utility in the US reported detecting chloroacetonitrile in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0017 mg/L.

DWI (2012) reported a UK study. The lowland water sources that were included in the survey formed more N-DBPs than the upland and groundwater sources. The six treatment works that applied ozone were associated with higher concentrations of HANs and HAcAms than non-ozone treatment works, although this was potentially confounded because all the ozone works were treating lowland source waters which may have had higher N-DBP formation potential. None of the N-DBPs exhibited consistent links with total trihalomethanes (THMs). There were no trends linking HNMs to either THMs or HAA9. The mean HAN concentration was 0.0032 mg/L (3.2 µg/L). The main HANs were dichloroacetonitrile, bromochloroacetonitrile and dibromoacetonitrile. Trichloroacetonitrile was not detected; chloroacetonitrile was rare.

### Removal methods

As chloroacetonitrile arises in waters principally as a disinfection by-product, the preferred method for minimising their formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See DWI (2010).

### Health considerations

The only known route of human exposure is through chlorinated drinking-water.

After oral administration, a small, significant increase in the proportion of mice with lung tumours and number of tumours per mouse was observed. However, there is inadequate evidence in experimental animals for the carcinogenicity of chloroacetonitrile; IARC (1999) considered that chloroacetonitrile is not classifiable as to carcinogenicity to humans (Group 3).

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. One of their findings was that for cell induced genomic DNA damage, the rank order from the most genotoxic to the least genotoxic of the DBP classes was haloacetonitriles > haloacetamides > halonitromethanes > haloacetaldehydes > haloacetic acids > >2C‑haloacids > halomethanes.

2-Chloroacetonitrile is cytotoxic and causes genotoxicity *in vitro*. 2-Chloroacetonitrile crosses the placental and fetal blood-brain barriers and induces oxidative stress that triggers apoptotic neurodegeneration in the fetal brain. Maternal exposure to 2‑chloroacetonitrile adversely affects mouse foetal livers as evidenced by the induction of oxidative stress, apoptosis and histopathological changes. Intrauterine exposure to low levels of 2-chloroacetonitrile decreases fetal body weight and induces malformations in the musculoskeletal system in mice. The lowest observed effect level appears to be 25 mg/kg/day based on reprotoxicity test. The evidence for carcinogenicity is currently insufficient (DWI 2010).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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WHO. 2017. *Guidelines for Drinking-water Quality: Fourth edition incorporating the first Addendum*. Geneva: World Health Organization. 631 pp. [http://www.who.int/water\_sanitation\_health/publications/drinking-water-quality-guidelines-4-including-1st-addendum/en/](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\INetCache\Content.Word\www.who.int\water_sanitation_health\publications\2011\dwq_guidelines\en\).

# Chloroanilines

There are 19 chloroanilines, as follows (with their CAS No. in brackets):

1 pentachloroaniline (sometimes called PCA):

2,3,4,5,6-pentachloroaniline (527-20-8).

3 tetrachloroanilines:

2,3,4,5-tetrachloroaniline (634-83-3), 2,3,5,6-tetrachloroaniline (3481-20-7) and 2,3,4,6-tetrachloroaniline (654-36-4).

6 trichloroanilines:

2,3,4-trichloroaniline (634-67-3 and 54686-91-8), 2,3,5-trichloroaniline (18487‑39-3), 2,3,6-trichloroaniline (88963-39-7), 2,4,5-trichloroaniline (636‑30‑6), 2,4,6-trichloroaniline (634-93-5 and 33663-50-2) and 3,4,5‑trichloroaniline (634-91-3).

6 dichloroanilines (dichlorobenzenamines):

2,3-dichloroaniline (608-27-5 and 27134-27-6), 2,4-dichloroaniline (554-00-7), 2,5-dichloroaniline (95-82-9), 2,6-dichloroaniline (608-31-1 and 51225-19-5), 3,4‑dichloroaniline (95-76-1) and 3,5-dichloroaniline (626-43-7 and 13330-18-2).

3 monochloroanilines:

2-chloroaniline (95-51-2 and 137-04-2), 3-chloroaniline (108-42-9 and 141-85-5) and 4-chloroaniline (106-47-8 and 4084-48-4). These are also called orthochloroaniline, metachloroaniline and parachloroaniline, alternatively o‑chloroaniline, m-chloroaniline and p-chloroaniline respectively.

Also possibly appearing in the environment are the (chloro)nitroanilines and (chloro)methylanilines.

Chloroaniline can be called aminochlorobenzene, chlorobenzenamine, chloro‑aminobenzene or chlorophenylamine. Chloroaniline is occasionally abbreviated to just chloraniline.

### Maximum Acceptable Value

Chloroanilines are not mentioned in the WHO Guidelines, and do not have a MAV in the DWSNZ.

### Sources to drinking-water

#### 1. To source waters

Chloroanilines are toxic aromatic compounds and potential environmental contaminants. Possible sources of chloroanilines in the environment include industrial discharges; the biodegradation of certain pesticides; and the chlorination of aniline precursors. Chlorinated anilines are used as intermediates in the manufacture of dyes, pharmaceuticals, and agricultural agents (eg, urea herbicides).

Because 3,4-dichloroaniline is found frequently in relation to pesticides, it has a datasheet in the pesticides section (the other dichloroanilines are discussed in that datasheet too). 3,4-Dichloroaniline is referred to in the datasheets for diuron, linuron, iprodione and propanil; 3,5-dichloroaniline is mentioned in the iprodione datasheet. Pentachloroaniline, a degradation product of quintozene (pentachloronitrobenzene), is not expected to be mobile. 4-Chloroaniline can be an impurity and metabolite of diflubenzuron (qv).

A Dutch study found the exposure levels of monochloroanilines and dichloroanilines were far below the tentative maximum acceptable risk levels, whereas chloroanilines other than monochloroanilines and dichloroanilines probably did not occur in the Netherlands’ environment. Measured concentrations in the River Rhine and its tributaries are roughly between 0.0001 and 0.001 mg/L (IPCS 2003).

#### 2. From treatment processes

There is some evidence that 4-chloroaniline may be formed during the chlorination of drinking-water (Stiff and Wheatland 1984 – quoted in IPCS 2003).

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

If released to soil, 4-chloroaniline is expected to have high to low mobility based upon Koc values ranging from 96–1,530. However, aromatic amines are expected to bind strongly to humus or organic matter in soils due to the high reactivity of the aromatic amino group, suggesting that mobility may be much lower in some soils. Volatilisation from moist soil surfaces is expected based upon an estimated Henry’s Law constant of 3.1 x 10-6 atm‑cu m/mole. However, adsorption to soil may attenuate volatilisation. The pKa of 4-chloroaniline is 3.98, indicating that it will primarily exist in its non-ionic form in the environment. 4-Chloroaniline is not expected to volatilise from dry soil surfaces based upon its vapour pressure. If released into water, 4-chloroaniline is expected to adsorb to suspended solids and sediment based on its Koc values. Volatilisation from water surfaces is expected based on its estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 36 and 260 days, respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column (EAWAG accessed February 2015).

If released to soil, 2,4-dichloroaniline is expected to have low mobility based upon a Koc of 525. Volatilisation from moist soil surfaces is expected to be an important fate process based upon an estimated Henry’s Law constant of 1.6 x 10-6 atm‑cu m/mole. If released into water, 2,4-dichloroaniline is expected to adsorb to suspended solids and sediment in the water column based upon the estimated Koc. Photolysis rate constants of 0.071/hr and 0.033/hr in water were determined for summer and winter conditions, respectively, corresponding to respective half-lifes of 10 and 21 hours. No microbial degradation of 2,4-dichloroaniline occurred during short term incubations (up to three days) in die-away tests using an estuarine water from the Skidaway River in Georgia. Volatilisation from water surfaces is expected to be an important fate process based on its estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 30 and 219 days, respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. An BCF of 94.7 suggests bioconcentration in aquatic organisms is moderate (EAWAG accessed February 2015).

If released to soil, 3,5-dichloroaniline is expected to have moderate mobility based upon a Koc of 309. 3,5-Dichloroaniline may undergo covalent chemical bonding with humic materials, which can result in its chemical alteration to a latent form and tight adsorption. Incubation of 3,5-dichloroaniline in covered beakers containing a sandy loam soil for 14 days yielded the azo compound 3,3’,5,5’-tetrachloroazobenzene. Volatilisation from moist soil surfaces is expected to be an important fate process based upon an estimated Henry’s Law constant of 1.58 x 10-4 atm‑cu m/mole. If released into water, 3,5‑dichloroaniline is expected to adsorb to suspended solids and sediment in the water column based upon the Koc. Using the Closed Bottle screening test, a 0 percent theoretical BOD (Biological Oxygen Demand) was observed over a 30‑day inoculation period using a sewage inoculum. Volatilisation from water surfaces is expected to be an important fate process based on its estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 30 and 219 days, respectively. When covalently bound in this latent form, leaching in soil systems is not generally expected to occur. An estimated BCF of 94 suggests the potential for bioconcentration in aquatic organisms is moderate. Hydrolysis is not expected to occur due to the lack of hydrolysable functional groups (EAWAG accessed February 2015).

In soil all monochloro- and some dichloroanilines are transformed to their corresponding dichloro- and tetrachloroazobenzenes. Other dichloroanilines and the trichloroanilines are stable in soil.

Chloroanilines are not expected to bioaccumulate, are not readily biodegraded, and evaporation is not an important process. Numerous studies on the biodegradation of 4-chloroaniline indicate it to be inherently biodegradable in water under aerobic conditions, whereas no significant mineralisation was detected under anaerobic conditions. The measured half-life of 4-chloroaniline in water is 151 days at a water depth of 1 m and a temperature of 20°C. 4-Chloroaniline is quite soluble in water: about 0.2 to 0.3 percent, or say 2500 mg/L. Pentachloroaniline water solubility is reported to be 0.03 mg/L.

Dechlorination of pentachloroaniline, 2,3,4,5-and 2,3,5,6-tetrachloroaniline, 2,3,4-, 2,3,5-, 2,4,5-, 2,4,6-, and 3,4,5-trichloroaniline, and 3,5-dichloroaniline (to a low extent) was observed but none of the five remaining dichloroanilines and three monochloroanilines were dechlorinated by microbial enrichment culture during batch assays (Okutman et al 2006).

### Typical concentrations in drinking-water

In the 1980s and 1990s, 4-chloroaniline was found in German drinking-water samples at concentrations between 0.000007 and 0.000013 mg/L; and in groundwater (in 1995–1996) from three sites in an industrialised area near Milan, Italy, at concentrations between 0.00001 and 0.00006 mg/L (positive results found in four of seven wells) (IPCS 2003).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Anilines chlorinated at the 2, 3, and 4 (ortho, meta, and para) positions have the same use patterns. All chloroaniline isomers are haematotoxic and show the same pattern of toxicity in rats and mice, but in all cases 4-chloroaniline shows the most severe effects. 4-Chloroaniline is genotoxic in various systems while the results for 2- and 3‑chloroaniline are inconsistent and indicate weak or no genotoxic effects. IPCS (2003) therefore focused only on 4-chloroaniline as the most toxic of the chlorinated anilines.

The RfD for p-chloroaniline was calculated at 0.004 mg/kg/d (USEPA (1995).

RIVM (1998) said although some anilines are known to have a mutagenic and/or carcinogenic potential, evaluation of data on carcinogenic and mutagenic effects for humans, which was conducted in the context of another RIVM project, revealed that only few data are available so at present it is not possible to include a profound human risk evaluation. For monochloroanilines an oral excess 10-4 life-time tumour risk of 0.0009 mg/kg bw is derived (chance of 1 in 10,000 to develop a tumour when exposed to 0.0009 mg/kg bw for life).

Pentachloroaniline, a metabolite of quintozene, is found in food.

IPCS (2003) stated if one applied the uncertainty factors of 10 (for use of a LOAEL rather than a NOEL) × 10 (for interspecies extrapolation) × 10 (for interindividual variability) to the LOAEL of 2 mg/kg body weight per day, one could derive a tolerable intake of 0.002 mg/kg body weight per day. The three main exposure routes are wearing dyed textiles next to the skin, using deodorant products (containing triclocarban) and using mouthwashes (containing chlorohexidine).

IARC (1993) considered that para-chloroaniline is possibly carcinogenic to humans (Group 2B). 4-Chloroaniline appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. USEPA (1997) states that p-chloroaniline (PCA or 4-chloroaniline), a metabolite of diflubenzuron, is a probable human carcinogen (Group B2). Several other pesticides either contain chloroanilines, or include chloroanilines in their breakdown products, eg, chlorpropham, dicloran, diuron, iprodione, linuron, propanil and quintozene.

3,4-Dichloroaniline is on the EC List of 66 Category 1 substances showing evidence of endocrine disrupting activity in at least one species using intact animals (EC 2015).

### Derivation of Maximum Acceptable Value

No MAV.

### References

EAWAG. Accessed February 2015. *Biocatalysis/Biodegradation Database: Select 1396 compounds*. <http://eawag-bbd.ethz.ch/index.html>.

EC. 2015. *Endocrine Disruptors Priority List* (last updated 25/03/2015). Brussels, Belgium: European Commission. Accessed July 2015. <http://ec.europa.eu/environment/chemicals/endocrine/strategy/substances_en.htm>.

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IUPAC. 2003. Endocrine disruptors in the environment: International Union of Pure and Applied Chemistry technical report. *Pure and Applied Chemistry* 75(5): 631–81. Available at: http://old.iupac.org/publications/pac/2003/pdf/7505x0631.pdf.

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Okutman, et al. 2006. Kinetics of the microbial reductive dechlorination of pentachloroaniline. *Environmental Science & Technology* 40(14): 4467–72.

RIVM. 1998. *Maximum Permissible Concentrations and Negligible Concentrations for Aniline Derivations*. Bilthoven, The Netherlands: National Institute of Public Health and the Environment. Report 601501003. See <http://rivm.openrepository.com/rivm/bitstream/10029/10115/1/601501003.pdf>.

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USEPA. 1997. *Reregistration Eligibility Decision (RED) Diflubenzuron*. EPA 738-R-97-008. 214 pp. <http://www.epa.gov/pesticides/reregistration/status.htm>.

WHO. 2003. 4-Chloroaniline. *Concise International Chemical Assessment Document (CICAD)* 48. International Programme on Chemical Safety (IPCS). 62 pp. http://www.who.int/entity/ipcs/publications/cicad/en/cicad48.pdf.

# Chlorobutanes

The four isomers are:

* 1-chlorobutane: CAS No. 109-69-3. Also called n-butyl chloride
* 2-chlorobutane: CAS No. 78-86-4. Also called sec-butyl chloride
* isobutyl chloride: CAS No. 513-36-0. Also called 1-chloro-2-methyl propane
* tert-butyl chloride: CAS No. 507-20-0. Also called 2-chloro-2-methyl propane.

### Maximum Acceptable Value

Chlorobutanes do not have a MAV in the DWSNZ, and are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

1-Chlorobutane is used as a solvent and as an intermediate for the synthesis of catalysts and other organic compounds. n-Butyl chloride was once used as an antihelmintic in veterinary medicine. 1-Chlorobutane can be found in foods wrapped in polyvinylidene chloride casing films.

2-Chlorobutane is used for manufacturing solvents, plasticisers, pesticides and rubber, resins and surfactants and pharmaceuticals.

iso-Butyl chloride is used in the manufacture of medicines, plasticisers, solvents, pesticides raw materials, dewaxing agents, and can be used in the rubber industry.

tert-Butyl Chloride is used in the synthesis of agrochemicals and other organic compounds.

#### 2. From the treatment processes

There do not appear to be any references to any of the chlorobutanes being disinfection by‑products.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

When 1-chlorobutane is released into water, the majority of the chemical is likely distributed into soil and sediment. If released into water, 1-chlorobutane is not readily biodegraded, however it has a very high vapour pressure so will tend to volatilise fairly rapidly. In groundwater, where volatilisation may not occur, n-butyl chloride may be lost by hydrolysis. Based upon hydrolysis rates for alkyl chlorides, the half-life is estimated to be between 6 hr and 38 days at neutral pH. Water solubility of 1‑chlorobutane is about 370 mg/L.

Water solubility of isobutyl chloride is about 30 mg/L.

tert-Butyl chloride breaks down in water to form tert-butyl alcohol (see butanol datasheet).

### Typical concentrations in drinking-water

One water utility in the US reported detecting 1-chlorobutane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.01 mg/L.

### Removal methods

Aeration seems to offer the best option.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

#### 1-Chlorobutane

In a 13-week repeated dose study, mortality and decrease of body weights were observed at the dose of 250 mg/kg/day or more, and these findings might be caused by its irritancy. At the highest dose (500 mg/kg/day), the effects to spleen (eg, hematopoiesis) were also seen. In a preliminary reproductive/developmental toxicity screening test, the external examination of pups revealed depression of viability index and body weight gain at the highest dose (300 mg/kg/day). All gestation animals which delivered pups had lack of care behaviour in the 12 mg/kg/day group. Salivation was observed in the lowest dose group (2.4 mg/kg/day). Therefore, the NOEL was less than 2.4 mg/kg/day for repeated dose toxicity and 60 mg/kg/day for F1 offspring.

In an NTP carcinogenicity assay in rats and mice, 1-chlorobutane showed no evidence of carcinogenicity for male and female rats at doses of 60 or 120 mg/kg/day (UNEP 1997). USEPA (1990a) considered 1-chlorobutane to be Class D: not classifiable as to human carcinogenicity.

#### 2-Chlorobutane

USEPA (1991) considered 2-chlorobutane to be Class D: not classifiable as to human carcinogenicity.

#### tert-Butyl chloride

USEPA (1990a) considered t-butylchloride to be Class D: not classifiable as to human carcinogenicity.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Environmental Working Group (EWG). Accessed 2010. *National Drinking Water Database – Chemical Contaminants*. <http://www.ewg.org/tap-water/chemical-contaminants>.

OECD. 2005. *SIDS Initial Assessment Report*: 1-Chlorobutane. 41 pp. See: http://www.inchem.org/documents/sids/sids/109693.pdf or <http://www.inchem.org/pages/sids.html>.

UNEP. 1997. 1-Chlorobutane. OECD *SIDS Initial Assessment Report*. 41 pp. See: [www.inchem.org/documents/sids/sids/109693.pdf](http://www.inchem.org/documents/sids/sids/109693.pdf).

USEPA. 1990a. 1-Chlorobutane. *Integrated Risk Information System (IRIS)*. See: <http://www.epa.gov/iris/subst/0415.htm>.

USEPA. 1990b. 2-Chlorobutane. *Integrated Risk Information System (IRIS)*. See: <http://www.epa.gov/iris/subst/0416.htm>. See also <http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showSubstanceList>.

USEPA. 1991. tert-Butylchloride. *Integrated Risk Information System (IRIS)*. http://www.epa.gov/iris/subst/0417.htm.

# Chlorodibromoacetic acid

CAS No. 5278-95-5. Chlorodibromoacetic acid is also known as dibromochloracetic acid. Refer also to the haloacetic acids datasheet.

### Maximum Acceptable Value

Chlorodibromoacetic acid does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Chlorodibromoacetic acid (CDBAA), is one of the nine haloacids often analysed in drinking-water, sometimes abbreviated in the US as HAA9. The others are monochloroacetic acid (MCAA), dichloroacetic acid (DCAA), trichloroacetic acid (TCAA), monobromoacetic acid (MBAA), dibromoacetic acid (DBAA) bromochloroacetic acid (BCAA), bromodichloroacetic acid (BDCAA), and tribromoacetic acid (TBAA).

#### 2. From the treatment processes

Chlorodibromoacetic acid does not often appear on lists of disinfection by‑products.

#### 3. From the distribution system

No known sources.

### Typical concentrations in drinking-water

224 water utilities in the US reported detecting chlorodibromoacetic acid in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.042 mg/L.

### Removal methods

Treatment processes that reduce the concentration of natural organic matter normally produce insignificant levels of DBPs; these include chemical coagulation and some membrane systems.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See DWI (2009) which discusses weaknesses and improvements in EPA Method 552.3. DWI (2011) includes a thorough discussion on the analysis of the 9 haloacetic acids.

### Health considerations

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. One of their findings was that a majority of the DBPs tested (75.8 percent) induced significant levels of genomic DNA damage. In this group, iodoacetic acid was the most genotoxic. The least genotoxic was chlorodibromoacetic acid.

### Derivation of Maximum Acceptable Value

No MAV.

### References

DEFRA. 2009. *Review of the Current Toxicological and Occurrence Information Available on Iodinated Disinfection By-Products*. WRc Ref: DEFRA 7883.03 DWI Ref: 70/2/233. 104 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70_2_233.pdf>.

DWI. 2009. *The Formation and Occurrence of Haloacetic Acids in Drinking Water*. DWI 70/2/194. 16 pp. http://www.dwi.gov.uk/research/completed-research/2000todate.htm.

DWI. 2011a. *Evaluation of Haloacetic Acid Concentrations in Treated Drinking Water*. Report No. WT1236. 111 pp. http://dwi.defra.gov.uk/research/completed-research/reports/DWI70\_2\_242.pdf.

DWI. 2011b. *Evaluation of Haloacetic Acid Concentrations in Treated Drinking Water*. Report No. UC8493. 86 pp. http://dwi.defra.gov.uk/research/completed-research/reports/DWI70\_2\_253.pdf.

Environmental Working Group (EWG). Accessed 2010. *National Drinking Water Database – Chemical Contaminants*. <http://www.ewg.org/tap-water/chemical-contaminants>.

Water Research Foundation. 2009. *Mammalian Cell Cytotoxicity and Genotoxicity of Disinfection By-Products*. Project 3089. The summary can be found in: http://www.waterresearchfoundation.org/research/TopicsAndProjects/projectProfile.aspx?pn=3089.

WHO. 2017. *Guidelines for Drinking-water Quality: Fourth edition incorporating the first Addendum*. Geneva: World Health Organization. 631 pp. [http://www.who.int/water\_sanitation\_health/publications/drinking-water-quality-guidelines-4-including-1st-addendum/en/](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\INetCache\Content.Word\www.who.int\water_sanitation_health\publications\2011\dwq_guidelines\en\).

# Chloroethane

CAS No. 75-00-3. Chloroethane is also known as ethyl chloride or monochloroethane, and has been called chlorene.

### Maximum Acceptable Value

Chloroethane does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Chloroethane was used primarily in producing the [petroleum](http://en.wikipedia.org/wiki/Gasoline) additive, [tetra-ethyl lead](http://en.wikipedia.org/wiki/Tetra-ethyl_lead). Some chloroethane is generated as a by‑product of [polyvinyl chloride](http://en.wikipedia.org/wiki/Polyvinyl_chloride) production. The only remaining industrially important use of chloroethane is in treating [cellulose](http://en.wikipedia.org/wiki/Cellulose) to make [ethylcellulose](http://en.wikipedia.org/wiki/Ethylcellulose), a thickening agent and binder in [paints](http://en.wikipedia.org/wiki/Paint), [cosmetics](http://en.wikipedia.org/wiki/Cosmetics), and similar products. Once used as a general anaesthetic.

Chloroethane is not a common surface water pollutant and levels in unfiltered surface water samples typically fall below the detection limit.

#### 2. From the treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

If released to soil, ethyl chloride is expected to have very high mobility based upon an estimated Koc of 24. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 1.11 x 10-2 atm‑cu m/mole. Ethyl chloride will volatilise from dry soil surfaces based upon its vapour pressure. It can leach through subsurface soil where it becomes a potential groundwater contaminant. In groundwater, chloroethane would probably be subject to chemical hydrolysis. Sufficient data are not available to establish the rate of chloroethane degradation in groundwater; it breaks down to ethanol and chloride. It may appear in groundwater as an anaerobic biodegradation product of chlorinated solvents such as 1,l,l-trichloroethane and cis-1,1-dichloroethene. If released into water, ethyl chloride is not expected to adsorb to suspended solids and sediment in water based upon the estimated Koc. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 0.9 hours and 3.2 days, respectively (EAWAG accessed February 2015).

Very soluble in water: about 5,700 mg/L.

### Typical concentrations in drinking-water

Based on the limited amount of information available on the occurrence of chloroethane in drinking-water, it can be concluded that extremely low levels of chloroethane may occur in some drinking-water supplies as a result of its formation during chlorination, due to contamination of rivers and lakes used as drinking-water supplies, or seepage into groundwater resulting from storage of chemical wastes or disposal at waste sites. However, there is not enough information available to indicate what levels of chloroethane occur in drinking-water under these circumstances.

Results of a 1982–1983 survey of 10 Canadian drinking-water supplies suggest that trace levels (<0.1 mg/L) of chloroethane may occur in some finished drinking-water supplies as a result of formation during the chlorination process.

Seventy-five water utilities in the US reported detecting chloroethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.013 mg/L.

### Removal methods

Aeration seems to offer the best option.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Although chloroethane is mutagenic to bacteria, it is the least toxic of the chloroethanes. The IARC considers chloroethane is not classifiable as to its carcinogenicity to humans (Group 3).

Chloroethane is an alkylating agent and is mutagenic to *Salmonella*. Chloroethane has not been shown to cause genotoxic effects in *in* *vivo* assays in mice. Oral data concerning the effects of chloroethane were not identified, therefore, no oral MRLs were derived. ACGIH considers chloroethane to be an animal carcinogen of unknown relevance to humans (ATSDR 1998).

### Derivation of Maximum Acceptable Value

No MAV.

The odour threshold for chloroethane in water is 0.02 mg/L.

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# Chloroform

CAS No. 67-66-3. Chloroform is also known as trichloromethane, trichloroform, methane trichloride, methenyl trichloride, or methyltrichloride.

### Maximum Acceptable Value

Based on health considerations, the concentration of chloroform in drinking-water should not exceed 0.4 mg/L.

In DWSNZ 2005, the MAV had been 0.2 mg/L.

Chloroform is one of the four trihalomethanes with a MAV in the DWSNZ. The others are bromodichloromethane, bromoform and dibromochloromethane. The sum of the ratio of the concentrations of these four trihalomethanes to each of their respective MAVs must not exceed one. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, section 10.2.5.3).

The maximum contaminant level for total trihalomethanes (USEPA 2006/2009/2011) is 0.08 mg/L. The lifetime health advisory for chloroform is 0.07 mg/L (USEPA (2006/2011) where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) do not have an individual guideline value for chloroform; they do though for total trihalomethanes (qv).

Chloroform is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Chloroform was used primarily as the starting material in the manufacture of the refrigerant fluorocarbon-22. It is an important extraction solvent for resins, gums, and other products and therefore may enter raw water as an industrial contaminant. Significant amounts are also produced as by-products in the bleaching of paper pulp. It was once used in cough mixtures and as an anaesthetic during surgery.

#### 2. From the treatment processes

Trihalomethanes, including chloroform, are most likely to be formed as by-products of the chlorination of drinking-water. Chlorine reacts with natural organic materials such as fulvic and humic acids to form chloroform, which is the most common trihalomethane. The amount of chloroform depends on temperature (concentrations of chloroform in chlorinated water in treatment plants and distribution systems are approximately twice as high during warmer months as during colder months), pH, chlorine concentration, the concentration of organic matter, contact time and bromide concentration. Chloroform is usually the most common THM.

#### 3. From the distribution system

No known sources. However, levels can increase as the chlorinated water moves from the water treatment plant through the distribution system due to the continued presence of a chlorine residual reacting with natural organic matter in the water. Further increases in concentrations of chloroform in water can occur in domestic hot water tanks.

### Form and fate in the environment

If released to soil, chloroform is expected to have very high to moderate mobility based upon Koc values of 34–196. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 3.67 x 10-3 atm‑cu m/mole. Chloroform may volatilise from dry soil surfaces based upon its vapour pressure. Under normal environmental conditions, chloroform is not expected to undergo biodegradation in soil. However, several studies have demonstrated that at low concentrations, chloroform can be anaerobically degraded by methanogenic bacteria in the presence of a primary substrate such as acetic acid. If released into water, chloroform is expected to adsorb to suspended solids and sediment based upon the Koc values. Reports of biodegradation of chloroform in aqueous environments have both supported and refuted anaerobic biodegradation. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 3.5 hours and 4.4 days, respectively. BCF values of 2.9–10.35 suggest bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important process based on estimated hydrolysis half-lives of 3,400 and 340 years at pHs 7 and 8, respectively (EAWAG accessed February 2015).

The vapour pressure of chloroform at 20°C has been reported around 210 hPa, and being >0.01 kPa, is considered to be a volatile organic compound. The partition coefficient = logKow = 1.97. Henry’s Law constant = 367 Pa.m3/mol at 25°C (EU 2007).

Chloroform can be photo-oxidised in air, but less likely in water. Chloroform is quite soluble in water (about 7,000–9,000 mg/L). Hydrolysis is not a significant degradation process in water. Volatilisation is the major removal mechanism for chloroform from water and contaminated soil. Biodegradation in surface water is unlikely, in groundwater it can occur but is slow, with a half-life ranging from weeks to years (main metabolite is dichloromethane which persists for at least 21 days; EU 2007). Chloroform does not adsorb very strongly to soil or sediments. Bioaccumulation in aquatic organisms may occur.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-waters between  
1987–1992 contained chloroform results from 370 samples representing 157 chlorinated supplies. Chloroform was detected in 288 samples in concentrations ranging from 0.0004–0.122 mg/L (0.4–122 g/L).

The P2 Chemical Determinand Identification Programme, sampled from 511 zones, found chloroform concentrations to range from “not detectable” (nd) to 0.141 mg/L, with the median concentration being 0.0068 mg/L (limit of detection = 0.004 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with chloroform at greater than the MAV, but four distribution zones supplied 540 people with >50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0005 mg/L chloroform in the raw water, 0.0023 to 0.0027 mg/L in the treated water and up to 0.005 mg/L in the distribution system.

Based on data from eight Canadian provinces, mean chloroform levels for 1994–2000 were generally less than 0.05 mg/L, with some single maximum or peak values in the 0.40 mg/L range.

17,972 water utilities in the US reported detecting chloroform in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.24 mg/L.

### Removal methods

Chloroform present in contaminated source waters (unlikely) can be removed by adsorption on to granular activated carbon, or by air stripping – particularly useful for contaminated groundwaters; see WHO (2005) for further information.

However, as chloroform arises in waters principally as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Trihalomethane concentrations in chlorinated water increase with increasing pH. Concentrations in the finished water can therefore be reduced by ensuring that high pH levels are not present once the water is chlorinated. It is important to ensure that efforts to manage THM levels through adjustment of pH do not increase the formation of haloacetic acids.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Chloroform can be removed by adsorption on to granular activated carbon, or by air stripping.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (APHA 6232B; EPA 551).

### Health considerations

Available studies indicate that gastrointestinal absorption is high for all trihalomethanes and because of their high lipophilicity, accumulation is higher in tissues with high lipid content, including body fat, liver and kidneys.

The use of chloroform as an anaesthetic in dentifrices, liniments, and in anti-tussives has been largely discontinued. Individuals may be exposed during showering to elevated concentrations from chlorinated tap water. Based on estimates of mean exposure from various media, the general population is exposed to chloroform principally in food (approximately 1 μg/kg bw per day), chlorinated drinking-water (approximately 0.5 μg/kg bw per day), and indoor air (0.3 to 1 μg/kg bw per day). Small amounts of chloroform have been found in a variety of foods, with some greater than 0.1 mg/100 kg; the highest concentrations of chloroform have frequently been measured in dairy products (from Health Canada 2006).

Chloroform is a central nervous system depressant. It can also affect the liver and kidney functions. A fatal dose will result in respiratory or cardiac arrest. Workers exposed to chloroform by inhalation at levels between 0.1 and 1.2 g/m3 for one or more years reported symptoms including nausea, lassitude, dry mouth, flatulence, thirst, depression, irritability and the impression of scalding urine. In another incidence, workers inhaling chloroform at similar levels for one to four years had an increased incidence of viral hepatitis and enlarged liver.

The genotoxicity of chloroform has been studied in a wide variety of assays, but the results are inconclusive. The weight of evidence for genotoxicity of chloroform is considered negative. The weight of evidence for liver tumours in mice is consistent with a threshold mechanism of induction. Although it is plausible that kidney tumours in rats may similarly be associated with a threshold mechanism, there are some limitations of the database in this regard. The most universally observed toxic effect of chloroform is damage to the centrilobular region of the liver. The severity of these effects per unit dose administered depends on the species, vehicle and method by which the chloroform is administered.

The results of mutagenicity assays that have been conducted with chloroform are mixed. By number, the majority of tests are negative, and many of the positive studies have been conducted under high exposure conditions that resulted in severe cytotoxicity (USEPA 2002). The weight of the evidence indicates that a mutagenic mode of action via DNA reactivity is not a significant component of the chloroform carcinogenic process. Studies in humans are inadequate to determine if chloroform is carcinogenic.

The International Agency Responsible for Research on Cancer considered that there is sufficient evidence in experimental animals for the carcinogenicity of chloroform, and has classified chloroform as Class 2B (possibly carcinogenic to humans). This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

In the Stage 1 D/DBPR, USEPA finalised an MCLG of zero for chloroform based on a weight of evidence evaluation of both the cancer and non-cancer effects and classified chloroform as a “likely human carcinogen”. The MCLG was based on linear default extrapolation until USEPA completed additional deliberations with the Agency’s Science Advisory Board on the scientific basis of the mode of action for chloroform. At the same time the Agency identified 0.07 mg/L as the MCLG in a situation where a non-linear approach was used in the evaluation of the cancer endpoint. For the Stage 2 D/DBPR, EPA proposed an MCLG for chloroform of 0.07 mg/L and then finalised the MCLG of 0.07 mg/L in 2006 based on the SAB’s conclusions that the nonlinear approach is most appropriate for the risk assessment for chloroform. The MCLG is based on an RfD of 0.01 mg/kg/day, derived using a benchmark dose level (BMDL) of 1.2 mg/kg/day for liver necrosis in dogs with an uncertainty factor of 100, adult tap water consumption of two litres/day for a 70 kg adult and a relative source contribution of 20 percent for drinking water exposure. USEPA concluded that chloroform is “*likely to be carcinogenic to humans”* only under high exposure conditions that lead to cytotoxicity and regenerative hyperplasia and that chloroform is “*not likely to be carcinogenic to humans”* under conditions that do not cause cytotoxicity and cell regeneration. Copied from USEPA (2016).

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 MRLs for chloroform are:

minimal risk level

0.3 mg/kg/day for acute-duration oral exposure (1–14 days)

0.1 mg/kg/day for intermediate-duration oral exposure (15–364 days)

0.01 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD (USEPA 2002 and 2006/2009/2011) for chloroform is 0.01 mg/kg/d, based on a LOAEL of 15 mg/kg/d and an uncertainty factor of 1,000. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.35 mg/L.

### Derivation of Maximum Acceptable Value

It is considered appropriate to use the tolerable daily intake (TDI) approach for calculating the guideline value for chloroform. In a recent assessment of chloroform (IPCS 2004), a dog study was chosen as the most appropriate study for risk assessment. In IPCS (2004), a TDI of 0.015 mg/kg of body weight per day was calculated as follows:

12 mg/L × 2 litres = 0.015 mg/kg of body weight per day

25 x 64

where:

* 12 mg/L is the 95 percent lower confidence limit for the 5 percent incidence of hepatic cysts, generated by PBPK modelling
* 25 is the uncertainty factor (10 for intraspecies differences in toxicokinetics and toxicodynamics and 2.5 for differences in interspecies toxicodynamics)
* 2 litres is the amount of drinking-water consumed per day
* 64 is the body weight in kg of an adult.

Therefore the WHO guideline value was derived as follows:

0.015 x 60 x 0.75 = 0.3375 mg/L

2 L/d

where:

* 0.015 mg/kg of body weight per day is the TDI
* 60 is the body weight in kg of an adult
* 0.75 is the allocation of the total daily intake to drinking-water
* 2 L/d is the drinking-water intake

Recalculating the above using a 70 kg body weight gives a rounded MAV of 0.4 mg/L. The new WHO guideline value represents an increase from their previous guideline value. The change is a result of the increase in the allocation of exposure in drinking-water from 50 percent to 75 percent to account for the fact that chloroform is used less (eg, as an anaesthetic) now than it was in 1993 when the original guideline was developed.

The derivation for the MAV in the 1995, 2000 and 2005 DWSNZ had been based on:

The MAV was based on extrapolation of the observed increase in male rats exposed to chloroform in drinking-water for two years, although it is recognised that chloroform may induce tumours through a non-genotoxic mechanism. Using the linearised multi-stage model, the concentration of chloroform in drinking-water which corresponds to an excess lifetime risk of one additional cancer per one hundred thousand (10-5) is a concentration of 0.2 mg/L.

That MAV was supported by a 7.5-year study in dogs, in which the MAV for chloroform in drinking-water was derived as follows:

15 x (6/7) mg/kg body weight/day x 70 kg x 0.5 = 0.225 mg/L (rounded to 0.2 mg/L)

2 L x 1000

where:

* lowest-observable-adverse-effect level = 15 mg/kg body weight per day, based on slight hepatotoxicity (increases in hepatic serum enzymes and fatty cysts) observed in beagle dogs ingesting 15 mg of chloroform per kg of body weight per day in toothpaste for 7.5 years (normalised for 6 days/week dosing)
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.5 based on estimates indicating that the general population is exposed to chloroform principally in food, drinking-water and indoor air in approximately equivalent amounts and that most of the chloroform in indoor air is present as a result of volatilization from drinking-water
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for evidence of possibly non-genotoxic carcinogenicity). No additional factor for the short duration of the study was incorporated. It was considered to be unnecessary because the compound was administered in corn oil in the critical study and available data indicate that the toxicity following administration in water may be an order of magnitude less.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The short-term, chronic and subchronic limits are 0.03 mg/L.

The WHO guideline value only considers exposure to THMs in water via the oral route. However, WHO accepts THMs are volatile chemicals, and therefore, exposure via the inhalation and dermal routes may be significant sources of exposure, particularly during bathing and showering, as increasing water temperature will increase the rate of volatilisation, and ventilation may be poor. WHO suggested that in colder countries with low rates of ventilation in houses or where the incidences of showering and bathing are high, this guideline value may be lowered (WHO 2005, in DWI 2010).

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# Chloromethane

CAS No. 74-87-3. Also called methyl chloride and monochloromethane.

### Maximum Acceptable Value

There is no MAV for chloromethane in the DWSNZ, and chloromethane is not mentioned in the WHO Guidelines.

The USEPA concluded on 22 September 2009 that chloromethane is known or anticipated to occur in PWSs and may require regulation. Therefore they added chloromethane to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a). Chloromethane is on the USEPA List of Priority Pollutants.

The USEPA (2006) established a lifetime health advisory of 0.03 mg/L, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity. This lifetime health advisory does not appear in USEPA (2011).

### Sources to drinking-water

#### 1. To source waters

Chloromethane is produced in industry, but it also occurs naturally, and most of the chloromethane that is released to the environment (estimated at up to 99 percent) comes from natural sources, the atmospheric burden being produced in oceans or by fires involving wood or other biomass. Chloromethane is always present in the air at very low levels. Most of the naturally occurring chloromethane comes from chemical reactions that occur in the oceans or from chemical reactions that occur when materials like grass, tobacco, wood, charcoal, and coal are burned. It is also released to the air as a product of some plants or from rotting wood, and from volcanoes.

Today, nearly all commercially produced chloromethane is used to make other substances, mainly silicones (72 percent of the total chloromethane used). Other products that are made from reactions involving chloromethane include agricultural chemicals (8 percent), methyl cellulose (6 percent), quaternary amines (5 percent), and butyl rubber (3 percent). Chloromethane is completely used up so that by the end of the process there is no or little chloromethane left to be released, disposed of, or reused. It is, however, found as a pollutant in municipal waste streams from treatment plants and industrial waste streams as a result of formation or incomplete removal. There are also some manufacturing processes for vinyl chloride that result in chloromethane as an impurity in the vinyl chloride end product.

Chloromethane can dissolve in water, and small amounts of chloromethane in air may go into surface waters or groundwater when it rains.

#### 2. From treatment processes

Chloromethane may be formed to a small extent in tap water that has been chlorinated (ATSDR 1998).

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Chloromethane breaks down very slowly (months to years) in sterile water, but some micro-organisms may break it down more quickly (in days). It does not stick to the soil. Most of the chloromethane in soil will volatilise to the air. The volatilisation half-life has been calculated to be 2.1 hours in a model river. The volatilisation half-lifes of methyl chloride in a pond and in a lake have been estimated to be 25 hours and 18 days respectively. Some may dissolve in water and move down to the groundwater where it has an estimated half-life of about four years. Chloromethane does not concentrate in sediments, or in animals and fish in the food chain.

If released to soil, methyl chloride is expected to have very high mobility based upon an estimated Koc of 14. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 8.82 x 10-3 atm‑cu m/mole. Methyl chloride may volatilise from dry soil surfaces based upon its vapour pressure. Field and laboratory results demonstrate that several halogenated aliphatics may biodegrade slowly under anaerobic conditions, but not under aerobic conditions. If released into water, methyl chloride is not expected to adsorb to suspended solids and sediment in water based upon the estimated Koc. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 46 minutes and three days, respectively. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low (EAWAG accessed February 2015).

Water solubility has been reported variously from about 4,000 to 9,000 mg/L. It decomposes to methanol and hydrogen chloride in water (USEPA 2001).

### Typical concentrations in drinking-water

Tests reported by 25,761 US public water suppliers in 38 states shows that between 1998 and 2003, people in 977 communities drank water containing chloromethane. The highest concentration was 0.135 mg/L, which is above the 0.03 mg/L level, the concentration in drinking-water that the USEPA does not expected to cause any adverse, non-carcinogenic health effects for a lifetime of exposure. The lifetime health-based limit (or Health Advisory) is based on exposure for a 70‑kg adult consuming two litres of water per day (EWG 2008).

742 water utilities in the US reported detecting chloromethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.04 mg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,848 drinking water samples for chloromethane between 2013 and 2015, and found 283 samples exceeded the minimum reporting level (MRL) of 0.2 µg/L.

### Removal methods

Aeration appears to offer the best option.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Because chloromethane evaporates quickly, it is usually not found in food, surface water, or soil, thus the main intake route for humans is by respiration. The air in some cities may contain up to about 1 to 2 ppb mainly due to releases from combustion.

Male mice that breathed air containing chloromethane (one million ppb) for two years developed tumours in their kidneys, but female mice and male and female rats did not develop tumours. It is not known whether chloromethane can cause sterility, miscarriages, birth defects, or cancer in humans. The Department of Health and Human Services has not classified chloromethane for carcinogenic effects. The International Agency for Research on Cancer (IARC) calls chloromethane a Group 3 compound, which means it cannot be determined whether or not it is a carcinogen because there is not enough human or animal data.

USEPA (2001) stated that the few studies that have examined methyl chloride’s potential carcinogenicity in humans have failed to demonstrate any association, and in one instance even indicated a lower cancer incidence than expected in workers chronically exposed to methyl chloride. Earlier the USEPA had considered chloromethane possibly carcinogenic to humans (ie, Group C) based on limited evidence of carcinogenicity in animals.

No association between occupational exposure to chloromethane and pancreatic cancer was found, but chloromethane produced chromosome aberrations in cultured mammalian cells. Cigarette smoke emits four magnitudes of chloromethane concentrations more than is typically found in the urban environment, and exposed cigarette smokers and those passively exposed to the smoke are potentially exposed to greater amounts of chloromethane than the otherwise general population (ATSDR 1998).

Chloromethane is not likely to cause cancer, birth defects or other reproduction problems at normally encountered exposure levels or at reasonably anticipated higher exposure levels. This conclusion is based on an integration of the voluminous toxicity data developed over the past decades and human experiences for well over 80 years of industrial use (OECD 2004).

Methyl chloride is genotoxic in in vitro systems in both bacteria and mammalian cells. Although the positive effects seen in a dominant lethal test most likely were cytotoxic rather than genotoxic, methyl chloride might be considered a very weak mutagen in vivo based on some evidence of DNA–protein crosslinking at higher doses (WHO 2001).

The reference dose or RfD (USEPA 2006/2009) is 0.004 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009) is 0.1 mg/L; these do not appear in USEPA (2011).

### Derivation of Maximum Acceptable Value

No MAV.

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# 4-chloro-3-methylphenol

There is a large number of chlorinated methylphenols (ie, chlorinated cresols), including the following 10 possible monochloro-monomethyl compounds:

|  |  |  |
| --- | --- | --- |
| **Chlorinated methylphenol** | **Chlorinated cresol** | **CAS no.** |
| 2-chloro-3-methylphenol | 2-chloro-3-cresol | 608-26-4 |
| 2-chloro-4-methylphenol | 2-chloro-4-cresol | 6640-27-3 |
| 2-chloro-5-methylphenol | 2-chloro-5-cresol | 615-74-7 |
| 2-chloro-6-methylphenol | 2-chloro-6-cresol | 87-64-9 |
| 3-chloro-2-methylphenol | 3-chloro-2-cresol | 3260-87-5 |
| 3-chloro-4-methylphenol | 3-chloro-4-cresol | 615-62-3 |
| 3-chloro-5-methylphenol | 3-chloro-5-cresol | 58291-77-3 |
| 3-chloro-6-methylphenol | 3-chloro-6-cresol | 5306-98-9 |
| 4-chloro-2-methylphenol | 4-chloro-2-cresol | 1570-64-5 |
| 4-chloro-3-methylphenol | 4-chloro-3-cresol | 59-50-7. This is one of the “priority pollutants” under the US Clean Water Act, under the name of 4‑chloro-m-cresol or chlorocresol. |

#### Synonyms

4-chloro-2-methylphenol has been called 2-methyl-4-chlorophenol, PCOC, 4-chloro-o-cresol and p-chloro-o-cresol (PCOC), or even chlorocresol sometimes.

4-chloro-3-methylphenol (one of the commoner chlorinated methylphenols) is also known as 3-methyl-4-chlorophenol, p-chloro-m-cresol (PCMC), 4-chloro-1-hydoxy-3-methyl benzene, 2-chloro-5-hydroxytoluene and CMK.

2-chloro-5-methylphenol can be called 6-chloro-3-methylphenol, 3-methyl-6-chlorophenol and 6-chloro-m-cresol.

Dichloro- and trichloro-compounds also exist, as do chlorinated dimethylphenols (the dimethylphenols are called xylenols, of which there are six isomers).

### Maximum Acceptable Value

There are no MAVs in the DWSNZ and WHO does not refer to chlorinated methylphenols or chlorinated cresols.

The New Jersey Department of Environmental Protection has established an interim groundwater quality criterion of 0.1 mg/L for 4-chloro-3-methylphenol.

The Danish EPA (2001) has set drinking-water limits for 2-chloro-6-methylphenol, 4‑chloro-2-methylphenol, and 4,6-dichloro-2-methylphenol of 0.0001 mg/L.

### Sources to drinking-water

#### 1. To source waters

The EU tonnage of 4-chloro-2-methylphenol (PCOC) for 1989 has been estimated as a total of 15,000 tons per annum based on the production volumes presented by the manufacturers and supported by the production and consumption figures of the phenoxyherbicides MCPA (4-chloro-2-methylphenoxy acetic acid), MCPB (4-chloro-2-methylphenoxy butyric acid) and MCPP (mecoprop 2,4-chloro-2-methylphenoxy-propionic acid). PCOC is their main degradation product.

More than 50 percent of the production volume of 4-chloro-3-methylphenol is used in metal working fluids. These fluids, used to lubricate and cool during metal grinding or in plant machinery, are rich in proteins which provide a source of nutrition for bacterial growth. The other major use is as a pharmaceutical preservative. For example, hand and body creams containing organic compounds in an aqueous phase may contain 4‑chloro-3-methylphenol to prevent micro-organisms degrading the ingredients. 4‑Chloro-3-methylphenol has been used as a fungicide and preservative for raw leathers, glues, gums and paints. Chlorocresol appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 as an ectoparasiticide (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). 3-Methyl-6-chlorophenol may be generated as an impurity during the production of 3-methyl-4-chlorophenol.

Mecoprop is broken down in soil to 4-chloro-2-methylphenol, with a half-life of  
7–9 days (WHO 2003).

Chlorinated cresols and chlorinated xylenols (eg, ‘Dettol’) are still commonly used as or in antiseptics. The main active ingredient in Dettol is actually [4-chloro-3,5-dimethylphenol](http://www.chm.bris.ac.uk/motm/dettol/cs.htm). Chlorinated cresols (usually 4-chloro-2-methylphenol) appear as a constituent of products such as Jeyes Fluid.

#### 2. From the treatment processes

Degradation of humic matter can produce complex phenols. Chlorination of potable water may result in inadvertent formation of small amounts of 4-chloro-3-methylphenol, and possibly some other chlorinated methylphenols. 3-Methyl-6-chlorophenol may be formed in chlorinated waters from the reaction of hypochlorite with phenolic impurities.

### Form and fate in the environment

4-Chloro-2-methylphenol: this substance is very toxic to aquatic organisms. The available biodegradation data are somewhat conflicting but based on a judgement of the balance of evidence the “realistic worst case” aerobic biodegradation half-life of PCOC in soil is estimated to be 21 days, whereas no biodegradation has been found under anaerobic conditions. The aerobic biodegradation half-life in surface waters is also estimated to be 21 days. The chemical has a low bioaccumulative potential. The predicted environmental concentrations are lower than the predicted no-effect levels for all environmental compartments. It is currently considered of low potential risk and low priority for further work (Inchem 1998). Water solubility 2300 mg/L at 20°C (OECD 2002). EU (2002) quotes: vapour pressure = 26.7 Pa at 20°C; partition coefficient = log Kow = 3.09; PCOC in water is not readily biodegradable; PCOC was resistant to chemical or biologically mediated changes (no abiotic or biotic degradation) during anaerobic degradation at 10 μg/L. PCOC is expected to adsorb to sediments and particulate materials in the water column depending on the pH.

4-Chloro-3-methylphenol: degradation in the atmosphere is rapid (days, by reaction with hydroxyl radicals). Releases to land are expected to biodegrade in soil but significant leaching may occur and if it reaches groundwater then it may be relatively persistent there; 4-chloro-3-methylphenol may biodegrade in water systems but detailed information is lacking (EA 2008). Water solubility is about 3800 mg/L. EC (2002a) states that 4-chloro-3-methylphenol is readily biodegradable, and does not strongly sorb to organic carbon in sediments and soils based on a low organic carbon water partition coefficient (log Koc = 1.25–1.7). Also, 4-chloro-3-methylphenol is degraded in soil with a half life of 1.4 to 21 days. Volatilisation is unlikely to represent a major removal process from the aquatic environment based on the Henry’s Law Constant of 4.52 x 10-2 to 2.72 x 10-1 Pa-m3 mol-1 (4.58 x 10-7 to 2.76 x 10-6 atm-m3 mol-1).

If released to soil, 3-methyl-6-chlorophenol is expected to have moderate mobility based upon the measured Koc values of structurally similar compounds ranging from 124 to 645, with most of these values falling within the Koc range for moderate mobility. Volatilisation from moist soil surfaces is expected to be a slow environmental fate process based upon an estimated Henry’s Law constant of 4.6 x 10-7 atm‑cu m/mole. 3-Methyl-6-chlorophenol is not expected to volatilise from dry soil surfaces based upon its vapour pressure. 3-Methyl-6-chlorophenol is expected to biodegrade in soil based on a half life of 2.1 days in basic sandy silt loam. If released into water, 3-methyl-6-chlorophenol is expected to adsorb very little to suspended solids and sediment based upon the measured Koc values of structurally similar compounds. 3-Methyl-6-chlorophenol is not expected to biodegrade in the aquatic environment based on a repetitive die-away closed bottle test using activated sludge that showed no biodegradation after 28 days. Volatilisation from water surfaces is expected to be an important fate process based upon this compound’s estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 96 days and 700 days, respectively. Water solubility is >1 mg/L. (NIH, accessed February 2016).

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

4-Chloro-3-methylphenol is most often measured using GC/ECD, FID and GC/MS.

### Health considerations

Technical p-chloro-m-cresol (99.90–99.97 percent) was tested in a chronic feeding/carcinogenicity study in male and female Wistar rats for 24 months. The systemic NOEL is less than 28 mg/kg/d based on poor general condition, decreased body weight and food efficiency, increased water intake, decreased urinary protein, changes in organ weights and histopathology of the kidney and brain (USEPA 1996). The most important source of direct exposure (in New Zealand conditions) is assumed to be use of phenoxy herbicides (containing PCOC as an impurity or breakdown product) where exposures of ca. 0.35 mg/kg/day may occur (OECD 2002).

4-Chloro-3-methylphenol was formerly used as a preservative and antifungal agent in eye drops. USEPA (2004) states: Since 4-chloro-3-methylphenol is not indicated to be present in effluent or ambient surface water monitoring data throughout West Virginia, the chemical has not been shown to be present in surface waters at levels above action levels that would be protective of human health, ie, 3 mg/L to protect from undesirable taste and odor problems; 5.25 mg/L to protect against non-cancer effects associated with “water + organism” exposure; and 26.6 mg/L to protect against non-cancer effects associated with “organism only” exposure.

The USEPA has determined that p-chloro-m-cresol is not classifiable as a human carcinogen, ie, Group D.

4-Chloro-3-methylphenol is on the EC List (Annex 15) of 66 Category 1 substances showing evidence of endocrine disrupting activity in at least one species using intact animals (EC 2002a).

### Derivation of Maximum Acceptable Value

No MAV.

Young et al (1996) report the 2-chloro-4-methylphenol taste threshold in drinking-water to be <0.00005 mg/L, and the odour threshold to be 0.00015 mg/L.

Young et al (1996) report the 4-chloro-2-methylphenol taste threshold in drinking-water to be <0.0025 mg/L, and the odour threshold to be 0.062 mg/L. The USEPA established an organoleptic effect criterion of 1.8 mg/L for 2-methyl-4-chlorophenol. Source: [Quality Criteria for Water, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

The USEPA established an organoleptic effect criterion of 0.02 mg/L for 3-methyl-6-chlorophenol. Source: [Quality Criteria for Water, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

Young et al (1996) report the taste and odour thresholds of 4-chloro-3-methylphenol in drinking-water to be as low as 0.002–0.003 mg/L. The USEPA established an organoleptic effect criterion of 3.0 mg/L for 3-methyl-4-chlorophenol. Source: [Quality Criteria for Water, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

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# 2-chloronaphthalene

2-Chloronaphthalene, CAS No. 91-58-7, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs.

Commercial polychlorinated naphthalenes (PCNs) are mixtures of up to 75 chlorinated naphthalene congeners plus by‑products and are often described by the total fraction of chlorine. They have been given a general CAS No. of 70776-03-3. Polychlorinated naphthalenes were added to the list of Stockholm Convention Persistent Organic Pollutants (POPs); <http://chm.pops.int/>. Therefore this datasheet discusses more than just 2-chloronaphthalene.

WHO (2001) discusses properties of 77 different chloronaphthalenes. NICNAS (2002) discusses eight groups of polychlorinated naphthalenes, from mono- to octachloronaphthalene. Sometimes 2-chloronaphthalene is called beta-chloronaphthalene. Refer also to the polynuclear aromatic hydrocarbons datasheet.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

Most industrially produced PCNs were mixtures of several isomers. Production was common in the early 20th century but fell off in the late 1970s. PCNs were contaminants in PCBs. They also result from various heating and burning processes; NICNAS (2002).

Monochloronaphthalenes and mixtures of mono- and dichloronaphthalenes have been used for chemical-resistant gauge fluids and instrument seals, as heat exchange fluids, as high boiling speciality solvents, for colour dispersions, as engine crankcase additives, and as ingredients in motor tune-up compounds. Monochloronaphthalenes have also been used as a raw material for dyes and as a wood preservative with fungicidal and insecticidal properties. 2-Chloronaphthalene has been detected in fly ash from municipal incinerators in the USA at levels up to 3 μg/kg.

Polychlorinated naphthalenes have been detected at 19 Finnish plywood plants. The source was the pesticide Basileum SP-70, which contains approximately 80 percent PCNs (mainly mono- and dichlorinated isomers) and 4 percent tributyltin oxide. The pesticide was mixed into the glues used to make the plywood, and monochloronaphthalene and dichloronaphthalene concentrations of 0.2–8 mg/m3 were detected in the glueing department (WHO 2001). PCNs make effective insulating coatings for electrical wires.

A polychloroprene polymer has been reported to comprise 3 percent trichloronaphthalene, 1 percent tetrachloronaphthalene, and 0.2 percent pentachloronaphthalene. Production ceased in 2002 (NICNAS 2002).

#### 2. From treatment processes

Levels of chloronaphthalene and dichloronaphthalene have been measured in two samples of Tsukuba (Japan) tap water after chlorination. The detection limit in the experiment was 0.003 ng/L, and the levels of both chloronaphthalene and dichloronaphthalene were below the detection limit in the raw water before chlorination. After chlorination, levels of 0.03–0.44 ng/L for chloronaphthalene and levels of not detected to 0.15 ng/L for dichloronaphthalene were measured (WHO 2001).

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

Monochloronaphthalenes appear to be readily degradable by soil and water micro-organisms under aerobic conditions (WHO 2001). Water solubility of 2‑chloronaphthalene is about 0.9 mg/L. The Log octanol/water partition coefficient is about 4.

Half-lifes for degradation of 2-chloronaphthalene by soil micro-organisms of 38 days in waste sludge, 59 days in slop oil sludge, and 70–104 days in wood preserving sludge were obtained (from WHO 2001).

Water solubility decreases as the number of chlorine atoms increases; monochloronaphthalenes from 0.1 to 0.3 mg/L, to 0.00008 mg/L for octachloronaphthalene.

### Typical concentrations in drinking-water

A single study on chlorinated tap water revealed chlorinated naphthalene concentrations of up to 0.15 ng dichloronaphthalene/litre and up to 0.44 ng monochloronaphthalene/litre (WHO 2001).

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

### Analytical methods

Refer also to the polynuclear aromatic hydrocarbons datasheet, and WHO (2001).

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

The USEPA (1990) quoted a reference dose or RfD of 0.08 mg/L for beta-chloronaphthalene, the critical effect being dyspnea, abnormal appearance, liver enlargement based on a mouse subchronic oral gavage study. The NOAEL was 250 mg/kg/d; LOAEL 600 mg/kg/d.

### Derivation of Maximum Acceptable Value

No MAV.

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# 3-chlorophenol

Note: 2-chlorophenol has a GV in the DWSNZ, so its datasheet appears in Part 2.5.

CAS No. 108-43-0. Also called 3-monochlorophenol, meta-chlorophenol, m-chlorophenol, 1-chloro-3-hydroxybenzene or 3-hydroxy-chlorobenzene. Sometimes spelt chlorphenol.

### Maximum Acceptable Value

There is no MAV in the DWSNZ; WHO does not mention 3-chlorophenol.

### Sources to drinking-water

#### 1. To source waters

3-Chlorophenol is manufactured in much smaller quantities than 2-chlorophenol and 4-chlorophenol.

#### 2. From treatment processes

Chlorophenols are most likely to occur in drinking-water as disinfection by-products through the reaction of naturally-occurring organic matter with chlorine, or due to the chlorination of water containing phenolic compounds from industrial discharge.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Aerobic micro-organisms in clay loam soils were able to degrade more than 70 percent of the 3-chlorophenol present (100 mg/kg) within 80 to 100 days (IPCS 1989).

Water solubility is about 2.6 percent.

### Removal methods

As this compound probably arises in New Zealand waters principally during water treatment as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/ flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products. The formation of chlorophenols can be reduced by the use of chlorine dioxide in place of chlorine.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Chlorophenols can be removed by adsorption on to activated carbon. The effectiveness of the processes is pH dependent. Greater adsorption occurs as the pH is lowered.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

### Analytical methods

#### Referee method

A referee method cannot be selected for 3-chlorophenol because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for 4-chlorophenol for the above reason. However, the following information may be useful:

1. Chlorophenols in water can be solvent extracted with dichloromethane (Method APHA 6410) and analysed by gas chromatography with mass spectrometry detection (Method APHA 6410 or EPA 8270). The detection limit for this method is 0.003 mg/L (3 g/L). Interference may come from contaminated reagents or glassware.

2. A more sensitive and specific method of analysis for chlorophenols is to solvent extract with dichloromethane and derivatise with pentafluorobenzyl ether and analyse by gas chromatography with electron capture detection (Method EPA 604 or APHA 6420B). The limit of quantification for this method is 0.0006 mg/L (0.6 g/L). The specificity of this method reduces the likelihood of interferences.

### Health considerations

Chlorophenols are well-absorbed after oral administration and they readily penetrate the skin. Chlorophenols do not appear to accumulate in body tissues in rats but are rapidly metabolised and eliminated from the body, principally in urine. Exposure to chlorophenols via tap water has been estimated to be less than 10 percent of total dietary exposure. The three monochlorophenols are expected to have similar toxicity characteristics.

IARC considers chlorophenols as a group to have limited evidence for human carcinogenicity (Group 2B).

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA established an organoleptic effect criterion of 0.0001 mg/L for 3‑chlorophenol. Source: [Quality Criteria for Water, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

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# 4-chlorophenol

Note: 2-chlorophenol has a GV in the DWSNZ, so its datasheet appears in Part 2.5.

CAS No. 106-48-9. Also called 4-monochlorophenol, para-chlorophenol, p‑chlorophenol or 4-hydroxy-chlorobenzene. Sometimes spelt chlorphenol.

### Maximum Acceptable Value

There is no MAV in the DWSNZ; WHO does not mention 4-chlorophenol.

### Sources to drinking-water

#### 1. To source waters

4-Chlorophenol may occur in raw water as an industrial contaminant, or through agricultural activity or residues where it was once used as a pesticide. It may be used as a precursor for the production of higher chlorophenols and dyestuffs. Chlorophenols are used commercially as preservatives, moth-proofing agents, germicides and anti-mildew agents. Also, chlorophenols have been used as antiseptics; 4-chlorophenol is still used as a dental antiseptic.

#### 2. From treatment processes

Chlorophenols are most likely to occur in drinking-water as disinfection by-products through the reaction of naturally-occurring organic matter with chlorine, or due to the chlorination of water containing phenolic compounds from industrial discharge.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Because 4-chlorophenol is water-soluble (about 2 – 3 percent), weakly acidic, and has a low vapour pressure, it is anticipated that volatilisation does not play a significant role in removing it from water. Photolytic breakdown of dilute solutions of monochlorophenols has been reported. Sorption is not significant for monochlorophenols. Biodegradation appears to be the primary removal mechanism of chlorinated phenols from surface waters. Aquatic biota may bioconcentrate chlorinated phenols with bioconcentration factors increasing with increasing chlorine substitution.

Aerobic micro-organisms in clay loam soils were able to degrade most of the 4‑chlorophenol present (100 mg/kg) within a few days (IPCS 1989).

If released to soil, 4-chlorophenol is expected to have very high to moderate mobility based upon a Koc range of 70 to 485.6. The pKa of 4-chlorophenol is 9.41, indicating that it will partially exist in the anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Mobility of 4-chlorophenol may also be affected by the amount of soil organic matter present as it will tend to irreversibly absorb. Volatilisation from moist soil surfaces is expected to be an important fate process for the neutral species based upon a Henry’s Law constant of 6.3 x 10-7 atm‑cu m/mole. 4-Chlorophenol is not expected to volatilise from dry soil surfaces based upon its vapour pressure. 4‑Chlorophenol degraded in clay and silt loams soils from 84-100 percent in 3-16 days and 22.2 percent and 35 percent in 1 and 10 weeks respectively in para-brown soil, suggesting that biodegradation of 4-chlorophenol in soils is variable depending on conditions. There may be decreased availability for biodegradation due to the potential of 4-chlorophenol to irreversibly absorb to soil organic matter. If released into water, 4‑chlorophenol may exhibit low to moderate absorption to suspended solids and sediment based on the Koc range. Biodegradation of 4-chlorophenol in water varies depending on the conditions. Complete removals have been reported in water after 13 days for acclimated water and 30 days in farm stream sediment, 44 percent degradation after five days and 33 percent after 25 days in non-acclimated water. A pKa of 9.41 indicates 4-chlorophenol will exist partially in the anion form at pH values of 5 to 9 and therefore volatilisation from water surfaces is not expected to be an important fate process (EAWAG accessed February 2015).

### Removal methods

As this compound arises in New Zealand waters principally during water treatment as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products. The formation of chlorophenols can be reduced by the use of chlorine dioxide in place of chlorine.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Chlorophenols can be removed by adsorption on to activated carbon. The effectiveness of the processes is pH dependent. Greater adsorption occurs as the pH is lowered.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

### Analytical methods

#### Referee method

A referee method cannot be selected for 4-chlorophenol because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for 4-chlorophenol for the above reason. However, the following information may be useful:

1. Chlorophenols in water can be solvent extracted with dichloromethane (Method APHA 6410) and analysed by gas chromatography with mass spectrometry detection (Method APHA 6410 or EPA 8270). The detection limit for this method is 0.003 mg/L (3 g/L). Interference may come from contaminated reagents or glassware.

2. A more sensitive and specific method of analysis for chlorophenols is to solvent extract with dichloromethane and derivatise with pentafluorobenzyl ether and analyse by gas chromatography with electron capture detection (Method EPA 604 or APHA 6420B). The limit of quantification for this method is 0.0006 mg/L (0.6 g/L). The specificity of this method reduces the likelihood of interferences.

### Health considerations

Chlorophenols are well-absorbed after oral administration and they readily penetrate the skin. Chlorophenols do not appear to accumulate in body tissues in rats but are rapidly metabolised and eliminated from the body, principally in urine. Exposure to chlorophenols via tap water has been estimated to be less than 10 percent of total dietary exposure. The three monochlorophenols are expected to have similar toxicity characteristics.

IARC considers chlorophenols as a group to have limited evidence for human carcinogenicity (Group 2B).

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.01 mg/kg/day for acute-duration oral exposure  
(1–14 days) to 4-chlorophenol.

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA established an organoleptic effect criterion of 0.0001 mg/L for 4‑chlorophenol. Source: [Quality Criteria for Water, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>

4-Chlorophenol has a higher taste and odour threshold than 2-chlorophenol: about 0.04 and 0.01 mg/L respectively (Young et al 1996).

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# Chloropicrin

CAS No. 76-06-2. The IUPAC and CAS name is trichloronitromethane. Also called nitrochloroform. Refer also to the halonitromethanes datasheet and DWI (2010).

### Maximum Acceptable Value

There are insufficient data to derive a MAV for chloropicrin in drinking-water.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set a guideline value for chloropicrin in drinking water.

### Sources to drinking-water

#### 1. To source waters

Chloropicrin may enter raw water as an industrial contaminant. It may be used as a reagent in the synthesis of organic chemicals, in the manufacture of methyl violet, as a fumigant (fungicide, nematicide and insecticide) for soil and stored grain.

Chloropicrin appears as a nematicide, fungicide, herbicide, insecticide and vertebrate toxic agent on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). Strawberry growers in New Zealand use it. It is sometimes been used with methyl bromide. The use of chloropicrin as a pesticide is no longer authorised within the EU.

#### 2. From treatment processes

Chloropicrin can be formed in water by the reaction of chlorine with humic acids, amino acids, and nitrophenols. The presence of nitrate increases the formation of chloropicrin.

Chloropicrin (trichloronitromethane) undergoes a slow degradation in the pH range of 6.1 to 8.5. Chloropicrin is rapidly reduced in the presence of pipe corrosion solids, and dissolved oxygen slows the reaction. The reaction rate was most dependent on water-soluble iron content. For most of utilities, there is an apparent linear relationship between chloropicrin and chloroform concentration. This suggests, chloropicrin, like chloroform, does not undergo significant degradation in distribution systems, and that it continues to form, as chloroform does. However, the slope of chloropicrin versus chloroform line varies a substantially between utilities. A higher slope and a higher formation of chloropicrin was observed at utilities using chloramine as final disinfectant. In general, the chloropicrin concentration increased with increasing water age; in one case it doubled through the system. WRF (2016).

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

The half-life of chloropicrin in sandy loam soil was 8 – 24 hours. Chloropicrin moves rapidly in soils within twelve inches of injection but may diffuse to a maximum depth of four feet in sandy soil. NSW Government (2013) reports that chloropicrin degrades relatively quickly in soils mainly due to microbial activity. The degradation is accelerated as soil temperature increases, but is relatively independent of changes in soil moisture. Under aerobic conditions the final product is carbon dioxide, in anaerobic conditions chloropicrin is converted to nitromethane.

Chloropicrin in water is reduced to chloroform when reducing agents are added to the water eg, to remove excess chlorine. The metabolite dichloronitromethane presents a risk of groundwater contamination.

The half-life of chloropicrin in water exposed to light was 31 hours with carbon dioxide, bicarbonate, chloride, nitrate and nitrite being the breakdown products.

Water solubility is about 600 mg/L.

NPIC (1994) quotes for chloropicrin a soil half-life of 1 day, water solubility of 2270 mg/L and a sorption coefficient (soil Koc) of 62. This resulted in a pesticide movement to groundwater rating of extremely low.

### Typical concentrations in drinking-water

No data are available on the concentration of chloropicrin in New Zealand drinking-water supplies. In a US study of 36 supplies suspected of having high disinfection by-product levels, the highest concentration of chloropicrin measured was 0.0056 mg/L (5.6 g/L).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0003 mg/L chloropicrin in the raw water, treated water and distribution system.

In a sampling of 1,386 wells in California between 1984 and 1989, no chloropicrin was detected. In a sampling of 15,175 wells in Florida, chloropicrin was found in three wells at 0.035–0.068 mg/L.

### Removal methods

No information is available on methods to remove chloropicrin from contaminated source waters.

As chloropicrin arises in water principally as a disinfection by-product, the preferred method for minimising its concentration is to reduce the formation of natural organic matter (NOM) coming into contact with the chlorine. The presence of nitrate also increases its formation. Therefore the removal of nitrate (difficult), or the selection of a source with low nitrate concentrations will help to reduce chloropicrin formation.

Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

### Analytical methods

#### Referee method

A referee method cannot be selected for chloropicrin because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for chloropicrin because a MAV has not been established. However, the following information may be useful.

APHA (2005) states that chloropicrin in water may be analysed by solvent extraction with methyl tert-butyl ether (MTBE) and analysis by gas chromatography with electron capture detection (Method EPA 551 1990). The limit of determination is approximately 0.00002 mg/L (20 ng/L).

### Health considerations

No long-term data are available on health effects in humans.

Decreased survival and body weights have been reported following long-term oral exposure in laboratory animals. Chloropicrin has been shown to be mutagenic in bacterial tests and in *in vitro* assays in lymphocytes. Because of the high mortality in a carcinogenesis bioassay and the limited number of end-points examined in the 78‑week toxicity study, the available data were considered inadequate to permit the establishment of a guideline value for chloropicrin.

Studies investigating the chronic toxicity of chloropicrin in mice and rats found that there was an association between high dose levels (46 and 50 mg/kg body weight per day respectively) and accelerated mortality. EU (2011) reports an ADI and ARfD of 0.001 mg/kg/d. Reaffirmed in EFSA (2013).

In humans, inhalation of chloropicrin at 0.002 mg/L for one minute caused pulmonary effects. Chloropicrin was used in [World War I](http://en.wikipedia.org/wiki/World_War_I) as a [chemical weapon](http://en.wikipedia.org/wiki/Chemical_weapon).

The Acceptable Daily Intake (ADI) adopted in Australia for chloropicrin is 0.001 mg/kg body weight, with a NOEL of 0.1 mg/kg bw. There is no ARfD.

Studies to date do not permit an evaluation of the carcinogenicity of chloropicrin because of the short survival time of dosed animals. Chloropicrin exhibited mutagenic activity in some tests with bacteria, and with human lymphocytes *in vitro*.

### Derivation of Maximum Acceptable Value

The are insufficient data to derive a MAV for chloropicrin at this time.

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# Chloroprene

CAS No. 126-99-8. Also called 2-chloro-1,3-butadiene, 2-chlorobutadiene, β‑chloroprene, or 2-chlorobuta-1,3-diene (C4H5Cl).

### Maximum Acceptable Value

There are insufficient data to derive a MAV for chloroprene in drinking-water. Chloroprene is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

There are no known natural occurrences of chloroprene in the environment. Because chloroprene has a propensity to spontaneously oxidise and form dimers, peroxides, and other oxygenated species, it usually occurs (industrially) in combination with inhibitors (eg, phenothiazine), stabilisers or antioxidants. 1-Chlorobutadiene may be a trace impurity. Phenothiazine was used as an [insecticide](http://en.wikipedia.org/wiki/Insecticide) in New Zealand, mainly as an [antihelminthic](http://en.wikipedia.org/wiki/Antihelminthic) in [livestock](http://en.wikipedia.org/wiki/Livestock), but is no longer approved.

Chloroprene (321,000 tonnes produced in 1989) is available commercially as crude β‑chloroprene with a minimum purity of 95 percent. The principal impurities are dichlorobutene and solvents, with smaller amounts of 1-chlorobutadiene (α‑chloroprene), chlorobutenes and dimers of both chloroprene and butadiene. It is used to produce the synthetic elastomer polychloroprene, called neoprene by DuPont (CAS No. 9010-98-4). The only other use accounting for a significant volume is the synthesis of 2,3-dichloro-1,3-butadiene, which is used as a monomer in selected copolymerisations with chloroprene.

The vulcanised products of polychloroprene have favourable physical properties and excellent resistance to weathering and ozone. Articles made with this rubber include electrical insulation and sheathing materials, hoses, conveyor belts, flexible bellows, transmission belts, sealing materials, O-rings, diving suits and other protective suits. Adhesive grades of polychloroprene are used mainly in the footwear industry. Polychloroprene latexes have been used for dipped goods (balloons, gloves), latex foam, fibre binders, adhesives and rug backing.

It is reported that dry polychloroprene no longer contains detectable chloroprene (detection limit 0.5 ppm). In polychloroprene latexes, residual chloroprene is less than 1 percent, varying with the manufacturing process and intended use. Chloroprene has been detected as an impurity at levels of several parts per million in commercial vinyl chloride in Italy and Japan, and in acrylonitrile in the USSR.

2-Chloro-1,3-butadiene was detected in 1 out of 204 samples of surface water taken from sites near heavily industrialised areas across the US during 1975/76. 2-Chloro-1,3-butadiene was identified in 2 out of 63 industrial effluents at a concentration of (0.01 mg/L).

#### 2. From treatment processes

None.

#### 3. From the distribution system

No known sources, other than the theoretical potential for chloroprene to leach from O-rings.

### Form and fate in the environment

Volatilisation is the primary mechanism of removal of chloroprene from water. Chemical hydrolysis, adsorption to suspended solids or sediments, or bioaccumulation in aquatic animals is not expected to occur. If released to soil, chloroprene should be susceptible to removal by rapid volatilisation and transport by leaching into groundwater. The half-life in water is estimated to be about 3 hours , and it is not expected to chemically hydrolyse, adsorb significantly to suspended solids or sediments, biodegrade, or bioaccumulate in aquatic organisms.

Water solubility has been reported from 250 – 480 mg/L at 20°C.

### Removal methods

Aeration seems to offer the best option.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See IARC (1999).

### Health considerations

There is sufficient evidence in experimental animals for the carcinogenicity of chloroprene; IARC conclude that chloroprene is possibly carcinogenic to humans (Group 2B); note that all animal tests were by inhalation, and the primary route of potential human exposure to chloroprene is inhalation. However, polychloroprene is not classifiable as to human carcinogenicity (Group 3).

A National Toxicology Program (NTP 1998) study concluded that chloroprene showed clear evidence of carcinogenic activity in both rats and mice. The USEPA (2000) classified chloroprene in Group D, not classifiable as to human carcinogenicity. USEPA (2010) however considered chloroprene is “*likely to be carcinogenic to humans*” by all routes of exposure.

The USEPA (2000) calculated a provisional Reference Dose ([RfD](http://earth1.epa.gov/ttn/atw/hlthef/hapglossaryrev.html#rfd)) of 0.02 mg/kg body weight per day (mg/kg/d) for chloroprene. USEPA (2010) however concluded the available data are inadequate to derive an oral RfD for chloroprene.

### Derivation of Maximum Acceptable Value

No MAV.

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# Chlorotoluenes

The three isomers (where the chlorine atom is attached to the benzene ring) are:

* o-chlorotoluene: CAS No. 95-49-8. Also called 2-chlorotoluene or  
  1-chloro-2-methylbenzene or o-tolyl chloride.
* m-chlorotoluene: CAS No. 108-41-8. Also called 3-chlorotoluene or  
  1-chloro-3-methylbenzene, or m-tolyl chloride.
* p-chlorotoluene: CAS No. 106-43-4. Also called 4-chlorotoluene or  
  1-chloro-4-methylbenzene, or p-tolyl chloride.

Dichlorotoluenes (dichloromethylbenzenes) exist as well, the commonest being 2,4‑dichlorotoluene (CAS No. 95-73-8) and 2,6-dichlorotoluene (CAS No. 118-69-4).

When the chlorine atom is attached to the methyl link rather than to the benzene ring, it is called an α-chlorotoluene. There is a monochloro-, dichloro- and trichloro- α‑chlorotoluene.

The monochloro α-chlorotoluene is called benzyl chloride, benzylchloride, chloromethyl benzene, chlorophenylmethane or α-tolyl chloride, and has the CAS No. 100-44-7.

The dichloro α-toluene is CAS No. 98-87-3, and is called dichlorotoluene, benzal chloride, benzyl dichloride, benzylene chloride, benzylidine chloride, chlorobenzal, (dichloromethyl)benzene, dichlorophenylmethane and dichlorotoluene.

The trichloro α-toluene is CAS No. 98-07-7, and can be called benzotrichloride, (trichloromethyl)benzene, α,α,α-trichlorotoluene, benzenyl chloride, benzenyl trichloride, benzylidyne chloride, benzyl trichloride, phenyl chloroform, phenyltrichloromethane, toluene trichloride and trichloromethylbenzene.

### Maximum Acceptable Value

The DWSNZ do not include a MAV for any of the above chlorotoluenes; WHO does not mention chlorotoluenes.

The USEPA concluded on 22 September 2009 that benzyl chloride is known or anticipated to occur in PWSs and may require regulation. Therefore they added benzyl chloride to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a).

The USEPA (2006/2011) established a lifetime health advisory of 0.1 mg/L for o‑chlorotoluene and p-chlorotoluene, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

### Sources to drinking-water

#### 1. To source waters

About 130,000 t/a chlorotoluenes are produced worldwide, of which about 60,000 to 70,000 t/a is 2-chlorotoluene, which is used as a solvent and a chemical intermediate in the manufacture of pesticides, dyes, and pharmaceuticals.

A mixture of isomeric chlorotoluenes is used as a solvent in “Splendor”, a formulation of the herbicide tralkoxydim – see datasheet in pesticides section.

2,4-Dichlorotoluene is used as an intermediate for pesticides, drugs and chlorinated-nitrated benzenes. 2,6-Dichlorotoluene is used as intermediate for pesticide and pharmaceuticals; no consumer use is reported.

Benzyl chloride is the most important chlorinated α-toluene, with 144,000 tonnes produced in 1989. More than two-thirds of the benzyl chloride produced is used in the manufacture of butyl benzyl phthalate, a plasticiser used extensively in vinyl flooring and other flexible poly(vinyl chloride) uses such as food packaging. Other significant uses are the manufacture of benzyl alcohol and benzyl chloride-derived quaternary ammonium compounds, each of which consumes more than 10 percent of the benzyl chloride produced. Benzylchloride is widely used in the production of other substances such as plastics, dyes, pesticides, lubricants, petrol, photographic developer, flavour products and pharmaceuticals. It has previously been used as an irritant gas in chemical warfare.

Benzal chloride is used almost exclusively for the manufacture of benzaldehyde and cinnamic acid.

Benzotrichloride is mostly used as a chemical intermediate, primarily for benzoyl chloride. Lesser amounts are used in the manufacture of benzotrifluoride, as a dyestuff intermediate, and in producing hydroxybenzophenone ultraviolet light stabilisers.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

The chlorotoluenes are neutral and stable compounds, therefore hydrolysis is not expected to be an important process in determining the environmental fate. They are also not readily biodegradable. A high volatility from water to air is indicated for 2‑chlorotoluene by the Henry constant; therefore it should mostly evaporate before biodegrading. In soil it is supposed that 2-chlorotoluene would be moderately mobile.

4-Chlorotoluene was detected in the River Rhine in Holland up to 0.002 mg/L.

A range of water solubilities have been reported: 2-chlorotoluene from 47 to 470 mg/L; and 4-chlorotoluene about 10 to 100 mg/L; although errors are apparent, these substances are quite soluble.

An estimated Koc of 4,800 suggests that 2,4-dichlorotoluene may have slight mobility in soil. Volatilisation from moist soil surfaces may occur based on an estimated Henry’s Law constant of 4.2 x 10-3 atm cu m/mol. 2,4-Dichlorotoluene was found to be susceptible to anaerobic biodegradation in soil slurry microcosms under methanogenic conditions; complete degradation required 130 days. The predominant product of this reaction was 4-chlorotoluene, a small amount of 2-chlorotoluene was also produced. 2,4-Dichlorotoluene is expected to adsorb to suspended matter in the water based on its estimated Koc value. This compound should volatilise from water surfaces given its Henry’s Law constant. Estimated half-lifes for a model river and model lake are four hours and five days, respectively. An estimated BCF value of 1000 suggests that 2,4‑dichlorotoluene may bioconcentrate in aquatic organisms (EAWAG, accessed February 2015). Water solubility is 25 mg/L.

Benzyl chloride water solubility is 525 mg/L at 25°C but is hydrolysed rapidly water (half life <12 h) to benzyl alcohol which is biodegradable (OECD). DWI (2014) quotes log Kow of 2.3, Henry’s Law constant of 0.000412 atm.m3/mole.

Benzal chloride is described as being insoluble in water, but hydrolyses to benzaldehyde under both acid and alkaline conditions.

Benzotrichloride is described as being insoluble in water, but hydrolyses in water. Benzoyl chloride decomposes in water.

α,α,α-Trichlorotoluene hydrolyses completely to hydrochloric acid and benzoic acid upon contact with moisture, hence its environmental impacts are largely related to benzoic acid (CAS No. 65-85-0) – see benzyl benzoate datasheet. Water solubility is about 100 mg/L.

### Typical concentrations in drinking-water

Eighteen water utilities in the US reported detecting o-chlorotoluene (2-chlorotoluene) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0066 mg/L.

Fourteen water utilities in the US reported detecting p-chlorotoluene (4-chlorotoluene) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.167 mg/L; this result was rather an outlier, the next highest was 0.0025 mg/L.

### Removal methods

Aeration seems to offer the best option.

### Health considerations

2-Chlorotoluene showed no mutagenic activity in bacterial and in mammalian cell test systems *in vitro*. Based on the observations in three-month studies on rat and dog, there is no indication that 2-chlorotoluene affects the reproductive organs.

UNEP (OECD 2004) quoted a NOEL for repeated dosing of 2-chlorotoluene by gavage to rats and dogs (three months) of 20 mg/kg bw.

The USEPA (1990/2006/2009/2011) developed a RfD (an estimate of a daily exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime) for 2-chlorotoluene of 0.02 mg/kg/d. This was based on studies that showed male rats developed a statistically significant decrease in mean body weight gain (15 percent and 22 percent, respectively) and an increase in adrenal weight. Increased heart and testes weights, an increase in white blood cell count, and a decrease in prothrombin time were observed in males at the 320 mg/kg/day dose level. At the 80 mg/kg/day dose, blood urea nitrogen was increased in males. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.7 mg/L.

Likewise, the reference dose or RfD (USEPA 2006/2009/2011) for p-chlorotoluene is 0.02 mg/kg/d, and the Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.7 mg/L.

Since there is no developmental toxicity study with p-chlorotoluene, the data from o‑chlorotoluene are taken into account to fill the data gap. The comparison of the two isomers showed a rather high degree of qualitative similarity with respect to available data on absorption, excretion and metabolism, toxicity after acute and repeated exposure. Overall, o-chlorotoluene and p-chlorotoluene have a similar toxicity profile (OECD 2005).

IARC (1999) stated that combined exposures to α-chlorinated toluenes (which includes benzal chloride, benzyl chloride and benzotrichloride) and benzoyl chloride are probably carcinogenic to humans (Group 2A).

The USEPA (1995) classified benzyl chloride as a B2 human carcinogen, ie, a possible carcinogen. However, as benzyl chloride is rapidly hydrolysed to benzyl alcohol in the water phase, health risks via the environment are assessed as benzyl alcohol exposure (OECD 2002). DWI (2014) states: Both negative and positive results have been reported for benzylchloride in *in vitro* genotoxicity assays. Overall the weight of evidence indicates that benzylchloride is genotoxic *in vitro*. However, the weight of evidence indicates that benzylchloride is non genotoxic *in vivo*. For Repeat Dose Toxicity and Carcinogenicity a NOAEL of 15 mg/kg bw/day (reported to be 6.4 mg/kg bw/day, adjusting to a seven day/week dosing regimen) was identified based on forestomach tumours. Based on this NOAEL a Tolerable Daily Intake (TDI) of 0.006 mg/kg bw/day (6 μg/kg bw/day; rounded) is derived.

There is only one key study on repeated dose toxicity of 2,4-dichlorotoluene. This chemical was studied for oral toxicity in rats according to the OECD combined repeated dose and reproductive/developmental toxicity test; the NOEL for this compound was indicated to be less than 12.5 mg/kg/day (OECD 2005).

In a repeated oral toxicity study on rats the NOEL for 2,6-dichlorotoluene was considered to be 30 mg/kg/day for male and 100 mg/kg/day for female, respectively (OECD 2002).

The USEPA (1990) classified benzotrichloride as a B2 human carcinogen, ie, a possible carcinogen.

α,α,α-Trichlorotoluene is readily absorbed from the gastrointestinal tract, distributed within the body, and excreted after metabolic transformation to hippuric acid mainly via the urine (OECD 2005). Because α,α,α-trichlorotoluene hydrolyses completely to hydrochloric acid and benzoic acid upon contact with moisture, its health effects relate mainly to those chemicals.

### Derivation of Maximum Acceptable Value

No MAV.

Based on the health protective concentration calculated, the Californian Office of Environmental Health Hazard Assessment recommends and supports an action level of 0.14 mg/L for 2-chlorotoluene in drinking-water.

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# Chrysene

Chrysene, CAS No. 218-01-9, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. Also called benzo[a]phenanthrene.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

The principal route of entry for PAHs to source water is via atmospheric deposition. Based on the literature reviewed, the presence of chrysene and benzo(k)fluoranthene may be indicators for coal combustion emissions. Whereas other PAHs are indicators of other combustion process: benzo(g,h,i)perylene, coronene and phenanthrene are indicators for motor vehicle emissions, pyrene and fluoranthene are associated with incineration and fluorene, fluoranthene and pyrene are associated with oil combustion (Environment Australia 2003).

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; benzo[a]pyrene (BaP) levels range from 6 to 35 mg/kg TEQ depending on land use. The equivalent BaP concentration is calculated as the sum of each of the detected concentrations of nine carcinogenic PAHs which are benz(a)anthracene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene and indeno(1,2,3-cd) pyrene, multiplied by their respective potency equivalency factors.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

Water solubility is about 0.002 mg/L. The partition coefficient (octanol/water) (logKow) is 5.73.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 and 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. Chrysene was not detected (less than 0.0000023 mg/L (2.3 ng/L)) in any of the New Zealand samples.

Twelve water utilities in the US reported detecting chrysene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00053 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation (ATSDR 1995).

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess. The relative carcinogenic potencies of various polynuclear aromatic hydrocarbons, based on bioassays by several routes of administration and related toxicological data, have been ranked in decreasing order as follows: dibenz[a,h]anthracene, benzo[a]pyrene, anthanthrene, indeno[1,2,3-cd]pyrene, benz[a]anthracene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, benzo[j]fluoranthene, cyclopentadieno[c,d]pyrene, benzo[g,h,i]perylene, chrysene and benzo[e]pyrene.

USEPA (1994) classified chrysene as B2, a probable human carcinogen.

IARC (2010) classified chrysene in Group 2B (possible human carcinogen).

Chrysene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

No MAV.

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# Creosotes, coal tars and coal tar pitches

Although not included amongst the 17 PAH “priority pollutants” under the USEPA Clean Water Act, some commercial products containing a range of PAHs are or were used widely. The substances covered in this datasheet are derived from coal, not petroleum.

CAS No. 8001-58-9 covers creosotes, CAS No. 8007-45-2 covers coal tars, CAS No. 61789-60-4 covers pitch, CAS No. 65996-93-2 covers coal tar pitches (high temperature), and CAS No. 92061-94-4 covers residues (coal tar), pitch distillation.

Refer also to the polynuclear aromatic hydrocarbons datasheet.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

Creosote, a complex mixture of hundreds of distinct compounds, including bi- and polycyclic aromatic hydrocarbons (PAHs), phenols, as well as heterocyclic, oxygen-, sulphur- and nitrogen-containing compounds. The chemical composition is influenced by the origin of coal and also by the nature of the distillation process, and as a result, the composition of different batches may vary to a great extent., It is a distillation by‑product of the coal gas industry; it was a popular wood preservative/stain in New Zealand. Coal tar creosote is still the most widely used wood preservative in the United States. Coal tar creosote is a thick, oily liquid that is typically amber to black in colour, and is a distillation product of coal tar. Over 100 components in creosote have been identified. It is used as a fungicide, insecticide, miticide, and sporicide to protect wood and is applied by pressure methods to wood products, primarily utility poles and railroad ties (USEPA 2007).

Coal tar and coal tar pitch are the by-products of the high-temperature treatment of coal to make coke or natural gas. They are usually thick, black or dark brown liquids or semi-solids with a smoky or aromatic odour. Coal tar products are ingredients in medicines used to treat skin diseases such as psoriasis. The main use (71.3 percent) in Europe of coal tar pitch is in the production of anodes, followed by electrodes (18 percent).

The major chemicals in coal tar creosote, coal tar, and coal tar pitch that can cause harmful health effects are polycyclic aromatic hydrocarbons (about 75 percent by weight), phenol, and cresols. About 300 chemicals have been identified in coal tar creosote, but as many as 10,000 other chemicals may be in this mixture; Table 4.7 in ATSDR (2002) identifies 199 chemicals found in coal tar pitch. EU (2008) lists dozens as well.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

Polynuclear aromatic hydrocarbons enter the environment through atmospheric deposition. Because of their low water solubility most polynuclear aromatic hydrocarbons are adsorbed to sediments and suspended solids in aquatic systems. Volatilisation may be important over periods exceeding one month. Most polynuclear aromatic hydrocarbons are susceptible to aqueous photolysis. Polynuclear aromatic hydrocarbons of 3 or fewer fused aromatic rings are biodegraded but for the larger polynuclear aromatic hydrocarbons this is minimal. Polynuclear aromatic hydrocarbons are adsorbed but not greatly accumulated by aquatic biota.

EU (2008) describes pitch as having a water solubility of 0.04 mg/L although it is quite variable, with a high value of 14 mg/L reported. The main PAHs found in elution tests were (in approximate order) fluoranthene, phenanthrene, acenaphthene, pyrene and fluorene; long term chrysene became the most prominent.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess.

USEPA (1988) classified creosote as B1; probable human carcinogen.

IARC (2010) classified coal tar and coal tar pitches in Group 1 (human carcinogen); and creosotes in Group 2A (probable human carcinogen).

Creosotes and bitumen appear on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

There are no reports of adverse reproductive or developmental outcomes in humans exposed to coal tar and coal tar products. Sperm counts and sperm characteristics were found to be unaffected in workers exposed to coal tar pitch volatiles. Women treated with coal tar for psoriasis or dermatitis (ages at treatment were 18–35 years) did not exhibit an increase in spontaneous abortions or congenital disorders in their offspring.

### Derivation of Maximum Acceptable Value

No MAV.

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# Cresol

CAS No. 1319-77-3, which covers the cresols as a group or mixture, (also called methylphenol, hydroxytoluene, methyl phenol, cresylic acid and tricresol). The three isomers are:

* o-cresol: CAS No. 95-48-7. Also called 2-methylphenol, 2-hydroxytoluene, o-toluol, 1-hydroxy-2-methylbenzene or ortho-cresylic acid/
* m-cresol: CAS No. 108-39-4. Also called 3-methylphenol, 3-hydroxytoluene, m‑toluol, 1-hydroxy-3-methylbenzene, or meta-cresylic acid.
* p-cresol: CAS No. 106-44-5. Also called 4-methylphenol, 4-hydroxytoluene, 4-cresol, p-tolyl alcohol, p-methylhydroxybenzene or para-cresylic acid.

Technical grade cresol contains approximately 20 percent o-cresol, 40 percent m-cresol and 30 percent p-cresol.

A mixture of meta- and para-cresol, m/p cresol or dicresol has a CAS No. of 15831‑10‑4. This mixture contains 60 to 75 percent m-cresol and 25 to 40 percent p‑cresol.

Cresols have also been called hydroxy methylbenzenes and toluols.

### Maximum Acceptable Value

The DWSNZ do not include a MAV for any of the above cresols; WHO does not mention cresol.

### Sources to drinking-water

#### 1. To source waters

These chemicals are not normally found in natural waters. They are a component of creosote and other coal tar products. o-Cresol is a natural component of crude oil. Cresols are natural products that are present in many foods. They have been detected in many plants (such as jasmine, easter lily, yucca, conifers, oaks, and sandalwoods), cheese flavour, and some other foods. They are present in wood, coal and tobacco smoke, volcanic gases, crude oil and car exhaust. In addition, cresols can be manufactured and used as industrial and household disinfectants and deodorisers, solvents, and as starting chemicals for making pesticides, plasticisers, resins and other chemicals. ‘Lysol’ which is still used as a rough antiseptic, is a mixture of cresols solubilised with soap or alkali. Cresols are used with xylenols overseas as a pesticide – see xylenol datasheet.

Sewage may contain 1 to 2 mg/L of cresols, and they may appear up to 0.002 mg/L in rainwater.

p-Cresol is an endogenous metabolite of the amino acid tyrosine and a normal constituent of animal (including human) urine with levels of human excretion ranging from 16 to 74 mg/24 hours (OECD 2005).

In a national study of organic contaminants in 139 US streams located in 30 states from 1999 to 2000, p-cresol was detected in 24.7 percent of the samples taken with a maximum concentration of 0.0005 mg/L and a mean concentration of 0.00005 mg/L.

#### 2. From the distribution system

No known sources.

### Form and fate in the environment

The cresols are very soluble in water: about 2–2.5 percent. This high solubility means they can readily find their way to groundwater, where they may persist for months. Cresols evaporate slowly from soil and water surfaces. They do not adsorb strongly to soils. The major degradation process is by biodegradation, which can be quite rapid in surface waters, from several hours to a few days. Any cresols entering anaerobic conditions will be very persistent.

If released to soil, o-cresol is expected to have high mobility based upon a log Koc of 1.34. Volatilisation from moist soil surfaces is expected to occur slowly based upon a Henry’s Law constant of 1.2 x 10-6 atm‑cu m/mole. o-Cresol is not expected to volatilise from dry soil surfaces based upon its extrapolated vapour pressure. This compound is expected to biodegrade rapidly based upon half-lifes of 1.6 and 5.1 days in two agricultural soils. If released into water, o-cresol is not expected to adsorb to suspended solids and sediment in the water column based upon the Koc value. o‑Cresol is expected to biodegrade in water based on a reported half-life of 50 days in southern California coastal waters and a half-life of 20 days in gasoline contaminated groundwater. Volatilisation from water surfaces is expected to be slow based upon this compound’s Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 21 and 235 days, respectively. o-Cresol is not expected to undergo hydrolysis since it lacks functional groups that hydrolyse under environmental conditions. o-Cresol absorbs light greater than 300 nm and photodegrades, but direct photolysis in sunlit surface waters is expected to occur at a much slower rate than biodegradation. An estimated BCF of 6 suggests the potential for bioconcentration in aquatic organisms is low (EAWAG accessed February 2015).

If released to soil, m-cresol is expected to have high mobility based upon a log Koc of 1.54. Volatilisation from moist soil surfaces is not expected to be an important environmental fate process based on a Henry’s Law constant of 8.6 x 10-7 atm‑cu m/mole. m-Cresol is not expected to volatilise from dry soil surfaces based upon its extrapolated vapour pressure. This compound is expected to biodegrade rapidly based upon half-lifes of 0.6 and 11.3 days in two agricultural soils. If released into water, m-cresol is not expected to adsorb to suspended solids and sediment in the water column based upon the Koc value. m-Cresol is expected to biodegrade in water based on reported half-lifes in the range of 2 to 29 days (aerobic water) and 15 to 49 days (anaerobic water). Volatilisation from water surfaces is not expected to be an important environmental fate process based on its Henry’s Law constant. m-Cresol is not expected to undergo hydrolysis since it lacks functional groups that hydrolyse under environmental conditions. m-Cresol absorbs light greater than 300 nm, but direct photolysis in sunlit surface waters is expected to occur at a much slower rate than biodegradation. Log BCF values of 1.3 and 1.03 reported for m-cresol in fish suggest that bioconcentration in aquatic organisms is low (EAWAG accessed February 2015).

If released to soil, p-cresol is expected to have moderate to high mobility based upon log Koc values of 1.69–2.81. Volatilisation from moist soil surfaces is expected to occur based upon a Henry’s Law constant of 1 x 10-6 atm‑cu m/mole. p-Cresol is not expected to volatilise from dry soil surfaces based upon its extrapolated vapour pressure. This compound is expected to biodegrade rapidly based upon half-lifes of one and 0.5 days in two agricultural soils. If released into water, p-cresol is not expected to adsorb to suspended solids and sediment in the water column based upon the log Koc values. p-Cresol is expected to biodegrade quickly in water under aerobic conditions based on complete degradation of p-cresol within four and six days in Lake Tahoe, CA water. Volatilisation from water surfaces is expected to occur based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 25 and 281 days, respectively. p-Cresol is not expected to undergo hydrolysis since it lacks functional groups that hydrolyse under environmental conditions. p‑Cresol possesses absorption bands with tails extending beyond 300 nm and can photodegrade, but direct photolysis in sunlit surface waters is expected to occur at a much slower rate than biodegradation. An estimated BCF of 6 suggests the potential for bioconcentration in aquatic organisms is low (EAWAG accessed February 2015).

### Removal methods

IEH (2014) reports cresols may be reduced in concentration by about 50 percent using conventional treatment and chlorine. Ozone and activated carbon should enhance this.

### Health considerations

Cresols are present in cigarette smoke. Cresols are also found in some foods, such as tomatoes, asparagus, cheese, butter, bacon, and smoked foods. Drinks can also contain cresols, such as coffee, black tea, wine, whiskey, brandy and rum. Cresols that enter the stomach can pass rapidly (in minutes) to the blood. Once in the blood, cresols can be distributed to many organs in the body. Most of the cresols that enter the body metabolise and leave in the urine within one day. WHO note that o-cresol is the most toxic isomer, followed by p-cresol and then m-cresol.

Oral exposure of o-cresol of up to 13 weeks of mice and rats resulted in mortality, tremors, reduced body weights, hematologic effects and increase in organ weights. An overall subchronic NOAEL of 50 mg/kg bw/day can be derived (OECD 1998).

According to the USEPA’s updated criteria for assessing the potential for a chemical to cause cancer, cresols fall in the category of chemicals for which there is “inadequate information to assess carcinogenic potential – Group C”. Animal studies suggest that cresols probably would not produce birth defects or affect reproduction in humans.

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for cresols of:

* 0.1 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.1 mg/kg/day for chronic-duration oral exposure (>364 days).

The Dutch National Institute for Public Health and the Environment (RIVM) has suggested a TDI of 0.05 mg/kg; reported in IEH (2014).

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA (IRIS 1990) derived oral reference doses of 0.05 mg/kg/day for each of the cresols based on NOAELs of 50 mg/kg/day for decreased body weights and neurotoxicity (myoclonus, tremors, laboured respiration) observed in Sprague-Dawley rats exposed by gavage for 90 days (TRL 1986) in an assessment conducted in 1989. An uncertainty factor of 100 (10 for interspecies and 10 for intraspecies variability) was applied to the NOAEL. In 1993 the RfD for 4-methylphenol was withdrawn.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for o-cresol is 0.03 mg/L, 0.03 mg/L for m-cresol and 0.003 mg/L for p-cresol.

IEH (2014) reports taste and odour thresholds:

* o-cresol, sweet, tar-like: 0.26 to 1.4 µg/L
* m-cresol, sweet, tar-like: 0.015 to 0.8 µg/L
* o-cresol, pungent, tar-like: 0.0027 to 0.2 µg/L.

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# Cumene

CAS No. 98-82-8. Also called isopropylbenzene, iso-propylbenzene, (1-methylethyl)-benzene and 2-phenylpropane. Has also been called cumol.

A structurally similar chemical is n-propylbenzene (CAS No. 103-65-1) which is also called propylbenzene, isocumene, or 1-phenylpropane.

The trimethylbenzenes (qv) comprise a benzene ring with a methyl group attached in three places. The propylbenzenes are made up of a benzene ring with a propyl group attached.

### Maximum Acceptable Value

Cumene and isocumene do not have a MAV in the DWSNZ, and are not mentioned in the WHO Guidelines.

The USEPA concluded on 22 September 2009 that n-propylbenzene is known or anticipated to occur in PWSs and may require regulation. Therefore they have added [it](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane) to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a).

IEH (2014) reports a taste and odour threshold of 0.00007 to 0.0001 mg/L for cumene.

### Sources to drinking-water

#### 1. To source waters

Cumene is a water-insoluble petrochemical used almost entirely (about 95 percent) in the manufacture of several chemicals, including phenol and acetone. A minor component is used in the manufacture of detergents. Crude oil typically contains 0.1 percent wt cumene but may contain up to 1 percent wt. It is about 0.3 percent of petrol and 0.9 percent of diesel.

Cumene has been reported at up to 0.0004 mg/L in surface waters, and 1.6 mg/L in groundwater under solvent storage tanks (EU 2001).

#### 2. From treatment process

No known sources.

### Forms and fate in the environment

Cumene readily volatilises into the atmosphere from water and dry soil. Cumene is expected to adsorb moderately to strongly to soil/sediments and to undergo biodegradation in water and soil. Water solubility about 50 mg/L. In water, it is subject to volatilisation (half-lifes of 1.2 hours in a model river and 4.4 days in a model lake).

EU (2001) reports vapour pressure = about 5 hPa; partition coefficient = logPow = 3.55; Henry’s law constant = 1010 Pa.m3/mol. The photo-oxidation of cumene has been measured at 0.4 to 5.1 hours.

IEH (2014) reports that cumene has a vapour pressure of 4.5 mm Hg and Henry’s law constant of 0.0115 atm‑cu m/mole indicate that volatilisation is likely to be slow from dry soils but may represent an important clearance mechanism for moist soil. Iso-propylbenzene is expected to possess a Koc value of 820 and as such is likely to exhibit poor mobility in soils. Additionally, this Koc value is likely to limit soil volatilisation through sorption of iso-propylbenzene to soil particles. Experimental inoculation of iso-propylbenzene with wastewater indicates the potential for rapid biodegradation in soil.

### Typical concentrations in drinking-water

Forty-three water utilities in the US reported detecting n-propylbenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.072 mg/L.

Fifty-two water utilities in the US reported detecting isopropylbenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.021 mg/L.

Cumene has been found between 1–30 mg/L in groundwater near petrol storage tanks.

### Removal methods

Because cumene adsorbs to suspended solids and sediment, any that does not volatilise will be reduced in concentration by treatment processes that remove particulate matter.

IEH (2014) reports that the concentration of iso-propylbenzene may be reduced by about 50 percent by conventional water treatment and chlorination; PAC will enhance this to nearer 100 percent.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

In humans, iso-propylbenzene exhibits anaesthetic-like CNS depression with symptoms including dizziness, drowsiness, loss of coordination and unconsciousness; the EU RAR cites a rat oral LD50 of 1,400 to 4,000 mg/kg (IEH 2014). A guidance value for cumene for oral exposure of 0.1 mg/kg body weight per day has been derived, based on the no-observed-adverse-effect level (NOAEL) of 154 mg/kg body weight per day for increased kidney weight in female rats in a six- to seven-month oral study; the NOAEL was adjusted for the dosing schedule, and a total uncertainty factor of 1000 was applied. No data are available with which to quantify human exposure to cumene. Most genotoxicity test data with cumene are negative (WHO 1999).

The oral reference dose or RfD (USEPA 1997/2006/2009/2011) for cumene is 0.1 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 4 mg/L.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit for cumene (isopropylbenzene) is 0.3 mg/L.

USEPA (1997) states that under the proposed 1996 *Guidelines for Carcinogen Risk Assessment*, it is concluded that the carcinogenic potential of cumene *cannot be determined* because no adequate data, such as well-conducted long-term animal studies or reliable human epidemiological studies, are available to perform any assessment.

IARC (2012) concluded that cumene is possibly carcinogenic to humans (Group 2B).

### Derivation of Maximum Acceptable Value

No MAV.

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# Cyanuric acid

CAS No. 108-80-5. The IUPAC name is 1,3,5-triazinane-2,4,6-trione. Also called isocyanuric acid, tricyanic acid, tricarbimide or 2,4,6-trihydroxy-1,3,5-triazine.

Usually added to water as sodium dichloroisocyanurate (CAS No. 2893-78-9), or NaDCC; or as the dihydrate (CAS 51580-86-0). The IUPAC name for sodium dichloroisocyanurate is 1,3-dichloro-1,3,5-triazinane-2,4,6-trione. It is also known as sodium dichloro-s-triazine trione and sodium troclosene.

Cyanuric acid is an analogue of melamine, as are ammelide, ammeline, trichloromelamine (a sanitiser, CAS 7673-09-8) and cyromazine (WHO 2009a). There is a datasheet for cyromazine in the pesticides section. Dichlorocyanuric acid has CAS No. 2782-57-2.

Although it is not mentioned in the WHO Guidelines (2017), trichloroisocyanuric acid (CAS No. 87-90-1) is also available, and is reported as being used in swimming pools. The IUPAC name is 1,3,5-trichloro-1,3,5-triazinane-2,4,6-trione, and other names include trichlorocyanuric acid, trichlor, isocyanuric chloride, chloreal, symclosene and TCCA.

### Maximum Acceptable Value

WHO (2017) has calculated a Guideline Value of 40 mg/L for cyanuric acid or 50 mg/L for sodium dichloroisocyanurate.

WHO 2004/2008 stated that the concentration in drinking-water will be well below that at which toxic effects are observed. Sodium dichloroisocyanurate is included in the [plan of work of the rolling revision](http://www.who.int/entity/water_sanitation_health/dwq/en/index.html) of the WHO Guidelines for Drinking-water Quality.

There is no MAV in the 2008 DWSNZ.

### Sources to drinking-water

#### 1. To source waters

These chemicals are not normally found in natural waters.

The chlorinated isocyanurates are registered for use in the US as disinfectants, sanitisers, algicides, fungicides, fungistats, bactericides, bacteriostats, microbiocides and microbiostats. These compounds are used primarily as disinfectants and algicides in swimming pools and to a lesser extent in industrial cooling water systems. Minor uses include sanitising and disinfecting food and non-food contact surfaces and sanitising laundry (USEPA 1992).

Cyanuric acid can be a by-product in the production of melamine (WHO 2009a).

#### 2. From treatment processes

Cyanuric acid is mainly used as a precursor to N-chlorinated cyanurates (1). Further chlorination gives [trichloroisocyanuric acid](http://en.wikipedia.org/wiki/Trichloroisocyanuric_acid), [C(O)NCl]3.

[C(O)NH]3 + 2 Cl2 + 2 NaOH → [C(O)NCl]2[C(O)NH] ……………(1)

NaDCC is the sodium salt of a chlorinated hydroxytriazine and is used as a source of free available chlorine (FAC) in the form of hypochlorous acid for the disinfection of water. It is widely used as a stable source of chlorine for the disinfection of swimming pools and in the food industry, because it is more stable in sunlight than most other sources of chlorine. It is also used to disinfect drinking-water, primarily in emergencies, when it provides an easy-to-use source of FAC.

The FAC content in anhydrous NaDCC (commercial product) is 62–64 percent, and the dihydrate has 55–56 percent FAC; noting that the FAC for elemental chlorine is 100 percent. Therefore, development of 1 mg/L of FAC requires approximately 1.6 mg of anhydrous NaDCC per litre and approximately 1.8 mg/L for the dihydrate.

When added to water, NaDCC (anhydrous or dihydrate) rapidly hydrolyses to release FAC and establishes a complex series of equilibria involving six chlorinated and four non-chlorinated isocyanurates. These equilibria are established in the order of seconds. The concentration of each species depends on the concentrations of total available chlorine (TAC = FAC plus “reservoir” chlorine, eg, as DCC−) and total isocyanurates, the pH and the values of the equilibrium constants (dependent on temperature and ionic strength). “Reservoir” chlorine refers to the bound chlorine of the various chloroisocyanurates. The latter function as reservoirs of rapidly released FAC, as the existing FAC depletes. Thus, if hypochlorous acid is consumed by reaction with organic material (oxidation), chloroisocyanurates will rapidly dissociate to release more hypochlorous acid. However, for the sake of simplicity, the overall hydrolysis reaction can be considered as:

C3N3O3Cl2Na + 2H2O ↔ C3N3O3H2Na + 2HOCl

producing sodium cyanurate and hypochlorous acid.

The distribution of the chemical species in aqueous solutions of NaDCC can be calculated from their hydrolysis and acid dissociation constants. As an example, dissolution of NaDCC to provide 1.0 mg/L of TAC at pH 7.0 gives the following: 48.1 percent TAC from hypochlorous acid, 26.8 percent TAC from monochlorocyanurate, 11.8 percent TAC from dichlorocyanurate, 12.8 percent TAC from hypochlorite and less than 1 percent from other chlorocyanurates and chlorocyanuric acids. In normal batch-type use of NaDCC, oxidative and microbiocidal demand will consume FAC until all available chlorine has been reduced, leaving only non-chlorinated isocyanurates/cyanurate (eg, cyanuric acid). As long as NaDCC is added to water to maintain a certain level of TAC or FAC, the various cyanurates will be present at levels dependent on the properties of the water (ie, pH, temperature, etc).

In calculating the FAC content, it has to be borne in mind that when chlorine hydrolyses:

Cl2 + H2O ↔ HOCl + HCl

only one chlorine atom ends up as available chlorine (HOCl), whereas with NaDCC, both chlorine atoms end up as available chlorine.

Section 3.2 of WHO (2009) discusses the use of sodium dichloroisocyanurate.

#### 3. From the distribution system

No known sources, other than when added as a disinfectant.

### Form and fate in the environment

Cyanuric acid is considered to be not readily biodegradable in laboratory studies, and is stable in water at pH 4 to 9. However, it does biodegrade in the environment, in both soil and ambient water systems, in which it degrades to carbon dioxide and ammonia so that it does not accumulate in the environment.

If released to soil, cyanuric acid is expected to have high mobility based upon an estimated Koc of 58. The pKa of cyanuric acid is 6.88, indicating that it will partially exist in the anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 8.7 x 10-15 atm‑cu m/mole. Cyanuric acid biodegrades readily under a wide variety of natural conditions, and particularly well in systems of either low or zero dissolved- oxygen levels, such as anaerobic activated sludge and sewage, soils, muds, and muddy streams and river waters, as well as ordinary aerated activated sludge systems with typically low (1 to 3 ppm) dissolved-oxygen levels. If released into water, cyanuric acid is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. BCF values of <0.5 suggest bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

Water solubility:

* isocyanuric acid is about 2700 mg/L
* sodium dichloroisocyanurate dehydrate is about 25 percent
* trichloroisocyanuric acid is about 1.2 percent.

A solution of trichloroisocyanuric acid has a pH of 4.4, and sodium dichloroisocyanurate has a pH of about 6.5; Pub Chem.

### Typical concentrations in drinking-water

Where NaDCC is used for the disinfection of drinking-water, exposure will be to both the chlorinated species and residual cyanuric acid, which is highly stable in drinking-water. In a flow-through system, the concentrations will directly relate to the quantities added (ie, the quantities sufficient to achieve adequate disinfection).

When used to disinfect water in a container or water storage tank, the concentration of cyanuric acid will depend on whether the concentrations are topped up, because the concentrations of cyanuric acid will continue to increase; however, there will be a limit to this if a free chlorine level is to be achieved, because the increasing levels of cyanuric acid would affect the equilibrium (see below). When used in emergency situations, the practice of “topping up” to maintain a free chlorine residual should be discouraged. In this case, it would be possible for the sodium cyanurate concentration to build up to undesirable levels. In such cases, it would be very desirable to monitor the concentration of sodium cyanurate.

Trace levels of cyanuric acid can be present in food and water from the use of dichloroisocyanurate in drinking-water, swimming pools and water used in food manufacturing. The Joint FAO/WHO Expert Committee on Food Additives (JECFA) estimated residues of cyanuric acid in water from the use of sodium dichloroisocyanurate as a disinfectant in water treatment. Using a conservative assumption that 1 mol of sodium dichloroisocyanurate would generate 1 mol of cyanuric acid, it was estimated that the cyanuric acid concentration would range from approximately 1.6 mg/L (equivalent to 1.0 mg/L of free chlorine) up to 3.2 mg/L (for a maximum dose of 2.0 mg/L of free chlorine). However, the review indicated that the use of sodium dichloroisocyanurate as a drinking-water disinfectant would be primarily for emergency situations (WHO 2009a).

It is not unusual for cyanuric acid to be maintained at about 50 mg/L in swimming pools.

### Removal methods

Because sodium dichloroisocyanurate is added to water intentionally, it is unlikely that one would want to remove it, or its end-products.

### Analytical methods

The main concern with NaDCC is monitoring the concentration of its decomposition product, sodium cyanurate (or cyanuric acid).

The commonest technique for determination of cyanuric acid is HPLC. Analysis by HPLC with UV detection at 225 nm gave a limit of detection of 0.1 mg/L in swimming pool water.

Reverse-phase liquid chromatography with UV detection at 213 nm was used to analyse water samples and gave a detection limit of 0.05 mg/L for concentrations in the range 0.5–125 mg/L.

Similar results were obtained using two different HPLC columns. Limits of detection of 0.001 and 0.09 mg/L were reported using derivatisation followed by gas chromatography with flame thermionic specific detection and mass spectrometry selective ion monitoring, respectively.

For application in the field, various types of test kit are available – typically marketed for swimming pool applications. Test kits with detection limits of 1 mg/L or lower are available.

### Health considerations

In contact with saliva of about pH 7.0, chlorinated isocyanurates react extremely rapidly, such that, at the concentrations required to deliver FAC at the levels typically used in drinking-water, no detectable chlorinated isocyanurate remains. The material that reaches the gastrointestinal tract is, therefore, the unchlorinated cyanuric acid. The relevant toxicological studies cited in WHO (2008) refer to this compound. WHO (2009a) reports a TDI of 1.5 mg/kg/d for cyanuric acid; available data indicate that simultaneous exposure to melamine and cyanuric acid is more toxic than exposures to each compound individually.

In studies in which 14C-labelled sodium cyanurate was administered in multiple doses of 5 mg/kg of body weight to rats, the sodium cyanurate was extensively absorbed and excreted unchanged in the urine, mainly within about six hours. Only 5 percent of the administered dose was detected in the faeces, and the radiolabel was not exhaled as 14CO2.

In humans, absorption and excretion of cyanuric acid have been studied in long-distance swimmers exposed by swimming in pools disinfected with chlorinated isocyanurates and in two volunteers given an unspecified solution of cyanuric acid orally. More than 98 percent of the administered dose was recovered unchanged in urine after 24 hours. The half-life of excretion was about three hours (WHO 2009a).

Both NaDCC and sodium cyanurate have low acute oral toxicity. Sodium cyanurate did not induce any genotoxic, carcinogenic or teratogenic effects. The USFDA permits a certain amount of cyanuric acid to be present in some [non-protein nitrogen additives](http://en.wikipedia.org/wiki/Chinese_protein_export_scandal#Non-protein_nitrogen_as_legitimate_and_illegitimate_feed_additive) used in animal feed and drinking-water.

The Acceptable Daily Intake (ADI) adopted in Australia for sodium cyanurate is 0.5 mg/kg body weight, with a NOEL of 50 mg/kg bw.

Sodium dichloroisocyanurate used for disinfecting drinking-water should be of adequate purity so that there is no increase in any inorganic or organic contaminants in the drinking-water.

USEPA (1992) states that carcinogenic effects were not noticed in two-year rat studies and two-year mouse studies involving doses up to 1500 mg/kg/d via their drinking water.

### Derivation of Maximum Acceptable Value

No MAV as at DWSNZ 2008.

If a MAV is to be adopted for cyanuric acid in a future DWSNZ based on WHO (2017) it would be calculated as follows:

154 mg/kg body weight per day x 70 kg x 0.8 = 43 mg/L (rounded to 40 mg/L)

2 L x 100

where:

* NOEL = 154 mg/kg body weight
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.8
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 100 for intra- and interspecies variation.

Likewise for sodium dichloroisocyanurate:

220 mg/kg body weight per day x 70 kg x 0.8 = 61.6 mg/L (rounded to 60 mg/L)

2 L x 100

where:

* NOEL = 220 mg/kg body weight
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.8
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 100 for intra- and interspecies variation.

The TDI determined was 2.2 mg/kg of body weight as sodium dichloroisocyanurate, and 1.54 mg/kg body weight for cyanuric acid. This was based on a NOEL of 154 mg/kg body weight per day (equivalent to 220 mg/kg body weight per day as anhydrous sodium dichloroisocyanurate) for urinary tract and cardiac lesions from a two-year study of rats exposed to sodium cyanurate and using an uncertainty factor of 100 for interspecies and intraspecies variation.

However, the controlling factors would be the level of free chlorine and the residue of cyanuric acid, particularly if there was topping up of chlorine in a static system under emergency conditions. The concentration of free chlorine should normally be such that it should not give rise to unacceptable tastes and should not normally exceed the MAV of 5 mg/L for free chlorine.

It should be noted that the amounts of NaDCC used should be the lowest consistent with adequate disinfection and that the concentrations of cyanuric acid should be managed to be kept as low as is reasonably possible.

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# Β-cyclocitral

CAS No. 432-25-7. Also called beta-cyclocitral, 2,6,6-trimethyl-1-cyclohexene-1-carboxaldehyde, 2,6,6-trimethyl-1(2)-cyclohexen-1-carboxaldehyde.

### Maximum Acceptable Value

There is no MAV for β-cyclocitral in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

β-Cyclocitral is formed by cyanobacteria in reservoirs and rivers. It is mainly produced during cell lysis or decomposition. It imparts an aromatic odour to water, reminiscent of grass, hay or dry wood at low concentrations, becoming more tobacco-like and fruity at higher concentrations. It may be found together with other carotenoid breakdown products such as beta-ionone.

β-Cyclocitral is a certified water flavour standard used to train professional tasters to recognise and scale the intensity of tobacco-like character; it is marketed by Aroxa.

Large amounts of the musty-tobacco-smelling pigment derivative β-cyclocitral are generated at cell damage or death by all *Microcystis* spp. via enzyme-mediated catalysis.

β-Cyclocitral is often present in eutrophic waters and is a well known source of airborne and drinking water malodour, and is derived from the catalytic breakdown of β-carotene. Evidence indicates that it is produced by the activation of a specific carotene oxygenase by all species of the bloom-forming cyanobacterium *Microcystis*, and acts as a grazer defence signal unique to the cyanobacterium *Microcystis* (Juttner et al 2010).

### Form and fate in the environment

β-Cyclocitral is short-lived in water and rapidly lost by volatilisation and chemical–photooxidative breakdown. As a result, *Microcystis* rarely causes significant long-lasting off-flavour in treated drinking water supplies because it does not produce either of the more resilient T & O compounds geosmin and 2-MIB (Watson et al 2008).

### Typical concentrations in drinking-water

β-Cyclocitral is normally found in eutrophic waters at the nanogram to microgram/L level, although it has been found at 0.75 mg/L in windblown surface scums.

### Removal methods

Aeration processes should reduce the concentration of β-cyclocitral in water. Chlorination seems to stop *Microcystis* producing β-cyclocitral, probably by inhibiting the enzyme process responsible for its production.

### Analytical methods

See Furtula et al (2004) and EFSA (2012).

### Health considerations

There are no reports on the health effects of β-cyclocitral.

The flavour threshold of beta-cyclocitral in water is reported by Aroxa as 150 ng/L (0.15 µg/L or 0.00015 mg/L). It has also been reported to be 19,000 ng/L (19 µg/L or 0.019 mg/L) by Furtula et al (2004); and 1–20 µg/L in WRc (2001).

### Derivation of Maximum Acceptable Value

No MAV as at DWSNZ 2008.

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# Cyclohexane

CAS No. 110-82-7. Also called hexahydrobenzene, hexamethylene, benzenehexahydride and hexanaphthene.

### Maximum Acceptable Value

There is no MAV for cyclohexane in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

The primary use of cyclohexane (in the US) accounting for approximately 58 percent of all use, is in the production of adipic acid, a nylon intermediate (USEPA 1992). Cyclohexane is also used in the production of caprolactam (CAS No. 105-60-2, hexahydro-2*H*-azepin-2-one), another nylon intermediate; it is also used to make resins and plastics. Small amounts of cyclohexane are used as a solvent for adhesives (around 10–15 percent), lacquers and resins; as a paint and varnish remover; as an intermediate in the manufacture of benzene (qv), cyclohexanone (CAS No. 108-94-1), and nitrocyclohexane; as fuel for camp stoves; and as an ingredient in fungicidal formulations. Cyclohexanone is used primarily as an intermediate in the production of nylon; it is also of the Substances in the JECFA Flavouring Group of Alicyclic Ketones, Secondary Alcohols and Related Esters.

Cyclohexane occurs naturally in crude oil and may be released to the environment from sites where petroleum products are refined, stored, and used (USEPA 1994). It is also released into the atmosphere from volcanos and tobacco smoke. The chemical is present in exhaust gases from motor vehicles.

Clean surface water samples generally contain <0.001 mg/L cyclohexane (EU 2004). A survey was carried out in the UK of 32 public and private supply boreholes in three major British aquifer systems in areas of sandstone, limestone and chalk. Cyclohexane was one of the most commonly occurring compounds found in the 32 samples collected. The overall average concentration was 0.022 μg/L and the maximum concentration was 0.08 μg/L.

### Form and fate in the environment

Cyclohexane is volatile (vapour pressure 77 mm Hg at 20°C; Henry’s Law constant, 0.195 atm.m3/mol at 25°C) and is expected to partition into the atmosphere from both water and soil. Cyclohexane has the potential to leach through soil into groundwater. Volatilisation and leaching are the primary removal mechanisms for cyclohexane in soil. The chemical is resistant to biodegradation under most conditions, unless other degradable hydrocarbons, such as oil and gasoline, are present. The primary route for the removal of cyclohexane from the aquatic environment is volatilisation (half-life in a model river is 2–3 hours and 3–4 days in a model lake).

EU (2004) quotes: water solubility = 58 mg/L at 25°C; vapour pressure = 10,300 Pa at 20°C; n-octanol/water partition coefficient = Pow = 3.44; based on the molecular structure of cyclohexane, hydrolysis is not expected to be an important fate process; overall, it can be concluded that cyclohexane is readily biodegradable in the aquatic environment.

If released to soil, cyclohexane is expected to have moderate mobility based upon an estimated Koc of 160. Volatilisation from moist soil surfaces is expected to be an important fate process based upon an experimental Henry’s Law constant of 0.15 atm‑cu m/mole. Cyclohexane may potentially volatilise from dry soil surfaces based upon its vapour pressure. Little mineralisation was detected in a soil biodegradation test and aqueous screening biodegradation tests. If released into water, cyclohexane is expected to adsorb to suspended solids and sediment in the water column based upon the estimated Koc. Cyclohexane is highly resistant to biodegradation and is catabolised chiefly by co-oxidation (use of other organic matter as a carbon and energy source). Volatilisation from water surfaces is expected to be an important fate process based on its estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are three hours and 3.6 days, respectively. An estimated BCF of 89 suggests the potential for bioconcentration in aquatic organisms is moderate. Hydrolysis is not expected to occur due to the lack of hydrolysable functional groups (EAWAG accessed February 2015).

Water solubility of cyclohexane is about 55 mg/L. Water solubility of caprolactam is >100 percent.

### Typical concentrations in drinking-water

A contaminated drinking water well in NY contained 0.54 mg/L of cyclohexane. While a public drinking water supply in East Anglia, England that was 210 m from a leaking petroleum storage tank contained 0.01 ppb of cyclohexane, the groundwater 10 and 110 m from the tank were 10 and <1 ppb, respectively. Cyclohexane was found in five of 14 waterworks sampled in the UK. See <http://www.speclab.com/compound/c110827.htm>.

Up to 80 percent of the cyclohexane can be stripped from the water in the shower box (Moya et 1999).

### Removal methods

Aeration processes should reduce the concentration of cyclohexane in water.

### Health considerations

Cyclohexane has low acute toxicity. There are insufficient data to calculate a RfD (USEPA 2003).

In 1999 the USEPA characterised cyclohexane as “Data are inadequate for an assessment of human carcinogenic potential”.

As a matter of interest, the RfD for caprolactum is 0.5 mg/kg/d (USEPA 1994a). IARC (1999) states that caprolactam is probably not carcinogenic to humans (Group 4).

IARC (1999) stated that cyclohexanone is not classifiable as to its carcinogenicity to humans (Group 3).

### Derivation of Maximum Acceptable Value

No MAV as at DWSNZ 2008.

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# Dialkyltins

The organotins are a large class of compounds that differ in their properties and applications. They can be divided into four groups with the general formulas R4Sn, R3SnX, R2SnX2 and RSnX3, where R is usually an organic group and X is an anion (eg, chloride, fluoride, oxide or hydroxide). In the case of the compounds used as heat stabilisers, the alkyl group can be methyl, butyl, octyl or dodecyl, and the most common are produced by the reaction of mono- and dialkyltin chlorides with mercaptoesters to give thioglycolates.

Some of the commoner dialkyltin compounds are:

dimethyltin dichloride CAS No. 753-73-1

dibutyltin dichloride CAS No. 683-18-1

dioctyltin dichloride CAS No. 3542-36-7.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for individual dialkyltins in drinking-water.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set a health-based guideline value for dialkyltins in drinking water.

### Sources to drinking-water

#### 1. To source waters

Of the large group of compounds known as the organotins the mono- and di- substituted compounds are the ones most likely to be found in water.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

The di-substituted organotins are employed as stabilisers in plastics, including PVC and CPVC water pipes, since around the 1960s. PVC is the only plastic in which organotin stabilisers are used. Stabilisers are used in all PVC products in order to avoid decomposition while heating during processing and also to reduce deterioration through exposure to ultraviolet light and weathering. Levels of organotins in rigid PVC are 1–1.5 percent. The minor use in flexible PVC is probably more in the range of  
0.8–1.2 percent due to the plasticiser present (WHO 2006).

The following has been copied from TSA (2002):

A number of studies have been conducted in North America on the presence of tin stabiliser compounds in water from new and existing PVC and CPVC pipe installations. These studies show that, while organotins have been detected, the levels are too low to cause a concern for effects on human health. The amounts detected are well below the safe Short Term Exposure Levels (STEL) and Maximum Allowable Levels (MAL) established by NSF Standard 61. For example, single time point testing results for organotin extraction from PVC and CPVC pipe, fittings and materials under Standard 61 showed average levels in CPVC at 0.01 mg/L, and in PVC at 0.006 mg/L – well below levels that would present a human health risk.

When tin stabilisers are detected in the water, it is usually just after new pipes have been installed. This is because residues of some of the materials used in the pipe formulation may be left on the pipe wall after manufacturing, and these residues – which may include tin – are quickly washed into the water as it flows through the pipes. After about 12 hours the levels of tin are negligible. After a few litres of water pass through the system, levels drop to the parts per trillion range – well below established safe levels.

Occasionally, regulators, customers and environmental organisations have expressed some concerns about tin stabilisers because they mistakenly equate these products with tributyltin (TBT) biocides. TBT is used primarily in marine antifoulant paints and is a registered pesticide in the US and Canada. Many countries have regulated its usage and application. TBT is never used as a stabiliser, and tin stabilisers do not have biocidal properties. Concern over TBT in the environment has raised concern about all organotin compounds. This concern is misplaced, as tin stabilisers have been safely used in vinyl products for many years without cause for concern.

It is important, therefore, to clearly distinguish between TBT and tin stabilisers on issues related to environmental and human health effects.

### Forms and fate in the environment

Unknown quantities of organotins may be released into air from factories that produce polyurethane or PVC resins stabilised with organotins.

Standard tests using the organotin compounds show ready biodegradation. However, there is some doubt as to whether this reflects full degradation or dissociation of the ligand. For the purposes of fate modelling and risk assessment, the compounds have been assumed to be “inherently” biodegradable, giving a default half-life of 150 days. Measured half-lifes in soils for dialkyltins are around 120–150 days in laboratory tests. Methyltins and butyltins in forest soils showed half-lifes ranging from six months to 15 years (WHO 2006).

There is little information given on the fate of organotins in the aquatic environment. WHO (2006) reports results of biodegradation according to respiration inhibition test OECD 301F:

dimethyltin 45 percent degradation in 28 days; 58 percent in 39 days

dibutyltin 35 percent degradation in 28 days; 56 percent in 74 days

dioctyltin 36 percent degradation in 28 days; 49 percent in 74 days.

### Typical concentrations in drinking-water

No data are available on the concentration of dialkyltins in New Zealand drinking-water supplies.

There is evidence for organotin stabilisers leaching from plastic pipes, with one overseas study reporting a concentration for dibutyltin sulphide of 0.1 mg/L in a static water that had been in contact with a plastic pipe. Studies by Health Canada found levels of monobutyltin and dibutyltin in potable water in PVC and CPVC pipes in the ng/L range; the maximum concentrations detected were 28.5 ng/L for monobutyltin and 53 ng/L for dibutyltin; note that 53 ng/L = 0.053 µg/L or 0.000053 mg/L.

### Removal methods

No information is available on processes that can be used to remove these compounds from water.

Consumer exposure to these compounds could be reduced by ensuring that new piping, from which organometallic compounds are likely to leach, are filled with water, the pipes left filled for several days, and then flushed. The process should be repeated a number of times. Monitoring the levels of the metals at the beginning of each cycle will indicate the extent to which the leaching is still occurring.

### Analytical methods

#### Referee method

A referee method cannot be selected for dialkyltins because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for dialkyltins because a MAV has not been established. However, the following information may be useful.

The organotins can be analysed using a solvent extraction procedure (Greaves and Unger 1988). They are extracted using a hexane-tropolone mixture and derivatised to form hexylbutyltins. Analysis is by gas chromatography with flame photometric detection. Limits of quantification are less than 0.000002 mg/L (2 ng/L).

### Health considerations

Available data suggest that organotins are poorly absorbed. Following oral administration in rodents they tend to be distributed primarily in the liver and kidney and excreted in the faeces.

The principal effect on rats fed diets containing dioctyltin dichloride for six weeks was a reduction in thymus weight. Rats administered a mixture of octyltin trichloride and dioctyltin dichloride in the diet had highly significant increased frequency of primary tumours of the thymus.

No evidence of mutagenicty was observed with dioctyltin dichloride and dibutyltin diacetate. Dibutyltin dichloride and dioctyltin dichloride have been reported to be positive in mammalian cell mutation assays *in vitro* in the absence of metabolic activation, and dibutyltin sulphide increased the incidence of chromosomal aberrations in rat bone marrow cells *in vivo*.

Neurotoxicity is the major end-point for the methyltins, with a NOAEL of approximately 0.6 mg/kg body weight based on neuropathology for dimethyltin. No neurotoxicity was found with dibutyltin or mono- and dioctyltins (WHO 2006).

Developmental toxicity is shown by the disubstituted methyl-, butyl-, and octyltins, but not by the corresponding monosubstituted compounds. The major reported effect is teratogenicity, with effects on fetuses shown at doses close to maternally toxic ones in most cases. NOAELs for dimethyltin, dibutyltin, and dioctyltin are 10 (10), 2.5 (1.0), and 45 (30) mg/kg body weight per day for teratogenicity (maternal toxicity NOAELs in parentheses). The vast majority of *in vivo* tests show no genotoxicity of mono- and dialkyltins (WHO 2006).

WHO (2011) states: The disubstituted compounds that may leach from PVC water pipes at low concentrations for a short time after installation are primarily immunotoxins, although they appear to be of low general toxicity.

ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.005 mg/kg/day for intermediate-duration oral exposure (15–364 days) for dibutyltin dichloride.

### Derivation of Maximum Acceptable Value

There are insufficient data available to propose MAVs for individual dialkyltins. Leaching of dialkyltins and the mono derivatives used as stabilisers in PVC and CPVC is normally controlled by product specification.

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# Diallyldimethylammonium chloride

CAS No. 7398-69-8. Also called N,N-dimethyl-N-2-propenyl-2-propen-1-aminium chloride, or DADMAC.

### Maximum Acceptable Value

There are insufficient data to derive a MAV for diallyldimethylammonium chloride in drinking-water.

### Sources to drinking-water

#### 1. To source waters

Also used in the food-grade paper industry.

#### 2. From treatment processes

A quaternary ammonium compound, used in the manufacture of polyDADMAC polymer used in water treatment. PolyDADMAC is produced from the diallyldimethylammonium chloride (DADMAC) monomer, which is made from allyl chloride and dimethylamine. Its use is covered by AWWA Std B451-04: Poly (diallyldimethylammonium chloride), which stipulates a maximum DADMAC content of 5.0 percent of active product weight. The monomer may comprise up to 1 percent of the polymer, and is the main impurity. Diallyldimethylammonium chloride has been reported to be a contaminant in the final product, along with diallylether and 5‑hexanal (Letterman and Pero 1990).

PolyDADMACs are exclusively cationic with maximum charge density, are often used as primary coagulants and generally used at higher dose rates than polyacrylamides. Another sub-class is the DADMAC-acrylamide copolymers, which have a higher molecular weight and lower charge density than polyDADMAC.

The Australian National Health and Medical Research Council recommends a maximum DADMAC content of 2 percent, and a maximum polyDADMAC dose rate of 10 mg/L. This equates to a maximum residual monomer level of 0.2 mg/L (quoted in Stockham and Morran 2000). BS EN 1408:1998 and the Scottish Government (2004) state that the poly(diallyldimethylammonium chloride) dose used must not exceed 10 mg/L of active ingredient; *Australian Drinking Water Guidelines*, NHMRC, Polydiallyldimethylammonium chloride fact sheet.

#### 3. From the distribution system

Disinfection by-products resulting from the use of polyDADMAC are discussed by Stockham and Morran (2000).

### Analytical methods

#### Referee method

A referee method cannot be selected for diallyldimethylammonium chloride because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended because a MAV has not been established. However, the following information may be useful: see Fen Jin et al (2005).

### Health considerations

A single oral dose of the polymer was administered to five male and five female rats at 5 mg/kg bodyweight. While no mortalities were observed, pilo-erection, hunched posture ,waddling, pallor of the extremities, and diarrhea were observed in all animals shortly after dosing. These effects were no longer apparent at Day 7, and terminal autopsy findings were normal.

In contrast to polyacrylamide, the toxicity of the DADMAC monomer is less than that of the polymer; DADMAC has a LD50 (mouse) of 7,100 mg/kg, while the polymer has an LD50 of 1,720 mg/kg (Stockham and Morran 2000).

### Derivation of Maximum Acceptable Value

There are insufficient data available to propose a MAV for diallyldimethylammonium chloride. The substance is usually controlled by product specification.

### References

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# 1,4-diaminobutane (putrescine)

CAS No. 110-60-1. The IUPAC name is butane-1,4-diamine. Commonly known as putrescine or putrescin. Some commercial preparations contain 2.7 g/kg 1,4‑diaminobutane, 91 g/kg trimethylamine hydrochloride and 211.3 g/kg ammonium acetate.

### Maximum Acceptable Value

There is no MAV for 1,4-diaminobutane in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

1,4-Diaminobutane is used in Europe as an insect attractant in orchards (fruit crops), citrus and other crops where *Ceratitis capitata* (Mediterranean fruit fly) causes damage (EFSA 2011). It is not listed NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2012 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)).

Putrescine is one of the five common biogenic amines, the others being tyramine, spermidine, spermine and cadaverine.

Putrescine, a polyamide, is a foul-smelling organic chemical that is related to [cadaverine](http://en.wikipedia.org/wiki/Cadaverine); both are produced by the breakdown of [amino acids](http://en.wikipedia.org/wiki/Amino_acid) in living and dead organisms and both are toxic in large doses. The two compounds are largely responsible for the foul odor of [putrefying](http://en.wikipedia.org/wiki/Putrefaction) flesh, but also contribute to the odour of such processes as [bad breath](http://en.wikipedia.org/wiki/Bad_breath) and bacterial [vaginosis](http://en.wikipedia.org/wiki/Vaginosis). They are also found some microalgae. Many bacteria that occur naturally in water can produce putrescine.

Putrescine is reacted with [adipic acid](http://en.wikipedia.org/wiki/Adipic_acid) to yield the [polyamide](http://en.wikipedia.org/wiki/Polyamide) [Nylon](http://en.wikipedia.org/wiki/Nylon)-4,6.

EA (2004) states that “Other odorous compounds are indole and skatole (from tryptophan), several amines (for example, putrescine, cadaverine and β‑phenylethylamine) and fatty acids (for example butyric, propionic and stearic acids).”

While volatile sulfur compounds are the principal causative agents of bad breath, the bacteria that live in our mouth produce other waste products too. Some of these have their own unique and unpleasant smell. A few of these waste by‑products (usually from anaerobic bacteria) are:

* cadaverine – the smell we associate with corpses
* putrescine – for much of the foul odour produced by decaying meat
* skatole – the characteristic smell of human faecal matter, and skunks
* isovaleric acid – the smell of sweaty feet.

### Forms and fate in the environment

1,4-Diaminobutane is volatile.

Water solubility is about 88 percent.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

The relevant short-term oral toxicity No Observed Adverse Effect Level (NOAEL) is 180 mg/kg bw/day, based on decreased food consumption (male rats), decreased body weight, and increased alanineaminotransferase and relative brain weight in females.

1,4-Diaminobutane did not show any genotoxic potential, however no long-term toxicity and carcinogenicity studies were submitted. In a developmental study of limited validity no teratogenicity was observed. For the representative use, the vapour releasing dispensers of the preparation are placed inside physical traps and the active substances, which are fully contained within the polymeric dispensers, do not come into contact with crops, therefore, an Acceptable Daily Intake (ADI) and an Acute Reference Dose (ARfD) were not established (EFSA 2011).

Til et al (1997) had reported the no-observed-adverse-effect level was 180 mg/kg body weight/day for tyramine, cadaverine and putrescine, 83 mg/kg body weight/day for spermidine and 19 mg/kg body weight/day for spermine.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Dibenz[a,h]anthracene

Dibenz[a,h]anthracene, CAS No. 53-70-3, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. Also called 1,2:5,6-dibenzoanthracene.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; benzo[a]pyrene (BaP) levels range from 6 to 35 mg/kg TEQ depending on land use. The equivalent BaP concentration is calculated as the sum of each of the detected concentrations of nine carcinogenic PAHs which are benz(a)anthracene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene and indeno(1,2,3-cd) pyrene, multiplied by their respective potency equivalency factors.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Typical concentrations in drinking-water

Twelve water utilities in the US reported detecting dibenz[a,h]anthracene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00025 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess. The relative carcinogenic potencies of various polynuclear aromatic hydrocarbons, based on bioassays by several routes of administration and related toxicological data, have been ranked in decreasing order as follows: dibenz[a,h]anthracene, benzo[a]pyrene, anthanthrene, indeno[1,2,3-cd]pyrene, benz[a]anthracene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, benzo[j]fluoranthene, cyclopentadieno[c,d]pyrene, benzo[g,h,i]perylene, chrysene and benzo[e]pyrene.

The USEPA (1990) classified dibenz[a,h]anthracene as B2; probable human carcinogen.

IARC (2010) classified dibenz[*a*,*h*]anthracenein Group 2A (probable human carcinogen).

Dibenz[*a*,*h*]anthraceneappears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Dibromoacetic acid

CAS No. 631-64-1. The IUPAC name is dibromoethanoic acid. Dibromoacetic acid is one of the USEPA’s 5 haloacids (HAA5); the others are monochloroacetic acid, dichloroacetic acid, trichloroacetic acid and monobromoacetic acid. Refer also to the haloacetic acids datasheet.

### Maximum Acceptable Value

WHO (2004 and 2011) states that the available data relating to dibromoacetate was considered inadequate to permit recommendation of a health-based guideline value.

The total maximum contaminant level (MCL) for the 5 haloacetic acids (USEPA 2006) is 0.06 mg/L.

### Sources to drinking-water

#### 1. To source waters

No known sources: no reported industrial uses.

#### 2. From treatment processes

Brominated acetic acids are formed during disinfection (with ozone) of water which contains bromide ions and organic matter. Bromide ions occur naturally in surface water and groundwater and exhibit seasonal fluctuations in concentrations. Bromide ion concentrations can increase due to saltwater intrusion resulting from drought conditions, or due to pollution. Bromide is introduced into New Zealand surface waters usually by wind blown seaspray.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

IARC (2002) describes dibromoacetic acid as “very soluble in water”. Health Canada (2008) quotes 211 percent; Log octanol/water partition coefficient = 1.22.

No other information available.

### Typical concentrations in drinking-water

ESR (2001) reported that 488 samples were tested for dibromoacetic acid and was found in 12 distribution zones above the detection limit of 0.005 mg/L, with a maximum concentration of 0.013 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found <0.0005 mg/L dibromoacetic acid in the raw water, and 0.0018 to 0.0026 mg/L in the treated water.

Brominated acetates generally are present in surface water and groundwater distribution systems at mean concentrations below 0.005 mg/L.

9,738 water utilities in the US reported detecting dibromoacetic acid in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.14 mg/L.

Drinking-water was studied in Israel because its source water (the Sea of Galilee, a freshwater lake, also called Lake Kinereth) has among the highest natural levels of bromide in the world for surface water (2 mg/L). The concentration of dibromoacetic acid was 12.5 μg/L (for chloramine plus chlorine dioxide disinfection); between 12 and 38.7 μg/L (for chlorination); and between 14.1 and 23.3 μg/L (for chlorination plus chlorine dioxide disinfection).

In comparison, water collected from 53 Canadian drinking-water treatment facilities in the winter of 1993 were tested for dibromoacetic acid. When bromide concentrations were very low (<0.01 mg/L), the water contained <0.01 μg/L dibromoacetic acid; when they were low (0.06 mg/L), the water contained 0.9 μg/L dibromoacetic acid; and when they were moderate (0.5 mg/L), the water contained 0.8 μg/L dibromoacetic acid.

### Removal methods

Brominated acetic acids arise in waters as a disinfection by-product, so the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the ozone.

Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps.

### Analytical methods

#### Referee method

A referee method cannot be selected for dibromoacetic acid because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

Liquid/liquid extraction, gas chromatography-electron capture detection (APHA 6251B; EPA 552.3). DWI (2011) includes a thorough discussion on the analysis of the nine haloacetic acids.

### Health considerations

Dibromoacetic acid is rapidly absorbed into the bloodstream from the gastrointestinal tract following oral exposure in rats; the blood concentration peaked at approximately one hour post-dosing. The database for dibromoacetic acid is considered inadequate for the derivation of a MAV. There are no systemic toxicity studies of subchronic duration or longer in humans. Available subchronic and chronic studies suggest that the liver is the target organ.

Available mutagenicity data suggest that dibromoacetate is genotoxic. Potential health impacts associated with dibromoacetic acid include endocrine toxicity.

NTP (2007) indicates that there was clear evidence of carcinogenic activity of dibromoacetic acid in mice, based on increased incidences of hepatocellular neoplasm (both sexes) and hepatoblastomas (male only). It also finds some evidence of carcinogenic activity in rats based on an increased incidence of malignant mesothelioma in males and an increased incidence and positive trend of mononuclear cell leukaemia in females.

Health Canada (2008) has classified dibromoacetic acid as Group II, probably carcinogenic to humans, based on sufficient evidence in animals and inadequate evidence in humans. Animal studies have shown links between exposure to DBA and tumours in several organs in both mice and rats. DBA has not been evaluated or classified by IARC.

Health Canada (2008) states: the estimated unit lifetime risks associated with ingestion of 0.001 mg/L of DBA in drinking water were estimated to range from 0.14 × 10−6 to 4.26 × 10−6. The unit risk range was derived from mesotheliomas observed in male rats (0.14 × 10−6) as its lower bound (least sensitive) and hepatocellular adenoma/carcinoma in male mice (4.26 × 10−6) as its upper bound (most sensitive). Using the most conservative concentration in drinking-water estimated for a 10−5 lifetime human cancer risk, a health-based target of 0.002 mg/L (rounded) is derived for DBA in drinking water.

This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

In Stage 1 and 2 D/DBPRs, USEPA did not set an RfD or MCLG for DBAA due to lack of appropriate data on the dose-response for relevant health effects. The 2007 NTP study on DBAA showed clear evidence of carcinogenicity in male and female mice and some evidence of carcinogenicity in male and female rats. These data suggest the need for a new assessment for DBAA. Copied from USEPA 2016.

IARC (2012) considered that dibromoacetic acid is possibly carcinogenic to humans (Group 2B).

### Derivation of Maximum Acceptable Value

No MAV.

IPCS (in WHO 2004a) states that a health-based value of 0.1 mg/L (rounded figure) may be derived but the database is considered to be inadequate for derivation of a guideline value.

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# Dibromoacetonitrile

CAS No. 3252-43-5. The IUPAC name is dibromoethanenitrile. Also called 2,2‑dibromoacetonitrile and dibromocyanomethane. Datasheets also exist for 2‑chloroacetonitrile, 2,2-dichloroacetonitrile, 2-bromo-2-chloroacetonitrile, 2,2,2‑trichloroacetonitrile, and bromoacetonitriles.

### Maximum Acceptable Value

Based on health considerations, the concentration of dibromoacetonitrile in drinking-water should not exceed 0.08 mg/L.

The sum of the ratio of the concentrations of dibromoacetonitrile and dichloroacetonitrile to each of their respective MAVs must not exceed one. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, Section 10.2.5.3).

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for haloacetonitriles in drinking water.

### Sources to drinking-water

#### 1. To source waters

Halogenated acetonitriles are not produced on an industrial scale.

#### 2. From treatment processes

Halogenated acetonitriles are produced during water chlorination or chloramination from naturally occurring substances, including algae, fulvic acid and proteinaceous material. In general, increasing temperature and/or decreasing pH have been associated with increasing concentrations of halogenated acetonitriles.

Ambient bromide levels appear to influence, to some degree, the speciation of halogenated acetonitrile compounds. Dichloroacetonitrile is by far the most predominant halogenated acetonitrile species detected in drinking-water from sources with bromide levels of 0.02 mg/L or less. In chlorinated or chloraminated water from sources with higher bromide levels (0.05–0.08 mg/L), bromochloroacetonitrile was the second most prevalent compound. However, none of the treated water from any of these sources had a dibromoacetonitrile concentration exceeding 0.0005 mg/L, including treated water from one source that had a much higher bromide level (0.17 mg/L).

#### 3. From the distribution system

No known sources. Dibromoacetonitrile hydrolyses in the distribution system, particularly at alkaline pHs and in the presence of chlorine. Dibromoacetonitrile has been reported in tap water to be generally 20–50 percent of that at the treatment plants, indicating that hydrolysis occurred during transport.

### Forms and fate in the environment

Haloacetonitriles are reported to undergo hydrolysis in water, yielding non-volatile products. Dibromoacetonitrile is very mobile in soil and is expected to leach; however it is not likely to be found in soil. IARC (2012) describes dibromoacetonitrile as “slightly soluble in water”.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 209 zones, found dibromoacetonitrile concentrations to range from “not detectable” (nd) to 0.0041 mg/L, with the median concentration being “nd” (limit of detection = 0.002 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with dibromoacetonitrile at >50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0003 mg/L dibromoacetonitrile in the raw water, 0.0007 to 0.0009 mg/L in the treated water, and up to 0.002 mg/L in the distribution system.

In the USA, dibromoacetonitrile was detected in groundwater and surface water distribution systems at mean concentrations of 0.00082 and 0.00075 mg/L, respectively, and at high bromate levels, dibromoacetonitrile was found at a concentration of 0.011 mg/L.

DWI (2012) reported a UK study. The lowland water sources that were included in the survey formed more N-DBPs than the upland and groundwater sources. The six treatment works that applied ozone were associated with higher concentrations of HANs and HAcAms than non-ozone treatment works, although this was potentially confounded because all the ozone works were treating lowland source waters which may have had higher N-DBP formation potential. None of the N-DBPs exhibited consistent links with total trihalomethanes (THMs). There were no trends linking HNMs to either THMs or HAA9. The mean HAN concentration was 0.0032 mg/L (3.2 µg/L). The main HANs were dichloroacetonitrile, bromochloroacetonitrile and dibromoacetonitrile. Trichloroacetonitrile was not detected.

Dibromoacetonitrile was found in post-chlorinated drinking water in Western Australia at 0.0266 mg/L, said to be the highest yet recorded (Liew et al 2016).

### Removal methods

As dibromoacetonitrile arises in waters as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

### Analytical methods

#### Referee method

Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551).

#### Some alternative methods

No alternative methods have been recommended for dibromoacetonitrile because no methods meet the required criteria.

### Health considerations

The only known route of human exposure is through chlorinated drinking-water. Haloacetonitriles are rapidly absorbed from the gastrointestinal tract and metabolised to single carbon compounds. Insufficient data are available to determine whether haloacetonitriles can accumulate in specific organs.

Dibromoacetonitrile is currently under test (WHO 2004 and 2011) for chronic toxicity in mice and rats. None of the available reproductive or developmental studies were adequate to use in the quantitative dose–response assessment. The data gap may be particularly relevant since cyanide, a metabolite of dibromoacetonitrile, induces male reproductive system toxicity, and due to uncertainty regarding the significance of the testes effects observed in the 14-day National Toxicology Program (NTP) rat study.

No data are available on the health effects of haloacetonitriles in humans.

Several short-term exposure studies of dibromoacetonitrile on rats have concluded that decreased body weight is the most sensitive end-point.

Dibromoacetonitrile was a direct-acting mutagen in tests on bacteria and induced DNA damage (sister chromatid exchange and DNA strand breaks) in mammalian cells.

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. One of their findings was that for cell induced genomic DNA damage, the rank order from the most genotoxic to the least genotoxic of the DBP classes was haloacetonitriles > haloacetamides > halonitromethanes > haloacetaldehydes > haloacetic acids > >2C‑haloacids > halomethanes.

IARC (1999) concluded that dibromoacetonitrile is not classifiable as to its carcinogenicity in humans (Group 3). However, IARC (2012) considered dibromoacetonitrile is possibly carcinogenic to humans (Group 2B).

DWI (2010) states:

2,2-Dibromoacetonitrile is cytotoxic and causes genotoxicity *in vitro*. The NTP (2008) concluded that under the conditions of the two-year drinking water studies there was clear evidence of carcinogenic activity of dibromoacetonitrile in male rats based on increased incidences of squamous cell papillomas or carcinomas of the oral cavity; adenomas in the glandular stomach of male rats were also considered to be exposure-related. There was some evidence of carcinogenic activity of dibromoacetonitrile in female rats based on an increased incidence of squamous cell papillomas of the oral cavity; increased incidences of basal cell or squamous cell neoplasms of the skin in female rats may have been related to dibromoacetonitrile exposure. There was clear evidence of carcinogenic activity of dibromoacetonitrile in male and female mice based on increased incidences of squamous cell papillomas or carcinomas of the forestomach. Increased incidences of neoplasms in the liver of male mice may have been related to dibromoacetonitrile exposure.

Based on decreased body weight and decreased testes weight and pathology in males, the NOAEL is 12 mg/kg of body weight per day and the LOAEL is 18 mg/kg of body weight per day (NTP 2000c).

Poon et al(2003) found that treatment effects occurred predominantly at 100 ppm and included in both sexes: increased kidney weights, histological changes in the thyroid and bone marrow, and increased peroxisomal enzyme activities; and in males: decreased serum and urinary uric acid levels and indication of oxidative stress. The lowest adverse effect level were around 11.1 and 12.1 mg/kg/day in the males and females, respectively and the NOAEL was therefore judged to be 10 ppm, equivalent to 1.11 and 1.21 mg/kg/day in the males and females, respectively.

NTP (2010) conducted a two-year bioassay for DBAN dissolved in drinking water in male and female F344 rats plus male and female B6C3F1 mice. As a result of this study, the NTP concluded that there was clear evidence of carcinogenicity in male rats, male mice and female mice. Some evidence for carcinogenicity was the finding for female rats. In the male rats at the high dose of 7 mg/kg/day there were two rare glandular stomach adenomas. The incidence of squamous epithelial hyperplasia of the tongue was significantly increased at a dose of 7 mg/kg/day in males while both males and females exhibited hyperkeratosis of the tongue at a dose of 4 mg/kg/day. Precancerous papilloma and keratoacanthoma of the skin displayed a positive trend across the 2, 4 and 8 mg/kg/day doses in females. Due to a low response, this finding was classified as equivocal for DBAN. Tumours were also evident in the forestomach of male and female mice (squamous cell papilloma or carcinoma, combined). They were significantly increased as compared to controls at the high dose of 13 mg/kg/day in males and 11 mg/kg/day in females. In males, hyperplasia of the stomach tissues was present even at the low dose of 4 mg/kg/day. Male mice of the 4 and 7 mg/kg/day dose groups had hepatocellular adenoma, hepatocellular carcinoma or hepatoblastoma (combined), theses finding were high in all dose groups and were classified as equivocal. Water intake was less than that of the control for both the rats and mice suggesting possible taste aversion for the treated drinking water. From USEPA (2016).

### Derivation of Maximum Acceptable Value

A tolerable daily intake approach been used to derive the MAV for dibromoacetonitrile in drinking-water using a no-observable-adverse-effects level determined for effects on body weight in a 90-day study in rats.

The MAV for dibromoacetonitrile in drinking-water was derived as follows WHO (2017):

11.3 mg/kg body weight per day x 70 kg x 0.2 = 0.0791 mg/L (rounded to 0.08 mg/L)

2 L x 1000

where:

* no-observable-adverse-effect level = 11.3 mg/kg body weight per day for decreased body weight in male rats in a 13-week drinking-water study
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000; 100 for intra- and interspecies variation and 10 for the short duration of the study.

The MAV for dibromoacetonitrile in the 1995 and 2000 DWSNZ was 0.2 mg/L, based on the following:

23 mg/kg body weight per day x 70 kg x 0.2 = 0.2 mg/L

2 L x 1000

where:

* no-observable-adverse-effect level = 23 mg/kg body weight per day for the effects of body weight in a 90-day corn oil gavage study in rats
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000; 100 for intra- and interspecies variation and 10 for the short duration of the study.

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# Dibromochloromethane

CAS No. 124-48-1. Also called chlorodibromomethane or DBCM.

### Maximum Acceptable Value

Based on health considerations, the concentration of dibromochloromethane in drinking-water should not exceed 0.15 mg/L.

Action to reduce THMs is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than THMs.

Dibromochloromethane is one of the four trihalomethanes with a MAV in the DWSNZ. The others are bromodichloromethane, bromoform and chloroform. The sum of the ratio of the concentrations of these four trihalomethanes to each of their respective MAVs must not exceed one. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, section 10.2.5.3).

The maximum contaminant level for total trihalomethanes (USEPA 2006/2009/2011) is 0.08 mg/L. The lifetime health advisory for dibromochloromethane is 0.06 mg/L (USEPA 2006/2011) where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) do not include a guideline value specifically for dibromochloromethane. They do have one though for total trihalomethanes (qv).

Chlorodibromomethane is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Brominated trihalomethanes, such as dibromochloromethane, may occur in raw water as an industrial contaminant and from human activity. They have been used as laboratory reagents, chemical intermediates, as fluids for mineral ore separation, as solvents for waxes, fats, and resins, and as flame retardants. Dibromochloromethane is now used only on a very small scale.

#### 2. From treatment processes

Trihalomethanes, including dibromochloromethane, are most likely to be formed as by-products of the chlorination of drinking-water. Naturally-occurring bromide is oxidised by chlorine to form bromine (hypobromous acid and hypobromite ion) which reacts with organic matter, such as humic and fulvic acids, in the water, with the result that the trihalomethanes found in the water show varying degrees of bromine incorporation. When full bromine substitution occurs, bromoform is produced; mixed substitution of chlorine and bromine results in dibromochloromethane and bromodichloromethane. The concentration of trihalomethanes produced depend upon: pH, organic matter concentration, chlorine dose, bromide concentration, contact time and temperature. Being a disinfection by-product, the USEPA (2007) regulates dibromochloromethane.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

In air, brominated trihalomethanes may be removed through oxidation with atmospheric hydroxyl radicals. Dibromochloromethane is quite soluble in water (about 3,000 mg/L). Volatilisation is a major removal mechanism for dibromochloromethane from water. Biodegradation occurs under anaerobic conditions. Hydrolysis is extremely slow. Bioaccumulation in aquatic organisms may occur. Brominated trihalomethanes are expected to be mobile in soil.

### Typical concentrations in drinking-water

The 1992 review of organic contaminants in New Zealand drinking-water supplies from 1987–1992 contained dibromochloromethane results from 370 samples representing 157 supplies. Dibromochloromethane was detected in 242 samples at concentrations ranging from 0.00023–0.029 mg/L.

The P2 Chemical Determinand Identification Programme, sampled from 511 zones, found dibromochloromethane concentrations to range from “not detectable” (nd) to 0.028 mg/L, with the median concentration being “nd” (limit of detection = 0.002 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with dibromochloromethane at >50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0003 mg/L dibromochloromethane in the raw water, 0.006 mg/L in the treated water, and up to 0.010 mg/L in the distribution system.

In the USA, monitoring data were collected over an 18-month period between July 1997 and December 1998 from approximately 300 water systems operating 501 plants and serving at least 100,000 people; the mean concentration of dibromochloromethane was 0.002 mg/L, the median was 0.005 mg/L and the 90th-ile was 0.013 mg/L. 16,561 water utilities in the US reported detecting dibromochloromethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.21 mg/L.

### Removal methods

Dibromochloromethane present in contaminated source waters can be removed by adsorption on to granular activated carbon, or by air stripping. DBCM removal using packed tower aeration depends on the air-to-water ratio, water loading rate, and packing depth; 59 percent removal from an inlet concentration of approximately 0.1 mg/L was achieved in pilot-scale studies.

However, as dibromochloromethane arises in waters principally as a disinfection by‑product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Trihalomethane concentrations in chlorinated water increase with increasing pH. Concentrations in the finished water can therefore be reduced by ensuring that high pH levels are not present once the water is chlorinated.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Dibromochloromethane can be removed by adsorption on to granular activated carbon, or by air stripping. Adsorption efficiency increases, and air stripping efficiency decreases, with increasing bromine substitution in the trihalomethane.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6210D, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551).

### Health considerations

The major route for human exposure to chlorodibromomethane is via drinking-water. Available studies indicate that gastrointestinal absorption is high for all trihalomethanes and because of their high lipophilicity, accumulation is higher in tissues with high lipid content, including body fat, liver and kidneys.

In a 90-day study in rats administered dibromochloromethane in drinking-water, mild to moderate histological changes in the liver and thyroid, and a significant increase in the severity of hepatic lesions, were observed at the highest dose.

Results of studies on the genotoxicity of trihalomethanes in bacteria have been inconsistent, with most reporting negative results.

In a US bioassay, DBCM induced hepatic tumours in female and possibly in male mice but not in rats. The genotoxicity of DBCM has been studied in a number of assays, but the available data are considered inconclusive. The International Agency for Research on Cancer has classified dibromochloromethane in Group 3 (not classifiable as to its carcinogenicity to humans). DBCM is mostly positive in genotoxicity tests employing closed systems to overcome the problem of volatility. The USEPA (2009/2011) quotes a health advisory of 0.08 mg/L for dibromochloromethane, representing a 10-4 cancer risk.

Dibromochloromethane was removed from the State of California EPA list of chemicals known to cause cancer or reproductive toxicity in October 1999.

In the Stage 1 D/DBPR, an MCLG of 0.06 mg/L for DBCM was established by USEPA based on a weight of evidence evaluation of both the cancer and non-cancer effects. At that time DBCM was classified as a “possible human carcinogen”. The MCLG was based on the RfD, an adult tap water consumption of two litres/day for a 70 kg adult, and an additional risk management factor of 10 to account for possible carcinogenicity. The assumed drinking water contribution to total exposure was 80 percent. At the time of the Stage 2 Rule an RfD of 0.02 mg/kg/day was derived based on a NOAEL of 30 mg/kg/day (adjusted to 21.4 mg/kg/day for a five-day/week exposure) for liver effects from the subchronic portion of a NTP (1985) study in rats and an uncertainty factor of 1000. USEPA used the chronic studies of the NTP (1985) study to determine a classification of “*suggestive evidence for cancer”*. No evidence of carcinogenicity was reported in rats, but there was equivocal evidence of carcinogenicity in male mice and some evidence of carcinogenicity in female mice based on an increased incidence of liver tumours. The RfD value did not change due to the lack of significant new health effects data. USEPA did not revise the MCLG for DBCM in the Stage 2 D/DBPR. Copied from USEPA 2016.

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 MRLs for chlorodibromomethane are:

minimal risk level

0.1 mg/kg/day for acute-duration oral exposure (1–14 days)

0.09 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD (USEPA 1991/2006/2009/2011) is 0.02 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.7 mg/L.

### Derivation of Maximum Acceptable Value

A tolerable daily intake approach has been used (derived on the basis of a NOAEL of 30 mg/kg of body weight per day) for the derivation of the MAV of dibromochloromethane in drinking-water. The no-observable-adverse-effects level used for the derivation of the MAV was determined for the absence of histopathological effects in the liver in a well-conducted and well-documented 90-day study in rats.

The MAV for dibromochloromethane in drinking-water was derived as follows:

30 x (5/7) mg/kg body weight per day x 70 kg x 0.2 = 0.15 mg/L

2 L x 1000

where:

* no-observable-adverse-effect level = 30 mg/kg body weight per day based on the absence of histopathological lesions in the liver in a 90-day study on rats (normalised for five days/week dosing in derivation). This NOAEL is supported by the results of long-term studies.
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for the short duration of the study). An additional uncertainty factor for potential carcinogenicity was not applied because of the questions regarding mice liver tumours from corn oil vehicles and inconclusive evidence of genotoxicity.

The MAV for dibromochloromethane in the 1995 and 2000 DWSNZ was 0.1 mg/L. The basis was the same as above, except the MAV had been rounded to one significant number.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for dibromochloromethane is 0.01 mg/L.

The WHO guideline value only considers exposure to THMs in water via the oral route. However, WHO accepts THMs are volatile chemicals, and therefore, exposure via the inhalation and dermal routes may be significant sources of exposure, particularly during bathing and showering, as increasing water temperature will increase the rate of volatilisation, and ventilation may be poor. WHO suggested that in colder countries with low rates of ventilation in houses or where the incidences of showering and bathing are high, this guideline value may be lowered (WHO 2005, in DWI 2010).

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# Dibromomethane

CAS No. 74-95-3. Also called 1,2-dibromomethane, methylene dibromide, or methylene bromide (incorrectly).

### Maximum Acceptable Value

There is no MAV for dibromomethane in the DWSNZ, and it is not mentioned in the WHO Guidelines.

Dibromomethane does not appear on any national lists of regulated substances.

### Sources to drinking-water

#### 1. To source waters

Dibromomethane is used as a solvent for fats, waxes, and resins, and as a refrigerant, a gauge fluid, and in fire extinguishers. It is used in the production of a range of pesticides. It is naturally produced by marine [algae](http://en.wikipedia.org/wiki/Algae).

Of the 377 and 282 representative samples of groundwater and surface water in New Jersey that were analysed for dibromomethane, 12 percent and 28 percent, respectively, contained dibromomethane. Ninety percent of the samples of both types contained (or less than) 0.0001 mg/L of dibromomethane. The maximum dibromomethane concentration in groundwater was 0.045 and that in surface water was 0.36 mg/L.

#### 2. From treatment processes

Dibromomethane appears on some lists of disinfection by‑products found in chlorinated water. It could be formed following treatment with ozone too.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

If released into water, dibromomethane would be lost primarily by volatilisation (half life 5.2 hours from a model river). Adsorption to sediment should not be significant. If released on soil, it will be expected to volatilise from the soil surface and leach into the ground.

Water solubility is about 1.2 percent.

### Typical concentrations in drinking-water

ESR (2001) reported that 173 samples were tested for dibromomethane and was found in six distribution zones above the detection limit of 0.0005 mg/L, with a maximum concentration of 0.0011 mg/L.

In a survey of 14 treated drinking water supplies of varied sources in England, dibromomethane was detected in seven supplies. These supplies were derived from both groundwater and surface water sources.

125 water utilities in the US reported detecting dibromomethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.007 mg/L.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

The acceptable daily intake is 1 milligram/kilogram bw (TOXNET).

### Derivation of Maximum Acceptable Value

No MAV.

### References

ESR. 2001. *A Report on the Chemical Quality of New Zealand’s Community Drinking Water Supplies*. Client Report FW0120, prepared as part of a Ministry of Health Contract for Scientific Services. 333 pp.

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TOXNET. <http://toxnet.nlm.nih.gov/cgi-bin/sis/search/a?dbs+hsdb:@term+@DOCNO+1334>.

# 2-2-dibromo-3-nitrilopropionamide

CAS No. 10222-01-2. The IUPAC name is 2,2-dibromo-2-cyanoacetamide. Also called DBNPA, 2,2-dibromo-2-carbamoylacetonitrile and dibromonitrilopropionamide.

### Maximum Acceptable Value

There is no MAV for 2-2-dibromo-3-nitrilopropionamide in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

DBNPA or 2,2-dibromo-3-nitrilopropionamide is a quick-kill [biocide](http://en.wikipedia.org/wiki/Biocide) (to control microorganisms including algae, bacteria, and fungi) that easily [hydrolyses](http://en.wikipedia.org/wiki/Hydrolyzes) under both [acidic](http://en.wikipedia.org/wiki/Acidic) and [alkaline](http://en.wikipedia.org/wiki/Alkaline) conditions. It is used for its instability in water as it quickly kills and then quickly degrades to form a number of products, depending on the conditions, including [ammonia](http://en.wikipedia.org/wiki/Ammonia), [bromine](http://en.wikipedia.org/wiki/Bromine) ions, [dibromoacetonitrile](http://en.wikipedia.org/w/index.php?title=Dibromoacetonitrile&action=edit&redlink=1), and [dibromoacetic acid](http://en.wikipedia.org/w/index.php?title=Dibromoacetic_acid&action=edit&redlink=1). DBNPA acts in a similar manner to the typical halogen biocides.

DBNPA is used in a wide variety of applications such as [papermaking](http://en.wikipedia.org/wiki/Papermaking) as a [preservative](http://en.wikipedia.org/wiki/Preservative) in [paper coating](http://en.wikipedia.org/wiki/Paper_coating) and [slurries](http://en.wikipedia.org/wiki/Slurry). It is also used as [slime control](http://en.wikipedia.org/wiki/Biocide) on [paper machines](http://en.wikipedia.org/wiki/Papermachine), and as a [biocide](http://en.wikipedia.org/wiki/Biocide) in [hydraulic fracturing](http://en.wikipedia.org/wiki/Hydraulic_fracturing) wells and in heat exchangers and cooling water. Used at up to 25 ppm in water systems.

#### 2. From the distribution system

No known sources.

### Form and fate in the environment

Because of its use pattern, DBNPA would not generally contaminate groundwater, but would likely contaminate surface waters by discharge or spill.

Hydrolysis and aqueous photolysis studies show that DBNPA degrades to an array of products: dibromoacetonitrile; dibromoacetamide; dibromoacetic acid; monobromoacetamide; monobromonitrilopropionamide; monobromoacetic acid; cyanoacetic acid; cyanoacetamide; oxoacetic acid; oxalic acid and malonic acid. The various pathways that generate these degradation products depend upon pH and the presence of light and nucleophiles.

As the pH increases from neutral the half-life of DBNPA decreases. For instance, at pH 5 the half-life of DBNPA is 67 days as opposed to 63 hours at pH 7 and 73 minutes at pH 9. Hydrolysis produces dibromoacetic acid (30.6 percent of applied) as the major degradate at pH 5, dibromoacetonitrile (54.5 percent) as the major degradate at pH 7, and dibromoacetonitrile (38.6 percent) as the major degradate at pH 9. Aqueous photolysis produces dibromoacetic acid (63.7 percent) as the major degradate at pH 5 (t½ = 14.8 hours) in exposed solutions and dibromoacetic acid (31.4 percent) in the dark control, and dibromoacetic acid as the major degradate in the exposed (66.5 percent) and unexposed (74.9 percent) solutions at pH 7 (t½= 6.9 hours). Additionally, the aqueous photolysis study indicate that:

1) at pH 9 (t½= 0.4 hours) in the solutions exposed to light, bromoacetamide was present at 14.6 percent and an unknown degradate at 61.4 percent;

2) in the unexposed solutions an unknown (possibly dibromoacetonitrile from retention times) was present at 51.7 percent and oxalic acid at 29.9 percent.

In the aerobic and anaerobic aquatic metabolism studies DBNPA degraded with a half-life of <4 hours. DBNPA and degradate concentrations decreased rapidly during the metabolism studies and the majority of the residues were found in the aqueous layer. The six degradates detected were oxalic acid, 2-cyanoacetamide, bromoacetamide, dibromoacetic acid, bromoacetic acid, and dibromoacetonitrile. Although DBNPA degrades into the same six degradates under aerobic or anaerobic aquatic metabolism conditions, the percent of individual degradates present during different periods of time varies with the type of metabolism.

In the anaerobic aquatic metabolism study 2-cyanoacetamide reached a maximum of 56.35 percent of applied by day 7, then decreased to undetectable levels at day 48. Dibromoacetic acid was at 27.3 percent at 0 hours then decreased to 17.0 percent by day 48. Oxalic acid reached 10.3 percent by day 2, and was at 5.4 percent by day 48. Bromoacetamide was at 2.3 percent by day 48. Dibromoacetonitrile reached 1.2 percent then decreased to undetectable levels by day 48. Bromoacetic acid reached 0.7 percent at day 14 then decreased to undetectable levels by day 48.

Three degradates were identified in the sediment phase: 2-cyanoacetamide; dibromoacetonitrile; and bromoacetamide. The major degradate was 2‑cyanoacetamide which reached 15.3 percent of applied by day 7, and decreased to 2.8 percent by day 48. Dibromoacetonitrile was detected once at 0.3 percent on day 2. Bromoacetamide was detected once at 0.2 percent on day 30.

In the aerobic aquatic metabolism study, the degradate found in highest concentration was dibromoacetic acid. This degradate reached 66.45 percent of applied at 0 hours, fell to 9.0 percent at hour 5, and was not detected at day 2. 2-Cyanoacetamide was present at 56.5 percent at hour 5 and fell to 2.3 percent at day 30. Oxalic acid was present at 7.9 percent at hour 5 and remained relatively constant throughout the experiment. Bromoacetic acid was detected once at 2.3 percent on day 14. Bromoacetamide was detected twice at 1.1 percent at hr 5 and 1.4 percent on day 7. Dibromoacetonitrile was detected at 1.8 percent at day zero, and 5.6 percent at day 5. Unknowns were detected at a maximum of 10.9 percent on day 15.

Three degradates were identified in the sediment layer: oxalic acid; 2-cyanoacetamide; and bromoacetamide. The degradate found in highest concentration in the sediment was 2-cyanoacetamide which reached 16 percent of the applied at day 2 and fell to 1.1 percent by day 30. Bromoacetamide was present at 0.2 percent at 0 hours, rose to 2.1 percent by day 2, and was not detected by day 30. Oxalic acid reached 4.5 percent at hour 5, then fell to 0.2 percent by day 30. Unknowns were detected during various times at 0.2 percent.

DBNPA is very soluble in water – about 1.5 percent. The Octanol/Water Partition Coefficient (Kow) is about 6.35 at pH 5 to 9; log Kow is 0.8. The Henry’s Law constant for 2,2-dibromo-3-nitrilopropionamide is estimated as 1.9 x 10-8 atm‑cu m/mole derived from its vapour pressure, 9.0 x 10-4 mm Hg and water solubility. This Henry’s Law constant indicates that 2,2-dibromo-3-nitrilopropionamide is expected to be essentially non-volatile from water and soil surfaces.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

In acute toxicity studies, DBNPA is moderately systemically toxic by the oral and inhalation routes (Toxicity Category II). In a subchronic toxicity study, rats were given DBNPA for 90 days by gavage at doses of 0, 5, 13, or 30 mg/kg/day. The NOEL was 5 mg/kg/day.

The USEPA (1994) has a concern for the potential effect of DBNPA on human developmental toxicity. In an oral developmental toxicity study in rabbits, DBNPA was observed to produce fetal structural alterations at a dose (30 mg/kg/day) which was not maternally toxic. The NOEL for developmental effects was 10 mg/kg/day and the maternal NOEL was 30 mg/kg/day.

### Derivation of Maximum Acceptable Value

No MAV.

### References

USEPA. 1994. *Reregistration Eligibility Decision (RED). 2,2-dibromo-3-nitrilopropionamide (DBNPA)*. EPA 738-R-94-026. 180 pp. <http://www.epa.gov/pesticides/reregistration/REDs/3056.pdf> or <http://www.epa.gov/pesticides/reregistration/status.htm>.

# 2,3-dibromopropan-1-ol

CAS No. 96-13-9. Also called 2,3-dibromo-propanol, 2,3-DBP, glycerol 1,2‑dibromohydrin, *beta*-dibromohydrin. Has also been called 1,2-dibromopropan-3-ol, allyl alcohol dibromide and 2,3-dibromopropyl alcohol.

2,3-Dibromopropan-1-ol belongs to the large, loosely defined group of chemicals called halohydrins. Chlorohydrins occur as well, eg, see datasheet for 1,3 dichloropropan-2-ol. Other halohydrins are listed in the halohydrin datasheet.

### Maximum Acceptable Value

There is no MAV for 2,3-dibromopropan-1-ol in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

2,3-Dibromopropan-1-ol has been used as an intermediate in the preparation of flame retardants, insecticides and pharmaceuticals. In particular, in the 1970s, the major use of 2,3-dibromopropan-1-ol was in the preparation of the flame retardant tris(2,3‑dibromopropyl) phosphate, which was used on textiles; production of this flame retardant other than for research purposes has been discontinued.

#### 2. From treatment processes

2,3-Dibromopropan-1-ol has been reported as a disinfection by-product when using ozone.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

If released to soil, 2,3-dibromopropanol is expected to have very high mobility based upon an estimated Koc of 4. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 6.3 x 10-8 atm‑cu m/mole. Limited data indicate that 2,3-dibromopropanol may biodegrade under aerobic conditions. If released into water, 2,3-dibromopropanol is not expected to adsorb to suspended solids and sediment in the water column based upon the estimated Koc. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. 2,3‑Dibromopropanol may slowly hydrolyse in basic waters although the rate of this process is expected to be slower than for biodegradation (EAWAG accessed February 2015).

Very soluble in water. 2,3-DBP is not expected to adsorb to suspended solids and sediments in water, and volatilisation is not expected to be an important process of elimination from water. The volatilisation half-life in a model river (1 m deep, flowing 1 m/s, and wind velocity of 3 m/s) is estimated at 107 days. The volatilisation half-life in a model lake (1 m deep, flowing 0.05 m/s, and wind velocity of 0.5 m/s) is estimated at 780 days.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

IARC (2000) evaluated the brominated analogue 2,3 dibromo-propanol (2,3 DBP) and considered that “there is sufficient evidence in experimental animals for the carcinogenicity of 2,3 dibromo propanol-1-ol” and in their overall evaluation commented “2,3-dibromopropan-1-ol is possibly carcinogenic to humans (Group 2B). Animal carcinogenicity data related to skin application in mice and rats. In mice it produced tumours of the skin at the site of application and forestomach in both males and females, and tumours of the liver in males. In rats it produced tumours of the skin at the site of application and of the digestive tract, including the mouth, oesophagus, forestomach and intestines, nasal mucosa and zymbal gland in both males and females, and tumours of the liver mammary gland and clitoral gland in females.

In assays, bromopropanols were found to be more toxic than their chlorinated counterparts; a finding consistent with the known reactivity of organohalides, ie, the toxicity of halopropanols increases as electronegativity increases and/or ease of hemolytic cleavage increases (COC 2001).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Dichloroacetic acid

CAS No. 79-43-6. Also called dichloracetic acid, or dichloroethanoic acid (IUPAC), or DCA. Dichloroacetic acid is one of the USEPA’s five haloacids (HAA5); the others are monochloroacetic acid, trichloroacetic acid, monobromoacetic acid and dibromoacetic acid. Refer also to the haloacetic acids datasheet. Sodium dichloroacetate has a CAS No. of 2156-56-1.

### Maximum Acceptable Value (provisional)

Based on health considerations, the concentration of dichloroacetic acid in drinking-water should not exceed 0.05 mg/L.

The WHO 2005 and 2011 guideline value was designated as provisional because the data were insufficient to ensure that the value was technically achievable under a wide range of circumstances.

The sum of the ratio of the concentrations of dichloroacetic acid, monochloroacetic acid and trichloroacetic acid to each of their respective MAVs must not exceed one. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, section 10.2.5.3).

The total maximum contaminant level (MCL) for the 5 haloacetic acids (USEPA 2006/2009/2011) is 0.06 mg/L. There is no lifetime health advisory for dichloroacetic acid (USEPA 2006/2009/2011).

The MAC in Canada is 0.01 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of dichloroacetic acid in drinking water should not exceed 0.1 mg/L.

### Sources to drinking-water

#### 1. To source waters

Dichloroacetic acid may occur in raw water as an industrial contaminant and through human activity. It is used as a chemical intermediate in the synthesis of organic materials, as an ingredient in pharmaceuticals (as a cauterising agent) and medicines, as a topical astringent, and as a fungicide. The commercial grade also contains monochloroacetic acid 0.2 percent maximum; trichloroacetic acid 1.0 percent maximum (IARC 2013). IARC (2013) states that dichloroacetic acid was formulated into pharmaceutical products by one company each in New Zealand.

Table 1.2 of IARC (2013) lists concentration of dichloroacetic acid found as a water pollutant and as a disinfection by-product.

#### 2. From treatment processes

Chlorinated acetic acids are formed from organic material during water disinfection when using chlorine or chloramine. Levels of dichloroacetic acid tend to decline with length of time in the distribution system, and concentrations tend to be higher in warmer seasons. Dichloroacetic acid occurs almost exclusively in the ionised form (dichloroacetate) in normal drinking water (pH range 6 to 9). Dichloroacetic acid is also found in chlorinated swimming pool water of neutral pH. Being a disinfection by-product, the USEPA (2007) regulates dichloracetic acid.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Dichloroacetic acid has a relatively short persistence (about four days) in pond waters. It has been reported as a degradation product of some pesticides (eg, dichlorvos), and chlorinated organic chemicals. It is very soluble in water (miscible). DCA has a very low vapour pressure and is not expected to volatilise from drinking water or contaminated environmental media to any appreciable extent. Log octanol/water partition coefficient = 0.92.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 488 zones, found dichloroacetic acid concentrations to range from “not detectable” (nd) to 0.072 mg/L, with the median concentration being “nd” (limit of detection = 0.005 mg/L). The Priority 2 Identification Programme found 5 distribution zones supplying drinking-water to a total of 3160 people with dichloroacetic acid at greater than the MAV, and 38 distribution zones supplied 68,123 people with >50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0005 mg/L dichloroacetic acid in the raw water, and 0.0014 to 0.0016 mg/L in the treated water.

Data for drinking-water supplies in the USA indicate that dichloroacetic acid was detected in groundwater and surface water distribution systems at mean concentrations of 0.007 and 0.017 mg/L, respectively; concentrations ranged from <0.001 to 0.1 mg/L in surface water distribution systems and from <0.001 to 0.07 mg/L in groundwater systems. Higher concentrations have been reported in swimming pool water, up to 0.4 mg/L.

12,002 water utilities in the US reported detecting dichloroacetic acid in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.18 mg/L.

### Removal methods

There are some reports that granular activated carbon may remove dichloroacetic acid from contaminated source waters.

As dichloroacetic acid arises in waters principally as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

The lower pH associated with higher coagulant doses can lead to increased dichloroacetic acid concentrations. Some control of dichloroacetic acid concentrations can be achieved by increasing the pH at which chlorination is carried out, but this may lead to increased formation of THMs.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

There is no information available as to how the chlorinated acetic acids may be removed from drinking-water, once formed.

### Analytical methods

#### Referee method

Ion Exchange Liquid-Solid Extraction and Gas Chromatography with Electron Capture Detection (EPA 552.1). DWI (2011) includes a thorough discussion on the analysis of the nine haloacetic acids. See also IARC (2013).

#### Some alternative methods

1. Micro Liquid–Liquid Extraction Gas Chromatographic Method (APHA 6251B; EPA 552.3).

### Health considerations

Administering a single oral dose of dichloroacetic acid (as the sodium salt) to young adult rats, found most distributed to the muscles (11.9 percent), liver (6.19 percent), gastrointestinal tract (3.74 percent), fat (3.87 percent) and kidney (0.53 percent). Other tissues, including plasma, spleen, heart, skin, bone, brain, lung and testes, accounted for 9.5 percent of the administered dose. In humans, the average half-life of the parent compound in the plasma was 0.43 hours. Urinary excretion of unchanged dichloroacetate was negligible after 8 hours , and cumulative excretion was less than 1 percent of the total dose in all subjects.

In several bioassays, dichloroacetate has induced hepatic tumours in mice. Data on genotoxicity are inadequate.

Diabetic patients treated orally with dichloroacetate for 6–7 days experienced mild sedation, but no other clinical evidence of adverse effects was noted during, or immediately after treatment. Biochemical effects included significantly reduced fasting blood glucose levels, decreases in plasma lactate and alanine, decreased plasma cholesterol levels, decreased triglyceride levels, elevated plasma ketone bodies and elevated serum uric acid levels. Oral or intravenous therapeutic doses are usually in the range of 25–50 mg/kg of body weight per day.

Dichloroacetate has been used to treat severe familial hypercholesterolaemia, resulting in significantly decreased total serum cholesterol levels. However, one patient experienced diminished deep tendon reflexes and decreased strength in all muscle groups of the lower extremities. After six months these conditions had improved, although serum cholesterol levels returned to their high levels.

In 2002 IARC reclassified dichloroacetic acid as Group 2B (possibly carcinogenic to humans), based on the absence of data on human carcinogenicity and sufficient evidence of its carcinogenicity in experimental animals; this was confirmed in IARC. 2013. This classification replaced an earlier Group 3 classification (not classifiable as to its carcinogenicity in humans) from 1995.

Dichloroacetic acid appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. Health Canada (2008) considers DCA to be a probable carcinogen to humans, based on sufficient evidence in animals and inadequate evidence in humans. Animal studies have shown links between exposure to DCA and liver tumours in both mice and rats.

The USEPA (2009/2011) quotes a health advisory of 0.07 mg/L for dichloroacetic acid, representing a 10-4 cancer risk.

Increased incidences of either liver tumours (adenomas and carcinomas) or preneoplastic lesions (eg, hyperplastic nodules and altered hepatic foci) were reported when mice and rats were exposed to drinking water containing 500–5000 mg DCA/L for periods ranging from 52 to 104 weeks (in Health Canada 2008). Using the most conservative concentration in drinking water estimated for a 10-5 lifetime human cancer risk, a health-based target of 0.01 mg/L is derived by Health Canada for DCA in drinking water.

In Stage 1 D/DBPR, USEPA established an MCLG of zero for DCAA based on a weight of evidence evaluation of both the cancer and non-cancer effects and classified DCAA as a “probable or likely human carcinogen”. The MCLG was based on several studies showing liver tumours in mice and rats from lifetime exposure to DCAA in drinking water. Insufficient evidence existed regarding the mode of carcinogenic action of DCAA; the low-dose extrapolation approach was used to be protective of public health. The RfD of 0.004 mg/kg/day was based on a LOAEL of 12.5 mg/kg/day for effects on the liver, brain and testis in dogs with the application of an uncertainty factor of 3000. USEPA did not revise the MCLG for DCAA in Stage 2 D/DBPR. Copied from USEPA 2016.

The reference dose or RfD (USEPA 2003 and 2003/2006/2009/2011) is 0.004 mg/kg/d, based on a LOAEL of 12.5 mg/kg-day and an uncertainty factor of 3000. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.1 mg/L.

### Derivation of Maximum Acceptable Value

Linear multistage model applied to combined data for carcinomas and adenomas in male mice exposed to doses up to 429 mg/kg body weight per day for up to two years (WHO 2017).

Tumour prevalence data from male mice were used to quantify the cancer risk from dichloroacetic acid. The combined data for carcinomas and adenomas in the mice exposed to doses of 0, 8, 84, 168, 315, or 429 mg/kg of body weight per day for up to two years were plotted using the USEPA’s Benchmark Dose software version 1.3.1. The slope factor of 0.0075 (mg/kg of body weight per day)-1 was derived from the BMDL10 using a linear multistage model of the dose–response data. If it is assumed that a 60‑kg person ingests two litres of water per day, the concentration of dichloroacetic acid in drinking-water associated with upper-bound excess lifetime cancer risks of 10-4, 10-5, and 10-6 are 0.4, 0.04, and 0.004 mg/L, respectively. This corresponds to 0.5, 0.05 and 0.005 mg/L (rounded) for 70 kg body weight.

The concentration associated with a 10-5 upper-bound excess lifetime cancer risk is usually identified as the health-based guideline for drinking-water when the contaminant is a carcinogen. However, it may not be possible to provide for adequate disinfection treatment of potable water and maintain dichloroacetic acid at levels of 0.05 mg/L or less. Accordingly, the MAV is provisionally established as 0.05 mg/L. The MAV is designated as provisional because the data on treatment are insufficient to ensure that the 0.05 mg/L value is technically achievable under a wide range of circumstances.

Difficulties in meeting a MAV must never be a reason to compromise adequate disinfection. It should be possible to achieve a dichloroacetic acid concentration at or below the 0.05 mg/L provisional MAV by appropriate control of the water treatment processes.

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# Dichloroacetonitrile

CAS No. 3018-12-0. IUPAC name is dichloroethanenitrile. Also called 2,2‑dichloroacetonitrile. Datasheets also exist for 2-chloroacetonitrile, 2‑bromo‑2‑chloroacetonitrile, 2,2-dibromoacetonitrile, 2,2,2-trichloroacetonitrile, and bromoacetonitriles.

### Maximum Acceptable Value (Provisional)

Based on health considerations, the concentration of dichloroacetonitrile in drinking-water should not exceed 0.02 mg/L.

The sum of the ratio of the concentrations of dibromoacetonitrile and dichloroacetonitrile to each of their respective MAVs must not exceed one. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, section 10.2.5.3).

The guideline value for dichloroacetonitrile is provisional due to limitations of the toxicological database (WHO 2004 and 2011).

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for haloacetonitriles in drinking water.

### Sources to drinking-water

#### 1. To source waters

No known sources.

#### 2. From treatment processes

Halogenated acetonitriles are produced during water chlorination or chloramination from naturally occurring substances, including algae, fulvic acid and proteinaceous material. In general, increasing temperature and/or decreasing pH have been associated with increasing concentrations of halogenated acetonitriles. Ambient bromide levels appear to influence, to some degree, the speciation of halogenated acetonitrile compounds. Dichloroacetonitrile is by far the most predominant halogenated acetonitrile species detected in drinking-water from sources with bromide levels of 0.02 mg/L or less.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Haloacetonitriles are reported to undergo hydrolysis in water, yielding non-volatile products. Dichloroacetonitrile is very mobile in soil and is expected to leach; however it is not likely to be found in soil.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 209 zones, found dichloroacetonitrile concentrations to range from “not detectable” (nd) to 0.008 mg/L, with the median concentration being “nd” (limit of detection = 0.002 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with dichloroacetonitrile at >50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0003 mg/L dichloroacetonitrile in the raw water, <0.0003 to 0.0003 mg/L in the treated water, and up to 0.0005 mg/L in the distribution system.

Data for drinking-water supplies in the USA indicate that dichloroacetonitrile is present in groundwater and surface water distribution systems at mean concentrations of 0.0009 and 0.0022 mg/L. The highest concentrations of dichloroacetonitrile found in US drinking-water from 1998 to 2003 ranged from 0.001–0.003 mg/L.

Fifty water utilities in the US reported detecting dichloroacetonitrile in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.005 mg/L.

DWI (2012) reported a UK study. The lowland water sources that were included in the survey formed more N-DBPs than the upland and groundwater sources. The six treatment works that applied ozone were associated with higher concentrations of HANs and HAcAms than non-ozone treatment works, although this was potentially confounded because all the ozone works were treating lowland source waters which may have had higher N-DBP formation potential. None of the N-DBPs exhibited consistent links with total trihalomethanes (THMs). There were no trends linking HNMs to either THMs or HAA9. The mean HAN concentration was 0.0032 mg/L (3.2 µg/L). The main HANs were dichloroacetonitrile, bromochloroacetonitrile and dibromoacetonitrile. Trichloroacetonitrile was not detected.

### Removal methods

As dichloroacetonitrile arises in waters as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

### Analytical methods

#### Referee method

Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551).

#### Some alternative methods

No alternative methods have been recommended for dichloroacetonitrile because no methods meet the required criteria.

### Health considerations

The only known route of human exposure for dichloroacetonitrile is through chlorinated drinking-water. Dichloroacetonitrile is well-absorbed from the gastrointestinal tract. Most is excreted in urine, with smaller amounts being eliminated in expired air and faeces. Its metabolites are detected in highest concentrations in liver, blood, muscle and skin. It may be formed in vivo following ingestion of chlorinated water.

Studies of pregnant rats administered high levels of dichloroacetonitrile during gestation report significantly increased foetal resorptions and decreased foetal weight and size. Malformations of the cardiovascular, digestive and urogenital systems were observed.

Potential health impacts associated with dichloroacetonitrile include neurotoxicity and respiratory toxicity.

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. One of their findings was that for cell induced genomic DNA damage, the rank order from the most genotoxic to the least genotoxic of the DBP classes was haloacetonitriles > haloacetamides > halonitromethanes > haloacetaldehydes > haloacetic acids > >2C-haloacids > halomethanes.

Assay results have indicated that dichloroacetonitrile is mutagenic. The International Agency for Research on Cancer has concluded that dichloroacetonitrile is not classifiable as to its carcinogenicity to humans (Group 3).

2,2-Dichloroacetonitrile is cytotoxic and causes genotoxicity *in vitro*. Data developed in subchronic studies provided some indication of NOAEL for the general toxicity of 2,2‑dichloroacetonitrile. The LOAEL is 8 mg/kg of body weight per day, based on increased relative liver weights. A Working Group for the WHO *Guidelines for drinking-water quality* considered 2,2-dichloroacetonitrile (WHO, 1993), and determined a TDI of 15 μg/kg of body weight for 2,2-dichloroacetonitrile based on a NOAEL of 15 mg/kg of body weight per day in a reproductive toxicity study in rats (DWI 2010).

### Derivation of Maximum Acceptable Value

Due to the lack of evidence of the carcinogenicity of dichloroacetonitrile, a tolerable daily intake approach has been used for the derivation of the PMAV for dichloroacetonitrile in drinking-water. A lowest-observable-adverse-effect level determined for adverse effects in a study in rats has been used as the basis of the derivation.

The PMAV for dichloroacetonitrile in drinking-water was derived as follows:

8 mg/kg body weight per day x 70 kg x 0.2 = 0.0187 mg/L (rounded to 0.02 mg/L)

2 L x 3000

where:

* lowest-observable-adverse-effect level = 8 mg/kg body weight per day for increased relative liver weight in male and female rats in a 90-day study
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 3000 (based on a factor of 10 each for intra- and interspecies variation, a factor of 3 for the short duration of the study, a factor of 3 for the use of a minimal LOAEL and a factor of 10 for database deficiencies. Due to the overlap of uncertainty factors, the product of three factors of 10 and two partial factors of 3 (actually the square root of 10) is 3000).

The MAV is designated as provisional because of the limitations of the database (ie, lack of long-term toxicity and carcinogenicity bioassays.

The MAV (provisional) for dichloroacetonitrile in the 1995 and 2000 DWSNZ had been 0.1 mg/L, based on the following:

15 mg/kg body weight per day x 70 kg x 0.2 = 0.1 mg/L

2 L x 1000

where:

* no-observable-adverse-effect level = 15 mg/kg body weight per day for foetal resorptions, decreases in foetal weight and size, and malformations of the cardiovascular, digestive, and urogenital systems in offspring in a teratology study in rats
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation, and 10 for the severity of the effects at doses above the NOAEL).

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# Dichloroanisoles

CAS No. 553-82-2. 2,4-Dichloroanisole. Also called 2,4-dichloro-1-methoxybenzene or 2,4-DCA or 1,3-dichloro-4-methoxybenzene. Methoxybenzenes are also known as phenyl methyl ethers, eg, 2,4-dichlorophenyl methyl ether.

CAS No. 1984-59-4. 2,3-Dichloroanisole or 2,3-dichloro-1-methoxybenzene or 2,3‑DCA.

CAS No. 1984-65-2. 2,6-Dichloroanisole or 2,6-dichloro-1-methoxybenzene or 2,6‑DCA.

CAS No. 36404-30-5. 3,4-Dichloroanisole or 3,4-dichloro-1-methoxybenzene or 3,4‑DCA.

CAS No. 33719-74-3. 3,5-Dichloroanisole or 3,5-dichloro-1-methoxybenzene or 3,5‑DCA.

A datasheet has also been prepared for the trichloroanisoles.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for any chlorinated anisoles; nor do the WHO Guidelines have a guideline value.

### Sources to drinking-water

#### 1. To source waters

The dichloroanisoles have been reported to give rise to tastes or odours in drinking-water. Haloanisoles are ranked among the most powerful odour compounds, with odour thresholds in the low part-per-trillion range.

Aerobic soil microbial methylation reactions can result in 2,4-dichloroanisole, being a major metabolite of 2,4-D, 2,4-DP-p – see datasheets and Allard et al (1987). The odour has been described as musty, sweet or fruity, with an odour threshold in water of about 0.0004 mg/L. Young et al (1996) reported an odour threshold in water of about 0.0002 mg/L, and a taste threshold in water of about 0.00008 mg/L.

2,6-Dichloroanisole odour has been described as musty, medicinal or phenolic, with an odour threshold in water of about 0.00004 mg/L. 2,4-Dichloroanisole odour has been described as musty, sweet, fruity or scented, with an odour threshold in water of about 0.0004 mg/L; in Saxby (1996), reporting earlier work.

Bromoanisoles can be produced as well in bromide-rich waters (Diaz et al 2004). Bromochloroanisoles can form too, causing odours (sometimes described as rubbery) at very low concentrations, eg, 2 to 30 ng/L (Diaz et al (2005).

#### 2. From treatment processes

The base compound, anisole (CAS No. 100-66-3) is also called methoxybenzene or phenyl methyl ether. It is manufactured to prepare fragrances. Because it is not thought to occur in water, disinfection processes are not likely to be the source of any chloroanisoles. Even if anisole appears in water, it only reacts with chlorine at low pH, much less than pH 3 to 4.

#### 3. From the distribution system

Reported in Dietrich (2006) is an observation that biofilm in the distribution system can cause the biomethylation of chlorophenols and bromophenols to form haloanisoles which have earthy and musty odours at concentrations less than 1 ng/L, ie, <0.000001 mg/L.

### Analytical methods

#### Referee method

Not needed.

### Health considerations

Chlorinated anisoles do not present a health risk at the concentrations found in drinking-water.

### Derivation of Maximum Acceptable Value

No MAV.

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# 1,2-dichlorobenzene

CAS No. 95-50-1. Also called ortho-dichlorobenzene or o-dichlorobenzene or 1,2-DCB. A long time ago, called *o*-dichlorobenzol.

Technical-grade 1,2-dichlorobenzene typically consists of 70 to 85 percent 1,2‑dichlorobenzene, <0.05 percent chlorobenzene and <0.5 percent trichlorobenzene, with the remainder as 1,3- and 1,4-dichlorobenzene. Pure-grade 1,2-dichlorobenzene consists of >99.8 percent 1,2-dichlorobenzene.

### Maximum Acceptable Value

Based on health considerations, the concentration of 1,2-dichlorobenzene should not exceed 1.5 mg/L in drinking-water.

The maximum contaminant level or MCL (USEPA 2006/2009/2011) is 0.6 mg/L. The USEPA (2006/2009/2011) also established a lifetime health advisory of 0.6 mg/L, where the lifetime health advisory is the concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The maximum acceptable concentration in Canada is 0.2 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on aesthetic considerations, the concentration of 1,2-dichlorobenzene in drinking water should not exceed 0.001 mg/L, and it would not be a health concern unless its concentration exceeded 1.5 mg/L.

1,2-Dichlorobenzene is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Dichlorobenzenes are used widely in industry and domestic products and therefore are likely to occur in source waters from industrial contamination and human activity. 1,2‑Dichlorobenzene is used primarily as a chemical intermediate for dyestuffs and herbicides (mainly 3,4-dichloroaniline products). It is also used in paint removers, and as a solvent for waxes, gums, resins, tars, rubbers, oils and asphalts, hence its application as a degreaser in grease traps and drains. The predominant use in Australia is as a sheep branding fluid (NICNAS 2001). 1,2-DCB is produced in large quantities as a by-product during the production of 1,4-DCB and can be released into the environment during the disposal of unused supplies.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

The dichlorobenzenes are expected to adsorb readily to soils with high organic content and are not expected to leach appreciably into groundwater (solubility of 1,2‑dichlorobenzene about 150 mg/L). In deep water, the major process for dichlorobenzene removal is likely to be adsorption to sediments. Volatilisation has been found to be the predominant removal process for chlorobenzenes from lakes and coastal seawater. Volatilisation half-life in shallow stream maybe <1 hour and up to 60 days for deep slow moving river (OECD 2005). Dichlorobenzenes may be biodegraded slowly under aerobic conditions but this is not likely to occur under the anaerobic conditions that may exist in lake sediments or groundwaters. Their log octanol-water partition coefficients (Kow) are moderately high, around 3.0 for all three isomers.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 294 zones, did not find any 1,2-dichlorobenzene at detectable concentrations (limit of detection = 0.0005 mg/L) (ESR 2001).

In a study in the USA on the contamination of 685 groundwaters, 1,2-DCB, 1,3-DCB, and 1,4-DCB were detected in 20, 19, and 19 samples at maximum concentrations of 6.8, 0.24, and 1.0 mg/L, respectively.

Two water utilities in the US reported detecting dichlorobenzenes (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.087 mg/L. 42 water utilities in the US reported detecting o-dichlorobenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.011 mg/L.

### Removal methods

Removal of 1,2-dichlorobenzene can be achieved by adsorption on to granular activated carbon, or air stripping.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction Gas Chromatographic/Mass Spectrometric Method (APHA 6410B).

### Health considerations

Sources of human exposure are predominantly air and food; inhalation is expected to be the major route for human exposure.

Dichlorobenzenes are absorbed almost completely from the gastrointestinal tract and distributed primarily to fat and fatty tissue because of their lipophilicity (solubility in fat) and to kidney, liver and lungs. They are metabolised rapidly by oxidation in the liver to chlorophenols and excreted in urine.

Dichlorobenzenes are of low acute oral toxicity in experimental animals. The major target organs are the kidney and liver.

Data concerning the health effects of exposure of humans to dichlorobenzenes are restricted to case reports of accidental exposure or misuse of the products. Reported acute effects following short-term exposure (all of which are reversible) include liver damage, blood disorders and disturbances to the immune system, the central nervous system, or the respiratory tract. Skin pigmentation and allergic dermatitis have followed skin contact.

Several oral studies of 1,2-dichlorobenzene in rats and mice ranging from 10 days to two years duration indicate that the adverse effects include increases in liver and kidney weights and hepatotoxicity. From these repeat dose studies, the NOAEL for non-neoplastic effects was 60 mg/kg bw, while the LOAEL was 120 mg/kg bw due to increased renal tubular regeneration in male mice (OECD 2005).

The balance of evidence suggests that 1,2-dichlorobenzene is not mutagenic in tests with bacteria, and there is no evidence for its carcinogenicity in rodents. The International Agency for Research on Cancer consider that there is evidence suggesting lack of carcinogenicity in experimental animals of ortho-dichlorobenzene, and that 1,2-dichlorobenzene is not classifiable as to its carcinogenicity to humans (Group 3).

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 MRLs for 1,2‑dichlorobenzene are:

minimal risk level

0.7 mg/kg/day for acute-duration oral exposure (1–14 days)

0.6 mg/kg/day for intermediate-duration oral exposure (15–364 days)

0.3 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD (USEPA 1991/2006/2009/2011) is 0.09 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 3 mg/L.

### Derivation of Maximum Acceptable Value

A tolerable daily intake approach has been used for the derivation of the MAV for 1,2‑dichlorobenzene in drinking-water. The MAV has been derived on the basis of a no-observable-no-adverse effects level determined in a two-year mouse gavage study.

The MAV for 1,2-dichlorobenzene in drinking-water was derived as follows:

60 x (5/7) mg/kg body weight/day x 70 kg x 0.1 = 1.5 mg/L

2 L/day x 100

where:

* no-observable-adverse-effect level = 60 mg/kg body weight per day for tubular regeneration of the kidney, identified in a two-year mouse gavage study (normalised for five days/week dosing in the derivation)
* average weight of adult = 70 kg
* average quantity of water consumed by an adult = 2 L per day
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* uncertainty factor = 100 (for intra- and interspecies variation).

The MAV in the 1995 and 2000 DWSNZ had been 1 mg/L, based on the same information as above but rounded down to one significant number.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for 1,2-dichlorobenzene is 0.6 mg/L.

The taste threshold for 1,2-dichlorobenzene has been reported at 0.001 mg/L, and from 0.002 to 0.01 mg/L for odour, and as such it is included in the aesthetic determinands in the DWSNZ.

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# 1,3-dichlorobenzene

CAS No. 541-73-1. Also called meta-dichlorobenzene or m-dichlorobenzene or 1,3‑DCB. Has previously been called m-dichlorobenzol and m-phenylene dichloride.

Commercial-grade 1,3-dichlorobenzene typically consists of 85 to 99 percent 1,3‑dichlorobenzene, <0.01 percent chlorobenzene and <0.1 percent 1,2-dichlorobenzene, with the remainder as 1,4-dichlorobenzene.

### Maximum Acceptable Value

There are insufficient data to derive a health based MAV for 1,3-dichlorobenzene in drinking-water.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on aesthetic considerations, the concentration of 1,3-dichlorobenzene in drinking-water should not exceed 0.02 mg/L, and that there is inadequate data to establish a health based value.

1,3-Dichlorobenzene is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Dichlorobenzenes are used widely in industry and domestic products and therefore are likely to occur in source waters from industrial contamination and human activity. 1,3‑Dichlorobenzene is a minor fumigant and insecticide and can be formed from incomplete combustion of waste. It can be used to make herbicides, insecticides, medicines, and dyes.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

The dichlorobenzenes are expected to adsorb readily to soils with high organic content and are not expected to leach appreciably into groundwater (solubility of 1,3‑dichlorobenzene about 110 mg/L). In water, the major processes for dichlorobenzene removal are likely to be adsorption to sediments and bioaccumulation in aquatic organisms. Evaporation from surface water may also be important. Dichlorobenzenes may be biodegraded under aerobic conditions but this is not likely to occur under the anaerobic conditions that may exist in lake sediments or groundwaters. Their log octanol-water partition coefficients (Kow) are moderately high, around 3.0 for all three isomers.

### Typical concentrations in drinking-water

No data are available on the concentration of 1,3-dichlorobenzene in New Zealand drinking-water supplies. In a US study of 685 groundwaters, 1,3-dichlorobenzene was detectable in 19 samples, with a maximum concentration of 0.24 mg/L being found.

In a study in the USA on the contamination of 685 groundwaters, 1,2-DCB, 1,3-DCB, and 1,4-DCB were detected in 20, 19, and 19 samples at maximum concentrations of 6.8, 0.24, and 1.0 mg/L, respectively.

Two water utilities in the US reported detecting dichlorobenzenes (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.087 mg/L. 14 water utilities in the US reported detecting m-dichlorobenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0026 mg/L.

### Removal methods

Removal of 1,3-dichlorobenzene can be achieved through adsorption on to activated carbon, or air stripping.

### Analytical methods

#### Referee method

A referee method cannot be selected for 1,3-dichlorobenzene because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for 1,3-dichlorobenzene because a MAV has not been established. However, the following methods are used to analyse for 1,3‑dichlorobenzene:

1. Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

2. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

3. Liquid–Liquid Extraction Gas Chromatographic/Mass Spectrometric Method (APHA 6410B).

### Health considerations

Sources of human exposure are predominantly air and food.

Dichlorobenzenes are absorbed almost completely from the gastrointestinal tract. Once ingested they are absorbed rapidly, primarily to fat and adipose tissue because of their lipophilicity, and to kidney, liver and lungs. They are rapidly metabolised by oxidation in the liver and excreted in urine.

Dichlorobenzenes are of low acute oral toxicity in experimental animals, however no data are available on chronic toxicity for 1,3-dichlorobenzene. The major target organs are the kidney and liver, and has also been reported to have thyroid and pituitary effects.

Data concerning the health effects of exposure of humans to dichlorobenzenes are restricted to case reports of accidental exposure or misuse of the products. Reported acute effects following short-term exposure (all of which are reversible) include liver damage, blood disorders and disturbances to the immune system, the central nervous system or the respiratory tract. Skin pigmentation and allergic dermatitis have followed skin contact.

1,3-Dichlorobenzene shows no mutagenic activity in tests with bacteria. Both IARC and the USEPA concluded that 1,2-DCB and 1,3-DCB are not classifiable as to human carcinogenicity. The IARC considers that there is inadequate evidence in experimental animals for the carcinogenicity of meta-dichlorobenzene, ie, is not classifiable as to its carcinogenicity to humans (Group 3).

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for 1,3-dichlorobenzene of:

* 0.4 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.02 mg/kg/day for intermediate-duration oral exposure (15–364 days).

The reference dose or RfD (USEPA 2006/2009/2011) for 1,3-dichlorobenzene is 0.09 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 3 mg/L. The values for m-dichlorobenzene were based on o-dichlorobenzene.

### Derivation of Maximum Acceptable Value

There are insufficient toxicological data to derive a MAV for this compound, but it should be noted that it is rarely found in drinking-water, and much less frequently than the other dichlorobenzenes.

The Australian Drinking-water Guidelines have set a maximum concentration guideline of 0.02 mg/L based on the aesthetic considerations of taste and odour. It is not included in the DWSNZ as an aesthetic determinand, probably because it is much less likely to be found than 1,2-dichlorobenzene and 1,4-dichlorobenzene.

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# 1,4-dichlorobenzene

CAS No. 106-46-7. Also called paradichlorobenzene or p-dichlorobenzene, p-DCB or 1,4-DCB. Sometimes previously called p-chlorophenyl chloride and paradichlorobenzol.

### Maximum Acceptable Value

Based on health considerations, the concentration of 1,4-dichlorobenzene in drinking-water should not exceed 0.4 mg/L.

The maximum contaminant level (USEPA 2006/2009/2011) is 0.075 mg/L. The USEPA (2006/2009/2011) also established a lifetime health advisory of 0.075 mg/L, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The maximum acceptable concentration in Canada is 0.005 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on aesthetic considerations, the concentration of 1,4-dichlorobenzene in drinking water should not exceed 0.0003 mg/L, and it would not be a health concern unless its concentration exceeded 0.04 mg/L.

1,4-Dichlorobenzene is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

There are no known natural sources of 1,4-dichlorobenzene. The main route to water is from urinals from where it may be washed into the sewer system. Dichlorobenzenes are widely used in industry and domestic products and therefore are likely to occur in source waters from industrial contamination and human activity. 1,4 Dichlorobenzene is often used in toilet blocks to deodorise air or more correctly to mask odours (usually mixed with a dye and perfume), and as a moth repellant. 1,4-DCB has been used as an insecticide on fruit and as an agent to control mould and mildew growth on tobacco seeds, leather, and some fabrics. It appears on ERMA’s Full List of ACVM approved veterinary medicines and pesticides (ectoparasiticide), as at 2009. Recently, using 1,4‑DCB to make polyphenylene sulfide (PPS) resins has become very important. It is also used in the manufacture of 2,5-dichloroaniline and pharmaceuticals. Its main use in Europe is in the manufacture of 1,4-dichloro-2-nitrobenzene, a precursor for dyes and pigments.

The highest concentration of 1,4-dichlorobenzene reported by EU (2004) for river water in Europe was 0.004 mg/L, most maxima were <0.001 mg/L.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

The dichlorobenzenes are expected to adsorb readily to soils with high organic content and are not expected to leach appreciably into groundwater (solubility of 1,4‑dichlorobenzene about 85 mg/L). Volatilisation is considered the dominant pathway of removal for paradichlorobenzene in soil. Much less of the compound is removed via photolysis, hydrolysis, oxidation, or biodegradation. Breakdown products for any paradichlorobenzene that does not volatilise include 1,4-dichlorobenzene dihydrodiol, 2,5-dichloro-*cis*,*cis*-muconic acid, 2-chloromaleyl-acetic acid, and 2‑chloroaceto-acrylic acid. NPIC.

If released to soil, 1,4-dichlorobenzene is expected to have moderate mobility in soils based on Koc values of 273 and 390. Volatilisation of 1,4-dichlorobenzene from dry soil surfaces is expected to be an important fate process based on its vapour pressure. Volatilisation from moist soil surfaces is expected based on the Henry’s Law constant of 2.7 x 10-3 atm‑cu m/mole at 20°C. 1,4-Dichlorobenzene is expected to biodegrade slowly in soils and water with reported biodegradation half-lifes of about a year or longer. If released to water, 1,4-dichlorobenzene is expected to adsorb to sediment and particulate matter based on the Koc values. Volatilisation from water surfaces is expected to be an important environmental fate process given this compound’s Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 4 and 120 hours , respectively. BCF values in the range of 33 to 720 suggest bioconcentration in aquatic organisms is moderate to high, provided the compound is not metabolised by the organism. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

In deep water, the major process for dichlorobenzene removal is likely to be adsorption to sediments. Volatilisation has been found to be the predominant removal process for chlorobenzenes from lakes and coastal seawater. An estimated half-life of 4.3 hours from a model river one metre deep flowing at 1 m/s with a wind velocity of 3 m/s at 20°C has been measured. Other volatilisation half-lifes ranging between <1 and 31 days have been reported (NICNAS 2000). Dichlorobenzenes may be biodegraded under aerobic conditions but this is not likely to occur under the anaerobic conditions that may exist in lake sediments or groundwaters.

Octanol-Water Partition Coefficient (log Kow): 3.5. Henry’s constant: 1.7 to 2.6 x 10-3 atm·m3/mol; 5.1 x 10-13 atm mole/m3. Soil Sorption Coefficient (Koc): 275 to 833 depending on soil type.

EU (2004) quotes: vapour pressure = about 165 Pa at 2°C and 1330 Pa at 54.8°C; Henry’s law constant = 262 Pa.m3/mol at 20↑8C; water solubility about 60–70 mg/L; partition coefficient = logPow = 3.4.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 294 zones, found 1,4-dichlorobenzene twice in one zone at detectable concentrations between 0.005 and 0.009 mg/L (limit of detection = 0.0005 mg/L) (ESR 2001).

In a US study of 685 groundwaters, 1,4-dichlorobenzene was detected in 19 samples with a maximum concentration of 1.0 mg/L being reported.

In a study in the USA on the contamination of 685 groundwaters, 1,2-DCB, 1,3-DCB, and 1,4-DCB were detected in 20, 19, and 19 samples at maximum concentrations of 6.8, 0.24, and 1.0 mg/L, respectively.

Two water utilities in the US reported detecting dichlorobenzenes (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.087 mg/L. 276 water utilities in the US reported detecting p-dichlorobenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.009 mg/L.

### Removal methods

Removal of 1,4-dichlorobenzene can be achieved through adsorption on to activated carbon or air stripping. Good destruction by ozone has been reported.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction Gas Chromatographic/Mass Spectrometric Method (APHA 6410B).

### Health considerations

Sources of human exposure are predominantly air and food.

Dichlorobenzenes are almost completely absorbed from the gastrointestinal tract. Once ingested, they are absorbed rapidly, primarily to fat and adipose tissue because of their lipophilicity, and to kidney, liver and lungs. They are metabolised rapidly by oxidation in the liver and excreted in urine.

Dichlorobenzenes are of low toxicity and the main target organs are the liver and kidneys. There is evidence that 1,4-dichlorobenzene increases the incidence of renal tumours in rats and the hepatocellular adenomas and carcinomas in mice after long-term exposure. The formation of kidney tumours in male rats is thought to be due to the presence of the protein, α2µ-globulin. Because α2µ-globulin is specific to the mature male rat, *p-*dichlorobenzene is not considered to present a carcinogenic risk to humans by this mechanism. The International Agency for Research on Cancer considers that there is sufficient evidence in experimental animals for the carcinogenicity of para-dichlorobenzene, and has placed 1,4-dichlorobenzene in Group 2B (possibly carcinogenic to humans). This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

Data concerning the health effects of exposure of humans to dichlorobenzenes are restricted to case reports of accidental exposure or misuse of the products. Reported acute effects following short-term exposure (all of which are reversible) include liver damage, blood disorders and disturbances to the immune system, the central nervous system, or the respiratory tract. Skin pigmentation and allergic dermatitis have followed skin contact.

1,4-Dichlorobenzene does not exhibit mutagenic activity in tests with bacteria or mammalian cells, and the relevance for humans of the tumours observed in animals is doubtful.

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 MRLs for 1,4‑dichlorobenzene are:

minimal risk level

0.07 mg/kg/day for intermediate-duration oral exposure (15–364 days)

0.07 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD (USEPA 2006/2009/2011) for 1,4-dichlorobenzene is 0.1 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 4 mg/L.

### Derivation of Maximum Acceptable Value

A tolerable daily intake approach has been used for the derivation of the MAV for 1,4‑dichlorobenzene. It has been derived on the basis of a lowest-observable-no-adverse effects level determined in a two-year toxicological study in which rats were exposed to 1,4-dichlorobenzene by oral administration.

The MAV for 1,4-dichlorobenzene in drinking-water was derived as follows:

150 x (5/7) mg/kg body weight/day x 70 kg x 0.1 = 0.375 mg/L (rounded to 0.4 mg/L)

2 L/day x 1000

where:

* lowest-observable-adverse-effect level = 150 mg/kg body weight per day for kidney effects observed in a two-year rat gavage (normalised for five days/week dosing in derivation)
* average weight of adult = 70 kg
* average quantity of water consumed by an adult = 2 L per day
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for the use of a LOAEL in place of a NOAEL and because the toxic end-point is carcinogenicity.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The cancer health risk limit for 1,4-dichlorobenzene is 0.01 mg/L.

Taste and odour thresholds for 1,4-dichlorobenzene have been reported at concentrations of about 0.011 and 0.005 mg/L respectively, Young et al (1996), and are significantly lower than the thresholds for 1,3-dichlorobenzene and 1,4‑dichlorobenzene. It is included in the DWSNZ as an aesthetic determinand.

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# 1,1-dichloroethane

CAS No. 75-34-3. Also called ethylidene chloride, ethylene dichloride or 1,1-DCA.

### Maximum Acceptable Value

There are insufficient data to derive a MAV for 1,1-dichloroethane in drinking-water.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set a drinking water guideline value for 1,1-dichloroethane.

The USEPA concluded on 22 September 2009 that 1,1-dichloroethane is known or anticipated to occur in PWSs and may require regulation. Therefore they added 1,1‑dichloroethane to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

1,1-Dichloroethane is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

1,1-Dichloroethane can be released to the aquatic environment as an industrial contaminant. It is used overseas in the commercial production of 1,1,1-trichloroethane and vinyl chloride, as a solvent in paints, and as a varnish and finish remover, and as a degreaser and cleaning agent and in ore flotation. It was used formerly as an anaesthetic. Occurrence in New Zealand waters is expected to be relatively low since vinyl chloride is not manufactured here, and 1,1-dichloroethane has not been identified, from a limited survey, as a solvent used by paint manufacturers in New Zealand.

DWI (2014) reports that concentrations of 1,1-dichlorethane in drinking water range from 0.105 to 24 μg/L, in groundwater from 0.0081 to 1,900 μg/L, in fresh surface water from 0 to 400 μg/L and in effluents from 0.5 to 6300 μg/L.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

1,1-Dichloroethane is volatile, so most of that released to the environment partitions to the atmosphere, where it is removed by photo-oxidation. 1,1-Dichloroethane is also quite soluble in water, about 5,000–6,000 mg/L, so is expected to be mobile in soil and could reach groundwater before volatilising. Biodegradation is not expected to be significant in aquatic systems.

If released to soil, 1,1-dichloroethane is expected to have very high mobility based upon a Koc of 30. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 5.62 x 10-3 atm‑cu m/mole. 1,1‑Dichloroethane may volatilise from dry soil surfaces based upon its vapour pressure. Halogenated aliphatic hydrocarbons are generally considered to be resistant to biodegradation. If released into water, 1,1-dichloroethane is not expected to adsorb to suspended solids and sediment based upon the Koc. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are three hours and four days, respectively. An estimated BCF of 5 suggests the potential for bioconcentration in aquatic organisms is low. The environmental hydrolysis half-life at 25°C and pH 7 is 61 years (EAWAG accessed February 2015). DWI (2014) quotes a Kow of 1.79.

### Typical concentrations in drinking-water

No data are available on the concentration of 1,1-dichloroethane in New Zealand drinking-water supplies. 132 water utilities in the US reported detecting 1,1‑dichloroethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.024 mg/L. Levels as high as 0.4 mg/L have been found in groundwater in the USA.

ATSDR (2013) reported that tests on 13,347 California groundwater sources of drinking water found 1,1-dichloroethane in 68 samples, ranging from 0.51 to 30 parts per billion (ppb or µg/L).

The maximum concentration found in 5336 samples from 2,322 groundwaters in the UK was 0.019 mg/L, mean 0.0002 mg/L (DWI 2008).

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,848 drinking water samples for 1,1-dichloroethane between 2013 and 2015, and found 835 samples exceeded the minimum reporting level (MRL) of 0.03 mg/L, and 244 samples contained >0.006 mg/L.

### Removal methods

DWI (2014) reports results from several studies:

In a pilot plant trial, the GAC absorption capacity for an influent concentration of 9 μg/L of 1,1-dichloroethane and an empty bed contact time of 2.5 minutes was 0.12 to 0.15 mg/g at saturation.

Ozone doses of 2 and 6 mg/L were ineffective at removing 1,1,-dichloroethane. A 1,1‑dichloroethane concentration of 15 μg/L treated with an ozone dose of 1.7 mg/L at pH 8.2 underwent 20 percent removal after 40 minutes.

1,1-Dichloroethane is also amenable to removal by aeration; a concentration of 6 μg/L 1,1-dichloroethane in groundwater underwent 83 percent removal following a 10‑minute contact time and an air to water ratio of 4:1.

WRF (2014) reports that 1,1-dichloroethane is characterised with a moderate Henry’s law constant (0.194 dimensionless air/water at 20°C). Low profile air stripping is very effective for 1,1-dichloroethane removal even at low temperatures and low air to water ratios (below 100). This VOC exhibited 100 percent removal rates at the three temperatures and air to water ratio of about 150. The very high removal efficiency of 99.6 and 99.8 was achieved at the relatively low air to water ratio of about 70 and temperatures of 4°C and 12°C. At the low air to water ratio of 53, the temperature effect became clear with 99.6 percent removal at 20°C, 99.18 percent removal at 12°C, and the lowest removal efficiency of 95.3 percent, was achieved at the worst case scenario of 4°C.

### Analytical methods

#### Referee method

A referee method cannot be selected for 1,1-dichloroethane because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for 1,1-dichloroethane because a MAV has not been established. However, the following methods are used to analyse for 1,1‑dichloroethane:

1. Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

2. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

1,1-Dichloroethane is metabolised rapidly by mammals to acetic acid and a variety of chlorinated compounds. It is of relatively low acute toxicity, and limited data are available on its toxicity from short-term and long-term studies.

A 13-week inhalation study with 1,1-dichloroethane reported elevated blood-urea nitrogen concentrations in cats, but not in rats, rabbits or guinea pigs. No other adverse effects were observed. A 78-week feeding study reported a marginally significant increase in the incidence of tumours of the mammary glands of female rats. No statistically significant increase in tumours was observed in male rats, or male and female mice.

1,1-Dichloroethane has exhibited mutagenic activity in tests with bacteria and mammalian cells. There is limited *in vitro* evidence of genotoxicity. One carcinogenicity study in mice and rats provided no conclusive evidence of carcinogenicity, although there was some evidence for an increased incidence of haemangiosarcomas in treated animals. USEPA (1990 and 1996) classified 1,1-dichloroethane C: possible human carcinogen. This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

In humans, inhalation exposures to high concentrations of 1,1-dichloroethane causes central nervous system depression. It has been used as an anaesthetic until its use was discontinued because of the problems associated with heart rhythm. Exposure to 1,1‑dichloroethane may occur through drinking-water, but from the point of view of the general population the greatest exposure is usually from the inhalation of ambient air.

DWI (2014) quotes for Repeat Oral Dose Toxicity and Carcinogenicity a time weighted average LOAEL of 475 mg/kg bw/day in rats based on dose-dependent increases in mortality and clinical signs of toxicity. From this LOAEL an oral Tolerable Daily Intake (TDI) of 0.475 mg/kg bw/day (475 μg/kg bw/day) is derived.

### Derivation of Maximum Acceptable Value

In view of the limited data base on toxicity and carcinogenicity no MAV for 1,1‑dichloroethane in drinking-water is proposed.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The short-term and subchronic limits are 0.4 mg/L. The chronic risk assessment advice (exposure greater than 10 percent of a lifetime) for 1,1‑dichloroethane is 0.08 mg/L.

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# 1,2-dichloroethane

CAS No. 107-06-2. Also called ethylene dichloride, dichloro-1,2-ethane, ethane dichloride or 1,2-DCA.

### Maximum Acceptable Value

Based on health considerations, the concentration of 1,2-dichloroethane in drinking-water should not exceed 0.03 mg/L.

The maximum contaminant level (USEPA 2006/2009/2011) is 0.005 mg/L. The maximum acceptable concentration in Canada is 0.005 mg/L, based on treatment and analytical achievability.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of 1,2-dichloroethane in drinking water should not exceed 0.003 mg/L.

The Prescribed Concentration or Value (PCV) for 1,2-dichloroethane in England and Wales is 0.003 mg/L. See Notes.

1,2-Dichloroethane is one of the “priority pollutants” under the US Clean Water Act. It also appears on the Rotterdam Convention (UNEP) list of chemicals in Appendix III (which effectively bans or severely restricts use of a chemical), see http://www.pic.int/home.php?type=s&id=77.

### Sources to drinking-water

#### 1. To source waters

1,2-Dichloroethane can be released to the aquatic environment as a result of human and industrial activity. About 90 percent of the world production is used in the manufacture of vinyl chloride for use in the plastics industry overseas, and for the manufacture of other chlorinated chemicals. Occurrence from these sources is likely to be low in New Zealand since all PVC is imported and only compounded here. It is also used as a solvent, and can be used as a lead scavenger in leaded petrol. It is no longer registered for use as a fumigant on agricultural products in many countries, including New Zealand.

#### 2. From treatment processes

Chloroethanes may be formed in small amounts by the aqueous chlorination of effluents.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Although 1,2-dichloroethane is soluble in water (about 8,000–9,000 mg/L) it is also volatile, so most of that released to the environment partitions to the atmosphere; adsorption to soil and sediments is not expected. Biodegradation can also be significant in aquatic systems, although 1,2-dichloroethane may persist for long periods in groundwater, where volatilisation is restricted.

If released to soil, 1,2-dichloroethane is expected to have very high mobility based upon a Koc of 33. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 1.18 x 10-3 atm‑cu m/mole. 1,2‑Dichloroethane may volatilise from dry soil surfaces based upon its vapour pressure. Biodegradation in soil or water is not expected to be an important environmental fate process based upon a variety of biodegradation test data. If released into water, 1,2-dichloroethane is not expected to adsorb to suspended solids and sediment based upon the Koc. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are four hours and four days, respectively. A BCF of 2 suggests bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 332 zones, found 1,2-dichloroethane in one zone at a concentration of 0.009 mg/L, with the median concentration being “nd” (limit of detection = 0.0005 mg/L) (ESR 2001).

1,2-Dichloroethane was detected frequently in treated drinking water and raw water samples taken from 30 treatment facilities across Canada in 1979. Mean concentrations in treated water were between 0.004 and 0.005 mg/L during August and September and less than 0.001 mg/L in November and December. The overall mean of 31 positive determinations was 0.0032 mg/L. Maximum concentrations of 0.03 and 0.011 mg/L were found during August and September and during November and December, respectively (Health Canada 1987).

174 water utilities in the US reported detecting 1,2-dichloroethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.015 mg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,848 drinking water samples for 1,2-dichloroethane between 2013 and 2015, and found 853 samples exceeded the minimum reporting level (MRL) of 0.03 µg/L.

### Removal methods

Some removal of 1,2-dichloroethane can be achieved by adsorption on to granular activated carbon, although the adsorption is relatively weak. Some removal is also achievable by air-stripping.

WRF (2014) reports that 1,2-dichloroethane is characterised with a low Henry’s law constant (0.0397 dimensionless air/water at 20°C). Low profile air stripping is very effective for 1,2-dichloroethane removal at high air to water ratios (about 600) at all examined temperatures. The removal efficiency was still high at air to water ratios of about 150–167. 100 percent removal was achieved at 20°C (150 air to water ratio), 99.5 percent removal efficiency at 12°C (167 air to water ratio), and 99 percent at 4°C (162 air to water ratio). Because of the low Henry’s law constant, the effect of temperature became more significant at lower air to water ratios. At air to water ratio of about 70, the removal efficiency dropped to 95.9 percent and 84.1 percent for the 12°C and the 4°C. The efficiency dropped further at the lowest air to water ratio of about 53 to 91.4 percent, 83.1 percent, and 59.9 percent at 25°C, 12°C, and 4°C respectively.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

Inhalation appears to be the primary route of exposure to 1,2-dichloroethane. 1,2‑Dichloroethane is absorbed readily through the lungs, skin and gastrointestinal tract. It appears to be distributed readily following oral or inhalation exposure, with accumulation in the liver and kidneys. 1,2-Dichloroethane crosses the blood-brain barrier and the placenta and it has been detected in human milk following occupational exposure. Excretion of absorbed 1,2-dichloroethane occurs rapidly, mainly in the urine and expired air.

In humans, acute oral exposure to 1,2-dichloroethane is reported to cause central nervous system, hepatic, gastro-intestinal, respiratory, renal and cardiovascular effects. Death following acute intoxication is most often attributed to cardiovascular or respiratory failure. Repeated inhalation exposures in the workplace result in anorexia, nausea, vomiting, weakness and fatigue, nervousness, epigastric pain and irritation of the respiratory tract and eyes.

1,2-Dichloroethane has exhibited mutagenic activity in tests with different strains of bacteria, and its metabolites are known to be strongly mutagenic. 1,2-Dichloroethane has been consistently genotoxic in numerous *in vitro* assays in prokaryotes, fungi, and mammalian (including human) cells. Similarly, results were consistently positive for genotoxic activity (as well as binding to DNA) in *in vivo* studies in rats, mice, and insects.

A 13-week feeding and drinking study with 1,2-dichloroethane using rats and mice reported increased kidney and liver weights at high doses (4,000 mg/L). No increase in the incidence of tumours or lesions was observed in mice or rats, but female rats exhibited an increase in the incidence of kidney lesions.

Male rats fed 1,2-dichloroethane five times per week for 78 weeks were reported to have a significant increase in tumours of the forestomach and circulatory system. The same study reported tumours of the mammary glands in female rats.

The lowest NOAEL for subchronic oral exposure by gavage is assumed to be 120 and 150 mg/kg bw/d for male and female rats, respectively, based on treatment-related effects in the forestomach and clinical symptoms, while the chronic oral NOAEL of about 25 mg/kg bw is equivalent to the highest dose administered to rats for two years in the diet (OECD 2004).

The International Agency for Research on Cancer has classified 1,2-dichloroethane in Group 2B (possibly carcinogenic to humans). It has been shown to produce statistically significant increases in a number of tumour types in laboratory animals, including the relatively rare haemangiosarcoma, and the balance of evidence indicates that it is relatively genotoxic. USEPA (1987) classified 1,2-dichloroethane B2: probable human carcinogen.

1,2-Dichloroethane appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. The USEPA (2009/2011) quotes a health advisory of 0.04 mg/L for 1,2-dichloroethane, representing a 10-4 cancer risk.

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.2 mg/kg/day for intermediate-duration oral exposure  
(15–364 days) to 1,2-dichloroethane.

### Derivation of Maximum Acceptable Value

As the balance of evidence indicates that 1,2-dichloroethane is potentially genotoxic and there are no suitable long-term studies on which to base a tolerable daily intake, the MAV for 1,2-dichloroethane in drinking-water was calculated using an extrapolation model. On the basis of haemangiosarcomas observed in male rats in a 78‑week gavage study, and applying the linearised multistage model, it was determined that a concentration of 1,2-dichloroethane in drinking-water of 0.03 mg/L corresponds to a life-time risk of one additional cancer per 100,000 (10-5).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The short-term and subchronic limits are 0.2 mg/L. The chronic limit is 0.06 mg/L. The cancer health risk limit for 1,2-dichloroethane is 0.001 mg/L.

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# 1,1-dichloroethene

CAS No. 75-35-4. Also called vinylidene chloride, vinylidene dichloride, 1,1‑dichloroethylene, asym-dichloroethylene, VDC or 1,1-DCE.

### Maximum Acceptable Value

Because 1,1-dichloroethene occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based MAV.

In the DWSNZ 2005, the MAV had been 0.03 mg/L.

The maximum contaminant level or MCL (USEPA 2006/2009/2011) is 0.007 mg/L. The maximum acceptable concentration in Canada is 0.014 mg/L.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of 1,1-dichloroethene in drinking water should not exceed 0.03 mg/L.

1,1-Dichloroethene is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

1,1-Dichloroethene can be released to the aquatic environment as an industrial contaminant. It is used as a monomer to produce polyvinylidene chloride copolymer plastics such as “Saran” and as an intermediate in the synthesis of other organic solvents, and for flame retardant coatings for fibre and carpet backing. Potential for occurrence in New Zealand source waters is expected to be low since there is no polymerisation of the monomer in this country. Technical vinylidene chloride is more than 99.6 percent pure. It is stabilised against oxidation and polymerisation by the addition of hydroquinone monomethyl ether (p-methoxyphenol; 180–220 mg/kg).

1,1-DCE can be found in the environment from release during its manufacture and use, from the breakdown of polyvinylidene (PVDC) products, and from the biotic or abiotic breakdown of 1,1,1-trichloroethane, tetrachloroethene, 1,1,2-trichloroethene, and 1,1‑dichloroethane. The principal sources of environmental exposure for humans are ambient air and contaminated drinking-water.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources, other than the breakdown of polyvinylidene (PVDC) products.

### Forms and fate in the environment

Although quite soluble in water (about 2,000–3,000 mg/L), most 1,1-dichloroethene released to the environment volatilises to the atmosphere, where it is oxidised rapidly, with a lifetime of about one to three days. Photolysis is also expected to occur. Volatilisation is the major removal mechanism for 1,1-dichloroethene in surface waters and soils. It is much more persistent in groundwaters where anaerobic biotransformation to vinyl chloride is expected to be important. It has a high vapour pressure of 65.8 kPa at 20°C and a Henry’s law constant of 0.19 atm·m3/mol. It has a log octanol-water partition coefficient in the range 1.66 to 2.13, indicating that it is unlikely to have significant potential for bioaccumulation.

The leaching of solvents into groundwater is a potential source of 1,1-DCE contamination. The presence of 1,1-DCE in 43 percent of the groundwater samples at the Gloucester, Ontario, landfill site (concentrations ranged from 0.9 to 60 µg/L) sampled in 1988 is thought to have resulted from the degradation of tetrachloroethylene and 1,1,1-trichloroethane, as 1,1-DCE is a known degradation product of these two compounds and was not known to have been disposed of at the site (Health Canada 1994).

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 332 zones, did not find any 1,1-dichloroethene at detectable concentrations (limit of detection = 0.0005 mg/L) (ESR 2001).

213 water utilities in the US reported detecting 1,1-dichloroethylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.015 mg/L.

In a 1985 to 1988 survey of water supplies in the four Atlantic provinces, 1,1-DCE was not detected in raw or treated drinking water samples taken from 151 sampling stations; the minimum quantifiable limit ranged from 0.5 to 1.0 µg/L. In a 1981 survey of surface water samples taken from Lake Ontario, 1,1-DCE was detected (detection limit 0.09 µg/L) in 11 of 82 samples taken; concentrations ranged from trace to 3.5 µg/L. Maximum concentrations were generally near 0.19 µg/L (Health Canada 1994).

The maximum concentration found in 5,818 samples from 2,322 groundwaters in the UK was 0.022 mg/L, mean 0.0002 mg/L (DWI 2008).

### Removal methods

Some removal of 1,1-dichloroethene can be achieved by adsorption on to granular activated carbon, although the adsorption is relatively weak. Some removal is also achievable by air-stripping.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

Following oral or inhalation exposure, 1,1-dichloroethene is absorbed almost completely, metabolised extensively, and excreted rapidly.

It is a central nervous system depressant and may cause liver and kidney toxicity in occupationally exposed humans. It causes liver and kidney injury in laboratory animals. Because of its volatility, there is potential for exposure in the home to airborne 1,1-DCE released from tap water.

A long-term study, in which rats were exposed to 1,1-dichloroethene in their drinking-water for two years, reported minimal swelling to liver cells, but no other adverse effects. No changes were observed in tissues taken from dogs after 97 days of exposure.

IARC has placed 1,1-dichloroethene in Group 3 (not classifiable as to its carcinogenicity to humans). It was found to be genotoxic in a number of test systems *in vitro* but was not active in the dominant lethal assay *in vivo*. It induced kidney tumours in mice in one inhalation study but was reported not to be carcinogenic in a number of other studies, including several in which it was given in drinking-water. There is no evidence that reproductive toxicity or teratogenicity is a critical effect for 1,1-DCE. No reproductive or developmental toxicity was observed at an oral exposure that caused minimal toxicity in the liver of the dams.

Under the 1986 cancer guidelines, USEPA assigned 1,1-DCE to Group C, a possible human carcinogen. USEPA (2002) concluded that 1,1-DCE exhibits *suggestive evidence* of carcinogenicity but not sufficient evidence to assess human carcinogenic potential following inhalation exposure in studies in rodents. Limited evidence of genotoxicity has been reported in bacterial systems with metabolic activation. The data for 1,1-DCE are *inadequate* for an assessment of human carcinogenic potential by the oral route, based on the absence of statistically or biologically significant tumours in limited bioassays in rats and mice balanced against the suggestive evidence in male mice in a single bioassay by inhalation and the limited evidence of genotoxicity. The USEPA (2009/2011) quotes a health advisory of 0.006 mg/L for 1,1-dichloroethene, representing a 10-4 cancer risk.

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.009 mg/kg/day for chronic-duration oral exposure (>364 days) for 1,1-dichloroethene.

The reference dose or RfD (USEPA 2002/2006/2009/2011) is 0.05 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 2 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

In 2003 IPCS developed a TDI for 1,1-dichloroethene based on the critical effect from oral exposure, which is minimal hepatocellular mid-zonal fatty change in female Sprague-Dawley rats. A benchmark dose was determined, and the BMDL10 was 4.6 mg/kg of body weight per day. An uncertainty factor of 100 (for inter- and intraspecies variation) was applied to the BMDL10 to give a TDI of 0.046 mg/kg of body weight.

If it is assumed that a 60‑kg adult drinks two litres of water per day, one could calculate a health-based value of 0.14 mg/L (rounded value) using a conservative (because exposure from food is low) default allocation of 10 percent of the TDI to drinking-water. The health-based value of 0.14 mg/L for a 60 kg person equates to a value of 0.16 mg/L for a 70 kg person. However, this health-based value is significantly higher than the concentrations of 1,1-dichloroethene that are normally found in drinking-water. It is therefore considered unnecessary to set a formal guideline value for 1,1‑dichloroethene in drinking-water (WHO 2011).

The MAV for 1,1-dichloroethene in the 1995, 2000 and 2005 DWSNZ was 0.03 mg/L, and had been derived as follows:

as 1,1-dichloroethene is not classifiable as to its carcinogenicity to humans, a tolerable daily intake approach had been used for the derivation of the MAV. The MAV for 1,1-dichloroethene in drinking-water had been derived on the basis of a lowest-observable-adverse effects level determined in a two-year toxicological study.

9 mg/kg body weight/day x 70 kg x 0.1 = 0.03 mg/L

2 L/day x 1000

where:

* lowest-observable-adverse-effect level = 9 mg/kg body weight per day in a two-year drinking-water study (for increased incidence of hepatic lesions) in female rats
* average weight of adult = 70 kg
* average quantity of water consumed by an adult = 2 L per day
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for the use of a LOAEL in place of a NOAEL and the potential for carcinogenicity).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The subchronic limit, and the chronic health-based value (exposure greater than 10 percent of a lifetime), for 1,1-dichloroethene is 0.2 mg/L.

The odour threshold for 1,1-dichloroethene in water is 1.5 mg/L.

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# 1,2-dichloroethene

CAS No. 540-59-0 (for the mixture). Also called 1,2-dichloroethylene, ethylene dichloride or 1,2-DCE.

CAS No. 156-59-2 (cis form). CAS No. 156-60-5 (for the trans form).

### Maximum Acceptable Value

Based on health considerations, the concentration of 1,2-dichloroethene in drinking-water should not exceed 0.06 mg/L (sum of cis and trans forms).

The maximum contaminant level or MCL (USEPA 2006/2009/2011) is 0.07 mg/L for the cis form and 0.1 mg/L for the trans form. The USEPA (2006) established the same lifetime health advisories where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity. USEPA (2011) does not include a lifetime health advisory for the cis form.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of 1,2-dichloroethene in drinking water should not exceed 0.06 mg/L.

1,2-Dichloroethene (trans form) is a “priority pollutant” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

1,2-Dichloroethene (cis-, and trans- isomers) can be released to the aquatic environment as an industrial contaminant. It is used primarily as an intermediate in the synthesis of chlorinated solvents and compounds. It has also been used as an extraction solvent. The cis-form of 1,2-dichloroethene is more frequently found as a water contaminant. The presence of the two isomers, which are metabolites of other unsaturated halogenated hydrocarbons, may indicate the simultaneous presence of more toxic organochlorine chemicals, such as vinyl chloride. Accordingly, their presence indicates that more intensive monitoring should be conducted.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Removal of 1,2-dichloroethene from air is mainly through reaction with hydroxyl radicals.

Volatilisation is expected to be the primary fate process in surface water and surface soils. Chemical hydrolysis and oxidation are probably not important environmental fate processes for 1,2-dichloroethene. It is quite soluble in water (cis about 3,500 mg/L; trans about 6,300 mg/L), so it may leach through subsurface soils to groundwater where its half life is 13–48 weeks. However, research suggests that most 1,2‑dichloroethene detections in groundwater involve biodegradation processes related to primary pollution from trichloroethylene or tetrachloroethylene. Anaerobic biodegradation may remove both isomers from groundwater.

If released to soil, cis-1,2-dichloroethylene is expected to have very high mobility based upon a reported Koc of 49. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 4.08 x 10-3 atm‑cu m/mole. cis-1,2-Dichloroethylene may volatilise from dry soil surfaces based upon its vapour pressure. Utilising the Japanese MITI test, 0 percent of the theoretical BOD was reached in four weeks indicating that biodegradation may not be an important environmental fate process. However, cis-1,2-dichloroethylene exhibited soil field biodegradation rates of 0.288 and 0.194/week. If released into water, cis‑1,2‑dichloroethylene is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. The concentration of cis-1,2-dichloroethylene was reduced to <2 percent and trace concentrations after 16 and 40 weeks, respectively, when incubated in a serum bottle at 17°C with methanogenic aquifer material obtained adjacent to a landfill site; this indicates that anaerobic biodegradation is an important environmental fate process in groundwater. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are three hours and four days, respectively. An estimated BCF of 8 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

If released to soil, trans-1,2-dichloroethylene is expected to have high mobility based upon a reported Koc of 59. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 9.38 x 10-3 atm‑cu m/mole. trans-1,2-Dichloroethylene may volatilise from dry soil surfaces based upon its vapour pressure. Utilising the Japanese MITI test, 0 percent of the theoretical BOD was reached in four weeks indicating that aerobic biodegradation may not be an important environmental fate process in soil. However, trans-1,2-dichloroethylene exhibited soil field biodegradation rates of 0.229 and 0.215/week. If released into water, trans-1,2-dichloroethylene is not expected to adsorb to suspended solids and sediment based upon the Koc. No biodegradation occurred in a river die-away test. However, under anoxic conditions using uncontaminated organic sediment from the Everglades, 73 percent of the chemical was lost in six months, suggesting that anaerobic biodegradation is an important environmental fate process in water. trans-1,2-Dichloroethylene had aquatic field biodegradation rates of 0.182 and 0.185/week. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are three hours and four days, respectively. An estimated BCF of 11 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 332 zones, did not find any 1,2-dichloroethene (ethylenedichloride) at detectable concentrations (limit of detection = 0.0005 mg/L) (ESR 2001).

Overseas it has been found in drinking-water supplies derived from groundwater at levels up to 0.12 mg/L (WHO 2004).

340 water utilities in the US reported detecting cis-1,2-dichloroethylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.032 mg/L.

Fifty-six water utilities in the US reported detecting trans-1,2-dichloroethylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.006 mg/L.

The maximum concentration of cis 1,2-dichloroethene found in 5999 samples from 2442 groundwaters in the UK was 1.1 mg/L, mean 0.0006 mg/L (DWI 2008).

The maximum concentration of trans 1,2-dichloroethene found in 5993 samples from 2442 groundwaters in the UK was 0.012 mg/L, mean 0.00025 mg/L (DWI 2008).

### Removal methods

Removal of 1,2-dichloroethene can be achieved by adsorption on to granular activated carbon, or by air stripping.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

There is little information on the adsorption, distribution, and excretion of 1,2‑dichloroethene. Toxicokinetic evidence shows they have a high affinity for lipids and blood, but little accumulation in tissues. However, based on analogy with 1,1‑dichloroethene, it would be expected to be adsorbed readily, distributed mainly in the liver, kidney, and lung, and excreted rapidly. The cis-isomer is metabolised more readily than the trans-isomer in *in vitro* systems.

Both isomers have been reported to cause increased serum alkaline phosphatase levels in rodents. In a three-month study in mice given the trans-isomer in drinking-water, there was a reported increase in serum alkaline phosphatase and reduced thymus and lung weights. It was also reported to cause transient immunological effects, the toxicological significance of which is unclear. Trans-1,2-dichloroethene also caused reduced kidney weights in rats, but at higher doses. Only one rat toxicity study is available for the cis-isomer. The toxic effects of the cis-isomer in rats were observed only at higher doses than for the trans-isomer in mice, but the toxicity was of a similar magnitude. There are limited data to suggest that both isomers may possess some genotoxic activity.

Inhalation of high concentrations (9500 ppm and above) of 1,2-dichloroethane by humans causes central nervous system depression. Neurological effects have been reported following exposure to low levels of trans-1,2-dichloroethane, including nausea, drowsiness, fatigue and vertigo. A burning sensation of the eyes was also reported. The trans-isomer is reportedly about twice as potent a central nervous system depressant as the cis-isomer, which was previously used as an anaesthetic.

Neither the USEPA nor IARC has classified 1,2-dichloroethene with respect to carcinogenicity.

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for cis-1,2-dichloroethane of:

* 1 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.3 mg/kg/day for intermediate-duration oral exposure (15–364 days).

As at July 2013 ATSDR quotes a minimal risk level (MRL) of 0.2 mg/kg/day for intermediate-duration oral exposure (15–364 days) to trans-1,2-dichloroethane.

The reference dose or RfD (USEPA 2006/2009) is 0.01 mg/kg/d for the cis form (changed to 0.002 in 2010/2011), and 0.02 mg/kg/d for the trans form (not changed in 2010/2011). The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009) is 0.35 mg/L for the cis form (changed to 0.07 in 2011), and 0.7 mg/L for the trans form (not changed in 2011).

### Derivation of Maximum Acceptable Value

Data on the trans-isomer were used to calculate a joint MAV for both isomers because toxicity for the trans-isomer occurred at a lower dose than for the cis-isomer and because data suggest that the mouse is a more sensitive species than the rat.

The MAV for 1,2-dichloroethene (sum of cis and trans) in drinking-water was derived as follows:

17 mg/kg body weight/day x 70 kg x 0.1 = 0.0595 mg/L (rounded to 0.06 mg/L)

2 L/day x 1000

where:

* no-observable-adverse-effect level = 17 mg/kg body weight per day, for increases in serum alkaline phosphatase levels and increased thymus weight, based on a 90‑day study in mice administered trans-1,2-dichloroethene in drinking-water
* average weight of adult = 70 kg
* average quantity of water consumed by an adult = 2 L per day
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* uncertainty factor = 1,000 (100 for intra- and interspecies variation and 10 for the short duration of the study).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The short-term limit is 0.02 mg/L; the subchronic limit is 0.01 mg/L; the chronic health risk limit (exposure greater than 10 percent of a lifetime) for cis‑1,2‑dichloroethene is 0.006 mg/L.

For trans-1,2-dichloroethene, the subchronic limit is 0.2 mg/L; the chronic health risk limit is 0.04 mg/L.

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# Dichloromethane

CAS No. 75-09-2. Also called methylene chloride, methylene dichloride, methane dichloride, or DCM.

### Maximum Acceptable Value

Based on health considerations, the concentration of dichloromethane in drinking-water should not exceed 0.02 mg/L.

The maximum contaminant level or MCL (USEPA 2004/2009/2011) is 0.005 mg/L.

The maximum acceptable concentration (MAC) in Canada is 0.05 mg/L. For drinking water supplies that occasionally experience short-term exceedances above the MAC, it is suggested that a plan be developed and implemented to address these situations. For more significant long-term exceedances that cannot be addressed through treatment, it is suggested that alternative sources of drinking water be considered.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of dichloromethane in drinking water should not exceed 0.004 mg/L.

Dichloromethane is one of the “priority pollutants” under the US Clean Water Act. Dichloromethane is one of the fourteen VOCs on the USEPA’s 3rd Chemical Contamination List (CCL3).

### Sources to drinking-water

#### 1. To source waters

Dichloromethane is a widely used organic solvent found in paints, insecticides, degreasing and cleaning fluids, and paint strippers (where it may be blended with additives including alcohols, acids, amines or ammonium hydroxide, detergents and paraffin wax). Dichloromethane has been used as an extraction solvent for spices and beer hops and for decaffeination of coffee. It has also found use as a carrier solvent in the textile industry, in the manufacture of photographic film and as a blowing agent for polymer foams. Dichloromethane is used as a solvent for vapour degreasing of metal parts and may also be blended with petroleum distillates and other chlorinated hydrocarbons for use as a dip-type cleaner in the metal-working industry, although consumption by this industry is declining because of recycling and recovery efforts on the part of end users.

The reduction in use of 1,1,1-trichloroethane because of the Montreal Protocol and clean air legislation may increase the use of dichloromethane. Dichloromethane is also used as a component of low-pressure refrigerants, in air-conditioning installations, as a low-temperature heat-transfer medium, and as a propellant in aerosols such as insecticides, hair sprays, shampoos, and paints.

Dichloromethane can be released to the aquatic environment via inadequate storage or disposal, or from the discharge of wastes from industries in which it is used.

Commercial grades of methylene chloride may contain 0.0001 to 1 percent of added stabilisers, such as ethanol, cyclohexane, and amylene (2-methyl-2-butene). Others may include phenol, hydroquinone, p-cresol, resorcinol, thymol, 1-naphthol or amines.

#### 2. From treatment processes

Dichloromethane may be introduced into water during chlorination, as it is a recognised impurity in commercial chlorine. Although not commonly identified as a disinfection by-product, some published work indicates that dichloromethane may also be formed during chlorination of aqueous organic material.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Most dichloromethane released to water and soil will vapourise to air, where it can persist for up to 500 days. It is very soluble in water, about 13,000 to 20,000 mg/L (IARC 2017 states 1.38 percent). Methylene chloride that is present in water is broken down slowly by reactions with other chemicals or by bacteria. Over 90 percent of the methylene chloride in the environment changes to carbon dioxide. It takes about one to six days for half the methylene chloride to break down in water. When methylene chloride is spilled on land, it attaches loosely to nearby surface soil particles. It moves from the soil into the air. Some may also move into groundwater.

If released to soil, dichloromethane is expected to have very high mobility based upon an estimated Koc of 24. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a estimated Henry’s Law constant of 3.25 x 10-3 atm‑cu m/mole. Dichloromethane may volatilise from dry soil surfaces based upon its vapour pressure. Biodegradation in soil may occur based on activated sludge studies. If released into water, dichloromethane is not expected to adsorb to suspended solids and sediment in water based upon the estimated Koc. Biodegradation is possible in natural waters but will probably be very slow compared with evaporation. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are one hour and four days, respectively. An estimated BCF of 2 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not an important degradation process under normal environmental conditions (EAWAG accessed February 2015).

At 25°C, it has a vapour pressure of 57.8 kPa, a log octanol/water partition coefficient of 1.25, and a unitless Henry’s Law constant of 0.25 (Health Canada 2011). A high sorption partition coefficient (log Koc) of 1.4 suggests that dichloromethane will be highly mobile in soil, and therefore may leach into groundwater. Since dichloromethane has a high vapour pressure and Henry’s Law constant, it tends to volatilise from water and soil into the atmosphere.

Dichloromethane has been found in surface water samples at concentrations ranging from 0.0001 to 0.74 mg/L. Levels are usually higher in groundwater because volatilisation is restricted, with concentrations as high as 3.6 mg/L having been reported. Mean concentrations in drinking-water were less than 0.001 mg/L.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 332 zones, did not find any dichloromethane at detectable concentrations (limit of detection = 0.008 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with dichloromethane at >50 percent of the MAV (ESR 2001).

Dichloromethane has been detected in drinking-water supplies in numerous cities in the United States, with reported mean concentrations generally below 0.001 mg/L. It has also been identified in commercially bottled artesian water. Water chlorination in treatment plants appears to increase both the concentration and the frequency of occurrence of dichloromethane in drinking-water supplies. Samples from 128 drinking-water wells in the United States showed that 3.1 percent of them had dichloromethane levels of 0.001 to 0.005 mg/L. Dichloromethane was detected in 98.4 percent of drinking-water samples from Santiago de Compostela, Spain, in 1987; the average concentration was 0.014 mg/L, with a range of 0.001 to 0.093 mg/L. DWI (2014) reports that concentrations of dichloromethane in drinking water range from 0.008 to 3,600 μg/L (3.6 mg/L).

The majority of samples obtained from drinking water supplies in Canada had dichloromethane concentrations below the detection limit. Approximately 5 percent of groundwater and surface samples obtained in Quebec from 2001 to 2005 contained dichloromethane concentrations above 1 μg/L, with maximum values of 290 and 170 μg/L in groundwater and surface water, respectively, and average concentrations of 2.6 μg/L in groundwater, 1.5 μg/L in surface water, and 1.9 μg/L in all samples (Health Canada 2011).

841 water utilities in the US reported detecting dichloromethane (methylene chloride) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.012 mg/L.

Dichloromethane was measured in more than 5,000 wells in the USA between 1985 and 2002; in 97 percent of samples, concentrations of dichloromethane were below maximum contaminant levels (MCLs). Dichloromethane was detected in 3 percent of samples, with concentrations ranging from 0.02 to 100 μg/L (IARC 2017).

### Removal methods

When dichloromethane in drinking-water results from chlorination, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

Dichloromethane can be removed by adsorption on to granular activated carbon, or by air stripping.

DWI (2014) quote reports that state:

* coagulation and flocculation is not effective at removing dichloromethane during drinking water treatment, with only 16 percent removal being reported
* ozone is ineffective at removing dichloromethane; ozone doses of 2, 6 and 20 mg/L gave dichloromethane removals of 4, 6 and 1 percent, respectively
* air stripping is effective at removing dichloromethane during drinking water treatment. In a packed tower, an influent concentration of 770 μg dichloromethane/L was 98 percent removed by air-stripping using an air to water ratio of 51.

WRF (2014) reports that dichloromethane is characterised with low Henry’s law constant (0.081 dimensionless air/water at 20°C). Low profile air stripping is very effective for dichloromethane removal even at low temperatures and low air to water ratios (below 100). This VOC exhibited almost 100 percent removal rates at the three temperatures and air to water ratios of about 150. The 100 percent removal rate was achieved even at air to water ratio as low as 70, except for the 4°C run which resulted in the high removal efficiency of 98 percent. At the low air to water ratio of 53, the temperature effect became clear with 100 percent removal at 20°C, 96.8 percent removal at 12°C, and a lowest removal efficiency of 85 percent, which remains relatively high, at the worst case scenario of 4°C.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2). See also IARC (2017).

### Health considerations

Exposure from drinking-water is likely to be insignificant compared with that from other sources, particularly indoor air where spray paint, aerosol air fresheners, deodorants and hair sprays are used.

Dichloromethane appears to be absorbed readily from the gastrointestinal tract with distribution primarily to the liver. It is metabolised to carbon monoxide, carbon dioxide and formic acid. Animal data indicate that dichloromethane is excreted primarily through the lungs.

Dichloromethane is of low acute toxicity (DWI 2014). The primary effect associated with acute exposure is depression of the central nervous system.

In humans, inhalation of a high concentration of dichloromethane has been associated with a variety of central nervous system effects, most notably narcosis. Acute exposure to levels of 300 ppm can impair sensory and motor functions. Epidemiological studies involving occupational exposure has failed to show a positive correlation between inhalation exposure and increased cancer incidence.

Dichloromethane has exhibited mutagenic activity in various test systems.

The USEPA (2009/2011) quotes a health advisory of 0.05 mg/L for dichloromethane, representing a 10-4 cancer risk. The reference dose or RfD (USEPA 1988/2006/2009/2011) is 0.06 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 2 mg/L.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of:

0.2 mg/kg/day for acute-duration oral exposure (1–14 days)

0.06 mg/kg/day for chronic-duration oral exposure (>364 days).

Dichloromethane appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. USEPA (2009) states that a concentration of 0.5 mg/L dichloromethane represents a 10-4 cancer risk.

An inhalation study in mice provided conclusive evidence of carcinogenicity, whereas a drinking-water study provided only suggestive evidence. The International Agency for Research on Cancer has placed dichloromethane in Group 2B (possibly carcinogenic to humans), however, the balance of evidence suggests that it is not a genotoxic carcinogen and that genotoxic metabolites are not formed in relevant amounts in vivo. Overall, the effect of dichloromethane on mammalian cells has only indicated weak genotoxicity (DWI 2014).

IARC (2017) states that there is limited evidence in humans for the carcinogenicity of dichloromethane. Positive associations have been observed between exposure to dichloromethane and cancer of the biliary tract and non-Hodgkin lymphoma. Their overall evaluation is that dichloromethane is probably carcinogenic to humans (Group 2A).

### Derivation of Maximum Acceptable Value

The balance of evidence suggests that dichloromethane is not genotoxic and therefore a tolerable daily intake approach has been taken for the derivation of the MAV for dichloromethane in drinking-water. The no-observable-adverse-effect level used was for hepatoxic effects in a two-year drinking-water study in rats.

The MAV for dichloromethane in drinking-water was derived as follows:

6 mg/kg body weight per day x 70 kg x 0.1 = 0.021 mg/L (rounded to 0.02 mg/L)

2 L x 1000

where:

* no-observable-adverse-effect level = 6 mg/kg body weight per day for hepatotoxic effects in a two-year drinking-water study in rats
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 reflecting concern for carcinogenic potential).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for dichloromethane is 0.005 mg/L.

The odour threshold for dichloromethane in water is 9.1 mg/L (ATSDR 2000). No taste threshold for dichloromethane in drinking water has been identified.

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# 1,3-dichloro-2-propanol

CAS No. 96-23-1. Also called 1,3-DCP, 1,3-dichloropropan-2-ol, α-dichlorohydrin, 1,3‑dichlorohydrin, 1,3-dicloro-2-hydroxypropane, 1,3-dichloroisopropanol, propylene dichlorohydrin and glycerol 1,3-dichlorohydrin. A member of the chloropropanols.

Two other important chloropropanols are (see datasheets):

* 3-monochloropropane-1,2-diol also called 3-MCPD or 3-chloro-1,2-propanediol or alpha-chlorohydrin (CAS No. 96-24-2), and
* 2,3-dichloro-1-propanol (2,3-DCP).

These chemicals belong to the large, loosely defined group of chemicals called halohydrins. Bromohydrins occur as well, see datasheet for 2,3 dibromopropan-1-ol. Other halohydrins are listed in the halohydrin datasheet.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 1,3-dichloro-2-propanol. The WHO Guidelines do not mention 1,3-dichloro-2-propanol.

NSF Standard 60 states that the allowable concentration of 1,3-dichloro-2-propanol in drinking water is 0.009 mg/L.

### Sources to drinking-water

#### 1. To source waters

Chloropropanol isomers are by-products of the production of hydrolysed vegetable proteins and when chloride ions react with lipid components in foods under a variety of conditions, including food processing, cooking, and storage. 1,3-Dichloropropan-2-ol is used in high volume as an intermediate in epichlorohydrin production and the production of 1,3-dichloropropene and 1,2,3-trichloropropane.

#### 2. From treatment processes

1,3-Dichloro-2-propanol and 3-MCPD can be present as a contaminant in epichlorohydrin/amine copolymers used as flocculants or coagulant aids in water treatment. These polyamine flocculants have been available for many years as approved products for use in water treatment provided the dose does not exceed 2.5 mg/L. Maximum levels of 3-MCPD in the flocculants were set at 40 mg/L to achieve maximum level of 0.1 mg/L 3-MCPD in treated drinking-water. (NB: these maximum levels of 3-MCPD in flocculants are now being reviewed following revised opinions by COC and COM in 2000 on the lack of *in vivo* genotoxicity of this substance and the possibility of setting an ADI for this substance.) However no mandatory levels for other chloropropanols (ie, 1,3-DCP or 2,3-DCP) have been set by the DWI. At the current maximum flocculant dosage rates and impurity levels in flocculants, concentrations in drinking-water may theoretically be in the 2 to 3 mg/L range (although no measurements in drinking-water have been carried out). Levels in 1991 when 1,3 DCP was first considered by COC were estimated to be 16 mg/L.

The following has been copied from DWI (2007):

“There are no statutory levels for 1,3-DCP or 2,3-DCP in drinking-water. 3-MCPD and DCPs are known to co-exist in polyamine flocculants, but the relationship between their contaminant levels is not currently understood. However, their levels in drinking-water are restricted indirectly by controlling the maximum dosing rates of polyamine flocculants to no more than 2.5 mg/L.

A European (CEN) standard for polyamine flocculants for water treatment (BS EN 1409) proposed maximum contaminant levels of 500 mg/L for 1,3-DCP and 1,000 mg/L for 2,3-DCP. These are not health-based values and may need to be reconsidered in the light of COC advice, and the approach taken for other genotoxic carcinogens.”

DWI (2013) states that BS EN 1409 limits 3-monochloropropane-1,2-diol, 1,3-dichloro-2-propanol and 2,3-dichloro-1-propanol to 40 mg/kg of active polyamine product.

There have been reports of halohydrins being formed after disinfection with chlorine and with ozone.

### Forms and fate in the environment

1,3-DCP is not expected to be adsorbed to suspended solids or sediments in water bodies. Because of the estimated low Henry’s Law Constant (6 x 10-7 atm-m3/mol) and the low experimental vapour pressure (0.75 mm Hg), volatilisation from dry soil and water surfaces is not expected. At neutral pH, the hydrolysis rate (0.0031 L/hr) corresponds to a half-life in water of 1.4 years. At pH 8, the rate of 850 L/mol-hr corresponds to a half-life of 34 days.

If released to soil, 1,3-dichloro-2-propanol is expected to have very high mobility based upon an estimated Koc of 4. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 6 x 10-7 atm‑cu m/mole. Based upon conflicting results with aerobic aqueous screening biodegradation tests, the importance of biodegradation in soil and water is unknown. If released into water, 1,3-dichloro-2-propanol is not expected to adsorb to suspended solids and sediment in the water column based upon the estimated Koc. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. 1,3-Dichloro-2-propanol is expected to have a hydrolysis half-life at pH 7 and 8 of approximately 1.4 years and 34 days, respectively (EAWAG accessed February 2015).

Water solubility of 1,3-DCP is about 10 percent.

### Typical concentrations in drinking-water

Drinking water treatment chemicals are tested for compliance with ANSI/NSF (American National Standards Institute/NSF International) Standard 60. In the period 1991–1999, NSF International found non-compliance nine times due to exceedance of the 9 ppb action level for dichloropropanols.

NSF (2010) reported the results of testing 112 samples of drinking water for 1,3‑dichloro-2-propanol; the median was 0.0006 mg/L and the range was <0.0001 to 0.008 mg/L.

### Removal methods

By product specification.

### Recommended analytical techniques

#### Referee method

Not required.

#### Some alternative methods

See DWI (2003); Matthew and Anastasio (2000). Also see IARC (2012).

### Health considerations

Exposure to 1,3-DCP may occur from ingestion of food to which hydrochloric acid-hydrolysed vegetable protein has been added, or drinking-water in which epichlorohydrin polyamine polyelectrolytes are used as flocculents and coagulants for water purification. Food is expected to be the main exposure. It is an undesirable contaminant in food; WHO (2007) expressed the opinion that its concentration in acid-hydrolysed vegetable protein should be reduced as far as technically achievable.

Based on the current evidence, the ANZFA Board agreed that a limit of 0.2 mg/kg for 3-MCPD and 0.005 mg/kg for 1,3-DCP would be appropriate and has recommended to the Australia New Zealand Food Standards Council that these levels be included in the Australia New Zealand Food Standards Code for soy and oyster sauces imported into or manufactured in Australia and New Zealand FSANZ (2001).

The following has been copied from DWI (2007):

“In 1991 the COC considered 1,3-DCP to be a genotoxic carcinogen. During 2001, the COM evaluated the mutagenicity of 1,3-DCP and 2,3-DCP and while there was evidence of genotoxicity *in vitro*, there were no suitable *in vivo* studies. The COM concluded that it would be prudent to regard 1,3-DCP and 2,3-DCP as potentially genotoxic *in vivo* and recommended that both compounds should be tested for genotoxicity *in vivo*, using the approach set out in the COM guidelines.

In light of this advice, and after evaluating carcinogenicity data, the COC concluded that it is prudent to assume that 1,3-DCP is a genotoxic carcinogen. After reviewing 2,3-DCP, COC took a precautionary approach and concluded that 2,3-DCP may possess genotoxicity *in vivo*, but that no conclusions could be drawn as to carcinogenicity. For both dichloropropanols the COC recommended that exposure should be reduced to as low as technologically feasible.”

The results (IPCS a) of a long-term toxicity/carcinogenicity study in rats treated at dose levels of 2.1, 6.3, or 19 mg 1,3-dichloro-2-propanol/kg bw/day in the drinking-water for 104 weeks indicated a carcinogenic effect. Induction of benign and malignant tumours of the liver, kidney, thyroid gland, and oral epithelia/tongue was observed in rats at the mid- and high-dose levels. Investigations on the genotoxic mechanisms of 1,3-dichloro-2-propanol indicate that the genotoxic effect of 1,3-dichloro-2-propanol depends on the chemical formation of epichlorohydrin, which has mutagenic activity.

IARC (2012) considered 1,3-dichloro-2-propanol is possibly carcinogenic to humans (Group 2B).

Although only a few studies of kinetics and metabolism and few short- and long-term studies of toxicity and of reproductive toxicity were available for evaluation, they clearly indicated that 1,3-dichloro-2-propanol was hepatotoxic and nephrotoxic, induced a variety of tumours in various organs in rats, and was genotoxic in vitro. The Committee therefore concluded that it would be inappropriate to estimate a tolerable intake of 1,3-dichloro-2-propanol. The Committee noted that the dose that caused tumours in rats (19 mg/kg bw per day) was about 20,000 times greater than the highest estimated intake of 1,3-dichloro-2-propanol by consumers of soy sauce (1 μg/kg bw per day), and that the available evidence suggested that in soy sauces 1,3-dichloro-2-propanol was associated with high concentrations of 3-chloro-1,2-propanediol, concentrations of the latter being approximately 50 times higher than those of 1,3-dichloro-2-propanol. Therefore, in the opinion of the Committee, regulatory control of the latter would obviate the need for specific controls on 1,3-dichloro-2-propanol (WHO 2007).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# 2,3-dichloro-1-propanol

CAS No. 616-23-9 and 82890-22-0. A member of the chloropropanols. Also called 2,3‑dichloropropanol or 2,3-DCP, and 1,2-dichloro-3-propanol.

Other important chloropropanols are 3-monochloropropane 1,2-diol (CAS No. 96‑24‑2) also called 3-MCPD (qv), 1,3 dichloro-2-propanol (1,3-DCP) – see previous datasheet.

These chemicals belong to the large, loosely defined group of chemicals called halohydrins. Other halohydrins are listed in the halohydrin datasheet.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2,3-dichloro-1-propanol. The WHO Guidelines do not mention 2,3-dichloro-1-propanol.

NSF Standard 60 states that the allowable concentration of 1,2-dichloro-3-propanol in drinking water is 0.009 mg/L.

### Sources to drinking-water

#### 1. To source waters

Chloropropanol isomers are by-products of the production of hydrolysed vegetable proteins. Also an intermediate in epichlorohydrin production. Uses include adhesives, sealants, paints and coatings.

#### 2. From treatment processes

2,3 Dichloro-1-propanol can be present as a contaminant in epichlorhydrin/amine copolymers used as flocculants or coagulant aids in water treatment. These polyamine flocculants have been available for many years as approved products for use in water treatment and thus 2,3 dichloro-1-propanol may be present in drinking water from their use. See previous datasheet for further discussion.

The following has been copied from DWI (2007):

“There are no statutory levels for 1,3-DCP or 2,3-DCP in drinking water. 3-MCPD and DCPs are known to co-exist in polyamine flocculants, but the relationship between their contaminant levels is not currently understood. However, their levels in drinking-water are restricted indirectly by controlling the maximum dosing rates of polyamine flocculants to no more than 2.5 mg/L.

A European (CEN) standard for polyamine flocculants for water treatment (BS EN 1409) proposed maximum contaminant levels of 500 mg/L for 1,3-DCP and 1,000 mg/L for 2,3-DCP. These are not health-based values and may need to be reconsidered in the light of COC advice, and the approach taken for other genotoxic carcinogens.”

### Forms and fate in the environment

2,3-Dichloro-1-propanol is more chemically stable than its isomer, 1,3-dichloro-2-propanol, and is therefore more difficult to degrade.

If released to soil, 2,3-dichloropropanol is expected to have very high mobility based upon an estimated Koc of 4. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 4 x 10-9 atm‑cu m/mole. Limited data indicate that 2,3-dichloropropanol may slowly degrade in soil. If released into [water](http://pubchem.ncbi.nlm.nih.gov/compound/water), 2,3-dichloropropanol is not expected to adsorb to suspended solids and sediment in the [water](http://pubchem.ncbi.nlm.nih.gov/compound/water) column based upon the estimated Koc. Volatilisation from [water](http://pubchem.ncbi.nlm.nih.gov/compound/water) surfaces is not expected to be an important fate process based upon this compound’s estimated Henry’s Law constant. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Its hydrolysis half-life at pH 7 and 8 is 1.4 years and 140 days, respectively. From NIH.

### Typical concentrations in drinking-water

Drinking water treatment chemicals are tested for compliance with ANSI/NSF (American National Standards Institute/NSF International) Standard 60. In the period 1991–1999, NSF International found non-compliance nine times due to exceedance of the 9 ppb action level for dichloropropanols.

NSF (2010) reported the results of testing 112 samples of drinking water for 1,2‑dichloro-3-propanol; the median was <0.001 mg/L and the range was <0.001 to 0.008 mg/L.

### Recommended analytical techniques

#### Referee method

Not required.

#### Some alternative methods

See DWI 2003; Matthew and Anastasio (2000).

### Health considerations

The following has been copied from DWI (2007):

“In 1991 the COC considered 1,3-DCP to be a genotoxic carcinogen. During 2001, the COM evaluated the mutagenicity of 1,3-DCP and 2,3-DCP and while there was evidence of genotoxicity *in vitro*, there were no suitable *in vivo* studies. The COM concluded that it would be prudent to regard 1,3-DCP and 2,3-DCP as potentially genotoxic *in vivo* and recommended that both compounds should be tested for genotoxicity *in vivo*, using the approach set out in the COM guidelines.

In light of this advice, and after evaluating carcinogenicity data, the COC concluded that it is prudent to assume that 1,3-DCP is a genotoxic carcinogen. After reviewing 2,3-DCP, COC took a precautionary approach and concluded that 2,3-DCP may possess genotoxicity *in vivo*, but that no conclusions could be drawn as to carcinogenicity. For both dichloropropanols the COC recommended that exposure should be reduced to as low as technologically feasible.”

The oral RfD for 2,3-dichloropropanol was calculated at 0.003 mg/kg/d (USEPA 1990).

### Derivation of Maximum Acceptable Value

No MAV.

### References

COC. 2001. *Committee on the Carcinogenicity of Chemicals in Food, Consumer Products and the Environment*. www.iacoc.org.uk/papers/documents/cc-01-06internet.pdf.

Department of Health. May 2001. *Carcinogenicity of 1,3-dichloropropan-2-ol (1,3 DCP) and 2,3-dichloropropan-1-ol (2,3 DCP) COC statement COC/01/S1*. See: http://www.advisorybodies.doh.gov.uk/coc/cocdcp.htm.

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# Dichloropropenes

CAS No. for cis-1,3-dichloropropene is 10061-01-5. CAS No. for trans‑1,3‑dichloropropene is 10061-02-6.

### Maximum Acceptable Value

The MAV for 1,3-dichloropropene (total of cis and trans forms) is 0.02 mg/L. See datasheet in the Pesticides Section.

The DWSNZ do not have a MAV for the other dichloropropenes. The WHO Guidelines do not mention the other dichloropropenes.

### Sources to drinking-water

#### 1. To source waters

The dichloropropenes isomers are:

* 1,1-dichloropropene (CAS No. 563-58-6)
* 1,2-dichloropropene (CAS No. 563-54-2)
* 1,3-dichloropropene (CAS No. 542-75-6) – see datasheet in pesticides section
* 2,3-dichloropropene (CAS No. 78-88-6)
* 3,3-dichloropropene (CAS No. 563-57-5).

1,3-Dichloropropene is used mainly in farming as a pesticide (see datasheet in pesticides section). Much less is known about the other dichloropropenes. 2,3‑Dichloropropene is used in industry to make other chemicals. No uses were found for 1,1-, 1,2-, or 3,3-dichloropropene. The older formulations of technical-grade 1,3‑dichloropropene called Telone II® included about 2.5 percent of 1,2‑dichloropropene, and the pesticide known as D-D contained about 25 percent 1,2‑dichloropropene with minor components including 2,3-dichloropropene, 3,3‑dichloropropene, 1,2,3-trichloropropane, trichloropropene, and allyl chloride (USEPA 2000).

Most of the information on dichloropropenes is for 1,3-dichloropropene. There is much less information for 2,3-dichloropropene, almost no information on 1,2‑dichloropropene, and no information on 1,1- and 3,3-dichloropropene.

#### 2. From treatment processes

No information, so probably not a disinfection by-product.

### Forms and fate in the environment

If released to soil, cis-1,3-dichloropropene is expected to have very high mobility based upon a range of Kocs from 20 to 42. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 2.7 x 10-3 atm‑cu m/mole for the cis form and 8.7 x 10-4 atm‑cu m/mole for the trans form. If released into water, neither 1,3-dichloropropene is expected to adsorb to suspended solids and sediment based upon the Kocs. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 1.3 and 102 hours (cis form) and 2 hours and 4.5 days (trans form). An estimated BCF of 8 suggests the potential for bioconcentration in aquatic organisms is low. cis-1,3-Dichloropropene degrades by chemical hydrolysis; at pH values of 5, 7, 9, the half-life of 1,3-dichloropropene was 13.5 days at 20°C (EAWAG accessed February 2015).

Information on the release, environmental fate and partitioning, concentrations in environmental media, and potential for human exposure is very limited for 1,1-, 1,2-, 2,3-, and 3,3-dichloropropene. Based on their physical and chemical properties, these substances are expected to behave similarly to 1,3-dichloropropene when they are released into the environment. However, hydrolysis of 1,1- and 1,2-dichloropropene is expected to be much slower than hydrolysis of the other dichloropropene isomers due to the inhibiting effect of the vinylic chlorine atoms. 1,1-Dichloropropene may be formed as a metabolite during the anaerobic degradation of higher chlorinated propenes.

Water solubility is:

* 1,1-dichloropropene – unknown
* 1,2-dichloropropene – 2700 mg/L
* 2,3-dichloropropene – 2150 mg/L
* 3,3-dichloropropene – unknown.

### Typical concentrations in drinking-water

1,1-Dichloropropene was detected in only 0.01 percent of 97,698 public water system samples collected in the US between 1993 and 1997. The source of 1,1-dichloropropene in these water samples is unknown. 1,1-Dichloropropene does not appear to be produced or used based on available data; therefore, direct release of this substance into the environment is not expected.

Nine water utilities in the US reported detecting 1,1-dichloropropene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.167 mg/L.

Ten water utilities in the US reported detecting 1,3-dichloropropene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.13 mg/L.

### Recommended analytical techniques

#### Referee method

Not required.

#### Some alternative methods

None required.

### Health considerations

The potential for human exposure to 1,1-, 1,2-, and 3,3-dichloropropene is expected to be low because these chemicals are not produced or used in high amounts. Higher amounts of 2,3-dichloropropene may be released from facilities where this substance is produced or used.

Their small molecular size and lipid solubility facilitate rapid absorption and distribution throughout the body. Metabolism, primarily in the liver, but also in other tissues, results either in detoxification and elimination, or bioactivation to more a toxic or mutagenic metabolite.

1,1-Dichloropropene is a contaminant of some source waters used to make drinking-water. No *in vivo* toxicity or toxicokinetic data were located for 1,1-dichoropropene. *In vitro* metabolism results of one study indicate that this isomer differs from 1,3‑dichloropropene and 2,3-dichloropropene in that conjugation to glutathione results in bioactivation to a mutagenic metabolite, rather than the production of innocuous mercapturic acid metabolites. Because of this and the fact that limited toxicological data were available for this compound, which is structurally similar to the rodent carcinogen 1,3-dichloropropene, 1,1-dichloropropene was placed on the Contaminant Candidate List of the USEPA. 1,1-Dichloropropene is significantly more mutagenic than 1,1-dichloroethene.

ATSDR has developed oral minimal risk levels (MRLs) for some chemicals – see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>. As at July 2013 MRLs for 1,3‑dichloropropene are:

* 0.04 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.03 mg/kg/day for chronic-duration oral exposure (> 364 days)

No information is available about the toxicokinetic properties of 1,2-dichloropropene. Toxicity information is limited to a brief summary of results of acute-duration studies in animals exposed at high or unreported exposure levels.

No data are available for developmental or carcinogenic effects of exposure to 2,3‑dichloropropene. The toxicokinetic properties of 2,3-dichloropropene appear to be similar to those of 1,3-dichloropropene.

No toxicity or toxicokinetic data were located for 3,3-dichloropropene.

### Derivation of Maximum Acceptable Value

No MAV for the dichloropropenes, other than 1,3-dichloropropene.

### References

ATSDR. 2008. *Toxicological Profile for Dichloropropenes*. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. http://www.atsdr.cdc.gov/toxprofiles/index.asp.

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WHO. 2017. *Guidelines for Drinking-water Quality: Fourth edition incorporating the first Addendum*. Geneva: World Health Organization. 631 pp. [http://www.who.int/water\_sanitation\_health/publications/drinking-water-quality-guidelines-4-including-1st-addendum/en/](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\INetCache\Content.Word\www.who.int\water_sanitation_health\publications\2011\dwq_guidelines\en\).

# 2,3-dichloropropionic acid

CAS No. 565-64-0. IUPAC Name: 2,3-dichloropropanoic acid, or 2,3-DCPA. Note that 2,2-dichloropropionic acid is the pesticide dalapon (qv).

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2,3-dichloropropionic acid. The WHO Guidelines do not mention 2,3-dichloropropionic acid.

### Sources to drinking-water

#### 1. To source waters

No information.

#### 2. From treatment processes

Stockham and Morran reported that the major DBPs from the chlorination of acrylamide monomer were found to be 2,3-dichloropropionic acid and monochloroacrylic acid.

### Recommended analytical techniques

#### Referee method

Not required.

#### Some alternative methods

None required.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Hasegawa R, Naitoh K, Kawasaki Y, et al. 1990. Acute and subacute toxicity studies on 2,3-dichloropropionic acid and chlorinated polyacrylamide in rats. *Water Research* 24(5): 661–6 (not sighted).

Stockham P, Morran J. 2000. Disinfection by-products from a poly(diallyldimethylammonium chloride) based polyelectrolyte used in water treatment. *CRC for Water Quality and Treatment*. Research Report No 4. Available at: http://www.waterquality.crc.org.au/programs/program2d.htm.

# Dicyandiamide

CAS No. 461-58-5. Also called DCD, DICY, dicyanodiamide, cyanoguanidine, 1‑cyanoguanidine (IUPAC name), and 2-cyanoguanidine.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for dicyandiamide. The WHO Guidelines do not mention dicyandiamide.

### Sources to drinking-water

#### 1. To source waters

Organic N is converted from urea to ammonium salts (NH4+) by a variety of bacteria, actinomycetes and fungi in a process known as mineralisation. Autotrophic bacteria (*Nitrosomonas*) convert the ammonium to nitrite (NO2-) and further modification by other bacteria (*Nitrobacter*) converts nitrite to nitrate (NO3-). This process of chemical oxidation is known as nitrification. Nitrogen in these forms is very soluble and is easily removed from the soil by leaching (AFBI 2005).

Dicyandiamide is a biodegradable nitrogen inhibitor that slows the rate that soil bacteria, eg, *Nitrosomas/Nitrobacter,* convert ammonia into nitrite/nitrate and nitrous oxide (MPI 2012). It is a [dimer](http://en.wikipedia.org/wiki/Dimer_(chemistry)) of [cyanamide](http://en.wikipedia.org/wiki/Cyanamide). DCD is the only product recognised nationally to mitigate greenhouse gas emissions in agricultures and is an ingredient in the products Eco-n and DCN. It is applied at the rate of about 10 kg/ha, mainly during autumn, winter and spring. DCD is not a biocide and has no effect on other soil microbial biomass. It acts specifically on the enzyme ammonia monooxygenase contained in *Nitrosomas* by blocking the site where ammonium is converted to nitrite.

Smith and Schallenberg (2013) sampled 15 streams and drains in a coastal lowland agricultural catchment in the Lower Taieri Plain and found measurable concentrations of DCD in many of the surface waters, with a maximum measured concentration of approximately 1 mg/L.

As at January 2013, it is voluntarily off the market in New Zealand because traces have been found in milk products – investigations are underway. It won’t be reintroduced until a minimum international standard regarding acceptable levels of DCD in food products is set. That process could take several years as it involves the World Health Organization and the 170-member Codex Committee on Food Contaminants.

Dicyandiamide is widely used in industries such as electronics, as a curing agent for epoxies, in pharmaceuticals and food packaging. It is still sometimes used as an intermediate in the production of melamine. It is also used as a slow [fertiliser](http://en.wikipedia.org/wiki/Fertilizer) (66 percent nitrogen). World production is about 40,000 t/pa.

Dimethylpyrazole-phosphate (DMPP) is a much superior nitrification inhibitor to DCD and is effective at lower concentrations AFBI 2005. N-(n-butyl) thiophosphoric triamide has been used as well – see datasheet.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

When applied at 10 kg/ha, DCD remains in the soil about 100 days (20 to 160 days in trials), depending on soil pH, moisture and organic matter content, temperature and rainfall. An estimated Koc of 6 suggests that DCD should have very high mobility in soil. This compound is not expected to volatilise from water surfaces given its estimated Henry’s Law constant. The potential for bioconcentration in aquatic organisms should be low based on BCF values of <0.3 and <3.1, measured in carp.

Dicyandiamide does not hydrolyse under environmental conditions regardless of pH. As less than 10 percent of the test substance is hydrolysed in 5 days, dicyandiamid is considered hydrolytically stable. It is not readily biodegradable under aerobic condition within 28 days (BOD = 0 percent). However, a prolonged study showed that this substance is completely biodegraded within 34 weeks under aerobic conditions, while two-thirds of the total is biodegraded within 60 weeks under anaerobic conditions (INCHEM 2003).

DCD biodegrades to carbon dioxide, ammonia and water.

Water solubility is about 4 percent.

### Health considerations

The oral LD50 is greater than 30 g/kg bw in female rats. A combined repeated dose toxicity study with the reproduction/developmental toxicity screening test [OECD TG 422] was conducted using SD rats at doses of 0 (vehicle: 3 percent gum arabic solution), 40, 200, and 1,000 mg/kg/day. The dosing period for males was 44 days, and females were dosed from 14 days before mating to day 3 of lactation. This substance had no effect on clinical signs, body weights, food consumption or necropsy findings. The organ weights were similar among all groups. No histopathological changes ascribable to this substance in these organs were found in either sex. The NOAEL for the repeat dose toxicity is considered to be 1,000 mg/kg/day for both sexes. The NOAEL for reproductive and developmental toxicity is also considered to be 1,000 mg/kg/day. The reverse mutation studies in bacteria [OECD TG 471 and 472] gave negative results. The *in vitro* chromosomal aberration test with Chinese hamster lung cells (CHL/IU) [OECD TG 473] with and without metabolic activation was also negative. Therefore, this substance is not genotoxic. A carcinogenicity study was conducted in male and female Fischer 344 rats fed diets containing this substance at 0, 2.5 and 5 percent (male: 837.2 and 1958.6, female: 1001.3 and 2169.2 mg/kg bw/day) for up to two years. The study did not suggest an association of the substance with an increased tumour incidence (INCHEM 2003).

Acute oral and intraperitoneal toxicity was low in various species of laboratory animals. Studies involving repeated administration gave indications of effects in the liver, kidneys or blood of rats. There was no clear evidence of carcinogenic potential in rats fed dicyandiamide in the diet. Dicyandiamide was generally non-mutagenic in Ames bacterial tests (BIBRA).

ECHA reported the that the long term derived no effect level (DNEL) for the general population via the oral route is 6.5 mg/kg bw/d. Also, the non-observable-effect level (NOEL) of dicyandiamide administered to male and female beagle dogs is greater than 900 mg/kg/day in the diet for four weeks followed by 625 mg/kg/day administered via capsule for an additional three weeks. In another study, the NOAEL was determined to be greater than 1,300 mg/kg bw/d, based on a subchronic daily administration of dicyandiamide to male and female Sprague Dawley rats via dietary admixture for 13 weeks.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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Environment Waikato. 2004. *Nitrification and Urease Inhibitors*. 27 pp. http://www.waikatoregion.govt.nz/PageFiles/2883/TR04-22.pdf.

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INCHEM. 2003. *SIDS Initial Assessment Report* – Cyanoguanidine. UNEP Publications. 75 pp. <http://www.inchem.org/documents/sids/sids/461585.pdf>.

MPI. 2012. Effect of dicyandiamide (DCD) on white clover growth and nitrogen fixation in ryegrass / white clover pasture. *MPI Technical Paper* 2013/33. 32 pp. <http://www.mpi.govt.nz/news-and-resources/publications>.

Smith I, Schallenberg M. 2013. Occurrence of the agricultural nitrification inhibitor, dicyandiamide, in surface waters and its effects on nitrogen dynamics in an experimental aquatic system. *Agriculture, Ecosystems & Environment* 164: 23–31, January. <http://www.sciencedirect.com/science/article/pii/S016788091200326X> or http://waesearch.kobv.de/uid.do?query=rss\_feeds\_1994335&ref=feed.

# Dicyclopentadiene

CAS No. 77-73-6. Also called DCPD, (3a,4,7,7a-tetrahydro-1H-4,7-methanoindenem 1,3-cyclopentadiene dimer, and tricyclo[5.2.1.02,6]deca-3,8-diene.

DCPD occurs as two stereo-isomers, endo-DCPD and exo-DCPD. Although the exo form is thermodynamically more stable, commercial DCPD products contain mostly the endo molecule since it forms at a much greater rate than the exo form; typically >90 percent of the mixture present in commercial CDPD is the endo form.

### Maximum Acceptable Value

There is no MAV for dicyclopentadiene in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

IEH (2014) selected for consideration all those substances reported as being involved in taste and odour incidents in a developed country, excluding those for which there was no evidence of UK production or import, as well as those already regulated to a limit value either lower than or close to the reported taste and odour threshold. Other prioritised substances were then categorised according to amounts used and their reported taste and odour threshold. This process gave a list of compounds from which substances formed during water treatment were excluded leaving 18 priority compounds. In terms of taste and odour nuisance, dicyclopentadiene was rated as very high.

DCPD products may be best characterised as highly reactive intermediates since they react readily with other monomers and find application in the production of resins, alkyds, acrylates, latexes, elastomers (eg, EPDM), fragrances and other specialty intermediates. DCPD is increasingly substituting phthalic anhydride in fibreglass-reinforced unsaturated polyesters for applications such as pleasure boat construction. DCPD is used in the production of hexachlorocyclopentadiene, an intermediate used to manufacture of two of the leading types of fire retardant chemical, chlorendic anhydride and dechlorane, and there is also some evidence that it can be used as a pesticide.

Dicyclopentadiene is a clear/whitish crystalline solid at room temperature, possessing a disagreeable camphor-like odour with a taste and odour threshold in water of between 0.01 to 0.25 μg/L (IEH 2014 and DWI 2014).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

### Forms and fate in the environment

DCPD shows low water-solubility but has moderate soil mobility. Release to water will result in water contamination where it is stable for five days at 25°C. It is not adsorbed by particulate matter. A vapour pressure of 2.29 mm Hg suggests that volatilisation from dry soils is likely to occur but is unlikely to be rapid. Dicyclopentadiene exhibits a Henry’s law constant of 6.2 x 10-2 atm‑cu m/mole so volatalisation from moist soil is expected to be an important process. Dicyclopentadiene is predicted to exhibit a Koc of 1800, indicating moderate mobility in soil. A field study indicates that degradation of dicyclopentadiene in soils is likely to be due to bacterial action and as such a degree of variability in degradation rates is expected due to variations in local microbiological characteristics. The results of the field and laboratory studies indicate that it is unlikely that biodegradation represents a major method of removal of dicyclopentadiene from soils. Removal of dicyclopentadiene from a modelled river and lake is likely to be rapid, with half-lifes of 3.4 hours and 4.6 days respectively. Whilst no published studies examine dicyclopentadiene oxidation in aqueous environments other olefins undergo rapid photochemically mediated reactions with reactive oxygenated compounds in water. The Koc value of dicyclopentadiene indicates potential adsorption from aqueous solution to suspended soils in an aquatic environment.

### Removal methods

IEH (2014) reports that conventional treatment is ineffective at removing DCPD from water but activated carbon may remove about 75 percent.

### Health considerations

The rat oral LD50 has been established as 0.35 mL/kg (equivalent to 0.326 mg/kg at 25°C (IEH 2014).

OECD (1998) reports a study of rats given dicyclopentadiene orally at 4, 20 or 100 mg/kg/day for 44 days. Histopathology showed hepatic necrosis and alteredrenal tubular epithelium in male rats. The authors quote sex-specific NOAEL and LOAEL levels of: NOAEL: 4 mg/kg/day for males and 20 mg/kg/day for females; and LOAEL: 20 mg/kg/day for males and 100 mg/kg/day for females (from IEH 2014, which also reports a TDI of 0.004 mg/kg/d). Consuming dicyclopentadiene in water at its taste and odour threshold is unlikely to result in health effects.

### Derivation of Maximum Acceptable Value

No MAV.

IEH (2014) reports a taste and odour threshold of 0.01 to 0.25 μg/L.

### References

Dow. 2010. *Dicyclopentadiene Products: A guide to product handling and use*. 26 pp. <http://msdssearch.dow.com/PublishedLiteratureDOWCOM/dh_046d/0901b8038046d43a.pdf?filepath=aromatics/pdfs/noreg/778-04301.pdf&fromPage=GetDoc>.

DWI. 2014. *National Assessment of the Risks to Water Supplies Posed by Low Taste and Odour Threshold Compounds*. Final Report Project WT1275. 202 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-281.pdf>.

IEH. 2014. *National Assessment of the Risks to Water Supplies Posed by Low Taste and Odour Threshold Compounds*. Final Report Project WT1275. Institute of Environment and Health, Cranfield University. 202 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-281.pdf>.

# Di(2-ethylhexyl)adipate

CAS No. 103-23-1. Also called bis(2-ethylhexyl)adipate, DEHA, hexanedioic acid, adipic acid bis(2-ethylhexyl) ester, or dioctyl adipate, and a large number of trade names.

### Maximum Acceptable Value

Because di(2-ethylhexyl)adipate occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based MAV.

In DWSNZ 2005, the provisional MAV had been 0.1 mg/L.

The 1993 WHO Guidelines had proposed a health-based guideline value of 0.08 mg/L for DEHA in drinking-water.

The maximum contaminant level (USEPA 2006/2009/2011) is 0.4 mg/L. The USEPA (2006/2011) also established a lifetime health advisory of 0.4 mg/L, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that the data for di(2-ethylhexyl)adipate are inadequate to determine a health-based guideline value.

### Sources to drinking-water

#### 1. To source waters

Di(2-ethylhexyl)adipate is found in raw water through human and industrial activity. It is used primarily as a plasticiser in the flexible vinyl industry and is widely used in flexible polyvinylchloride (PVC) food film (cling film) and utensils, so may leach from tubing, dishes, containers, etc. DEHA can be added to cellophane (a cellulose-based material) too. Di(2-ethylhexyl)adipate is commonly blended with di(2-ethylhexyl) phthalate (see datasheet) and di(isooctyl) phthalate in PVC and other polymers. It is also used as a solvent, in hydraulic fluids and for aircraft lubrication. It is important in the processing of nitrocellulose and synthetic rubber, in plasticising polyvinyl butyral, cellulose acetate butyrate, polystyrene and dammar wax and in cosmetics (cellulose-based liquid lipsticks).

A survey of 23 major rivers and lakes in the USA showed that 7 percent of 82 samples contained DEHA at levels ranging from 0.0003 to 0.001 mg/L.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Di(2-ethylhexyl)adipate may leach from PVC pipes used in the distribution system.

### Forms and fate in the environment

Because of its low water solubility (about 0.005 mg/L or up to about 0.8 mg/L if emulsified), di(2-ethylhexyl)adipatereleased into the environment would be expected to partition to solids (biota, sediment, soil). Biodegradation is likely to be a significant removal mechanism from the aquatic environment. Model experiments with acclimated activated sludge systems have shown essentially complete biodegradation of relatively high concentrations (~20 mg/L) of di(2-ethylhexyl) adipate to carbon dioxide and water in 35 days.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 348 zones, did not find any di(2-ethylhexyl)adipate at detectable concentrations (limit of detection = 0.002 mg/L) (ESR 2001).

Overseas studies have detected DEHA at concentrations between 0.000001 mg/L (1 ng/L) to 0.0001 mg/L (100 ng/L) in treated drinking water.

215 water utilities in the US reported detecting di(2-ethylhexyl) adipate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.083 mg/L.

### Removal methods

No information is available on processes that can be used to remove di(2‑ethylhexyl)adipate from water. Processes that remove particulate matter may offer the best option.

### Analytical methods

#### Referee method

Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry (EPA 525).

#### Some alternative methods

1. Liquid–Liquid Extraction or Liquid-Solid Extraction and Gas Chromatography with Photoionisation Detection (EPA 506).

### Health considerations

As a consequence of its use in PVC films, food, particularly fatty foods such as cheese and meat, is the most important source of human exposure (up to 20 mg/day). Reformulation of PVC film reflecting the use of less di(2-ethylhexyl) adipate necessitated a more recent evaluation which suggested that the maximum daily intake of di(2-ethylhexyl) adipate in the United Kingdom was 8.2 mg in the mid-1990s (IARC 2000).

DEHA is of low short-term toxicity; however, dietary levels above 6,000 mg/kg of feed induce peroxisomal proliferation in the liver of rodents. This effect is often associated with the development of liver tumours. DEHA induced liver carcinomas in female mice at very high doses but not in male mice or rats. It is not genotoxic. IARC concluded that there is limited evidence that di(2-ethylhexyl)adipate is carcinogenic in mice, and has placed DEHA in Group 3, ie, not classifiable as to its carcinogenicity in humans. The USEPA (2009/2011) quotes a health advisory of 3 mg/L for di(2-ethylhexyl)adipate, representing a 10-4 cancer risk.

Di(2-ethylhexyl)adipate is absorbed readily when given orally to rats and mice. It is distributed widely in the body, with highest levels of metabolites reported in fatty tissue, liver and kidney. Transplacental transport of di(2-ethylhexyl)adipate has been reported.

Repeated-dose toxicity studies (up to 90-days) in rats and mice with DEHA in feed showed reduced body weight gains at levels of approximately 400 mg/kg and higher in rats and approximately 600 mg/kg and higher in mice (NOAELs of 189 mg/kg in rats and 451 mg/kg in mice). *In vitro* genotoxicity studies have been negative for mutations, unscheduled DNA synthesis and DNA interactions in bacterial and mammalian systems. *In vivo* genotoxicity studies have also been negative (two mouse micronucleus assays). DEHA has been evaluated for carcinogenicity in mice and rats, and there was no evidence of carcinogenicity in rats but there was evidence of liver cancer in female mice (significant incidence) and male mice (less significant). Tumours in mice were observed at high concentrations (3,222 mg/kg for females and 2,659 mg/kg in males). A one-generation reproductive toxicity test was performed in rats and there were no effects on reproduction although the body weight gains of pregnant dams and first generation pups was reduced at a dose level of approximately 3,222 mg/kg. A developmental toxicity study performed with DEHA in rats (animals treated orally via DEHA in feed on days 6–15 of gestation) demonstrated reduced maternal body weight gain at the highest dose (1,080 mg/kg/d). There was evidence of pre-implantation fetal loss at the highest dose, but no gross, skeletal, or visceral abnormalities. A NOAEL for developmental toxicity was determined in rats at an estimated oral dose of 170 mg/kg/d, based on slight fetotoxicity from reduced ossification which was not statistically significant (OECD 2005).

The acute oral toxicity of di(2-ethylhexyl)adipate is low. No data exists on the effects of ingested di(2-ethylhexyl)adipate in humans. Short-term mouse and rat toxicity studies have demonstrated that high dietary levels of the compound induce liver toxicity, which is often associated with the development of liver tumours, particularly in female mice.

Di(2-ethylhexyl)adipate has not exhibited mutagenic activity when applied to strains of bacteria or to mammalian cells.

The reference dose or RfD (USEPA 1992/2006/2009/2011) is 0.6 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 20 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

WHO (2004/2011) considered a health-based value of 0.08 mg/L can be calculated for DEHA on the basis of a TDI of 0.28 mg/kg of body weight based on fetotoxicity in rats and allocating 1 percent of the TDI to drinking-water. However, because DEHA occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value.

The MAV for di(2-ethylhexyl)adipate in the 1995, 2000 and 2005 DWSNZ had been 0.1 mg/L, based on the following:

Although di(2-ethylhexyl)adipate is carcinogenic in mice, the toxicity profile and lack of evidence of mutagenicity of di(2-ethylhexyl)adipate support the use of a tolerable daily intake approach to deriving a MAV for di(2-ethylhexyl)adipate in drinking-water. The provisional MAV in earlier DWSNZ had been derived on the basis of a no-observable-adverse-effects level determined in a 90-day toxicological study in rats, as follows:

28 mg/kg body weight per day x 70 kg x 0.01 = 0.098 mg/L (rounded to 0.1 mg/L)

2 L x 100

where:

* no-observable-adverse-effect level = 28 mg/kg body weight per day in a fetotoxicity study in rats
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.01
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 100 for intra- and interspecies variation.

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# Di(2-ethylhexyl) phthalate

CAS No. 117-81-7. Also called bis(2-ethylhexyl)phthalate, DEHP, 1,2‑benzenedicarboxylic acid, diethylhexyl phthalate, bis(2-ethylhexyl) ester, dioctyl phthalate (DOP), bis(2-ethylhexyl)-1,2-benzenedicarboxylate and others, plus a variety of trade names.

Phthalates other than di(2-ethylhexyl)phthalate are discussed in a datasheet titled Phthalates.

### Maximum Acceptable Value

Based on health considerations, the concentration of di(2-ethylhexyl)phthalate in drinking-water should not exceed 0.009 mg/L (9 g/L).

The maximum contaminant level (USEPA 2006/2009/2011) is 0.006 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of di(2-ethylhexyl) phthalate in drinking water should not exceed 0.01 mg/L.

Di(2-ethylhexyl)phthalate is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Di(2-ethylhexyl)phthalate is found in raw water through human and industrial activity. It is used primarily as a plasticiser in many flexible PVC products and in vinyl chloride co-polymer resins (where it can occasionally reach even 40 percent by weight). DEHP is the most widely used plasticiser and appears in a wide range of consumer products; about 95 percent is used as a plasticiser for PVC. DEHP may leach from tubing, dishes, food containers, etc. It is also used as a replacement for polychlorinated biphenyls in dielectric fluids for low voltage electrical capacitors.

The information collected by NICNAS (2010) identified that in Australia DEHP is imported as a component of perfumery and cosmetic products of unidentified origin with typical concentrations of approximately 0.05 percent. Some businesses indicated phasing out of DEHP in cosmetic applications following the ban of DEHP for use in cosmetics in the European Union (EU). However, given the absence of regulatory measures limiting the use of DEHP in cosmetics in Australia its potential use in these applications cannot be excluded. International use of DEHP in children’s toys and childcare articles is reducing.

World consumption of phthalates in the early 1990s was estimated to be 3.25 million tonnes, of which di(2-ethylhexyl)phthalate accounted for approximately 2.1 million tonnes.

It has been found in surface water, groundwater and drinking-water in concentrations of a few micrograms per litre; in polluted surface water and groundwater, concentrations of hundreds of micrograms per litre have been reported. It is also a component of diesel exhaust.

EU (2008) reports that surface water concentrations in unpolluted lakes ranged from  
0–0.013 μg/L (mean 0.009 μg/L, n=6). Levels of DEHP in surface waters from urban areas ranged between 0.010 and 0.33 μg/L. In a 1996-98 study carried out in Denmark, concentrations found in water were <0.2 to 0.87 μg/L, with the highest levels observed during increased water flow.

In groundwater associated with non-specified contaminated land, measured concentrations of DEHP ranged between 0.05 and 45 μg/L. In one study agricultural soil was treated with STP sludge prior to groundwater sampling; DEHP levels in groundwater were between 0 and 510 μg/L. In a monitoring study performed on DEHP in private and public water supply boreholes in UK DEHP was found in seven out of 11 samples. The average measured concentration was 0.07 μg/L (EU 2008).

Some customers request the additive Bisphenol A in the range of 0.025 to 0.5 percent.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Di(2-ethylhexyl)phthalate may leach from PVC pipes used in the distribution system. PVC cables buried in soil lost about 1.2 percent DEHP per annum (EU 2008).

### Forms and fate in the environment

Di(2-ethylhexyl)phthalate is insoluble in water (about 0.03 mg/L) but may be transported in the aquatic environment as complexes with humic substances or sorption on to particulate matter and biota. Since di(2-ethylhexyl)phthalate is lipophilic, it tends to be absorbed on to sediment, which serves as a sink. Volatilisation and photolysis are not expected to be important removal mechanisms of di(2‑ethylhexyl)phthalate from the aquatic environment, but aerobic biodegradation and bioaccumulation will be significant.

DEHP can break down in the presence of other chemicals to produce mono(2‑ethylhexyl)phthalate (MEHP) and 2-ethylhexanol. Many of the properties of MEHP are like those of DEHP, and therefore its fate in the environment is similar.

EU (2008) quotes: vapour pressure = 0.000034 Pa at 20°C; partition coefficient n-octanol/water = logPow = 7.5; Henry’s law constant = 4.43 Pa.m3/mol. The abiotic hydrolysis of DEHP to mono(2-ethylhexyl)phthalate (MEHP) and 2-ethylhexanol is expected to be very slow; an estimated half-life of approximately 2,000 years has been reported. The low photo-oxidative degradation of DEHP is not surprising since the resistance of DEHP to photo-oxidation in general is one of the characteristics that make it suitable as a plasticiser in durable products. In oligotrophic water, no mineralisation was observed during 60 days; while in eutrophic waters 35–71 percent was mineralised at 29°C after 40 days; the main degradation product MEHP is of toxicological relevance. Under anaerobic conditions, the screening data indicate that DEHP is persistent to biodegradation in sludge.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 348 zones, found di(2-ethylhexyl)phthalate concentrations to range from “not detectable” (nd) to 0.007 mg/L (a single sample), with the median concentration being “nd” (limit of detection = 0.002 mg/L); 121 people received drinking-water with >50 percent of the MAV (ESR 2001).

The Watercare Services’ 2013 Annual Water Quality Report states that the mean concentration of di(2-ethylhexyl)phthalate in 13 samples of the water leaving the Waikato Water Treatment plant was 0.0012 mg/L, and the maximum was 0.0023 mg/L.

Overseas studies have detected DEHP in drinking-water on a few occasions at concentrations from 0.00005 mg/L (50 ng/L) to 0.01 mg/L. Frequently even found in rainwater, up to 0.003 mg/L.

Thirty-seven water utilities in the US reported detecting bis(2-ethylhexyl) phthalate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 1.09 mg/L; this single result was somewhat of an outlier, the next highest was 0.05 mg/L. 1620 water utilities in the US reported detecting di(2-ethylhexyl) phthalate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.018 mg/L. It is not known why EWG reported these separately – they are synonyms.

### Removal methods

No information is available on processes that can be used to remove di(2‑ethylhexyl)phthalate from water. However, because it sorbs strongly to soils, any treatment process that removes turbidity should be reasonable effective at reducing the concentration of di(2-ethylhexyl)phthalate from water.

### Analytical methods

#### Referee method

Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry (EPA 525).

Detection and quantification of very low levels of DEHP are seriously limited by the presence of this compound as a contaminant in almost all laboratory equipment and reagents. Plastics, glassware, aluminium foil, cork, rubber, glass wool, solvents and Teflon sheets have all been found to be contaminated (IARC 2012).

#### Some alternative methods

1. Liquid–Liquid Extraction or Liquid-Solid Extraction and Gas Chromatography with Photoionisation Detection (EPA 506).

### Health considerations

In general, food will be the main exposure route, followed by inhalation, then water which may be about 1 percent. The European Union has temporarily banned the use of six phthalates, including di(2-ethylhexyl)phthalate, in toys and other articles intended for children aged under three years of age and designed to be put in the mouth. Several countries in Europe also have proposed, or are considering, restrictions on use of phthalates as plasticisers in PVC toys and baby care items.

In rats, di(2-ethylhexyl)phthalate is well-absorbed from the gastrointestinal tract after oral administration. Absorption is lower in humans and 11 to 25 percent of an ingested dose was found in urine. Mono(2-ethylhexyl)phthalate and its metabolites are extensively distributed throughout the body in rodents. The highest levels were found in the liver and fatty tissue. No or little accumulation occurs in rats.

The acute oral toxicity of di(2-ethylhexyl)phthalate is low. Liver and testes appear to be the main target organs in di(2-ethylhexyl)phthalate toxicity. The most striking effect in short-term toxicity studies is the proliferation of hepatic peroxisomes. The available data suggest that primates, including humans, are far less sensitive to this effect than rodents.

Tests on mice, rats, guinea pigs and ferrets have reported testicular effects, consisting of atrophy, tubular degeneration and inhibition or cessation of spermatogenesis. In similar studies, other effects reported included suppression of fertility, foetal mortality, foetal resorption, decreased foetal weight, neural tube effects and skeletal disorders.

Di(2-ethylhexyl)phthalate has not exhibited mutagenic activity when applied to strains of bacteria or to mammalian cells. In long-term oral carcinogenicity studies, hepatocellular carcinomas were found in rats and mice.

The International Agency for Research on Cancer (IARC 2000) concluded that di(2‑ethylhexyl)phthalate is not classifiable as to its carcinogenicity to humans (Group 3). In making this overall evaluation, the Working Group took into consideration that

(a) di(2-ethylhexyl)phthalate produces liver tumours in rats and mice by a non-DNA-reactive mechanism involving peroxisome proliferation

(b) peroxisome proliferation and hepatocellular proliferation have been demonstrated under the conditions of the carcinogenicity studies of di(2‑ethylhexyl)phthalate in rats and mice

(c) peroxisome proliferation has not been documented in human hepatocyte cultures exposed to di(2-ethylhexyl)phthalate nor in the liver of exposed non-human primates.

Therefore, the mechanism by which di(2-ethylhexyl)phthalate increases the incidence of hepatocellular tumours in rats and mice is not relevant to humans. However, the 2011 Working Group concluded that the human relevance of the molecular events leading to DEHP-induced cancer in several target tissues (eg, liver and testis) in rats or mice could not be ruled out, resulting in the evaluation of DEHP as a Group 2B agent (possibly carcinogenic to humans), rather than Group 3 (IARC 2012).

The USEPA (2009/2011) quotes a health advisory of 0.3 mg/L for di(2‑ethylhexyl)phthalate, representing a 10-4 cancer risk.

Di(2-ethylhexyl)phthalate appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for di(2-ethylhexyl)phthalate of:

* 0.1 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.06 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD (USEPA 1991/2006/2009/2011) is 0.02 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.7 mg/L.

In NIH (2006), the NTP concluded that there is serious concern that certain intensive medical treatments of male infants may result in DEHP exposure levels that adversely affect development of the male reproductive tract. DEHP exposure from medical procedures in infants was estimated to be as high as 6,000 μg/kg bw/day. There is concern for adverse effects on development of the reproductive tract in male offspring of pregnant and breastfeeding women undergoing certain medical procedures that may result in exposure to high levels of DEHP. There is concern for effects of DEHP exposure on development of the male reproductive tract for infants less than one year old. Diet, mouthing of DEHP-containing objects, and certain medical treatments may lead to DEHP exposures that are higher than those experienced by the general population. There is some concern for effects of DEHP exposure on development of the reproductive tract of male children older than one year. As in infants, exposures of children to DEHP may be higher than in the general population. There is some concern for adverse effects of DEHP exposure on development of the male reproductive tract in male offspring of pregnant women not medically exposed to DEHP. Although DEHP exposures are assumed to be the same as for the general population, the developing male reproductive tract is sensitive to the adverse effects of DEHP. There is minimal concern for reproductive toxicity in adults exposed to DEHP at 1–30 μg/kg bw/day. This level of concern is not altered for adults medically exposed to DEHP.

Di-(2-ethylhexyl)phthalate is on the EC List of 66 Category 1 substances showing evidence of endocrine disrupting activity in at least one species using intact animals (EC 2015).

### Derivation of Maximum Acceptable Value

Based on the absence of evidence for genotoxicity and the suggested relationship between prolonged proliferation of liver peroxisomes and the occurrence of heptacellular carcinomas, a tolerable daily intake approach was taken to derive the MAV.

The MAV for di(2-ethylhexyl)phthalate in drinking-water was derived as follows:

2.5 mg/kg body weight/day x 70 kg x 0.01 = 0.00875 mg/L (rounded to 0.009 mg/L)

2 L x 100

where:

* no-observable-adverse-effect level = 2.5 mg/kg body weight per day based on peroxisomal proliferation in the liver in rats
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.01
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 100 for intra- and interspecies variation (although the mechanism for heptacellular tumour induction is not fully resolved, using a NOAEL derived from the far most sensitive species with respect to the particularly sensitive end-point of peroxisomal proliferation justifies the use of this uncertainty value).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to valuate potential human health risks from exposures to chemicals in drinking water. The acute, short-term, subchronic and chronic for di(2‑ethylhexyl)phthalate are 0.006 mg/L, and for cancer: 0.007 mg/L.

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# N,N-diethyl-m-toluamide (DEET)

CAS No. 134-62-3. N,N-diethyl-m-toluamide is the IUPAC name. The commercial product contains other isomers, usually less than 5 percent; the o- and p-isomers are highly repellent but less effective than the m-isomer. Also known as diethyltoluamide and N,N-diethyl-3-methylbenzamide.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for N,N-diethyl-m-toluamide. The WHO Guidelines do not mention N,N-diethyl-m-toluamide.

### Sources to drinking-water

#### 1. To source waters

N,N-diethyl-m-toluamide is a member of the N,N-dialkylamide family of chemicals.

DEET has been used as a plasticiser and solvent for resin and film formers; it can damage certain rubber, plastic, vinyl, or elastic materials such as contact lenses, eyeglass frames and lenses, watch crystals, combs, painted and varnished surfaces, nail polish, and certain synthetic or treated fabrics. DEET does not damage natural fibres including cotton and wool (NPIC).

The main use of DEET is as a repellant against biting and flying insects such as biting flies, mosquitoes and midges. It has been estimated that approximately 30 percent of the US population uses DEET annually as a personal insect repellent.

DEET has been found in water up to 0.0001 mg/L where wastewater is thought to contribute to stream-flow (DWI 2014).

### Forms and fate in the environment

DEET is moderately mobile in soil and is stable to hydrolysis at normal pH levels. Although it is generally described as insoluble, the California EPA (2000) states that its water solubility is 2 to 3 mg/mL, ie, 2,000 to 3,000 mg/L. And EC (2010) states “The solubility of DEET in water is high (11.2 g/L with no pH control). The pH dependency of the solubility was not assessed, but DEET is not considered to be able to dissociate at environmentally relevant pH”.

EU (2010): Fate and effects data are only provided for the parent structure. DEET is considered to be ready biodegradable and no major (>10 percent) transformation products were formed in studies of hydrolysis and aquatic phototransformation. DEET has a vapour pressure of 0.23 Pa (25°C) and a Henry’s law constant of 3.93E-3 Pa\*m3/mol (2.1 x 10-8 atm‑cu m/mole). The substance is predicted to have an atmospheric half-life of 0.63 days (15.2 hours). Thus an accumulation of DEET in air and long range transport is unlikely. DEET is hydrolytically stable under acidic, basic and neutral conditions, and photolytically stable in sterile distilled water. DEET is considered to be ready biodegradable and causes only minor inhibitory effects on (STP) microbial activity.

Because the substance will primarily end up in sewage treatment plants before any major release to the environment, final environmental exposure will to a large extent depend on whether households are connected to STPs equipped with at least secondary (biological) treatment. DEET has a water solubility of 11.2 g/L (25°C) and its log Pow is 2.4 (22°C). If released into water, DEET is expected to adsorb to suspended solids and sediment based upon the estimated Koc. Based on the calculated BCFs for aquatic and terrestrial organisms, DEET is considered to have very little or no potential to bioaccumulate. DEET has a Koc of 43.3, suggesting that it is very mobile in soil and therefore could leach to the groundwater. However, DEET will not be directly emitted to soil and exposure via this route is therefore expected to be negligible.

If released to soil, DEET is expected to have moderate mobility based upon an estimated Koc of 300. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 2.1 x 10-8 atm‑cu m/mole. Based on limited data, this compound should not readily biodegrade under either aerobic or anaerobic conditions in either soil or water. If released into water, DEET is expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. BCF values of 0.8–2.4 suggest bioconcentration in aquatic organisms is low. DEET is stable to hydrolysis at environmental pHs of 5, 7, and 9 (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

DEET has been measured at 0.012 µg/L (DWI 2014).

### Recommended analytical techniques

#### Referee method

Not required.

#### Some alternative methods

The content of DEET in soil is determined by LC-MS/MS using 1 transition, with a LOQ of 0.01 mg/kg. A HPLC-UV method was provided for water, which was validated for data collection in ecotoxicological studies. It has a LOQ of 3 mg/L, which is low enough to cover the effect concentration of the most sensitive aquatic organisms (ie, 15 mg/L). The method does not comply with requirements for drinking water and no confirmatory method was provided (EU 2010).

### Health considerations

Up to 56 percent of DEET applied topically penetrates intact human skin and 17 percent is absorbed into the bloodstream. Blood concentrations of about 3 mg per litre have been reported several hours after DEET repellent was applied to skin in the prescribed fashion. DEET is also absorbed by the gut.

DEET was metabolised completely in all oral and dermal treatment groups with little or no parent compound excreted in the urine. DEET is extensively metabolised to 2 major metabolites, m-[(N,N-diethylamino)carbonyl] benzoic acid and m‑[(ethylamino)carbonyl] benzoic acid. Setting of an ADI is not considered necessary, since exposure to DEET is via direct application to skin (EU 2010).

Both male and female rats seem to be the most sensitive animals tested to-date orally, from which a NOAL of 100 mg/kg/d is possibly indicated. Some tests on dogs yielded similar results.

The USEPA classified DEET as “Group D – not classifiable as to human carcinogenicity” based on inadequate evidence of carcinogenicity, or lack of data in laboratory animals and humans.

As at October 2015 and August 2017 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for DEET of:

* 1 mg/kg/day for intermediate-duration oral exposure (15–364 days).

### Derivation of Maximum Acceptable Value

No MAV.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The short-term, chronic and subchronic health risk limits are 0.2 mg/L

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USEPA. 1998. *Reregistration Eligibility Decision (RED): DEET*. EPA738-R-98-010. 134 pp. http://www.epa.gov/oppsrrd1/REDs/0002red.pdf.

# Dimethylamine

CAS No. 124-40-3. Also called N-methylmethanamine and N,N-dimethylamine.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for dimethylamine. The WHO Guidelines do not mention dimethylamine.

NSF Standard 60 states that the allowable concentration of dimethylamine in drinking water is 0.12 mg/L.

### Sources to drinking-water

#### 1. To source waters

Dimethylamine is a gas which is very soluble in water: 350 g per 100 mL.

Dimethylamine is a precursor to several industrially significant compounds. It reacts with [carbon disulphide](http://en.wikipedia.org/wiki/Carbon_disulfide) to give dimethyl[dithiocarbamate](http://en.wikipedia.org/wiki/Dithiocarbamate), a precursor to a family of chemicals widely used in the [vulcanisation](http://en.wikipedia.org/wiki/Vulcanization) of [rubber](http://en.wikipedia.org/wiki/Rubber). The solvents [dimethylformamide](http://en.wikipedia.org/wiki/Dimethylformamide) and [dimethylacetamide](http://en.wikipedia.org/wiki/Dimethylacetamide) are derived from dimethylamine. It is raw material for the production of many [agrichemicals](http://en.wikipedia.org/wiki/Agrichemical) and [pharmaceuticals](http://en.wikipedia.org/wiki/Pharmaceutical), such as dimefox and [diphenhydramine](http://en.wikipedia.org/wiki/Diphenhydramine) respectively (although not on ERMA’s list under those names). The [surfactant](http://en.wikipedia.org/wiki/Surfactant) [lauryl dimethylamine oxide](http://en.wikipedia.org/w/index.php?title=Lauryl_dimethylamine_oxide&action=edit&redlink=1) is found in [soaps](http://en.wikipedia.org/wiki/Soap) and cleaning compounds.

Several pesticides, eg, MCPA and 2,4-D, are sold in New Zealand as the dimethylamine salt.

#### 2. From treatment processes

About 25 percent of the production of dimethylamine is used in the manufacture of water treatment chemicals. Polydiallylydimethyl ammonium chloride (polydadmac) and epichlorohydrin-dimethylamine (EPI-DMA) are established coagulants in the treatment of drinking water; both contain small amounts of dimethylamine. AWWA Standard B452-06 (revised 2014) regulates the residue of dimethylamine in EPI-DMA.

Health Canada (2000) commissioned NSF International to review contaminant occurrences from treatment chemicals. Several organic substances were listed, with dimethylamine being the most persistent offender. It is an impurity in polyelectrolytes. Because of its frequency in drinking-water, Health Canada proposed a maximum level of 0.05 mg/L, whereas NSF proposed 0.12 mg/L.

Chlorination of water containing dimethylamine and ammonium ions produces many products, one being N-nitroso-dimethylamine (NDMA). Ozonation can also produce NDMA (Andrzejewski et al 2008).

### Forms and fate in the environment

If released to soil, dimethylamine is expected to have moderate mobility based upon an average Koc value of 434.9 calculated from data of 5 soils. The pKa of dimethylamine is 10.73, indicating that this compound will exist almost entirely in the cation form in the environment; volatilisation from moist soil surfaces is not expected to be an important fate process based upon its cationic state. Dimethylamine may volatilise from dry soil surfaces based upon its vapour pressure. Dimethylamine was biodegraded 69–89 percent in three Saskatchewan soils during a seven-day incubation period. If released into water, dimethylamine is expected to adsorb to suspended solids and sediment based upon the Koc of 508 in lake sediment. Dimethylamine is expected to biodegrade in water surfaces based on a half-life of 1.6 days in Vistula River water (Warsaw, Poland) following a 0.3-day lag period. A pKa of 10.73 indicates dimethylamine will exist almost entirely in the cation form at pH values of 5 to 9 and therefore volatilisation from water surfaces is not expected to be an important fate process. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

If released into water, dimethylamine volatilises slowly, estimated half-life of 35 hours in a model river. Biodegradation is rapid, half-life about 1.5 days, and is the most important degradation process in water – direct photolysis and adsorption are not important.

### Typical concentrations in drinking-water

NSF (2010) reports levels from 0.004 to 0.12 mg/L dimethylamine in drinking water that has been treated with polyamine coagulant aids. The median concentration was 0.021 mg/L.

NSF (2010) reports levels from <0.0004 to 0.10 mg/L dimethylamine in drinking water that has been treated with polyDADMAC coagulant aids. The median concentration was 0.012 mg/L.

### Removal methods

Use product specification and dosage limits.

### Recommended analytical techniques

#### Referee method

Not required.

#### Some alternative methods

None required.

### Health considerations

Dimethylamine is important because it is a precursor of nitrosodimethylamine, a suspected human carcinogen.

The major human intake of dimethylamine would appear to be from the consumption of seafood, with squid reported to contain 1,000–2,000 ppm. It has also been detected in tobacco smoke.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Andrzejewski, et al. 2008. N-nitrosodimethylamine (NDMA) formation during ozonation of dimethylamine-containing waters. *Water Research* 42(4–5): 863–70.

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USEPA. 1990. Dimethylamine. *Integrated Risk Information System (IRIS)*. <http://www.epa.gov/iris/subst/0228.htm> or <https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=228>.

# Dimethylpyrazole

CAS No. 2820-37-3. Also called 3,4-dimethylpyrazole, 3,4-dimethyl-1H-pyrazole and 4,5-dimethylpyrazole. Usually sold/used as dimethylpyrazole phosphate or DMPP – CAS No. 202842-98-6. One product is marketed under the name of Entec.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for dimethylpyrazole, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

There are three nitrification inhibitors currently available on a commercial basis, dimethylpyrazole-phosphate (DMPP), dicyaniamide (DCD) and nitrapyrin (N-Serve). Dimethylpyrazole-phosphate requires fewer applications than DCD but the cost may be higher. DCD has higher water solubility and can be applied in liquid form and is also less volatile and can be used in conjunction with solid fertilisers, acting as a slow release fertiliser. Some research has shown that DMPP performed better in lighter soils and remained more effective after heavy rainfall than DCD, and DMPP had greater plant compatibility, and showed potential for further investigation having displayed favourable results in tests at low application rates of 0.5–1.5 kg/ha (AFBI 2005).

Dimethylpyrazole phosphate has been reported to produce no phytotoxic effects and consequent yield reduction in white clover. There are no reports of its use in New Zealand as at January 2013.

### Forms and fate in the environment

The USEPA has determined that any use of the substance resulting in surface water concentrations exceeding 19 ppb (0.019 mg/L) may cause significant adverse environmental effects.

Water solubility is described as miscible.

### Recommended analytical techniques

#### Referee method

No MAV.

### Health considerations

Dimethylpyrazole phosphate is said to have undergone thorough toxicology and ecotoxicology tests, but evidence of this is hard to find.

Ravensdown has a material safety data sheet (MSDS) for 3,4-dimethylpyrazole: see <http://www.ravensdown.co.nz/SafetyDatasheets/DNP-MSDS.pdf>.

### Derivation of Maximum Acceptable Value

No MAV.

### References

AFBI. 2005. *Opportunities to Reduce Nitrate Leaching from Grazed Grassland: A Summary of Research Findings in New Zealand*. Northern Ireland: Agri-Food and Biosciences Institute, Global Research Unit. 22 pp. <http://www.afbini.gov.uk/gru-report3-reduce-nitrate-leaching.pdf>.

USEPA. 2012. *Significant New Use Rules on Certain Chemical Substances*. 77 FR 61132, page 61132. <https://www.federalregister.gov/articles/2012/10/05/2012-23993/significant-new-use-rules-on-certain-chemical-substances> and http://www.gpo.gov/fdsys/pkg/FR-2012-10-05/pdf/2012-23993.pdf.

# 4,6-dinitro-o-cresol

Dinitrocresols are a group of organic chemicals that can contain up to 18 individual compounds. 4,6-Dinitro-o-cresol is the most important.

CAS No. 534-52-1. Also called 2-methyl-4,6-dinitrophenol, 2,4-dinitro-orthocresol, 3,5‑dinitro-2-hydroxytoluene, 4,6-dinitro-2-methyl phenol, DNC and DNOC.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 4,6-dinitro-o-cresol, and it is not mentioned in the WHO Guidelines.

4,6-Dinitro-o-cresol is on the USEPA List of Priority Pollutants. It also appears on the Rotterdam Convention (UNEP) list of chemicals in Appendix III (which effectively bans or severely restricts use of a chemical), see <http://www.pic.int/home.php?type=s&id=77>.

### Sources to drinking-water

#### 1. To source waters

2,6-Dinitro-p-cresol is used as an intermediate for synthesis of fungicides and biologically active compounds, dyes and pharmaceuticals, and as a polymerisation inhibitor for vinyl aromatic compounds.

4,6-Dinitro-o-cresol is listed in Table 1 of ERMA’s Summary of Approvals of Substances Transferred under the Hazardous Substances (Chemicals) Transfer Notice 2006 (with amendments), as at 24 June 2008 (<http://www.ermanz.govt.nz/hs/transfer/summaryapprove.html>, then select Summary of Approvals: Chemicals.

4,6-Dinitro-o-cresol has been used as an acaricide, fungicide, herbicide and insecticide overseas, usually as a salt. DNOC is a non-systemic stomach poison and contact insecticide, and is strongly phytotoxic for broad-leaved plants. It was formerly used in Australia for pre-harvest desiccation and the control of broad leaf plants; its use was revoked in 1990. Its use as a pesticide in the US was cancelled in 1991.

### Forms and fate in the environment

4,6-Dinitro-o-cresol is usually broken down within two months by organisms in the soil. It is reasonably mobile in sandy soils. Direct volatilisation from water will not be significant.

The partitioning of DNOC from water to solids present in the water transports the compound from the water phase to suspended solids and sediment. The adsorption of DNOC from water by suspended solids and sediment is pH dependent, and the adsorption increases as the pH of the solution decreases.

Water solubility about 150 mg/L.

Typical concentrations in drinking-water

### Recommended analytical techniques

#### Referee method

No MAV.

### Health considerations

#### Acute poisoning

Most of what is known about how DNOC can affect health comes from old reports from doctors who prescribed DNOC for patients who wanted to lose weight. DNOC has not been used as a diet pill for almost 60 years because of the harmful effects to those patients. The amount of DNOC that the patients took in pill form was as low as 0.35 milligram of DNOC per kilogram of body weight per day (mg/kg/day). DNOC increases the basal metabolic rate, which can increase the pulse and heart rates, and cause profuse sweating and fever. These effects can occur after breathing in, swallowing, or having skin contact with DNOC for a short period.

DNOC also may make it difficult to breathe and causes headaches, drowsiness, dizziness, and weight loss. DNOC stains the whites of the eyes and the skin yellow, and can cause mild damage to the stomach, the kidneys, and the liver. If swallowed for long periods, DNOC may cause cataracts in eyes and skin rashes.

#### Chronic exposure

Because DNOC is moderately nonpolar, it should be easily absorbed by oral, inhalation, and dermal routes. Although its distribution in human tissues is not well documented, animal data suggest that DNOC is distributed to most tissues including the lungs, heart, liver, kidney, brain, spleen, and muscle. DNOC and its metabolites are eliminated primarily via the urine in humans and animals, and elimination is slower in humans than in animals. DNOC appears to be metabolised to less toxic metabolites that are readily eliminated via the urine.

As at July 2013 and Aug 2018 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for 4,6-dinitro-o-cresol of:

* 0.004 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.004 mg/kg/day for intermediate-duration oral exposure (15–364 days).

### Derivation of Maximum Acceptable Value

No MAV.

### References

ATSDR. 2010. *Toxicological Profile for Dinitrocresols*. Atlanta, GA: Agency for Toxic Substances & Disease Registry, Department of Health and Human Services. See: http://www.atsdr.cdc.gov/toxprofiles/tp63.html.

IPCS. 2000. Dinitro-ortho-cresol; *Environmental Health Criteria* 220. INCHEM. International Programme on Chemical Safety. http://www.inchem.org/documents/ehc/ehc/ehc200.htm.

# Dinitrotoluenes

There are six possible isomers of dinitrotoluene; 2,4-dinitrotoluene is the commonest.

* 2,3-dinitrotoluene. CAS No. 602-01-7
* 2,4-dinitrotoluene. CAS No. 121-14-2
* 2,5-dinitrotoluene. CAS No. 619-15-8
* 2,6-dinitrotoluene. CAS No. 606-20-2
* 3,4-dinitrotoluene. CAS No. 610-39-9
* 3,5-dinitrotoluene. CAS No. 618-85-9.

The CAS No. for the mixture of isomers is 25321-14-6. This technical mixture contains approximately 77 percent 2,4-dinitrotoluene and 19 percent 2,6-dinitrotoluene. To a lesser extent the isomers 2,3-dinitrotoluene (1.5 percent), 2,5-dinitrotoluene (0.7 percent), 3,4-dinitrotoluene (2.4 percent) and 3,5-dinitrotoluene (0.04 percent) are contained in the technical mixture. There may also be small amounts of trinitrotoluene, cresols, mononitrobenzene, and ortho-, meta-, and para-, mononitrotoluenes.

Dinitrotoluene is also called DNT, dinitrotoluol and methyl dinitrobenzene.

Note that 4-nitrotoluene (no datasheet) is on the EC List of 66 Category 1 substances showing evidence of endocrine disrupting activity in at least one species using intact animals (EC 2015).

### Maximum Acceptable Value

The DWSNZ do not have a MAV for any dinitrotoluenes, and they are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Dinitrotoluenes (which are semi-volatile organic compounds or SVOCs) are not known to occur as natural products. Well over 95 percent of the 1.5 million tonnes produced annually is used to make [toluene diisocyanate](http://en.wikipedia.org/wiki/Toluene_diisocyanate), which is then used to produce flexible [polyurethane](http://en.wikipedia.org/wiki/Polyurethane) foams. Some is used to produce TNT, in plasticisers sometimes with dibutyl phthalate, and in the production of some dyestuffs. The EU Directive 2003/34/EC (EU 2003) bans the use of DNT in consumer products in the EU market.

Dinitrotoluenes are only expected to be found in the environment near manufacturing facilities. Concentrations of 2,4-dinitrotoluene and 2,6-dinitrotoluene in the River Elbe at the Czech border have been found at <0.001 mg/L and reduce further downstream.

### Forms and fate in the environment

2,4-Dinitrotoluene is expected to adsorb slightly to suspended solids and sediment; volatilisation from water surfaces is not expected based upon its Henry´s Law constant of 5.4 x 10-8 atm‑cu m/mole, vapour pressure and water solubility. Photolysis is probably the most significant removal process for 2,4-dinitrotoluene in water, while hydrolytic degradation is not to be expected (EU 2008).

If released to soil, 2,4-dinitrotoluene may have high to low mobility based upon an experimentally determined Koc range of 57-2000 in various soils. Leaching of 2,4‑dinitrotoluene has been observed in soil column transport studies and the compound has been detected in groundwater samples beneath ammunition plants indicating 2,4-dinitrotoluene can leach from soil. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon a measured Henry’s Law constant of 5.4 x 10-8 atm‑cu m/mole at 25°C. 2,4-Dinitrotoluene may undergo direct photolysis on soil surfaces based on observed photolysis of 2,4-dinitrotoluene in water. Biodegradation of 2,4-dinitrotoluene is expected to be an important fate process in soil and water based on results of various screening tests that demonstrated microbial degradation. If released into water, 2,4-dinitrotoluene is expected to be susceptible to adsorption to suspended solids and sediment in water based upon the Koc range of 57-2000 in various soils. Volatilisation from water surfaces is not an important fate process based on its Henry’s Law constant. An estimated BCF of 9 suggests the potential for bioconcentration in aquatic organisms is low. Photolysis of 1.0 ppm 2,4‑dinitrotoluene in distilled and natural waters gave half-lifes of 43 hours in distilled water and 2.7, 9.6, and 3.7 hours in river, bay and pond waters, respectively. Hydrolysis is not expected to occur due to the lack of hydrolysable functional groups (EAWAG accessed February 2015).

In surface waters, from photodegradation measurements a half-life of 1 day was derived for (predominantly) direct photolysis under the radiative conditions of latitude 40°N. In surface waters, with regard to the geographical conditions in Germany and the low light intensity in natural water bodies, the half-life of 2,4-dinitrotoluene for direct photolysis is calculated to be 20 days in a natural water body (surface layer: 6.5 days). Under environmental aquatic conditions where no adaptation of the microorganisms can be assumed, no biodegradation of DNT is expected. In organic soil a DT50 for 2,4‑dinitrotoluene of 7 days and a DT90 of 191 days was determined. A test on leaching from three different type of soils is available for 2,4-dinitrotoluene. After two days of leaching no 2,4-dinitrotoluene was found in the leachates (INCHEM 2004).

2,6-Dinitrotoluene has a low soil Koc, 1.28 to 1.86, so should be mobile, ie, adsorption to sediments is not likely. Nor is it likely to volatilise. The soil half-life of 2,4‑dinitrotoluene is 25 days at 22°C, and 20 days for 2,6-dinitrotoluene (USEPA 2008a).

2,4-Dinitrotoluene and 2,6-dinitrotoluene have a low affinity for organic particulate matter (Koc 1.65 and Koc 1.96, respectively) (USEPA 2008). Both biodegrade in water, in aerobic and anaerobic conditions.

Water solubilities:

* 2,4-dinitrotoluene: 300 mg/L, 22°C (USEPA 2008a); 270 mg/L (IARC 1996); 166 mg/L, 25°C (INCHEM 2004)
* 2,5-dinitrotoluene: 258 mg/L, 25°C (INCHEM 2004)
* 2,6-dinitrotoluene: 180 mg/L, 20°C (IARC1996); 145 mg/L, 25°C (INCHEM 2004).

The vapour pressure for 2,4-dinitrotoluene is 0.0051 mm Hg at 20°C, and the log octanol/water partition coefficient (log Kow) is 2.0 (USEPA 2000). For the isomers mixture a log Kow of 2.00 was calculated (INCHEM 2004).

### Typical concentrations in drinking-water

One water utility in the US reported detecting 2,6-dinitrotoluene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the concentration being 0.0045 µg/L. Some quite high concentrations have been reported in groundwater near ammunition dumps.

### Removal methods

Treatment processes that remove particulate matter may help reduce the concentration of dinitrotoluenes in river water. Neither ozone nor chlorine are very effective, nor more than 17 percent removal. Activated carbon should be the most effective process.

### Health considerations

Technical grade DNT is moderately toxic following oral administration to rats, with LD50 values of 268 to 660 mg/kg bw reported. Chronic feeding of technical grade DNT to rats led to haematological changes (especially methemoglobinemia), and toxicity to liver, kidney, adrenal glands and testes in rats. At the lowest administered dose of 3.5 mg/kg bw/day signs of hepatotoxicity became obvious. No NOAEL can be derived for repeated dose toxicity; INCHEM. 2004. Most human exposure is via inhalation and dermal contact in the workplace.

The Reference Dose ([RfD](http://www.epa.gov/ttnatw01/hlthef/hapglossaryrev.html#rfd)) for 2,4-dinitrotoluene is 0.002 milligrams per kilogram body weight per day (mg/kg/d) based on effects on the central nervous system, blood, and liver in dogs (USEPA 2000 and 2008).

An RfD of 0.001 mg/kg/day was established for 2,6-DNT, based on neurotoxicity, Heinz body formation, biliary tract hyperplasia, liver and kidney histopathology, and death in beagle dogs that were fed gelatin capsules containing 2,6-DNT daily for up to 13 weeks (USEPA 2008).

A daily dose of 35 mg/kg bw DNT led to testicular degeneration and hypospermatogenesis after 52 weeks of exposure. The NOAEL for changes on reproductive organs was 3.5 mg/kg bw/day (INCHEM 2004).

Technical grade DNT is mutagenic in bacterial test systems in the presence and absence of metabolic activation, but it shows no mutagenic or genotoxic activity in mammalian cells *in vitro*. Technical grade DNT shows no mutagenic activity in the mouse bone marrow micronucleus assay and in mouse dominant lethal and spot tests. However, a distinct activity of DNT to induce DNA repair in the liver of rats is reported. Additionally, DNA binding properties in various rat organs, mainly rat liver were demonstrated for 2,4-dinitrotoluene and 2,6-dinitrotoluene isomers. Gut flora may play an important role in activation of DNT to reactive metabolites. Overall, technical grade DNT shows the potential to induce genotoxic changes *in vivo* (INCHEM 2004).

The USEPA has not classified 2,4-dinitrotoluene for potential carcinogenicity. However, they classified the 2,4-/2,6-dinitrotoluene mixture as a Group B2, probable human carcinogen with an oral cancer slope factor of 0.68 (mg/kg/d) (USEPA 2000, 2008). This determination was based on significant increases in hepatocellular carcinoma and mammary gland tumours in female rats fed a DNT mixture (98 percent 2,4-DNT with 2 percent 2,6-DNT) in the diet in a two-year study. Concentrations of 5 μg/L, 0.5 μg/L, and 0.05 μg/L are associated with carcinogenic risks of 10-4, 10-5, and 10-6 respectively. The health reference level (HRL) for both 2,4- and 2,6-DNT is 0.05 μg/L (USEPA 2008).

The NOAEL for male and female beagle dogs was 0.2 mg/kg b.w./day of 2,4‑dinitrotoluene on the basis of neurotoxicity (incoordination and paralysis) derived from the 24-month study. According to the NOAEL/LOAEL values derived from chronic toxicity studies, both dogs and rats appear to be sensitive species to 2,4-dinitrotoluene toxicity. Nevertheless, the rat study with exposure duration up to 24 months is considered more appropriate for the risk characterisation of repeated dose toxicity than the 24-month dog study. Thus the critical effects for risk characterisation derived from the 24-month rat study were presence of hyperplastic foci in the liver and atrophy of seminiferous tubules. Therefore, the relevant LOAEL for risk characterisation is considered to be 0.57 mg/kg b.w./day derived from the 24-month rat study (EU 2008).

IARC (1996) classified 2,4-dinitrotoluene and 2,6-dinitrotoluene as possibly carcinogenic to humans (Group 2B). 3,5-Dinitrotoluene is not classifiable as to its carcinogenicity to humans (Group 3).

USEPA (2008a) reports that health advisories (HAs) were determined for 1-day, 10-day, and longer term (up to seven years) exposures:

The one-day HA for 2,4-DNT is 0.5 mg/L, and the 10-day HA is 1.0 mg/L. The longer term HA for 2,4-DNT for the 10‑kg child is 0.3 mg/L; for the 70‑kg adult, it is 1.0 mg/L. The drinking water equivalent level (DWEL) is 0.1 mg/L.

The one-day HA for 2,6-DNT is 0.4 mg/L, and the 10-day HA is 0.4 mg/L. The longer term 2,6-DNT HA for the 10‑kg child is 0.4 mg/L; for the 70‑kg adult, it is 1.0 mg/L. The DWEL is 0.04 mg/L.

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for 2,4-dinitrotoluene of:

* 0.05 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.007 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.001 mg/kg/day for chronic-duration oral exposure (>364 days).

The corresponding values for 2,6-dinitrotoluene are:

* 0.09 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.004 mg/kg/day for intermediate-duration oral exposure (15–364 days).

In 2016 they added:

* for 2,3-dinitrotoluene: 0.09 mg/kg/day for acute-duration oral exposure (1–14 days)
* for 2,5-dinitrotoluene: 0.007 mg/kg/day for acute-duration oral exposure (1–14 days)
* for 3,4-dinitrotoluene: 0.03 mg/kg/day for acute-duration oral exposure (1–14 days)
* for 3,5-dinitrotoluene: 0.03 mg/kg/day for acute-duration oral exposure (1–14 days).

2,6-Dinitrotoluene is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

### Derivation of Maximum Acceptable Value

No MAV.

### References

ATSDR. 2013. *Draft Toxicological Profile for Dinitrotoluenes*. Atlanta, GA: Agency for Toxic Substances & Disease Registry, Department of Health and Human Services. 305 pp. See: <http://www.atsdr.cdc.gov/toxprofiles/index.asp>.

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# 1,4-dioxane

CAS No. 123-91-1. Also called dioxane, paradioxane, p-dioxane, diethylene dioxide, diethylene-1,4-dioxide, 1,4-dioxacyclohexane, glycol ethylene ether, diethylene ether and dioxyethylene ether. Not to be confused with dioxins which are an entirely different group of chemicals.

### Maximum Acceptable Value

Based on health considerations, the concentration of 1,4-dioxane in drinking-water should not exceed 0.05 mg/L.

The USEPA concluded on 22 September 2009 that 1,4-dioxane is known or anticipated to occur in PWSs and may require regulation. Therefore they added 1,4-dioxane to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a).

### Sources to drinking-water

#### 1. To source waters

1,4-Dioxane is used mainly as a stabiliser in chlorinated solvents but this use is falling as alternatives to chlorinated solvents are being developed. It is also used as a solvent for cellulose acetate, ethyl cellulose, benzyl cellulose, resins, oils, waxes, oil and spirit-soluble dyes, as well as for electrical, agricultural and biochemical intermediates, and for production of adhesives, sealants, pesticides, magnetic tapes, detergents, personal care products and cosmetics, pharmaceuticals, rubber chemicals and surface coatings.

The commercial product may be stabilised by addition of a small amount of 2,6-tert-butyl-p-cresol.

1,4-Dioxane is a trace contaminant of some chemicals used in cosmetics, detergents, and shampoos, but this is more carefully controlled today.

In Japan, 1,4-dioxane is used as a solvent and surface-treating agent for artificial leather, and was formerly used as a stabiliser for trichloroethylene. The concentration of 1,4-dioxane in surface water over 7 years in Japan ranged from not detectable to 0.04 mg/L. One groundwater sample of 25 contained 0.08 mg/L. There was a high correlation between the concentrations of 1,4-dioxane and 1,1,1-trichloroethane.

### Forms and fate in the environment

It is difficult to remove 1,4-dioxane from water or to decompose it in water because of its very high water solubility, low to moderate volatility from water, and low rates of photolysis. Its high water solubility (described as being miscible with water), and high mobility in soil, means it can readily find its way into groundwater. 1,4-Dioxane may be more persistent in groundwater where volatilisation is hindered.

EU (2002) quotes: vapour pressure = 40hPa at 20°C; partition coefficient = logPow = about -0.3; Henry’s law constant = 4.3 Pa.m3/mol at 20ºC; 1,4-dioxane does not contain any hydrolysable groups and ethers are generally classified as resistant to hydrolysis; from standardised OECD and non-standardised tests it can be concluded that 1,4‑dioxane is not biodegradable.

1,4-Dioxane may volatilise from dry soil surfaces based on its vapour pressure. 1,4‑Dioxane biodegrades very slowly in water and soils and is considered recalcitrant. 1,4‑Dioxane is not expected to undergo hydrolysis or to biodegrade readily in the environment. Therefore, volatilisation is expected to be the dominant removal process for moist soil and surface water. Based on a Henry’s Law constant of 4.8×10-6 atm-m3/mole, the half-life for volatilisation of 1,4-dioxane from a model river is 5 days and that from a model lake is 56 days (USEPA 2010).

If released to soil, 1,4-dioxane is expected to have very high mobility based upon a Koc of 29. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 4.8 x 10-6 atm‑cu m/mole. 1,4-Dioxane may also volatilise from dry soils based upon it vapour pressure. 1,4-Dioxane is very slow to biodegrade and is considered recalcitrant in the environment. If released into water, 1,4-dioxane is not expected to adsorb to suspended solids and sediment based upon the Koc value. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 5 and 56 days, respectively. BCF values in the range of 0.2-0.7 measured in fish, suggests bioconcentration in aquatic organisms is low. Hydrolysis and photolysis in sunlit surface waters are not expected to be important environmental fate processes for 1,4-dioxane since it lacks functional groups that hydrolyse or absorb light under environmentally relevant conditions (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

1,4-Dioxane was found at a concentration of 0.0002 to 0.0015 mg/L in tap water samples from six cities in Kanagawa, Japan, during 1995–1996. Twenty water utilities in the US reported detecting 1,4-dioxane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.008 mg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,810 drinking water samples for 1,4-dioxane between 2013 and 2015, and found 4,197 samples exceeded the minimum reporting level (MRL) of 0.07 µg/L, and 341 water supplies contained >0.35 µg/L.

### Removal methods

1,4-Dioxane is not removed to any appreciable extent by air stripping, coagulation or oxidation by chlorine or potassium permanganate; granular activated carbon can achieve about 50 percent removal. 1,4-Dioxane was effectively removed by biological activated carbon treatment. 1,4-Dioxane is not effectively oxidised by ozone alone, but the rate can be increased by addition of hydrogen peroxide. UV/hydrogen peroxide treatment has been used too.

### Recommended analytical techniques

#### Referee method

None suggested.

#### Some alternative methods

None suggested. Solvent extraction and GC-MS analysis can give a quantification limit of 0.003 mg/L with good specificity. However, this technique requires a large volume of sample, about one litre, for analysis (WHO 2007).

USEPA Method 1624 can be used to determine 1,4-dioxane in water and in municipal and industrial discharges by isotopic dilution GC-MS. In this method, isotopically labelled 1,4-dioxane-d8 is added to the sample as an isotope dilution standard. The samples are then introduced into the GC using a purge-and-trap methodology. 1,4‑Dioxane is separated by GC and detected by MS. The labelled compounds serve to correct for the variability of the analytical technique. The detection limit for this method is 0.01 mg/L (USEPA 2001).

### Health considerations

The public health risk assessmentconcluded that the main potential source of exposure to the general public is from exposure to consumer products containing 1,4‑dioxane as an impurity (NICNAS 1998). 1,4-Dioxane is well-absorbed via the oral and inhalation routes. In rats, more than 95 percent is taken up from the gastrointestinal tract following administration of up to 1,000 mg/kg of body weight. Uptake (on a mg/kg of body weight basis) is approximately five to eight times greater in rats than in humans. Metabolism in rats and humans appears to be similar, with the vast majority of the dose being rapidly excreted in urine as β-hydroxyethoxyacetic acid (HEAA) and small amounts of unchanged 1,4-dioxane being eliminated in urine and expired air.

#### Acute poisoning

Oral LD50 values are in the range of 5,400 to 7,300 mg/kg of body weight in rats, 5,900 mg/kg of body weight in mice, 3,300 to 4,000 mg/kg of body weight in guinea-pigs and 2000 mg/kg of body weight in rabbits. The main acute effects at near-lethal doses in experimental animals (rats, mice, guinea-pigs, rabbits or dogs) are central nervous system depression (eg, narcosis) and severe gastric, pulmonary, hepatic and renal lesions.

#### Chronic exposure

Sherman rats of both sexes received 100, 1,000 or 10,000 mg of 1,4-dioxane per litre in their drinking-water for 716 days. The 10,000 mg/L group exhibited decreased body weight gain, survival rate and water consumption. Other histopathological data for animals receiving 1,000 or 10,000 mg/L pointed to renal tubular epithelial and hepatocellular degeneration and necrosis. The NOAEL was 100 mg/L.

1,4-Dioxane caused hepatic and nasal cavity tumours in rodents in most long-term oral studies conducted. Tumours in the peritoneum, skin and mammary gland were also observed in rats given a high dose. Lung tumours were specifically detected after intraperitoneal injection.

A RfD of 0.03 mg/kg-day was derived in USEPA (2010/2011) based on liver and kidney toxicity in rats exposed to 1,4-dioxane in the drinking-water for two years. The RfD was derived by dividing the NOAEL of 9.6 mg/kg-day by a composite UF of 300; the composite UF includes factors of 10 for animal-to-human extrapolation and for inter-individual variability, and an UF of 3 for database deficiencies. USEPA (2011) reports a DWEL of 1 mg/L.

IARC classified 1,4-dioxane in 1999 as Group 2B (possibly carcinogenic to humans). This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. USEPA (2010) states that 1,4-dioxane is “likely to be carcinogenic to humans” based on evidence of liver carcinogenicity in several 2-year bioassays conducted in three strains of rats, two strains of mice, and in guinea pigs. Studies in humans found no conclusive evidence for a causal link between occupational exposure to 1,4-dioxane and increased risk for cancer; however, only two studies were available and these were limited by small cohort size and a small number of reported cancer cases.

The USEPA (2009) has a Health Advisory for p-dioxane of 0.3 mg/L for a 10-4 cancer risk, and has classed it in Group 2B: probable human carcinogen with sufficient evidence in animals but inadequate or no evidence in humans; this was changed to 0.035 mg/L (USEPA 2011).

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for 1,4-dioxane of:

* 5 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.5 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.1 mg/kg/day for chronic-duration oral exposure (>364 days).

### Derivation of Maximum Acceptable Value

Although only a possible weak genotoxic potential has been suggested for 1,4‑dioxane, the compound clearly induces multiple tumours in various organs. Based on calculations using the linearised multistage model for estimating cancer risk for the most sensitive sites found in rats exposed to 1,4-dioxane in drinking-water (nasal carcinomas and hepatic tumours), drinking-water concentrations of 0.088 and 0.054 mg/L respectively, were found to be associated with an upper-bound excess lifetime cancer risk of 10-5 without body surface correction.

On the other hand, if it is considered that 1,4-dioxane is not genotoxic in humans at low doses, the TDI approach can be used for derivation of the guideline value. For a non-cancer end-point (including renal tubular epithelial and hepatocellular degeneration and necrosis), a TDI of 96 μg/kg of body weight per day can be calculated by applying an uncertainty factor of 100 (for inter- and intra-species variation) to a NOAEL of 9.6 mg/kg of body weight per day from a long-term drinking-water study in rats. For a cancer end-point (hepatocellular tumours), a TDI of 16 μg/kg of body weight per day can be calculated by applying an uncertainty factor of 1,000 (100 for inter- and intraspecies variation, 10 for non-genotoxic carcinogenicity) to the NOAEL of 16 mg/kg of body weight per day from a long-term drinking-water study in rats; WHO. 2017. The equivalent concentration in drinking-water is calculated to be 0.048 mg/L based on 10 percent allocation of the lower TDI from the cancer end-point.

As similar values of 0.054 and 0.048 mg/L were derived with two different approaches, a rounded figure of 0.05 mg/L is considered to be the appropriate guideline value for 1,4-dioxane. Based on the approach used above, it is probably not appropriate to adjust this value for a 70 kg body weight.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The subchronic limit is 0.3 mg/L, the chronic limit is 0.1 mg/L, and subchronic health risk limits are 0.007 mg/L, and a limit of 0.001 mg/L was set for cancer.

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# Dioxins

CAS No. for 2,3,7,8-tetrachlorodibenzo-1,4-dioxin (TCDD) is 1746-01-6. Also called 2,3,7,8-tetrachlorodibenzo-para-dioxin or tetradioxin.

Dioxin is a fairly loose description covering the polychlorinated dibenzo-*p*-dioxins, and for some people, dioxins include the polychlorinated dibenzofurans as well, eg, 2,3,4,7,8-pentachlorodibenzofuran, CAS No. 57117-31-4 – also called 2,3,4,7,8-PeCDF.

### Maximum Acceptable Value

No MAV has been derived because of the large number of compounds of differing toxicities in this group.

The WHO Guidelines do not mention dioxins.

The maximum contaminant level (MCL) for the dioxin 2,3,7,8-tetrachlorodibenzo-1,4-dioxin (TCDD) (USEPA 2006/2009/2011) is 0.00000003 mg/L (3 x 10-8 mg/L).

The dioxin 2,3,7,8-tetrachlorodibenzo-1,4-dioxin (TCDD) is a ‘priority pollutant’ under the US Clean Water Act.

Polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF)appeared in the original list of 12 Persistent Organic Pollutants (POPs) under the Stockholm Convention; see <http://chm.pops.int/>.

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are two chemically similar groups of chlorinated aromatic compounds. PCDDs can have up to eight chlorine substituents leading to 75 possible congeners; the PCDF group comprises 135 possible congeners.

Chlorinated dibenzo-p-dioxins (CDDs) are commonly referred to as polychlorinated dioxins. These compounds have varying harmful effects. The CDD family is divided into eight groups of chemicals based on the number of chlorine atoms in the compound. The group with one chlorine atom is called the mono-chlorinated dioxin(s). The groups with two through eight chlorine atoms are called di-chlorinated dioxin (DCDD), tri-chlorinated dioxin (TrCDD), tetra-chlorinated dioxin (TCDD), penta-chlorinated dioxin (PeCDD), hexa-chlorinated dioxin (HxCDD), hepta-chlorinated dioxin (HpCDD), and octa-chlorinated dioxin (OCDD). The chlorine atoms can be attached to the dioxin molecule at any one of eight positions. The name of each CDD indicates both the number and the positions of the chlorine atoms. For example, the CDD with four chlorine atoms at positions 2, 3, 7, and 8 on the dioxin molecule is called 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8‑TCDD. 2,3,7,8-TCDD is one of the most toxic of the CDDs to mammals and has received the most attention. Thus, 2,3,7,8-TCDD serves as a prototype for the CDDs. CDDs with toxic properties similar to 2,3,7,8-TCDD are called “dioxin-like” compounds.

Of the 135 PDCF compounds, those that contain chlorine atoms at the 2,3,7,8-positions of the parent dibenzofuran (CAS No. 132-64-9) molecule are especially harmful.

Dioxins and dioxin-like polychlorinated biphenyls (PCBs) are listed as a “priority contaminant” in the Ministry for the Environment’s *Toxicological Intake Values for Priority Contaminants in Soil* (MfE 2011a).

### Sources to drinking-water

#### 1. To source waters

Dioxins can be produced by chlorination reactions with organic matter. As a result they are present as contaminants in chlorophenoxy herbicides (WHO 1996), and the timber preservative pentachlorophenol (Severn 1980), and in the effluent from wood pulp operations when chlorine is used as a bleach. There are also releases of dioxins and dibenzofurans into the environment from incineration of municipal refuse, exhaust from vehicles fuelled with diesel or leaded petrol, fossil fuel combustion, disposal of industrial wastes, chlorophenol wood treatment, and accidental fires involving transformers containing PCBs. They are produced naturally from the incomplete combustion of organic material by forest fires or volcanic activity. PCP solutions used in New Zealand contained high levels of hexa-, hepta-, and octa-chlorinated dioxins. These dioxins are toxic, but considered less so than the highly toxic tetra-chlorinated dioxin that has caused health concerns in Paritutu, New Plymouth where 2,4,5-T was formulated. Traces of PCDFs are believed to form in the chloralkali process for chlorine production.

The total amount of dioxin remaining in soil collectively from the 255 sawmill sites in New Zealand was estimated at November 2002 to be:

* 80−250 g dioxin toxic equivalents from NaPCP use
* 172 g dioxin toxic equivalents from PCP in oil use.

On a site-by-site basis, risks to human health and the environment from soil contaminated by PCP and dioxin are associated predominantly with the sites (approximately 35) that were relatively large users of PCP (MfE 2008).

The main sources of dioxins to water in New Zealand now are wastewater treatment and landfills (MfE 2000 and 2011); overall, emissions have reduced significantly between 1998 to 2008.

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; TCDD levels range from 0.00012 to 0.0014 mg/kg TEQ (120 to 1400 ng/kg) depending on land use, and 0.00009 to 0.0012 mg/kg TEQ for dioxin-like PCBs. For dioxins and dioxin-like PCBs the total toxicity is assessed as a toxic equivalency (TEQ) to 2,3,7,8-TCDD using toxic equivalency factors (TEF). The TEQ is defined as the sum of the products of the concentration of each compound multiplied by the value of its TEF.

USEPA (2007) reported a study of soils in the US:

Total CDDs averaged 1,585 pg/g (1582 ng/kg). Total CDFs averaged 47 pg/g. Levels of the TCDD homologues were the lowest, with an average concentration of 0.2 pg/g. Levels of the OCDD homologue were the highest, with an average concentration of 1,482 pg/g. The range of concentrations found was similar to the range across five published studies on CDD/CDF levels in soils from rural areas of North America.

Müeller et al (2004) reported results of analysis of 29 dioxin-like chemicals in aquatic sediment cores from 62 sampling locations in Australia. Dioxin-like chemicals were found in all aquatic sediments analysed, with middle bound concentrations ranging from 0.002 to 520 pg TEQ/g dry weight. Considering all sediment samples from freshwater locations, the median concentration was 0.2 pg TEQ/g dry weight. Homologue and congener profiles for the PCDD/PCDF were strongly dominated by OCDD with the 1,2,3,4,6,7,8-heptachloro dibenzodioxin usually the congener with the second highest concentration. For most sediment samples, PCDD/PCDF dominated the mixture of dioxin-like chemicals present, accounting for more than 80 percent of the total TEQ.

### Forms and fate in the environment

The term dioxins covers a large number of structurally related compounds, but within this group one of the most toxic and environmentally stable compounds is 2,3,7,8‑tetrachlorodibenzo-1,4-dioxin (TCDD), often termed simply dioxin. TCDD is the most studied member of the dioxin family, and much of the information about the environmental fate of the dioxins is based on what is known about it.

Dioxins have a very low solubility in water (a fraction of a mg/L – TCDD: 0.0000013 mg/L), and consequently, if present in water, a large percentage of it is associated with sediments and suspended matter (IPCS 1989). Decomposition by sunlight can occur, but the rate of decomposition will decrease with water depth, and may contribute little to loss of TCDD attached to bottom sediments (USEPA b). The rate of decomposition decreases as the chlorine content of the dioxin increases, and is also influenced by the pattern of chlorine substitution within the compound and is also found to be faster in dioxins containing less chlorine substitution (IPCS 1989). Some loss may take place by volatilisation, but this too is expected to be hindered markedly by adsorption to sediment (USEPA b).

TCDD is generally very resistant to biodegradation (IPCS 1989) – half-life in surface soil ranging from 1 to 25 years.

In summary, sediments and soils are the main sinks for dioxins in the environment.

### Typical concentrations in drinking-water

The data on dioxin concentrations in drinking waters are very limited. Exposure to these compounds in the general population probably occurs mainly through the food chain (IPCS 1989).

A set of three samples was taken from the Hamilton supply because it draws its water from the Waikato River downstream of timber processing operations. The raw water sample showed detectable concentrations (of the order of pg/L). Lower concentrations were found in the two samples from the distribution system. Once toxicities were taken into account, the toxicities of the dioxins in these samples were determined to be not greatly different from purified laboratory water (Nokes 1992).

The levels of CDFs in most drinking-waters are below the level that can be measured. CDFs were found in drinking-water of one of the 20 water supplies in New York State at a concentration of 3.4 parts of CDF in a quadrillion parts of water. Four water utilities in the US reported detecting 2,3,7,8-TCDD (dioxin) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 5 x 10-9 mg/L.

One water utility in the US reported detecting dibenzofuran in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00005 mg/L. (USEPA (1990) classified dibenzofuran as D: not classifiable as to human carcinogenicity.)

### Removal methods

The USEPA has approved granular activated carbon as a means of removing dioxins from drinking waters (USEPA a). While no information about the effectiveness of the combination of chemical coagulation, flocculation, sedimentation and filtration has been found, the tendency for dioxins to adsorb to particulate matter indicates that these processes should be able to lower dioxin concentrations.

Dioxin reduction can be achieved by the use of UV irradiation and ozone together in the laboratory (Vollmuth and Niessner 1997). Concentrations start to decrease rapidly after the first two minutes of treatment.

### Recommended analytical techniques

#### Referee method

N/A.

#### Some alternative methods

N/A.

### Health considerations

#### Acute poisoning

The USEPA found that acute exposures at concentrations greater than the US maximum contaminant level (MCL) of 3 x 10-8 mg/L can cause liver damage, weight loss, atrophy of the thymus gland and immunosuppression. Acute effects also include persistent chloracne (IPCS 1989).

For short-term exposure, the USEPA considers a “safe” level for a 10 kg child consuming one litre of water per day to be 1 x 10-6 mg/L for a one-day exposure, and 1 x 10-7 mg/L for a 10-day exposure.

#### Chronic exposure

Excluding occupational or accidental exposures, most background human exposure to PCDDs and PCDFs occurs as a result of eating meat, milk, eggs, fish and related products, as PCDDs and PCDFs are persistent in the environment and accumulate in animal fat. Mean background levels of 2,3,7,8-tetrachlorodibenzo-para-dioxin (2,3,7,8‑TCDD) in human tissues today are in the range of 2 to 3 ng/kg fat (IARC 1997). Available data suggest that these levels have decreased by a factor of 3 to 5 since the late 1970s, when the development of gas chromatography/mass spectrometry methodology first permitted these extremely low levels of PCDDs in tissues and the environment to be measured accurately.

Based on limited data, the sum of the mean background levels of the penta- and hexachlorinated PCDF congeners commonly found in human tissues is generally in the range of 10 to 100 ng/kg fat, and the PCDF contribution to tissue international toxic equivalent (I-TEQ) values is typically of the same order of magnitude as that of the PCDDs. Between the mid-1980s and mid 1990s, mean tissue levels of total PCDFs and PCDDs (measured as I-TEQ) in the general population have decreased by two- to three-fold. Five-fold higher tissue levels have been found in subpopulations consuming large amounts of PCDF-contaminated fish. Accidental exposures to PCDFs have led to tissue levels one or more orders of magnitude higher than background levels.

Dioxin has the potential to cause a number of reproductive health effects through long-term exposure, ranging from reduced fertility to birth defects (ie, endocrine disruption). There is also some evidence for dioxins being capable of causing cancer at concentrations more than the USEPA’s MCL.

Once in the body dioxins accumulate in fat and persist for many years. The highest amounts are found in the liver and adipose tissue. In the blood dioxins bind to lipids and lipoproteins and serum TCDD levels are highly correlated with adipose tissue TCDD levels when both are expressed on a lipid weight basis. Dioxins are eliminated mainly in faeces with only small amounts eliminated in urine. Some is eliminated in breast milk. The half-life of TCDD in humans is uncertain but an average of 7–11 years is generally accepted. Levels for the New Zealand general population are at the low end of the range of levels reported internationally, and reducing (MoH 2004, updated 2014).

The IARC (1997) considered 2,3,7,8-tetrachlorodibenzo-para-dioxin to be carcinogenic to humans (Group 1). In making the overall evaluation, the Working Group took into consideration the following supporting evidence:

i) 2,3,7,8-TCDD is a multi-site carcinogen in experimental animals that has been shown by several lines of evidence to act through a mechanism involving the Ah receptor

ii) this receptor is highly conserved in an evolutionary sense and functions the same way in humans as in experimental animals

iii) tissue concentrations are similar both in heavily exposed human populations in which an increased overall cancer risk was observed and in rats exposed to carcinogenic dosage regimens in bioassays.

IARC (2012) considered both 2,3,7,8-tetrachlorodibenzo-para-dioxin and 2,3,4,7,8‑pentachlorodibenzofuran to be carcinogenic to humans (Group 1).

Other polychlorinated dibenzo-para-dioxins are not classifiable as to their carcinogenicity to humans (Group 3). Dibenzo-para-dioxin is not classifiable as to its carcinogenicity to humans (Group 3). The International Agency for Research into Cancer (IARC 1997) concluded that polychlorinated dibenzofurans are not classifiable as to their carcinogenicity to humans (Group 3).

USEPA (1987) classified hexachlorodibenzo-p-dioxins (HxCDD) as Class B2: probable human carcinogen. USEPA (1990) classified brominated dibenzofurans Class D: not classifiable as to human carcinogenicity.

The USEPA (2009/2011) quotes a health advisory of 0.00000002 mg/L (2 x 10-8) for 2,3,7,8-TCDD, representing a 10-4 cancer risk.

TCDD, PCDDs and PCDFs appear on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

The NOEL and LOEL for PCDD/Fs are between 0.001–14 mg/kg body weight and between 0.001–43 mg/kg body weight, respectively. However, these values are strongly dependent on the used compound, the test species (eg, mouse or rat), the test system, and the route of application. In vitro tests showed similar results, and in all investigations performed, TCDD had the highest antiestrogenic potency (IUPAC 2003).

MfE (2011a) states: Dioxins and dioxin-like polychlorinated-biphenyls (PCBs) are considered to be threshold contaminants, with developmental effects on the reproductive system in male offspring of exposed pregnant females considered the most sensitive toxicity endpoint. These effects are also considered to be protective against carcinogenic effects of dioxins. The maximum monthly intake value of 30 pg TEQ/kg determined by the Ministry of Health is recommended, for consistency between New Zealand agencies. Further it is recommended that toxic equivalency factors (TEFs) developed by WHO[[1]](#footnote-1) for individual dioxins and dioxin-like PCBs are used to calculate toxic equivalent doses (TEQs), as these are based on the latest re-evaluation by WHO, and thus are likely to become the international standard. Inhalation exposure to dioxins and dioxin-like PCBs is likely to be negligible on contaminated sites, due to their low volatility. Dermal absorption of these compounds is dependent on the physicochemical properties of the individual congeners. It is recommended that dermal factors of 0.02, 0.05 and 0.07 are used as conservative estimates of dermal absorption of PCDDs, PCDFs and dioxin-like PCBs, respectively. Dietary intake is the primary source of background exposure to dioxins and dioxin-like PCBs and was estimated to be 0.33 pg/kg bw/day or 10.0 pg I-TEQ/kg bw/month for an adult, and is extended to children.

The following substances are on the EC List of 66 Category 1 substances (EC 2015) showing evidence of endocrine disrupting activity in at least one species using intact animals: 1,2,3,7,8 pentachlorodibenzodioxin, 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) and 2,3,4,7,8 pentachlorodibenzofuran.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for 2,3,7,8-tetrachlorodibenzo-p-dioxin of:

* 0.0000002 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.00000002 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.000000001 mg/kg/day for chronic-duration oral exposure (>364 days).

As at July 2013 ATSDR quotes a minimal risk level (MRL) for 2,3,4,7,8-pentachloro-dibenzofuran (CAS No. 57117-31-4) of:

* 0.000001 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.00000003 mg/kg/day for intermediate-duration oral exposure (15–364 days).

The reference dose or RfD (USEPA 2006/2009/2011) for the dioxin 2,3,7,8‑tetrachlorodibenzo-1,4-dioxin (TCDD) is 0.000000001 mg/kg/d (1 x 10-9). The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.00000004 mg/L (4 x 10-8).

EFSA (2015) reviewed the guidance values established by several public health organisations.

Dioxins have been measured 10-yearly in New Zealand breast milk. The 2008 survey found that the mean dioxin and furan levels had declined by 40 percent over the 10‑year period between 1998 and 2008 to a mean toxic equivalence (TEQ) of 3.54 pg/g lipid (Massey University 2010).

2,3,7,8-Tetrachlorodibenzo-p-dioxin is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

### Derivation of Maximum Acceptable Value

There is inadequate data to enable derivation of a MAV or MAVs.

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# Diphenylamine

CAS No. 122-39-4. Also called *N*-phenylbenzenamine (CAS name), N,N-diphenylamine, anilinobenzene, DPA and even DFA.

### Maximum Acceptable Value

There is no MAV in the DWSNZ, and the WHO Guidelines do not refer to diphenylamine.

Diphenylnitrosamine (an impurity of technical grade diphenylamine), is classified as a probable human carcinogen. The carcinogen 4-aminodiphenyl may be present as an impurity in the commercial product.

### Sources to drinking-water

#### 1. To source waters

Diphenylamine has a fairly wide range of industrial uses, often as an anti-oxidant in the production of other substances, eg rubber. With [sulfur](http://en.wikipedia.org/wiki/Sulfur), it yields [phenothiazine](http://en.wikipedia.org/wiki/Phenothiazine), a precursor to certain pharmaceuticals and one-time pesticide.

It has also been used overseas as a fungicide, eg, long-term exposure of apples to low temperatures in controlled-atmosphere storage commonly induces a physiological disorder known as scald. Diphenylamine is registered in a number of countries for post-harvest application to apples for reducing scald during storage.

Diphenylamine is registered for post-harvest use on pears in Australia as a dip at a concentration of 0.037–0.26 kg ai/hl and a minimum contact time of 10–30 seconds. The New Zealand Food Safety Authority states that as at May 2014, diphenylamine is not registered in New Zealand. By 2011 its use in the EU was banned. Diphenylamine seems to have been used in the 1970s; Pipfruit New Zealand stated that none of the main producers used diphenylamine in the 2012/13 summer crop. Imports of diphenylamine have fallen since 2005 (<http://www.indexmundi.com/trade/imports/?country=nz&commodity=292144>).

Diphenylamine is used in lubricant oils in a concentration of 1 percent. Diphenylamine antioxidants control oxidation, keeping engines running longer and more smoothly by preventing oil thickening. The antioxidant properties increase the life of engine oil by reducing breakdown that can result from the formation of sludge. Their antioxidant properties also allow them to be used in other functional fluids.

### Forms and fate in the environment

When diphenylamine was incubated in a loam soil at a nominal rate of 10 mg/kg under aerobic conditions at 25°C in the dark for 12 months, diphenylamine initially disappeared rapidly, but after about seven days the disappearance was quite slow. After 12 months 15 percent of the dose remained as diphenylamine.

The mobility of diphenylamine has been described as ranging from immobile to low mobility.

Water solubility is about 40 mg/L. The octanol/water partition coefficient is relatively high at Kow = 3860, or log Kow = 3.6 at 25°C. Partition coefficient = Log Pow 3.4, and a high vapour pressure of Kow (6.39 x 10-4 torr). It exhibits very low persistence in direct water [photolysis](http://en.wikipedia.org/wiki/Photolysis) experiments in the laboratory and is moderately volatile. It is very toxic to aquatic organisms. The half-life of diphenylamine in anaerobic water was approximately 60 days.

Diphenylamine appears to be very labile in the environment, with aerobic soil metabolism, and aqueous photolysis having important roles in the dissipation of the molecule. Under aerobic soil metabolism conditions, diphenylamine is rapidly transformed to dimers and polymers (half-life <1 day). In addition, when exposed to light in aqueous media, transformation is rapid (half-life 4.39 hours) (USEPA 1998a).

Direct photodegradation of diphenylamine in water showed a half-life of 1.9 hours in summer and 33.1 h in winter are calculated (50° of latitude, clear sky, clean water near the surface, values integrated over the whole day. Assuming a daily sunlight period of 12 hours an overall mean half-life of 1.46 day results (EU 2008).

### Health considerations

Diphenylamine was first evaluated in 1969 (IPCS 1970) when an ADI of 0.025 mg/kg bw was estimated. Its toxicology was reviewed by the 1998 JMPR, which allocated an ADI of 0–0.08 mg/kg bw and concluded that an acute RfD was unnecessary. Taking together the data from all animal studies with repeated oral application, the value of 7.5 mg/kg bw/d was proposed as NOAEL for adverse effects after chronic exposure from a two-year carcinogenicity study in rats. This NOAEL is based on haematological and histological effects at dietary levels equal or greater than 25 mg/kg bw/d in female rats (LOAEL). This study was the basis for establishing the actual acceptable daily intake (ADI) of 0–0.08 mg/kg bw/d by the JMPR (1998) too (EU 2008).

The short-term [NOAEL](http://en.wikipedia.org/wiki/NOAEL) of 9.6–10 mg/kg bw/day was derived from 90-day rat, 90-day dog and one-year dog studies, and the long-term NOAEL (two-year rat study) was 7.5 mg/kg bw/day. The [Acceptable Daily Intake](http://en.wikipedia.org/wiki/Acceptable_Daily_Intake) of diphenylamine was 0.075 mg/kg bw/day based on the two-year rat study, applying a safety factor of 100. An ARfD was not deemed necessary (EFSA 2011).

As at May 2014, the USEPA *Human Health Benchmarks for Pesticides,* see: <http://water.epa.gov/drink/standards/hascience.cfm>, quotes a RfD of 0.10 mg/kg/d. The USEPA chronic (lifetime) HHBP (Human Health Benchmark for Pesticides) in drinking water for diphenylamine is 0.70 mg/L. (No acute one day value available.)

The Acceptable Daily Intake (ADI) adopted in Australia for diphenylamine is 0.02 mg/kg body weight, with a NOEL of 0.5 mg/kg bw. There is no ARfD.

USEPA (1998) states that DPA generally is of low acute toxicity and has been classified as “Not Likely “ as a human carcinogen.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Diphenylether

CAS No. 101-84-8. Also called diphenyl oxide or DPO; sometimes spelt diphenyl ether.

### Maximum Acceptable Value

Diphenylether does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

Diphenylether has a taste and odour threshold of 0.015 to 0.6 μg/L (IEH (2014).

### Sources to drinking-water

#### 1. To source waters

IEH (2014) selected for consideration all those substances reported as being involved in taste and odour incidents in a developed country, excluding those for which there was no evidence of UK production or import, as well as those already regulated to a limit value either lower than or close to the reported taste and odour threshold. Other prioritised substances were then categorised according to amounts used and their reported taste and odour threshold. This process gave a list of compounds from which substances formed during water treatment were excluded leaving 18 priority compounds.

Diphenyl ether has the following uses: heat transfer agent (sometimes blended with biphenyl); chemical intermediate (for surface-active agents and high temperature lubricants); and perfumery – particularly soap and detergents to impart floral fragrances.

Diphenylether has a disagreeable geranium-like odour.

#### 2. From treatment processes

No known sources.

### Form and fate in the environment

Diphenyl ether is expected to have moderate mobility in soil. The substance is inherently biodegradable when assessed using a modified semi-continuous activated sludge test and screening studies (using effluent from a domestic wastewater treatment plant) found >70 percent degradation within 20 days. However, the substance was degraded by only 6.3 percent after 28 days in an OECD TG 301C test. Based on these results, diphenyl ether is judged not readily biodegradable. Based on the Henry’s Law constant, volatilisation is considered moderate. The rate of hydrolysis is considered negligible and the substance is expected to have moderate persistence and low bioaccumulation potential. Diphenyl ether exhibits a Koc of 1950, suggesting that it is likely to exhibit low mobility in soils. A vapour pressure of 0.02 mm Hg indicates that diphenyl ether is likely to be readily volatilised from soil; and is consistent with diphenyl ether’s common usage as a fragrance compound. Volatilisation half-lifes of 8 and 89 hours for a modelled river and pond indicate that volatilisation is an important mechanism in the removal of diphenyl ether from water sources. The Koc value of diphenyl ether suggests that adsorption to sediment may occur in water; a recalculation of volatilisation from a modelled pond suggests that absorption to sediment may slow volatilisation to give a half-life of 27 days. From IEH (2014).

### Removal methods

Conventional treatment and chlorine may reduce the concentration by about 50 percent, and ozone by 70 percent. The use of granular activated carbon (GAC) and powdered activated carbon (PAC) will enhance the removal (IEH 2014).

### Health considerations

In humans, acute toxic effects of diphenyl ether include severe degenerative hepatic and renal lesions. However, the effects are transitory unless very large quantities are consumed. Rats given diphenyl ether by oral gavage at 400 mg/kg/day for two months exhibited irritation of the GI-tract and reductions in hepatic and renal functions. Rats were fed a diet containing diphenyl ether at 200, 1,000 or 5,000 mg/kg feed for 13 weeks, followed by a four-week period of withdrawal of treatment. At 1000 mg/kg or above, reduced body weight gain and changes in organ weights were noted; no such changes were seen at 200 mg/kg diet and this was suggested as a NOAEL. A project specific TDI of 0.2 mg/kg/day was calculated by the application of an uncertainty factor of 1,000 to this NOAEL. This indicates that controlling diphenyl ether levels in water supplies at its taste and odour threshold is likely to prevent any toxic effects upon consumption. From IEH (2014).

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2014. *National Assessment of the Risks to Water Supplies Posed by Low Taste and Odour Threshold Compounds*. Final Report Project WT1275. 202 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-281.pdf>.

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# 2,4-di-tert-butylphenol

CAS No. 96-76-4. Also called 2,4-di-t-butylphenol, 2,4-DTBP, 2,4-bis(1,1‑dimethylethyl)phenol or 2,4-di-tert-butylhydroxybenzene.

### Maximum Acceptable Value

2,4-Di-tert-butylphenol does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

2,4-Di-tert-butylphenol is a known degradation product of the antioxidant tris(2,4‑di‑butylphenol)phosphite, eg, Irgafos 168s. According to the EC, 10,000 to 50,000 tonnes pa of 2,4-di-tert-butylphenol is made or imported in Europe where it is used as an intermediate for production of phenolic antioxidants and UV stabilisers.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

2,4-Di-tert-butylphenol has been reported to be a major migrating component from most brands of HDPE pipes. Concentrations ranged from 0.05 to 5 micrograms per litre, and took at least 40 successive days of leaching to halve. The threshold odour number during testing was 4, ie, the leach water needed to be diluted four-fold before the odour was no longer perceptible.

2,4-Di-tert-butylphenol has also been found to leach from polypropylene and cross-linked polyethylene (PEX) pipes, and from polyolefin bottles. Migration rates increase with increasing pH and temperature. The presence of 2,4-di-tert-butylphenol has been one of the reasons for water supply pipes failing the leach test for NSF Std 61.

2,6-Di-tert-butylphenol is detected sometimes as well.

### Form and fate in the environment

2,4-Di-tert-butylphenol is persistent in the environment and may take weeks to months to degrade.

The water solubility of 2,4-di-tert-butylphenol is about 12 mg/L.

### Removal methods

No information is available.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

The no-effect concentration of 2,4-di-tert-butylphenol and the related substance 2,6‑di-tert-butylphenol has been estimated to be 0.00045 mg/L based on the lowest short-time effect and an assessment factor of 1000 (DTI 2008).

2,6-Di-tert-butylphenol appears on endocrine disruptor lists, so 2,4-di-tert-butylphenol is also a likely endocrine disruptor.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# EDD and EMD

2-EDD: CAS No. 768-58-1. Also called 2-ethyl-5,5-dimethyl-1,3-dioxane.

2-EMD: CAS No. 4359-46-0. Also called 2-ethyl-4-methyl-1,3-dioxolane.

### Maximum Acceptable Value

Neither has a MAV in the DWSNZ, and they are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

2-EDD and 2-EMD have been involved in several incidents of contamination of drinking water resulting in major taste and odour issues. Both 2-EDD and 2-EMD are extremely odorous, with thresholds of 0.01 μg/L, with the possibility that sensitive individuals may be able to detect them at a level as low as 0.005 μg/L. The odour of 2-EDD has been variously described as walnutty, latex paint, varnish, chlorinous, earthy, musty, creeky/decaying vegetation, nutty, sewage, fishy/algal, marshy/sulphurous, rotten, methanol/piney, sweet and chemical. At its threshold concentration, 2-EMD’s odour has been described as sweet while at higher concentrations terms such as solvent sweet, sickly sweet, toluene and medicinal are used.

Incidents have been reported in Philadelphia (1992), Worcester, affecting supplies drawn the River Severn (1994), a Spanish groundwater (1993). In September 2003, a major treatment works supplying several million consumers in South America had to be shut down due to many consumer complaints. In 2010 two water supplies feeding NE London were affected. Maximum values reported during this event were 0.026 μg/L of 2-EDD and 0.186 μg/L of 2-EMD, which affected 2 million consumers for up to two months. These maximum levels are about 10,000 times less than health-based action levels (DWI 2011a).

All incidents were traced back to resins manufacturing plants, mostly polyester resins, either directly or via effluent from public wastewater plants. These contaminates relate to the mixing of wastes containing aldehydes and glycols from industrial sources, mainly the three parent substances propylene glycol (CAS No. 57-55-6), neopentyl glycol (CAS No. 126-30-7) and propanal (CAS No. 123-38-6). The issues relating to the entry of these substances into water sources relate to reactions between chemicals in the industrial wastes rather than to either the production or downstream use of primary industrial chemicals per se.

The UK DWI states “All water and wastewater companies must ensure that any waste which may be a source of 2-EDD and 2-EMD is closely monitored and any resin manufacturer should be classified as high risk. Waste where the risk is high or unacceptable and cannot be mitigated should not be accepted where it could subsequently enter a raw water source however remote”.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

DWI (2001) reports tastes and odours caused by 2-EDD in conjunction with polyethylene pipes.

### Form and fate in the environment

Little information is available on the environmental fate of 2-EDD and 2-EMD once formed from industrial wastes. DWI (2001) provides some data on 2-EDD; it has a Log Kow of 2.63 and a water solubility of 455 mg/L (25°C).

### Removal methods

Both substances pass readily through many sewage treatment processes and are not removed by the drinking water treatments of coagulation, flocculation or clarification. Chlorine may reduce the concentration by about 35 percent. Ozone may halve the concentration. The use of granular activated carbon (GAC) and powdered activated carbon (PAC) are, however, reasonably effective in reducing the concentration of 2‑EDD and 2-EMD (IEH 2014).

### Analytical methods

#### Referee method

See DWI (2011).

### Health considerations

No specific health effects are known. Drinking water would become aesthetically unacceptable before any health issues arise: … *the reported concentrations are several orders of magnitude lower than the levels expected to cause health effects in humans.* DWI. 2011. DWI (2011a) reports Health Protection Agency (HPA) health-based action levels of 2.5 mg/L for 2-EDD and 1.4 mg/L for 2-EMD.

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2001. *Factors Causing Off-taste in Waters, and Methods and Practices for the Removal of Off-taste and its Causes*. DETR/DWI 5008/1. 69 pp. Available at: <http://dwi.defra.gov.uk/research/completed-research/reports/0820.pdf>.

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# EDTA

CAS No. 60-00-4. Also called edetic acid or ethylenediamine tetraacetic acid. The IUPAC name is 2-[2-(bis(carboxymethyl)amino)ethyl-(carboxymethyl)amino]acetic acid. CAS name is N,N’-1,2-ethanediylbis[N-(carboxymethyl)-glycine.

Tetrasodium EDTA has CAS No. 64-02-8; trisodium EDTA has CAS No. 150-38-9; disodium EDTA has CAS No. 139-33-3. The disodium EDTA dihydrate CAS No. is 6381‑92-6.

The technical grade of the tetrasodium salt is usually only about 74 percent pure; 3 percent is nitrilotriacetic acid (NTA) (EU 2004).

### Maximum Acceptable Value

Based on health considerations, the concentration of EDTA in drinking-water should not exceed 0.7 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of ethylenediamine tetraacetic acid in drinking water (as the free acid) should not exceed 0.25 mg/L.

### Sources to drinking-water

#### 1. To source waters

EDTA may enter source water as an industrial contaminant where it is used to mitigate against water hardness in boilers and industrial applications. It is used widely in many industrial processes, in agriculture, to control metals that destabilise cosmetics and pharmaceuticals, as an ingredient to sequester trace metals that cause rancidity in foods and vitamins, and in drugs for chelation therapy; it is the drug of choice to treat lead poisoning in humans and domestic animals. EDTA is also used in laundry detergents (banned in some countries), photochemicals, water softening, electroplating, and in the production of textiles and paper. About 260 tonnes per annum are used in the UK.

Cu-, Fe-, Mn-, Mg-, Mo- and Zn-EDTA complexes are mixed into fertilisers if there is a lack of trace elements in agricultural soil. The metals are applied in chelate form in order to prevent their precipitation as biological inactive compounds and to allow their conversion through the soil medium to the root zone for uptake into the plant. The herbicide 2,4-D may contain 1 percent EDTA (EU 2004).

DWI (2014) identified EDTA as a risk to drinking water from personal care products and domestic cleaning products.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

EDTA is only poorly degraded in the aquatic environment and is not adsorbed to particulate matter, ie, it is persistent in the water phase. It is not expected to be degraded in sewage treatment systems. For instance an OECD screening test indicated 10 percent degradation with municipal wastewater and EDTA concentration between 7 and 50 mg/L after 19 days. It has been reported at 5 mg/L in sewage effluent and 0.5 mg/L in surface water (DWI 2014).

The partition coefficient = logPow = about -4. Due to the ionic structure under environmental relevant pH conditions, no adsorption on to the organic fraction of soils or sediments is expected. Henry’s law constant = 1 x 10-20 Pa.m3/mol, suggesting that volatilisation will not occur. EDTA is resistant to hydrolysis over a wide pH range. A medium half-life for agricultural used soil of 300 days can be deduced (EU 2004).

It exists in the environment as metal complexes with the potential of mobilising potentially toxic heavy metals in water, thus increasing their concentration in water supplies. From overseas data this is not likely to be of health concern for the concentrations of heavy metals and EDTA found. Water solubility is quoted about  
400–1,000 mg/L. The sodium salts are extremely soluble.

### Typical concentrations in drinking-water

No data are available on the concentration of EDTA in New Zealand drinking-water supplies.

Overseas data show EDTA to be present in surface waters generally at concentrations below 0.07 mg/L, although higher concentrations (0.9 mg/L) have been measured; detected in drinking-water prepared from surface waters at concentrations of  
0.01–0.03 mg/L. DWI (2014) reports finding EDTA at 0.09 mg/L in drinking water.

The analysis of 47 samples of 14 water supply companies in The Netherlands confirmed the presence of EDTA in drinking water prepared from surface water, with or without dune filtration or bank filtration, in concentrations of 0.010 to 0.030 mg/L. From EU (2004).

### Removal methods

Based on measurements in river water (Elbe) and bank filtrate, a very slow degradation of EDTA during bank filtration was found under both aerobic and anaerobic conditions. The elimination rate was 20 percent after one to eight days in a both aerobic and anaerobic zone, 47 percent after 20 to 40 days and 53 percent after 150 to 300 days under anaerobic conditions. The EDTA concentration in the river was 0.015 mg/L (50 percentile values). In a test slow sand filter, no EDTA elimination was observed (EU 2004).

It is not removed by coagulation or filtration or chlorination. Up to 80 percent removal can be achieved by activated carbon and ozone (DWI 2014). Oxalic acid, glyoxylic acid, iminodiacetic acid, glycine, nitrate and ammonium were identified as ozone reaction products (EU 2004).

Measurements at WTPs showed different results: at Düsseldorf-Flehe, the EDTA concentration decreased from 0.028 mg/L in the Rhine to 0.004 mg/L in the drinking water (after bank filtration, ozone oxidation, filtration, and charcoal adsorption), ie, an elimination of 86 percent. In water from Lake Constance, the decrease was from 0.0025 mg/L in the lake to 0.0015 mg/L (elimination 40 percent) after sieve, ozone oxidation, and sand filtration. From EU (2004).

### Analytical methods

#### Referee method

Reverse Phase Ion Pair Liquid Chromatography (Bergers and de Groo 1994 *Wat Res* 28(3): 639).

#### Some alternative methods

No alternative methods have been recommended for EDTA because no methods meet the required criteria.

### Health considerations

CaNa2EDTA is poorly absorbed from the gut. It is medically inert, and no accumulation in the body has been found.

The long-term toxicity of EDTA is complicated by its ability to chelate essential and toxic metals, both in water and in animals. Toxicity data are therefore equivocal and difficult to interpret.

Long-term feeding studies in rats and dogs gave no evidence of interference with mineral metabolism in either species. Adverse effects on mineral metabolism and nephrotoxicity was seen only after parenteral administration of high doses. EDTA has been found to be both [cytotoxic](http://en.wikipedia.org/wiki/Cytotoxicity) and weakly [genotoxic](http://en.wikipedia.org/wiki/Genotoxicity) in laboratory animals.

High doses of EDTA tested on animals in the USA did not reveal any carcinogenicity.

A vast clinical experience with respect to the use of EDTA in the treatment of metal poisoning has demonstrated its safety in humans. The major human health problem due to oral exposure to EDTA appears to be zinc deficiency as a consequence of zinc complexed by EDTA. It has also been suggested that EDTA may enter kidney cells and, by interfering with zinc metabolism, exacerbate the toxicity of cadmium. Concern has been expressed over the ability of EDTA to complex, and therefore reduce the availability of, zinc. However, this is of significance only at elevated doses substantially in excess of those encountered in the environment.

### Derivation of Maximum Acceptable Value

In 1973, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) proposed an Allowable Daily Intake for calcium disodium edetate (CaNa2EDTA) as a food additive of 2.5 mg/kg body weight (1.9 mg/kg as the free acid). However, JECFA recommended that no sodium edetate should remain in food.

The MAV was derived for EDTA (as the free acid) as follows:

1.9 mg/kg body weight per day x 70 kg x 0.01 = 0.665 mg/L (rounded to 0.7 mg/L)

2 L

where:

* allowable daily intake = 1.9 mg/kg
* average body weight = 70 kg
* allocation of allowable daily intake to drinking-water = 0.01
* average quantity of water consumed per day = 2 L.

The MAV in the 1995 DWSNZ had been based on the body weight of a 10 kg child and allocating 10 percent of the daily intake to drinking-water, and a UF of 10, as follows:

1.9 mg/kg body weight per day x 10 kg x 0.1 = 0.2 mg/L

1 L x 10

where:

* allowable daily intake = 1.9 mg/kg
* average body weight of a child = 10 kg
* allocation of allowable daily intake to drinking-water = 0.1
* average quantity of water consumed per day by a 10 kg child = 1 L
* uncertainty factor = 10 to reflect that the JECFA allowable daily intake has not been considered since 1974, and concern over zinc complexation.

### References

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# Endocrine disrupting compounds

DWI (2001) quotes an internationally agreed working definition of endocrine disrupters:

“An endocrine disrupter is an exogenous substance or mixture that alters function(s) of the endocrine system and consequently causes health effects in an intact organism, or its progeny, or (sub) populations.”

### Maximum Acceptable Value

The DWSNZ do not include a MAV and the WHO Guidelines do not discuss endocrine disrupting compounds as a separate topic. Pharmaceuticals (eg, antibiotics, endocrine disruptors) in drinking-water are included in the [plan of work of the rolling revision](http://www.who.int/entity/water_sanitation_health/gdwqrevision/en/index.html) of the WHO Guidelines for Drinking-water Quality.

Datasheets (qv) exist for some individual determinands considered to be endocrine disrupting compounds.

EFSA (2018): This 170-page guidance document is an early version, published to ensure availability of guidance on the recommended assessment approach for Endocrine Disruptors by 7 June 2018, ie, the date by which the Commission Delegated Regulation (EU) 2017/2100 becomes applicable. This early version will be subject to a final editorial consistency check and typeset, but there will be no material change to the document. It will be replaced by the final typeset version published in the *EFSA Journal* which is scheduled for mid‐July 2018. It describes how to perform hazard identification for endocrine-disrupting properties by following the scientific criteria which are outlined in Commission Delegated Regulation (EU) 2017/2100 and Commission Regulation (EU) 2018/605 for biocidal products and plant protection products, respectively.

### Sources to drinking-water

#### 1. To source waters

The function of the endocrine system and how it can be disrupted is discussed in IUPAC (2003). Endocrine disrupting compounds can enter raw water from sewage, effluent from municipal and some industrial wastewater treatment plants, and from areas with high densities of animals or where manure is spread. The concentrations of various endocrine disrupting compounds in natural water appear in IUPAC (2003).

AWWA (2008) examined the toxicological relevance of trace levels of endocrine disrupting compounds and pharmaceuticals (qv) in drinking-water supplies. A sampling program was carried out at 20 drinking-water treatment plants and four wastewater treatment plants across the US. Twenty-three of the 62 target compounds were detected at least once in finished drinking-water by chemical assay methods, and eleven were detected in more than 20 percent of drinking-water samples. The herbicide atrazine occurred most frequently (83 percent of drinking-water samples), followed by meprobamate (78 percent, an anti-anxiety drug) and phenytoin (56 percent, an anti-epileptic drug). Only two finished drinking-water samples and one distribution system sample gave a positive response to the E-screen bioassay, whereas all foods and beverages tested other than bottled water gave positive results on the E‑screen bioassay.

The occurrence of endocrine disrupting compounds in Canadian wastewater and receiving waters was reported in Alberta Environment (2005).

17-alpha-Estradiol (CAS No. 50-28-2), estriol (CAS No. 50-27-1), estrone (CAS No. 53‑16-7), ethinyl estradiol (CAS No. 57-63-6), equilenin (CAS No. 517-09-9), equilin (CAS No. 474-86-2), mestranol (CAS No. 72-33-3) and norethindrone or 19‑norethisterone (CAS No. 68-22-4) are included in the USEPA (2009) third Contaminant Candidate List because their occurrence or anticipated occurrence is likely at levels of concern to human health.

Tremblay et al (2011) reported studies by Gadd at 18 New Zealand dairy farms, analysing steroid estrogens and their conjugates, as well as estrogenic activity using the E-Screen bioassay. This study highlighted the importance of measuring conjugates and 17α-estradiol (the dominant form of estrogen excreted by cattle). Concentrations of steroid estrogens were elevated in dairy shed effluents with potential to cause environmental effects if discharged directly to the aquatic environment with minimal dilution. The study also suggested that when disposed of on land, there is a possibility for water-soluble conjugated steroids to leach through soils to the aquatic environment and pose a problem if hydrolysed at a later time. Gadd also assessed the reduction of estrogens from dairy effluent using the two pond system and advanced pond system treatment options. The results suggested a 50–100 percent decrease in total steroid concentrations and 62–100 percent decrease in estrogenic activity. Estrogenic steroid hormones have been detected in groundwater and stream waters of intensively farmed dairy catchments. It was noted that estrogenic activity of the effluent at times exceeded suggested guideline values for protection of freshwater fish. Estrogenic steroid hormones are discussed further in Auckland City (2016).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

DWI (2001) tested compounds leaching from two replacement GRP tanks, and two tanks relined with a butyl rubber liner. Samples from the four sites were analysed for the following selection of known potential endocrine disrupting groups of chemicals:

* alkyl phenols – nonyl phenyl, octyl phenol
* bisphenols – bisphenol-A, bisphenol-F and their corresponding diglycidyl ethers
* phthalates – bis 1-octylphthalate, bis nonylphthalate, bis 2-ethylhexylphthalate, bis ethylphthalate, bis butylphthalate, bis iso-butylphthalate, benzylbutylphthalate, bis hexylphthalate, and bis methylphthalate
* styrene.

Results of the analysis of the targeted analytes indicated that there is negligible risk to the consumer from these potential endocrine disrupting chemicals following correctly installed or refurbished water storage tanks.

### Form and fate in the environment

The findings from two New Zealand laboratory studies into the sorption and degradation of estrogens and their conjugates suggested that soil type was important in determining the fate of these chemicals. Dissolved organic carbon facilitated transport of these hormones and needs to be considered when assessing the leaching risk for these compounds in the environment. Microcosm laboratory experiments were conducted in three pasture soils from New Zealand to study the aerobic degradation and metabolite formation kinetics of estrogen sulfate conjugates, which are excreted by livestock in urine, confirming that soil type was important as degradation was shown to be different between the three soils. Field and modelling studies have been undertaken to characterise the transport of estrogens through representative soils in New Zealand following land application of animal waste. Results confirmed that estrogens were transported mainly via preferential/macropore flow and also via an enhanced transport, probably mediated by colloids (taken from Tremblay et al 2011).

### Typical concentrations in drinking-water

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 11,795 drinking water samples for 17β-estradiol between 2013 and 2015, and found four samples exceeded the minimum reporting level (MRL) of 0.0004 µg/L, and one water supply contained >0.0009 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 11,796 drinking water samples for 17α-ethynlestradiol between 2013 and 2015, and found four samples exceeded the minimum reporting level (MRL) of 0.0009 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 11,796 drinking water samples for estriol between 2013 and 2015, and found four samples exceeded the minimum reporting level (MRL) of 0.0008 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 11,796 drinking water samples for equilin between 2013 and 2015, and found zero samples exceeded the minimum reporting level (MRL) of 0.004 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 11,796 drinking water samples for estrone between 2013 and 2015, and found zero samples exceeded the minimum reporting level (MRL) of 0.002 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 11,795 drinking water samples for testosterone between 2013 and 2015, and found 72 samples exceeded the minimum reporting level (MRL) of 0.0001 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 11,796 drinking water samples for 4-androstene-3,17-dione between 2013 and 2015, and found 101 samples exceeded the minimum reporting level (MRL) of 0.0003 µg/L.

### Removal methods

Raw water supplies likely to contain endocrine disrupting compounds usually include treatment processes such as ozone dosage and activated carbon adsorption. The USEPA (2001) discusses removal techniques for groups of pesticide residues, highly chlorinated compounds, alkylphenols and alkylphenol ethoxylates, and plastic additives. AWWA (2008) discusses some advanced oxidation processes.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See individual datasheets where relevant.

### Health considerations

EC (2002) categorises (in Table 6) 40 manmade chemicals or groups of chemicals into high, medium or low level of concern.

Oestrone, 17b-oestradiol and 17a-ethinyloestradiol are on the EC List (Annex 15) of 66 Category 1 substances showing evidence of endocrine disrupting activity in at least one species using intact animals (EC 2002a).

Most of the endocrine disrupting compounds with pesticide and industrial chemical origin have individual MAVs which protect consumers from health effects. The alkylphenols and alkylphenol ethoxylates have a non-ionic detergent origin, and are not reported to present a problem in New Zealand; in wastewater treatment plants and in the environment, alkylphenol ethoxylates can be microbially degraded, and toxic metabolites such as p-nonylphenol and p-nonylphenol ethoxylates are formed. It is these transformation products, not the parent compounds, that exhibit estrogenic activity (IUPAC 2003).

In a US study (AWWA 2008) most of the target pharmaceuticals potential toxicity was assessed by comparing levels detected in water with calculated Acceptable Daily Intakes (ADIs). This approach is commonly used to determine acceptable levels for chronic term exposure to non-carcinogenic environmental contaminants. The ADI was calculated by taking the highest daily dose for which no adverse effect was observed in a long-term animal study, then applying safety factors (usually 100-fold or more) to derive a dose which was expected to be safe for long-term exposure in humans. Additional safety factors were applied where the quality of data was limited. For several of the pharmaceuticals where there was evidence of carcinogenic effects in animals, an alternative and more conservative approach was used. The ADI for each pharmaceutical (expressed as mg per kg body weight per day) was then converted to an acceptable Drinking Water Equivalent Level (DWEL) assuming a body weight of 70 kg and a drinking water intake of two litres per day.

The highest estrogenic activity measured in source water (raw water) by the E-screen assay was similar to that found in green tea and only one-quarter of that found in coffee. Using chemical assay methods, the major estrogenic hormones estrone, estradiol and ethynylestradiol found in wastewater have not been detected in finished drinking-water samples in this study or other published studies from the US, yet they can be detected in human breast milk and cows milk (AWWA 2008).

Among the 11 pharmaceuticals detected in finished drinking-water or distribution system water, the anti-psychotic drug risperidone had the lowest margin of safety (170), followed by the anticonvulsants phenytoin (210) and carbamazipine (670). The minimum margins of safety for the remaining eight pharmaceuticals which were detected and the nine pharmaceuticals which were not detected ranged from greater than 2,700 to greater than 40 million. For the three EDCs which were detected in finished drinking-water or distribution system water, atrazine had the lowest minimum margin of safety (3), while the herbicide linuron (8400) and the industrial chemicals 4‑nonylphenol (16,000) and bisphenol A (72,000) had higher margins (AWWA 2008).

The compounds of greatest hormonal activity in sewage effluent are the natural estrogens and androgens. Present data indicate that estrogenic contamination of drinking-water is very unlikely to result in physiologically detectable effects in consumers; pesticide, detergent and industrial contamination remain issues of concern (Falconer 2006). The commoner anthropogenic compounds that have been implicated with endocrine disruption that have a datasheet in these Guidelines are:

|  |  |
| --- | --- |
| **Organic determinands** | **Pesticides** |
| bisphenol A | DDT and isomers |
| dibromacetic acid | diuron |
| di(2-ethylhexyl)phthalate | endosulfan |
| dioxin | linuron |
| PAHs | methoxychlor |
| PCBs | propazine |
| tributyltin oxide |  |

In a UK study (DWI 2012) the initial literature review identified 509 articles suggesting 325 potential EDCs could be present in water bodies of potential relevance. Through application of a customised prioritisation scheme, this candidate list was reduced to 159 potential EDCs that were then subject to a multistage modelling process to estimate their environmental fate and behaviour and extent of potential removal by water treatment processes prevalent in the UK. Thirty-five of these chemicals were predicted to have highest (worst case) concentrations (ie, following conventional treatment processes) ≥100 ng/L; these modelled levels were used to estimate potential intakes (as mg/kg bw/d) via drinking water for three population subgroups: adults (>18 years), toddlers (1 to 2 years) and infants (0 to 1 years), based on standard default assumptions. The extent of the risk posed by such a worst case intake was then determined by establishing the margin of safety (MOS) between this intake and either an established authoritative health-base criteria value (eg, tolerable daily intake) or using a study-specific exposure limit derived from the available hazard data; in either case, the value was based on what was considered to be the most sensitive endpoint irrespective to its relevance to the endocrine system. For endocrine-active pharmaceuticals considered to be of potential concern, a study-specific exposure limit was determined on the basis of the minimum therapeutic dose, using clinical judgement.

Comparison of predicted worst-case drinking water intakes against the hazard profile for the 35 chemicals subject to detailed modelling showed a very high MOS (>100) for 21 chemicals, even using worst case assumptions, and these were not considered to warrant further study. A further eight chemicals had MOS of 10 to 100 and hence were considered of doubtful importance and, hence not to warrant further consideration. For six chemicals (p-benzylphenol, dibutyl phthalate (qv), 4-nitrophenol (qv), digoxin, fluticasone and salbutamol), the MOS were ≤10, and hence were considered to warrant a more detailed consideration to establish the likely ‘real world’ situation, as opposed to the estimates derived here from the use of highly conservative ‘worst case’ assumptions throughout the modelling process.

Furthermore, a précis of current scientific understanding with regard to the risks posed by complex mixtures of EDCs was prepared and an indicative estimate made of the potential risk that might arise from a mixture containing those substances identified here that possess oestrogenic activity , each at their predicted worst-case level. Importantly, it was found that even such an extreme worst case combined intake, when expressed in terms of an equivalent oestradiol intake, did not raise a significant health concern.

### Derivation of Maximum Acceptable Value

No MAV. See individual datasheets where relevant.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The short-term limit for 17α‑ethinylestradiol is 0.0000005 mg/L; the chronic and subchronic health risk limits are 0.0000002 mg/L.

The short-term limit for mestranol is 0.0000007 mg/L; the chronic and subchronic health risk limits are 0.0000002 mg/L.

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# Epichlorohydrin

CAS No. 106-89-8. IUPAC name chloromethyloxirane. Also called epoxypropane, 3‑chloro-1,2-propylene oxide, chloropropylene oxide, chloromethylethylene oxide, chloromethyl oxirane, 1-chloro-2,3-epoxypropane, 1,2-epoxy-3-chloropropane, 2,3‑epoxypropyl chloride or glycidyl chloride. Can be called epichlorhydrin and alpha-epichlorhydrin too.

### Maximum Acceptable Value (provisional)

Based on health considerations, the concentration of epichlorohydrin in drinking-water should not exceed 0.0005 mg/L (0.5 g/L).

The guideline value is considered to be provisional because of the uncertainties surrounding the toxicity of epichlorohydrin and the use of a large uncertainty factor in deriving the WHO guideline value.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of epichlorohydrin in drinking water should not exceed 0.0005 mg/L (which is below the limit of determination; improved analytical procedures are required for this compound).

NSF Standard 60 states that the allowable concentration of epichlorohydrin in drinking water is 0.002 mg/L.

The Prescribed Concentration or Value (PCV) for epichlorohydrin in England and Wales is 0.0001 mg/L. See Notes.

USEPA (2006) states “when epichlorohydrin is used in drinking water systems the combination (or product) of dose and monomer level shall not exceed that equivalent to a epichlorohydrin-based polymer containing 0.01 percent monomer dosed at 20 mg/L”.

### Sources to drinking-water

#### 1. To source waters

Epichlorohydrin may enter raw water as a contaminant from a wide range of industrial uses. It is used mainly in the manufacture of glycerine (glycerol) (less common today), and unmodified epoxy resins, and also in the manufacture of elastomers, water treatment polymers, epoxy and phenoxy resins, surfactants, ion exchange resins, plasticisers, dyestuffs, pharmaceutical products, oil emulsifiers, lubricants and adhesives. Papers treated with epichlorohydrin-based wet-strength resins may be used in food contact, such as in tea bag paper, coffee filters; other consumer paper products are paper tissues and towels.

#### 2. From treatment processes

Epichlorohydrin can enter drinking-water supplies through the use of polyamine flocculating agents containing epichlorohydrin, although this is not a significant source in New Zealand at this time (Gregor et al 1993). The New Zealand standard (NZWWA 1999) states that epichlorhydrin levels shall not exceed 5 mg/kg of active polymer. Impurities in epichlorohydrin include 1,2,3-trichloropropane, 1,3-dichloro-2-propanol, 2,3-dichloro-1-propanol and 3-monochloropropane 1,2-diol – see datasheets, and the Epi-DMA datasheet. 1,2,3-Trichloropropene has been recorded as an impurity of epichlorhydrin too.

#### 3. From the distribution system

Epichlorohydrin may enter drinking-water through the leaching of epichlorohydrin from epoxy resin coatings on pipes and fittings.

### Form and fate in the environment

Epichlorohydrin is very soluble in water, where it hydrolyses. Its half-lifes in neutral, acidic and alkaline solutions at room temperature are 148, 79 and 62 hours, respectively.

### Typical concentrations in drinking-water

No data are available on the concentration of epichlorohydrin in New Zealand drinking-water supplies. NSF (2010) reported the results of testing 112 samples of drinking water; the median was <0.0004 mg/L and the range was <0.0004 to 0.0009 mg/L.

### Removal methods

Epichlorohydrin may arise from flocculating agents, or from water treatment resins. Unacceptable levels of the substance might therefore be reduced by consideration of product quality and the dosage being used for treatment of the water.

No information is available on processes that might reduce epichlorohydrin concentrations in water, although aeration is unlikely to be successful.

### Analytical methods

#### Referee method

Gas Chromatography with Electron Capture Detection, Pesselman and Feit 1988 *J Chrom* 439: 448–52.

#### Some alternative methods

No alternative methods have been recommended for epichlorohydrin because no methods meet the required criteria. However, the following information may be useful:

Epichlorohydrin in water can be determined by a purge and trap gas chromatographic procedure with mass spectrometry (USEPA 1987), or flame ionisation detection (AWWA Standard 1989). The limit of quantification is 0.01 mg/L.

### Health considerations

Epichlorohydrin is rapidly and extensively absorbed following oral administration and may be absorbed following both inhalation and skin contact. Following oral administration and inhalation, epichlorohydrin metabolites are excreted rapidly in the urine and expelled air.

Epichlorohydrin is a strong irritant and acutely toxic following oral, percutaneous, subcutaneous and respiratory exposure. Death is due to effects on the central nervous system and the respiratory centre.

Acute toxic responses following skin contact in humans are characterised by an initial redness and itching or burning sensation. With time, the redness intensifies and the tissue becomes swollen and blistered. The initial symptoms following inhalation are local irritation, burning sensation of eyes and throat, swelling of the face, nausea, vomiting, and severe headache.

In a case-study, long-term effects due primarily to damage of the liver and kidney were still present two years after exposure. In epichlorohydrin workers, increased incidences of chromatid and chromosomal breaks in peripheral lymphocytes and decreases of blood cell counts were observed.

An epidemiological study was undertaken for 863 workers, with probable exposure to epichlorohydrin at two chemical plants. All cancer, leukaemia, and most other causes of death were related to estimated levels of exposure to epichlorohydrin. The most consistent relationship was between exposure level and heart disease.

In animal studies, epichlorohydrin induced squamous cell carcinomas in the nasal cavity by inhalation and forestomach tumours by the oral route. It has been shown to be genotoxic *in vitro* and *in vivo*. The International Agency for Research on Cancer has placed epichlorohydrin in Group 2A (probably carcinogenic to humans).

The USEPA (2009/2011) quotes a health advisory of 0.3 mg/L for epichlorohydrin, representing a 10-4 cancer risk. Epichlorohydrin appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

The reference dose or RfD (USEPA 2006/2009/2011) is 0.002 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.07 mg/L.

### Derivation of Maximum Acceptable Value

Although epichlorohydrin is a genotoxic carcinogen, the use of the linear multistage model for estimating cancer risk was considered inappropriate because tumours are seen only at the site of administration where epichlorohyrin is highly irritating.

A tolerable daily intake approach was therefore taken for the derivation of the MAV for epichlorohydrin in drinking-water. The lowest-observable-adverse-effect level used in the derivation was determined for forestomach hyperplasia in a two-year study in rats by gavage.

The provisional MAV for epichlorohydrin in drinking-water was derived as follows:

2 x (5/7) mg/kg body weight per day x 70 kg x 0.1 = 0.0005 mg/L (0.5 g/L)

2 L x 10,000

where:

* lowest-observable-adverse-effect level = 2 mg/kg body weight/day for forestomach hyperplasia in a two-year study in rats by gavage (normalised for five days/week dosing in the derivation)
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 10,000 (100 for intra- and interspecies variation and 10 reflecting carcinogenicity and 10 for the use of a LOAEL instead of a NOAEL).

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# Epi-DMA

This is the cationic polyamine polymer called epichlorhydrin-dimethylamine or poly(epichlorhydrin-dimethylamine) that can be used to assist in the coagulation of water, dosage usually up to mg/L when used as the primary coagulant.

Refer to the datasheets for information about the following impurities or by-products:

* epichlorohydrin
* dimethylamine
* glycidol
* 1,3-dichloro-2-propanol
* 2,3-dichloro-2-propanol
* ethylenediamine
* N-nitrosodimethylamine
* 3-monochloropropane-1,2-diol

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# Ethylbenzene

CAS No. 100-41-4. Can be called (rarely) phenylethane, ethylbenzol, α-methyltoluene and EB.

### Maximum Acceptable Value

Based on health considerations, the concentration of ethylbenzene in drinking-water should not exceed 0.3 mg/L.

The maximum contaminant level or MCL (USEPA 2006/2009/2011) is 0.7 mg/L. The USEPA (2006/2009/2011) also established a lifetime health advisory of 0.7 mg/L, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on aesthetic considerations (taste and odour), the concentration of ethylbenzene in drinking water should not exceed 0.003 mg/L. Ethylbenzene would not be a health concern unless the concentration exceeded 0.3 mg/L.

Ethylbenzene is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

The primary source of ethylbenzene in the environment is the petroleum industry. Ethylbenzene occurs naturally as a component of crude oil, and is present in petrol (1.5–3.1 percent by weight). It is about 0.8 percent of aviation fuel and <0.2 percent of diesel no. 2. Ethylbenzene may occur naturally, as it has been found in orange peel, parsley leaves, dried legumes and other foodstuffs.

It is produced commercially by the alkylation of benzene with ethylene, and by fractionation of petroleum. It is used almost entirely in the production of styrene. It is a major component of commercial xylene and is used commercially in paints, glues, insecticides, and as a solvent. Therefore ethylbenzene may also be found in source waters as an industrial contaminant. It is also found in asphalt, and in contaminated coal tar and coal gas industrial sites.

EU (2007) reports typical concentrations of ethylbenzene of 0.01 µg/L in surface water, 0.02 µg/L in seawater, 0.03 µg/L in rainwater, 1.1 µg/L in snow, and 1 to 2 µg/L in drinking water. Concentrations have been falling over the past 10–15 years. Groundwater can be very high if contaminated.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Because of its high vapour pressure (9.3 hPa at 20°C) and reasonably fairly low solubility in water (about 150–180 mg/L), ethylbenzene will disperse in the atmosphere if released. Photolysis and biodegradation of ethylbenzene in soil, activated sludge and water, under aerobic conditions, has been reported. Hydrolysis is not important. Biodegradation is temperature dependant: half-life in water can be <1 day in the summer, two to three weeks in other seasons. However, it is much more resistant in anaerobic conditions. Ethylbenzene has a log octanol-water partition coefficient (Kow) of 3.15.

If released to soil, ethylbenzene is expected to have moderate mobility based upon an estimated Koc of 520. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 7.88 x 10-3 atm‑cu m/mole (617 Pa.m3/mol). Ethylbenzene may volatilise from dry soil surfaces based upon its vapour pressure. Biodegradation in soil takes place via nitrate-reducing processes. If released into water, ethylbenzene may adsorb to suspended solids and sediment in water based upon the estimated Koc. Biodegradation in a gasoline contaminated aquifer ranged from 10–16 days under aerobic conditions. Ethylbenzene was degraded in eight days in groundwater and 10 days in seawater as a component of gas oil. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 1.1 and 99 hours, respectively. Measured BCFs of 0.67 to 15 suggest the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to occur due to the lack of hydrolysable functional groups (EAWAG accessed February 2015).

Ethylbenzene has a biodegradation half-life of 15 days in surface water, 30 days in soil, and 300 days in sediment. A log Koc of 2.64 indicates a moderate mobility in soil (EU 2007).

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 301 zones, found ethylbenzene concentrations in two zones ranging from 0.0011 to 0.0018 mg/L, with the median concentration being “nd” (limit of detection = 0.0005 mg/L) (ESR 2001).

It has been found in contaminated groundwater at 0.3 mg/L. Ethylbenzene was listed as one of the 58 most frequently detected chemicals associated with groundwater contamination in the United States. It was detected in over 4 percent of the surface water samples and 11 percent of the groundwater samples analysed. In Canada, in a study of 30 water treatment plants, concentrations in drinking-water were below 0.001 mg/L. Surface water samples usually contain less than 0.001 mg/L.

1,050 water utilities in the US reported detecting ethylbenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.054 mg/L.

The maximum concentration found in 9,057 samples from 2634 groundwaters in the UK was 0.028 mg/L, mean 0.0006 mg/L (DWI 2008).

### Removal methods

Ethylbenzene can be removed from water by adsorption on to granular activated carbon or more cost-effectively by air stripping. Significant removal is also expected by ozonation.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

Ethylbenzene, in liquid form, is absorbed readily by humans via the skin and via the intestinal tract, and the vapour is readily absorbed when inhaled. It can be stored in fat and is metabolised to mandelic and phenylglyoxalic acids and excreted in urine. It can cross the placenta.

Ethylbenzene has a low acute toxicity via the oral route. No data are available on the human health effects after oral exposure, and inhalation data are limited to short-term studies.

A six-month feeding study using rats reported enlargement of the liver and kidney at high doses (400 mg/kg body weight per day). Liver effects were also observed in a number of inhalation studies. No longer-term studies are available.

No carcinogenicity data on ethylbenzene are available and the compound has been found to be non-mutagenic in a number of tests. The USEPA has classified ethylbenzene as Group D (not classifiable as to human carcinogenicity), due to the lack of animal bioassays and human studies (IRIS 2007). IARC (2000/2006) has classified ethylbenzene as a Group 2B carcinogen (possibly carcinogenic to humans). This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.4 mg/kg/day for intermediate-duration oral exposure  
(15–364 days) to ethylbenzene.

The reference dose or RfD (USEPA 1991/2006/2009/2011) is 0.1 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 3 mg/L.

### Derivation of Maximum Acceptable Value

A tolerable daily intake approach has been used for the derivation of a MAV for ethylbenzene in drinking-water. A no-observable-adverse-effect level was determined in a limited six-month study in rats, based on observed heptatoxicity and neprotoxicty.

The MAV for ethylbenzene in drinking-water was derived as follows:

136 x (5/7) mg/kg body weight per day x 70 kg x 0.1 = 0.34 mg/L (rounded to 0.3 mg/L)

2 L x 1000

where:

* no-observable-adverse-effect level = 136 mg/kg body weight per day based on its heptatoxicity and nephrotoxicity observed in a limited six-month study in rats (normalised for 5 days/week dosing in derivation)
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1,000 (100 for intra- and interspecies variation and 10 for the limited data base and short duration of the study).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The short-term, subchronic and chronic limits are 0.05 mg/L.

Taste and odour thresholds for ethylbenzene have been reported at 0.08 and 0.002 mg/L respectively. The aesthetic objective in Canada is not greater than 0.0024 mg/L.

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# Ethylenediamine

CAS No. 107-15-3. Also called ethane-1,2-diamine (IUPAC name), 1,2-diaminoethane, 1,2-ethanediamine and EDA. Sometimes spelt ethylene diamine.

### Maximum Acceptable Value

There is no MAV for ethylenediamine in the DWSNZ, and it is not mentioned in the WHO Guidelines.

NSF (2010) states that the allowable concentration of ethylenediamine in drinking water is 2 mg/L.

### Sources to drinking-water

#### 1. To source waters

Ethylenediamine is not known to occur naturally. It is a by‑product of polyamine polyelectrolytes (see datasheet).

Ethylenediamine is used in large quantities for production of many industrial chemicals, including ethylenebisdithiocarbamate pesticides and dyestuffs. It is used as a [corrosion inhibitor](https://en.wikipedia.org/wiki/Corrosion_inhibitor) in paints and [coolants](https://en.wikipedia.org/wiki/Antifreeze_(coolant)).

Ethylenediamine readily complexes with metals, similar to EDTA (qv), so has many similar uses.

#### 2. From treatment processes

NSF Standard 60 regulates the impurities and by-products in polyamine polyelectrolytes (see datasheet); this includes ethylenediamine.

### Form and fate in the environment

Ethylenediamine is miscible with water.

Leaching through soil profiles to groundwater is not expected (WHO 1999).

If released to soil, ethylenediamine is expected to have slight mobility based upon an average Koc of 4766. Volatilisation from moist soil surfaces is not expected to be an important fate process based on a Henry’s Law constant of 1.73 x 10-9 atm‑cu m/mole. Ethylenediamine may volatilise from dry soil surfaces based upon its vapour pressure. However, adsorption to soil may attenuate volatilisation. If released into [water](http://pubchem.ncbi.nlm.nih.gov/compound/water), ethylenediamine is expected to adsorb to suspended solids and sediment in [water](http://pubchem.ncbi.nlm.nih.gov/compound/water) based upon the average Koc value. Volatilisation from [water](http://pubchem.ncbi.nlm.nih.gov/compound/water) surfaces is not expected based on its Henry’s Law constant. Based on aerobic screening studies, biodegradation is expected to be the most important degradation process. Ethylenediamine may form stable complexes with metal ions (NIH).

### Typical concentrations in drinking-water

NSF (2010) reports the following after testing 112 samples of drinking water, presumably that had been treated with a polyamine polyelectrolyte. Then mean concentration found was <0.001 mg/L, and the range was <0.001 to 0.002 mg/L.

### Health considerations

The acute toxicity of ethylenediamine (LD50, rat, oral range from 637 mg/kg to 1850 mg/kg; LD50, rat, inhalation >29 mg/L and LD50, rabbit, dermal 560 mg/kg) is considered to be low to moderate. Ethylenediamine was rapidly excreted with most of the material eliminated in the urine within 24 hours. The Lowest-Observable-Adverse-Effect-Level (LOAEL) is 100 mg/kg/day with a No-Observable-Effect-Level (NOEL) of 20 mg/kg/day observed in the chronic dietary feeding study (INCHEM 2001).

USEPA (1991) classified ethylene diamine as Class D: not classifiable as to human carcinogenicity.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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WHO. 1999. 1,2-Diaminoethane (Ethylenediamine). *Concise International Chemical Assessment Document (CICAD)* 15. 34 pp. <http://www.who.int/ipcs/publications/cicad/en/cicad15.pdf>.

# Ethylene glycol

CAS No. 107-21-1. Also called ethane-1,2-diol (IUPAC name), 1,2-ethanediol, mono ethyl glycol, glycol alcohol, MEG and monoethylene glycol.

### Maximum Acceptable Value

There is no MAV for ethylene glycol in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Ethylene glycol is primarily used as a raw material in the manufacture of polyester fibres and fabric industry, and polyethylene terephthalate resins (PET) used in bottling. A small percentage is used in industrial products and applications, eg, [antifreeze](http://en.wikipedia.org/wiki/Antifreeze). About 10 million tonnes are produced annually. It is used in the natural gas industry to remove water vapour from natural gas before further processing or delivery in long pipelines.

A major use of ethylene glycol is as a medium for [convective heat transfer](http://en.wikipedia.org/wiki/Convective_heat_transfer), eg, in automobiles and liquid cooled computers. Ethylene glycol is also commonly used in chilled water [air conditioning](http://en.wikipedia.org/wiki/Air_conditioning) systems that place either the chiller or air handlers outside, or systems that must cool below the freezing temperature of water. In [geothermal heating](http://en.wikipedia.org/wiki/Geothermal_heating)/cooling systems, ethylene glycol is the [fluid](http://en.wikipedia.org/wiki/Fluid) that transports heat through the use of a [geothermal heat pump](http://en.wikipedia.org/wiki/Geothermal_heat_pump). It is used as a [de-icing](http://en.wikipedia.org/wiki/De-icing) fluid for aircraft. Ethylene glycol is also used as a [preservative](http://en.wikipedia.org/wiki/Preservative) for biological specimens during [dissection](http://en.wikipedia.org/wiki/Dissection) as a safer alternative to [formaldehyde](http://en.wikipedia.org/wiki/Formaldehyde).

Ethylene glycol has been identified in the edible fungus *Tricholoma matsutake* and has been identified as a metabolite of ethylene, a natural plant growth regulator (WHO 2002).

Releases (untreated) to water have gone up significantly since 1994. Releases then were reported at 91 tonnes, mostly from the paper products and the primary steel industry sectors. A sharp increase in releases occurred in 2003 and continued increases were reported up to 2005. Total 2005 releases to water were reported as 572 tonnes, with oil and gas accounting for 446 tonnes (78 percent). The paper products sector, including pulp mills, reported a significant drop. While this sector was previously reported as the biggest contributor of releases to water, it accounted for only eight tonnes (1.4 percent) in 2005. Iron and steel mills accounted for 44 tonnes (8 percent). The highest reported releases of ethylene glycol to the environment are to land resulting from aircraft deicing/anti-icing operations, with subsequent release to the aquatic environment. However, in recent years, management practices at Canada’s major airports have improved with the installation of new ethylene glycol application and mitigation facilities or improvements to existing ones. In 2005 Canadian airports released 6,745 tonnes of ethylene glycol. The mean concentration in Airport stormwater is usually about 20–25 mg/L (Environment Canada 2014).

Ethylene and propylene glycol concentrations up to 19,000 mg/L (1.9 percent) were detected in stormwater runoff at the Salt Lake City Airport in Utah, airport runoff was found to contain up to 3,100 mg/L (0.3 percent) at the Toronto International Airport in Canada, and up to 5,050 mg/L (0.5 percent) at the Denver Airport in Colorado. The USEPA estimated that 21 million gallons of aircraft deicing fluid (including both ethylene and propylene glycol-based fluids) are discharged to surface waters per year in the United States with an additional two million gallons discharged to publicly owned treatment works. These releases are expected to decrease as source reduction technologies and recycling/recovery systems are improved. Airports that have updated equipment and collection systems have achieved a 70 percent collection efficiency on average. Ethylene glycol that is released onto the ground when used in aircraft de-icing fluid may contaminate nearby groundwater. Groundwater samples collected from a perched water table at the Ottawa Airport in Canada contained 415 mg/L of ethylene glycol. Stated in ATSDR (2010).

### Form and fate in the environment

Once released into the environment, ethylene glycol partitions mainly into surface water or groundwater. It does not bioaccumulate or persist in the environment, primarily due to biodegradation. Half-lifes are estimated to typically range from 0.35 to 3.5 days in air, 2 to 12 days in water, 4 to 24 days in groundwater and 2 to 12 days in soil, but may exceed these ranges, depending on environmental conditions. Ethylene glycol has been found to biodegrade rapidly in the aquatic environment and therefore has the potential to induce depletion of the dissolved oxygen in receiving waters (Environment Canada 2014).

Ethylene glycol has a low vapour pressure (0.089 mm Hg at 25°C) and is miscible with water. If released to the atmosphere (eg, as vapours generated at elevated temperatures), ethylene glycol should exist almost entirely in the vapour phase. The high solubility of ethylene glycol in water ensures that at least partial removal of the compound will occur by wet deposition. The low Henry’s law constant value for this compound (6.0 x 10-8 atm-m3/mole) suggests that ethylene glycol released to surface water will not partition to the atmosphere via volatilisation. Ethylene glycol is not expected to adsorb to sediment or soil particulates based on an estimated Koc value of 0.2–1. Based on the low Koc value, ethylene glycol is expected to have a very high mobility in soil and could leach into groundwater. The low octanol/water partition coefficient (Kow) value of -1.36 suggests that bioconcentration and biomagnification of ethylene glycol are not likely to occur. In river die-away tests ethylene glycol was added to river water at concentrations ≤10 mg/L; it was completely biodegraded after 3 days at 20°C and after 14 days at 8°C. Ethylene glycol appears to be completely degraded within 1–2 weeks under anaerobic conditions (ATDSR 2010).

If released to soil, ethylene glycol is expected to have very high mobility based upon an estimated Koc of 0.2. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon a Henry’s Law constant of 6.00 x 10-8 atm‑cu m/mole. Ethylene glycol is not expected to volatilise from dry soil surfaces based upon its vapour pressure. Ethylene glycol is biodegraded in soil 97–100 percent in 2–12 days. If released into water, ethylene glycol is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. In a river die-away test, degradation was complete within three days at 20°C and 5–14 days at 8°C. Volatilisation from water surfaces is not expected to be an important fate process based on its Henry’s Law constant. A BCF of 10, reported for ethylene glycol in fish, Golden ide (*Leuciscus idus melanotus*), after three days of exposure suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

No data were identified concerning the presence or concentrations of ethylene glycol in drinking-water in Canada or elsewhere (WHO 2002).

### Health considerations

Ethylene glycol has low acute toxicity in experimental animals following oral exposure. Upon ingestion, ethylene glycol is oxidised to [glycolic acid](http://en.wikipedia.org/wiki/Glycolic_acid) (see datasheet) which is, in turn, oxidised to [oxalic acid](http://en.wikipedia.org/wiki/Oxalic_acid), which is toxic (see HPA 2014 for further details). Ethylene glycol and its toxic by‑products first affect the [central nervous system](http://en.wikipedia.org/wiki/Central_nervous_system), then the heart, and finally the kidneys. Ingestion of sufficient amounts (made possible by its sweet taste) can be fatal if untreated. Antifreeze products now tend to use [propylene glycol](http://en.wikipedia.org/wiki/Propylene_glycol) in place of ethylene glycol because propylene glycol possesses an unpleasant taste and is converted in the body to [lactic acid](http://en.wikipedia.org/wiki/Lactic_acid).

The Reference Dose ([RfD](http://www.epa.gov/ttnatw01/hlthef/hapglossaryrev.html#rfd)) for ethylene glycol is 2.0 mg/kg body weight per day based on kidney toxicity in rats. The USEPA has not classified ethylene glycol for carcinogenicity (USEPA 2000).

A tolerable intake of 0.05 mg/kg body weight per day has been derived for this substance, based on a benchmark dose of 49 mg/kg body weight per day calculated for non-neoplastic renal effects in animals and an uncertainty factor of 1,000. However, this tolerable intake is uncertain, owing primarily to lack of information on progression of renal lesions in the most sensitive animal model (WHO 2002).

Environment Canada (2014) derived a tolerable intake of 1.2 mg/kg body weight per day based on a BMD05 of 120 mg/kg-bw/day for the incidence of compound-induced crystal nephropathy in male Wistar rats after dietary exposure to ethylene glycol for 12 months, using 100 as the default uncertainty factor (x 10 for interspecies variation, x 10 for intraspecies variation).

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of:

* 0.8 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.8 mg/kg/day for intermediate-duration oral exposure (15–364 days).

Ethylene glycol has not been carcinogenic in a two-year bioassay in rats and mice, in primarily limited, early bioassays. In the limited number of identified *in vitro* and *in vivo* studies, ethylene glycol has not been genotoxic. Ethylene glycol is teratogenic, inducing primarily skeletal variations and external malformations, sometimes at doses less than those that are maternally toxic, with mice being more sensitive than rats (WHO 2002).

### Derivation of Maximum Acceptable Value

No MAV.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The acute and short-term limits are 4 mg/L; the subchronic and chronic health risk limits are 2 mg/L.

### References

ATSDR. 2010. *Toxicological Profile for Ethylene Glycol*. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine. 305 pp. <http://www.atsdr.cdc.gov/toxprofiles/index.asp>.

EAWAG. Accessed February 2015. *Biocatalysis/Biodegradation Database: Select 1396 compounds*. <http://eawag-bbd.ethz.ch/index.html>.

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# 2-ethylhexanoic acid

CAS No. 149-57-5. Also called 3-heptanecarboxylic acid, 2-ethylcaproic acid, 2‑butylbutanoic acid, 3-heptanecarboxylic acid, EHA, and (rather loosely) hexanoic acid.

### Maximum Acceptable Value

There is no MAV for 2-ethylhexanoic acid in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

This [carboxylic acid](http://en.wikipedia.org/wiki/Carboxylic_acid) is widely used to prepare metal derivatives that are soluble in nonpolar organic solvents. These lipophilic metal-containing derivatives are used as driers and [catalysts](http://en.wikipedia.org/wiki/Catalyst) in [polymerisations](http://en.wikipedia.org/wiki/Polymer) such as in the production of PVC, pharmaceuticals and dyes, and in some pesticide formulations. Another source is from the metabolism of [bis(2-ethylhexyl)phthalate](http://en.wikipedia.org/wiki/Bis(2-ethylhexyl)_phthalate) (DEHP) where the two ester bonds are hydrolysed and the resulting [2-ethylhexanol](http://en.wikipedia.org/wiki/2-Ethylhexanol) is [oxidised](http://en.wikipedia.org/wiki/Oxidized) to 2-ethylhexanoic acid. 2‑Ethylhexanoic acid is also used as a flavouring substance in food categories according to Commission Regulation EC No. 1565/2000 in FGE.06.

2-Ethylhexanoic acid is the active ingredient in the wood preservative agent Sinesto B which is used in New Zealand.

#### 2. From treatment processes

2-Ethylhexanoic acid has been found in drinking-water as an extractant from one or more drinking water system components evaluated under NSF/ANSI 61, or as a contaminant in drinking-water treatment chemicals evaluated under NSF/ANSI 60.

#### 3. From the distribution system

See above.

### Form and fate in the environment

Water solubility is about 25 mg/L.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Contamination with 2-ethylhexanoic acid (2-EHA) was found during studies by Würzburg University in 28 out of 35 samples of baby food and fruit juices for infants. The contamination stemmed from the lids of jars and bottles in which the foods and fruit juices had been packed. Salts of 2-ethylhexanoic acid are used as stabilisers during the production of sealing compounds in order to render the seals thermo-stable. The acid probably comes from these salts (BfR 2004).

2-Ethylhexanoic acid produced signs of liver effects in two-week and subchronic feeding studies in F-344 rats and B6C3F1 mice. These effects included increased absolute and/or relative liver weight, hepatocyte hypertrophy, and changes in cholesterol and triglyceride levels and were most likely due to peroxisome proliferation, which was positively identified. Oral 2-ethylhexanoic acid was a developmental toxin in rats, producing an increased incidence of skeletal malformations and variations, as well as dilated lateral ventricles of the brain with or without tissue compression. Developmental toxicity was specific to the (R)-enantiomer, the (S)-enantiomer being largely inactive when tested in mice.

An oral RfD (oral reference dose) for 2-ethylhexanoic acid has been established at 0.1 mg/kg/d, based on a NOAEL of 100 mg/kg/d following developmental toxicity studies in rats and rabbits. A total allowable concentration has been set at 0.7 mg/L from a developmental toxicity study in rats, for a 70 kg adult drinking 2 L/day with a 20 percent relative source contribution for drinking water; and a short term exposure level of 0.07 mg/L derived from a developmental toxicity study in rats, for a 10 kg child drinking 1 L/day (NSF 2008).

Juberg et al (1997), in feeding studies, found the no-observed-adverse-effect level (NOAEL) for male rats was 61 mg/kg/day and the no-observed-effect level (NOEL) for female rats was 71 mg/kg/day.

### Derivation of Maximum Acceptable Value

No MAV.

### References

BfR. 2004. *2-Ethylhexanoic Acid in Baby Food and Fruit Juices packed in Glass Containers*. 5 pp. www.bfr.bund.de/cm/245/2\_ethylhexanoic\_acid\_in\_baby\_food.pdf.

Juberg, et al. 1997. 2-Ethylhexanoic Acid: Subchronic Oral Toxicity Studies in the Rat and Mouse. *Food and Chemical Toxicology* 36(5): 429–36.

NSF. 2008. *2-Ethylhexanoic Acid, Oral Risk Assessment Document*, NSF International. 1 December: 99 pp. See summary at: <http://www.techstreet.com/standards/NSF/Ethylhexanoic_Acid?product_id=1601225>.

# Ethyl tert-butyl ether

CAS No. 637-92-3. Also called ETBE, ethyl-tertiary-butyl ether, ethyl-t-butylether, tert‑butyl ethyl ether or 2-ethoxy-2-methylpropane.

### Maximum Acceptable Value

There is no MAV for ethyl tert-butyl ether in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

The major use of ethyl tertiary-butyl ether (ETBE) is as a petrol additive (gasoline ether oxygenate or GEO) with production and consumption for this purpose increasing markedly in the 1990s in most parts of the developed world. Because of the relative scarcity of future low priced, domestically produced methanol for MTBE (qv) production and the likely trend toward lower volatility gasoline, ethyl tertiary-butyl ether has emerged as a probable fuel blending candidate for the future. IEH (2014) reports that 2.2 million tonnes of ETBE was consumed within the EU in 2011. Methyl tert-amylether (qv) has been used for some years too (IEH 2014).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

ETBE has been identified in drinking-water in cross-linked polyethylene (PEX) pipes as a major contributor to taste and odour values; panellists could detect MTBE and ETBE at levels as low as 0.005 mg/L; ETBE has been found from 0.023 to 0.2 mg/L (EDAW 2009).

### Forms and fate in the environment

Koc values for GEOs range from 9 (lowest recorded value for ETBE) to 160 (highest recorded value for TAME) indicating very high mobility in soil. In general GEOs exhibit low vapour pressures (<250 mm Hg) and thus may be readily volatilised from dry soils. Henry’s law constants of less than 1 x 10-3 indicate that volatilisation from wet soil is likely to also be a major process in determining environmental fate. No evidence indicating rapid biodegradation of GEOs in soil was found, suggesting that this is not an important fate process. Volatilisation of GEOs from water sources is expected to take three to four days from a modelled lake and three to four hours from a modelled river. GEO Koc values suggest that sorption of GEOs to suspended solids in an aqueous environment is unlikely (from IEH 2014).

Water solubility is >1 percent; API (2007).

### Typical concentrations in drinking-water

Two water utilities in the US reported detecting ethyl-t-butyl ether (ETBE) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0007 mg/L.

### Removal methods

IEH (2014) reports that conventional treatment is ineffective at removing ETBE from water but activated carbon may remove about half.

### Health considerations

The European Commission (EC) European chemical substances information system report an ETBE rat oral LD50 of >2000 mg/kg bw. Rats orally exposed to TAME exhibited increased adrenal weights with a LOAEL of 125 mg/kg/day. Due to the structural similarities between GEOs this LOAEL value has been assumed for ETBE and MTBE. A project specific TDI of 0.125 mg/kg/day was calculated through the application of an uncertainty factor of 1,000 to this LOAEL.

Examination of maximum taste and odour thresholds and TDI values indicated that ETBE is unlikely to exert toxic effects at or below its taste and odour threshold (IEH 2014).

### Derivation of Maximum Acceptable Value

No MAV.

IEH (2014) and DWI (2014) report a taste and odour threshold of 1 to 106 μg/L.

### References

API. 2007. *Technical Protocol for Evaluating the Natural Attenuation of MtBE*. API Publication 4761. 186 pp. <http://www.api.org/environment-health-and-safety/clean-water/ground-water/~/media/15A5D109BB1A44A9AD58EB186A805B3F.ashx>.

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# Fluoranthene

CAS No. 206-44-0. See also the datasheet for polynuclear aromatic hydrocarbons.

### Maximum Acceptable Value

WHO (2011) states that fluoranthene occurs in drinking-water at concentrations well below those of health concern. WHO (2004) had stated that under usual conditions the presence of fluoranthene in drinking-water does not represent a hazard to human health. For this reason, the establishment of a guideline value for fluoranthene is not deemed necessary.

In DWSNZ 2005, the provisional MAV had been 0.004 mg/L (4 μg/L).

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

Fluoranthene is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the US Clean Water Act.

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; benzo[a]pyrene (BaP) levels range from 6 to 35 mg/kg TEQ depending on land use. The equivalent BaP concentration is calculated as the sum of each of the detected concentrations of nine carcinogenic PAHs which are benz(a)anthracene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene and indeno(1,2,3-cd) pyrene, multiplied by their respective potency equivalency factors.

### Sources to drinking-water

#### 1. To source waters

Polynuclear aromatic hydrocarbons (PAHs) are a class of diverse organic compounds containing two or more fused aromatic rings of carbon and hydrogen atoms. They are ubiquitous pollutants formed from the combustion of fossil fuels and are always found as a mixture of individual compounds.

PAHs have been detected in a variety of foods as a result of the deposition of airborne PAHs and in fish from contaminated waters. PAHs are also formed during some methods of food preparation, such as char-broiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient and indoor air.

Owing to their low solubility and high affinity for particulate matter, PAHs are not usually found in water in notable concentrations.

Only a small number of PAHs are produced commercially. Fluoranthene is one of these, it is used as an intermediate in the production of fluorescent dyes.

PAHs in the environment are almost always derived from anthropogenic activities. The largest amount of PAHs enter the environment via the atmosphere from incomplete combustion processes, such as processing of crude oil and coal, industrial use of mineral oil products and coal (including related contaminated soils), heating, fires, incineration of refuse, vehicle traffic, tobacco smoking and volcanic activities. PAHs are also found in bitumen, coal tar and creosote, heating oils, vehicle fuels, lubricating and cutting oils, and printing colour oils.

#### 2. From treatment processes

No known sources.

#### 3. Distribution system

Fluoranthene is the most commonly detected PAH in drinking-water and is associated primarily with coal tar linings of cast iron or ductile iron distribution pipes (not common now in New Zealand).

Observations by one UK water company suggested that the pesticide 2,4‑dichlorophenoxyacetic acid (2,4-D) can be formed as a disinfection by-product in water distribution systems. It noticed that concentrations of 2,4-D increased whilst concentrations of fluoranthene decreased. Studies could not demonstrate the pathway for this to happen. The report also discusses the interaction between chlorine and a range of PAHs (DWI 2015).

### Forms and fate in the environment

PAHs reach the hydrosphere mainly by dry and wet deposition and road runoff but additionally from industrial wastes containing PAHs, and leaching from creosote-impregnated wood. PAHs are adsorbed strongly to the organic fraction of sediments and soils, and hence leaching of PAHs from the soil surface layer to groundwater is assumed to be negligible. However, their presence in groundwater has been reported, mainly at contaminated sites.

In laboratory experiments with soil samples, the calculated half lives for selected PAHs varied widely, from about 100 days to a couple of years. For pure water, the photodegradation half-life appears to be in the range of hours (Mill et al 1981, Mill and Mabey 1985 both cited in WHO 1998), whereas the half life increases dramatically when sediment/water partitioning is taken into account (Zepp and Schlotzhauer 1979, cited in WHO 1998).

The principal route of entry to source water is via atmospheric deposition. Based on the literature reviewed, the presence of chrysene and benzo(k)fluoranthene may be indicators for coal combustion emissions. Whereas other PAHs are indicators of other combustion process: benzo(g,h,i)perylene, coronene and phenanthrene are indicators for motor vehicle emissions, pyrene and fluoranthene are associated with incineration and fluorene, fluoranthene and pyrene are associated with oil combustion (Environment Australia 2003).

In summary, it can be concluded that sediments and soils are the main sinks for PAHs in the environment.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 346 zones, found fluoranthene in one zone at a concentration of 0.0002 mg/L (5 percent of the then MAV), with the median concentration being “nd” (limit of detection = 0.0002 mg/L) (ESR 2001).

Thirteen water utilities in the US reported detecting fluoranthene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0025 mg/L.

### Removal methods

An investigation by the UK Department for Environment (1993) into methods to reduce the rate of leaching of fluoranthene from the coal tar lining of distribution pipes found that of the remedial methods investigated only the partial covering of the lining material with woven nylon hose was shown to be effective.

Specific information about the removal of fluoranthene by coagulation/flocculation is unavailable, however, PAHs are hydrophobic (Faust and Aly 1983) and removal by adsorption to floc is therefore likely to achieve good removal.

Fluoranthene is removed very effectively by adsorption to activated carbon (Faust and Aly 1983).

PAHs in water can be oxidised by chlorination and ozonation. Pyrene was the most rapidly degraded PAH. Benz[a]anthracene, benzo[a]pyrene, and perylene were also highly degraded. Indeno[1,2,3-c,d]pyrene and benzo[g,h,i]pyrene were intermediate with respect to relative degradation. Benzo[k]fluoranthene and fluoranthene were the most slowly degraded of the compounds tested. A variety of complex end-products have been identified (ATSDR 1995).

Oxidation by ozone at neutral and acidic pH values can be achieved (Camel and Bermond 1998).

### Analytical methods

#### Referee method

Liquid-Solid Extraction Gas Chromatographic/Mass Spectrometric Method (EPA 525).

#### Some alternative methods

1. Liquid–Liquid Extraction High Performance Liquid Chromatographic Method (APHA 6440B; EPA 550).

2. Liquid-Solid Extraction High Performance Liquid Chromatographic Method (EPA 550.1).

3. Liquid–Liquid Extraction Gas Chromatographic/Mass Spectrometric Method APHA 6410B).

### Health considerations

PAHs are absorbed in experimental animals and humans through the pulmonary tract, the gastrointestinal tract, and the skin. Oral administration of fluoranthene to rats caused peak concentrations of these compounds in blood after one to two hours. In general, little is known about the metabolism of most PAHs, particularly in non-rodent species. PAH metabolites and their conjugates are excreted predominantly via the faeces and to a lesser extent in the urine. The excretion of urinary metabolites is a method used to assess internal human exposure to PAHs.

#### Acute poisoning

The oral LD50 for fluoranthene in the rat is about 2,000 mg/kg of body weight (Smyth et al 1962, cited in WHO 1998).

#### Chronic exposure

The MAV is determined on the basis of health effects from chronic exposure. Male and female mice were given fluoranthene by gavage for 13 weeks at 0, 125, 250 or 500 mg/kg of body weight per day and then sacrificed and autopsied (USEPA 1988). All treated mice exhibited nephropathy, increased salivation, and increased liver enzyme levels in a dose-dependent manner. At doses of 250 and 500 mg/kg of body weight per day, statistically increased serum glutamate-pyruvate transaminase (SGPT) levels and increased absolute and relative liver weights were noted, as well as compound-related microscopic liver lesions (indicated by pigmentation) in 65 and 87.5 percent of the mice, respectively.

The oral RfD for fluoranthene was calculated at 0.04 mg/kg/d (USEPA 1993).

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.4 mg/kg/day for intermediate-duration oral exposure  
(15–364 days) to fluoranthene.

The USEPA has determined that acenaphthene, anthracene, fluoranthene, fluorene and pyrene are not classifiable as to human carcinogenicity based on no human data and inadequate data from animal bioassays. Fluoranthene is classified by the International Agency for Research on Cancer (IARC) as Group 3: not classifiable as to carcinogenicity to humans. The US Department of Health and Human Services has classified coal tar and coal tar pitches as known to be human carcinogens (quoted in ATSDR 2009).

### Derivation of Maximum Acceptable Value

No MAV.

However, WHO (2003) states:

It is recommended, as before, that the use of coal-tar-based and similar materials for pipe linings and coatings on storage tanks be discontinued; and the monitoring of levels of individual indicator PAHs (including fluoranthene and BaP) and not just total PAHs in drinking-water continue, with the objective of detecting where coal-tar-based linings are deteriorating, so that they can be replaced in a timely manner by new pipes.

The DWSNZ 2005 provisional MAV had been derived as follows: a tolerable daily intake approach has been used for the derivation of the MAV for fluoranthene in drinking-water. The NOAEL was identified on the basis of increased serum glutimate pyruvate transaminase levels, kidney and liver pathology, and clinical and haematological changes, as follows (note that WHO 2011 states that a health-based value of 0.004 mg/L can be calculated on the same basis):

125 mg/kg body weight per day x 70 kg x 0.01 = 0.004 mg/L

2 L x 10,000

where:

* no observable adverse effect level = 125 mg /kg body weight per day for increased serum glutamate–pyruvate transaminase levels, kidney and liver pathology, and clinical and haematological changes in a 13-week oral gavage study in mice
* average weight of adult = 70 kg
* average quantity of water consumed by an adult = 2 L per day
* proportion of tolerable daily intake allocated to drinking-water = 1 percent (because there is significant exposure from food)
* uncertainty factor = 10,000 (100 for interspecies and intraspecies variation, 10 for the use of a subchronic study and inadequate database, and 10 because of clear evidence of co-carcinogenicity with benzo[a]pyrene in mouse skin-painting studies.

The only time that WHO had a guideline value for other than benzo[a]pyrene was in their 1971 International Standards, which stated that some PAHs are known to be carcinogenic and that the concentrations of six representative PAH compounds (fluoranthene, 3,4-benzofluoranthene, 11,12-benzofluoranthene, 3,4-benzopyrene, 1,12-benzopyrene and indeno[1,2,3-cd]pyrene) should not in general exceed 0.0002 mg/L.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit for fluoranthene is 0.3 mg/L. The subchronic limit is 0.2 mg/L, and the chronic limit is 0.07 mg/L.

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# Fluorene

Fluorene, CAS No. 86-73-7, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. Also called 9H-fluorene and o-biphenylmethane.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

The principal route of entry to source water is via atmospheric deposition. Based on the literature reviewed, the presence of chrysene and benzo(k)fluoranthene may be indicators for coal combustion emissions. Whereas other PAHs are indicators of other combustion process: benzo(g,h,i)perylene, coronene and phenanthrene are indicators for motor vehicle emissions, pyrene and fluoranthene are associated with incineration and fluorene, fluoranthene and pyrene are associated with oil combustion (Environment Australia 2003).

Fluorene has been detected in rain, snow and fog samples.

Fluorene is used as a chemical intermediate in many chemical processes, in the formation of polyradicals for resins, and in the manufacture of dyestuffs.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

If released to soil or water, fluorene will biodegrade readily (aerobically) in the presence of acclimated microbes; microbial adaptation is an important fate process. Biodegradation can be slow in pristine soils or waters (or under conditions of limited oxygen). Strong adsorption to soil and water sediment is an important transport process; fluorene has been detected in numerous, widespread sediment samples. The half-life of fluorene in soil has been reported to range from 2 to 64 days (EAWAG accessed February 2015).

Water solubility is about 2 mg/L.

### Typical concentrations in drinking-water

Twelve water utilities in the US reported detecting fluorene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.009 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation (ATSDR 1995).

### Analytical methods

See polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

The US Environmental Protection Agency has determined that acenaphthene, anthracene, fluoranthene, fluorene and pyrene are not classifiable as to human carcinogenicity based on no human data and inadequate data from animal bioassays.

IARC (2010) classified fluorene in Group 3 (not classifiable as to carcinogenicity).

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) has developed oral minimal risk levels (MRLs) for some PAHs:

|  |  |  |
| --- | --- | --- |
| **PAH** | **mg/kg/day** | **duration** |
| fluorene | 0.4 | intermediate (15–364 days) |

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA has a reference dose or RfD of 0.04 mg/L for fluorene and a Drinking Water Equivalent Level or DWEL of 1 mg/L.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limits (exposure greater than 10 percent of a lifetime) for fluorene is 0.3 mg/L.

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# Formaldehyde

CAS No. 50-00-0. Also called methanal (IUPAC), or as an aqueous solution: formalin. Has been called methylene oxide, methyl oxide, formic aldehyde, methyl aldehyde, oxomethane, oxymethylene, and various trade names. In solid form, formaldehyde is marketed as trioxane (CH2O)3, and its polymer, paraformaldehyde, with 8 to 100 units of formaldehyde.

### Maximum Acceptable Value

It is not considered necessary to set a formal guideline value for formaldehyde in view of the significant difference between the expected concentrations of formaldehyde in drinking-water and the tolerable concentration.

In DWSNZ 2005, the MAV had been 1 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of formaldehyde in drinking water should not exceed 0.5 mg/L.

Health Canada (2003) states that it is not considered necessary to establish a maximum acceptable concentration (MAC) for formaldehyde in drinking water.

The USEPA concluded on 22 September 2009 that formaldehyde is known or anticipated to occur in PWSs and may require regulation. Therefore they added formaldehyde to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a). The USEPA (2006/2011) established a lifetime health advisory of 1 mg/L, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

### Sources to drinking-water

#### 1. To source waters

Formaldehyde may enter raw water from a great variety of human activities. Its primary industrial use is in the production of urea-formaldehyde, phenolic, melamine, pentaerythritol, and polyacetal resins and glues used in the building industry. It is also used in the production of intermediates in the chemical industry, such as acetylene chemicals and hexamethylene, and in cosmetics, fungicides, textiles and embalming fluids (embalming fluids may contain 5 to 30 percent formaldehyde).

Formaldehyde is a colourless, highly flammable gas that is sold commercially as  
30–50 percent (by weight) aqueous solutions. Methanol or other substances are usually added to the solution as stabilisers, to reduce polymerisation. Formaldehyde has been used for many years in consumer goods to deter spoilage caused by microbial contamination. It has been used as a preservative in household cleaning agents, dishwashing liquids, fabric softeners, shoe-care agents, car shampoos and waxes, and carpet-cleaning agents. In addition, reported use of formaldehyde in fish farming and in animal husbandry may lead to a significant local environmental exposure. Generally, the formaldehyde content in these products is less than 1 percent.

Formaldehyde appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2015 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)) as a antimicrobial.

Formaldehyde is also a component of diesel exhaust (motor vehicles are probably the largest direct human source of formaldehyde in the environment) and is a product of forest fires. Trucks burning diesel have been reported to produce 0.2 to 32 mg of formaldehyde per km (Environment Australia 2003).

Rainwater contains 0.11–0.17 mg/L formaldehyde, with peaks as high as  
0.31–1.38 mg/L (IPCS 1991).

Concentrations of formaldehyde in drinking water range from 20 to <100 μg/L; in groundwater from <50 to 690 000 μg/L; in fresh surface water from ≤9 to 133 μg/L; in marine surface water it has not been detected, and in effluents levels range from detected but not quantified to 325 μg/L (DWI 2014).

#### 2. From treatment processes

Formaldehyde is produced as a disinfection by-product through the reaction between ozone and naturally-occurring organic substances, such as humic and fulvic acids. For three plants in the US using ozone, formaldehyde levels in influent ranged from <1.0 to 3.2 µg/L, while concentrations as high as 31 µg/L were detected in the ozonated drinking water. It can also result from chlorination and ozonation of humic matter. Stockham and Morran found formaldehyde in water treated with polyDADMAC after ozonation. Awad et al (1993) observed the formation of formaldehyde, glyoxal, and acetaldehyde, and the reduction of 8 to 12 carbon hydrocarbons when irradiating reclaimed wastewater with UV from low pressure mercury arc lamps. Formaldehyde increased from a background level of 0.0035 mg/L to 0.006 and 0.01 mg/L after an applied dose of 45 and 147 mWs/cm2. Similar reactions may occur in fresh water when using UV disinfection.

#### 3. From the distribution system

Leaching of formaldehyde may occur from polyacetal plastic fittings if these are used in the distribution system or plumbing. In water lines, an interior protective coating generally separates the water from the polyacetal resin. If a break occurs in the coating, however, the water may come in direct contact with the resins, resulting in a continuous release of formaldehyde into the water via hydrolysis of the resin surfaces. The resultant concentrations of formaldehyde vary depending on the residence time of water in the pipes; levels may approximate 20 µg/L in occupied dwellings with normal water usage or reach 100 µg/L in unoccupied dwellings or after a few days of no water usage (Health Canada 2003).

### Forms and fate in the environment

If released to soil, formaldehyde is expected to have very high mobility based upon an estimated Koc of 37. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon a Henry’s Law constant of 3.4 x 10-7 atm‑cu m/mole. Formaldehyde volatilises from dry soil surfaces because it is a gas. If released into water, formaldehyde is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Formaldehyde readily biodegrades under both aerobic and anaerobic conditions in the environment. Formaldehyde in aqueous effluent was degraded by activated sludge and sewage in 48-72 hr. In a die-away test using water from a stagnant lake, degradation was complete in 30 and 40 hours under aerobic and anaerobic conditions, respectively. Volatilisation from water surfaces is not expected to be an important fate process based on its Henry’s Law constant. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Formaldehyde is not expected to undergo hydrolysis in the environment because of the lack of hydrolysable functional groups (EAWAG accessed February 2015).

Formaldehyde is a reactive, extremely water soluble, colourless gas. In surface water it is hydrated and found largely in the form of methylene glycol and its oligomers, including low molecular mass poly(oxymethylene)glycols HO(CH2O)nH (n = 1–8). It is expected to biodegrade to low levels in a few days (slightly slower in anaerobic conditions). It is expected to oxidise to formic acid, and may polymerise to paraformaldehyde. Highly soluble in water: about 50 percent. Formaldehyde has a log Kow of 0.35, log Koc of 0.7 to 1.6, and a Henry’s law constant of 3.27 × 10-7atm·m3/mol.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from four zones (supplies using ozone treatment), has not found any formaldehyde at detectable concentrations (limit of detection = 0.01 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with formaldehyde at >50 percent of the MAV (ESR 2001).

Concentrations of up to 0.03 mg/L have been found in ozonated drinking-water (WHO 2005/2011).

Three water utilities in the US reported detecting formaldehyde in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 48 mg/L.

### Removal methods

Formaldehyde is reported to be rapidly hydrolysed to glycol. Therefore it is not expected to persist in raw water. There is little information available on methods of removing formaldehyde from water if it is present in the source water through contamination.

Based on a Henry’s Law constant of 3.37 x10-7 atm.m³/mol at 25°C, volatilisation from water surfaces is not expected to be significant, and therefore, formaldehyde is unlikely to undergo substantial removal by air stripping.

However, as formaldehyde arises in waters predominantly as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the ozone. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all other treatment steps, ie, avoiding preoxidation wherever possible.

Pilot studies have shown that biologically active media are able to reduce the concentration of formaldehyde in ozonated water. Biologically active media in conjunction with preozonation is therefore likely to offer a means of oxidising organic matter and removing the by-products of ozonation.

Drinking-water treated with ozone is unlikely to contain formaldehyde at concentrations exceeding 0.05 mg/L, so will be a minor source of exposure. It is uncertain whether boiling will have a significant impact on the concentration of formaldehyde in drinking-water. Formaldehyde is considered to be highly soluble in water, which suggests it will be very unlikely to volatilise from water.

### Analytical methods

#### Referee method

Dinitrophenylhydrazine Derivatization and High Performance Liquid Chromatography (EPA 554).

#### Some alternative methods

Gas chromatography with electron capture detector (APHA 6252B; EPA 556).

### Health considerations

The general population is exposed to formaldehyde mainly by inhalation, with 20-a-day smokers receiving about 1 mg/day by this route. People are also exposed in food, from the use of urea–formaldehyde foam and pressed wood products in housing, and from the use of cosmetics containing formaldehyde.

Formaldehyde is present in almost all common foods (with the highest concentrations occurring in some fruits and marine fish), and adult dietary intake is estimated at 11 mg/day. Drinking water would contribute less than 10 percent of total intake.

Ingested formaldehyde is absorbed readily by the gastrointestinal tract. It is metabolised rapidly to formic acid and subsequently to carbon dioxide and water. It is distributed primarily in muscle, with lower levels in the intestines, liver and other tissues.

Exposure of the skin to formaldehyde at levels higher than those encountered in drinking-water has been associated with irritation and allergic contact dermatitis. The presence of formaldehyde in some types of water filters has been associated with outbreaks of haemolytic anaemia in dialysis unit patients.

A number of epidemiological studies have looked at the effects of inhalation of formaldehyde. No effects could be directly attributed to long-term occupational exposure, but studies among exposed workers have reported elevated incidences of a number of cancers including nasal, buccal, nasopharyngeal, skin, prostate and colon cancers.

There was no evidence of tumour-promoting activity when formaldehyde was applied to mouse skin, but rats and mice exposed to formaldehyde by inhalation exhibited an increased incidence of carcinomas of the nasal cavity. Ingestion of formaldehyde in drinking-water for two years caused stomach irritation in rats.

Formaldehyde has demonstrated mutagenic activity when applied to cells *in vitro* but not when applied *in vivo*.

Formaldehyde interacts with proteins, DNA and RNA *in vitro*. DWI (2014) states that overall, the data indicate that formaldehyde is genotoxic *in vitro.* The data from *in vivo* studies indicate that formaldehyde is genotoxic, but only in the tissues of initial contact.

There is some evidence that formaldehyde is a carcinogen in humans exposed by inhalation, although there is little evidence that formaldehyde is carcinogenic by the oral route. In 2004 the International Agency for Research on Cancer (IARC 2006) reclassified formaldehyde from Group 2A (probably carcinogenic to humans) to Group 1 (carcinogenic to humans), by inhalation. The weight of evidence indicates that formaldehyde is not carcinogenic by the oral route.

This chemical (in the gaseous form) appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of:

* 0.3 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.2 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD (USEPA 1990/2006/2009/2011) is 0.2 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 7 mg/L.

DWI (2014) states that for Repeat Oral Dose Toxicity and Carcinogenicity a NOAEL of 260 mg/L (reported to be 15 mg/kg bw/day) was identified based on histopathological changes in the gastrointestinal system due to irritancy. Based on this, WHO derived a tolerable concentration of 2.6 mg/L. The WHO concluded that due to the high reactivity of formaldehyde, effects in the tissue of first contact following ingestion are more likely to be related to the concentration of the formaldehyde consumed rather than total intake.

### Derivation of Maximum Acceptable Value

No MAV.

Owing to formaldehyde’s high reactivity, effects in the tissue of first contact following ingestion are more likely to be related to the concentration of the formaldehyde consumed than to its total intake (WHO 2011). IPCS (2002) and repeated in WHO (2017) established a tolerable concentration of 2.6 mg/L (3 mg/L for 70 kg body weight) for ingested formaldehyde based on the NOEL (no-observed-effect-level) of 260 mg/L for histopathological effects in the oral and gastric mucosa of rats administered formaldehyde in their drinking-water for two years, using an uncertainty factor of 100 (for inter- and intraspecies variation).

Although a health-based value could be derived on the basis of this tolerable concentration, it is not considered necessary to set a formal guideline value for formaldehyde in view of the significant difference between the expected concentrations of formaldehyde in drinking-water and the tolerable concentration.

The DWSNZ 2005 MAV had been derived as follows: although formaldehyde is considered to be probably carcinogenic to humans by inhalation, the weight of evidence indicates that formaldehyde is not carcinogenic by the oral route. Therefore a tolerable daily intake approach has been used for the derivation of a MAV for formaldehyde in drinking-water. A no-observable-adverse-effect level determined from a two-year study in rats has been used as the basis of the derivation.

15 mg/kg body weight per day x 70 kg x 0.2 = 1.05 mg/L (rounded to 1 mg/L)

2 L x 100

where:

* no-observable-adverse-effect level = 15 mg/kg body weight per day from a two-year drinking-water study in rats (for a variety of effects, including increased relative kidney weights in females and an increased incidence of renal papillary necrosis in both sexes)
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 100 (for intra- and interspecies variation). No account was taken of potential carcinogenicity from the inhalation of formaldehyde from various indoor water uses, such as showering.

The USEPA has a draft Lifetime Health Advisory for formaldehyde in drinking-water of 1 mg/L.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for formaldehyde is 1 mg/L.

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# Furan

CAS No. 110-00-9. Also called divinylene oxide, 1,4-epoxy-l,3-butadiene, furfuran, and oxacyclopentadiene.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for furan. The WHO Guidelines do not have a guideline value for furan.

### Sources to drinking-water

#### 1. To source waters

Furan, a highly volatile cyclic ether, occurs in oils obtained by the distillation of pine wood containing rosin. It has also been identified in volatile emissions from sorb trees.

Furan is an intermediate in the manufacture of tetrahydrofuran, pyrrole and thiophene and in the formation of lacquers and solvent for resins. It is also used in the production of pharmaceuticals, agricultural chemicals and stabilisers.

Furan has been detected in one of 63 industrial effluents at <0.01 mg/L, and during the combustion of coal, and in engine exhausts, wood smoke and cigarette smoke.

### Forms and fate in the environment

If released to soil, furan is expected to have high mobility based on its estimated Koc of 80. Volatilisation from moist soil surfaces is expected to be an important fate process based on a Henry’s Law constant of 5.4 x 10-3 atm‑cu m/mole. Furan may volatilise from dry soil surfaces based on its vapour pressure. Using the Japanese MITI test, 4 percent of the theoretical BOD was reached in 8 weeks indicating that biodegradation is not an important environmental fate process. If released into water, furan is expected to adsorb to suspended solids and sediment based on its estimated Koc. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 2.5 hours and 3.3 days, respectively. BCFs of 0.9–1.5 and <3.2–13 measured in fish at furan concentrations of 1 and 0.1 mg/L, respectively, suggest bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since furan lacks functional groups that hydrolyse under environmental conditions (NIH).

Octanol:water partition coefficient (P): log P, 1.34 (IARC 1995). Water solubility 1 percent at 25°C.

### Health considerations

The major route of exposure to furan in the human population is through consumption of heat-treated foods and beverages. Furan is found in heat-treated commercial foods and is produced through [thermal degradation](https://en.wikipedia.org/wiki/Thermal_degradation) of natural food constituents. Notably, it can be found in roasted coffee, instant coffee, and processed baby foods. WHO (2011) lists the range of concentrations for several foods.

Furan is mutagenic in animals and is reasonably anticipated to be a human carcinogen based on evidence of carcinogenicity in experimental animals.

Repeated administration of furan to mice and rats leads to liver necrosis, liver-cell proliferation and bile-duct hyperplasia; in rats, prominent cholangiofibrosis develops. The IARC (1995) classified furan as possibly carcinogenic to humans, Group 2B.

Furan is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

The European Commission asked EFSA for a scientific evaluation on the risk to human health of the presence of furan and methylfurans (2-methylfuran, 3-methylfuran and 2,5-dimethylfuran) in food. They are formed in foods during thermal processing and can co-occur. Furans are produced from several precursors such as ascorbic acid, amino acids, carbohydrates, unsaturated fatty acids and carotenoids, and are found in a variety of foods including coffee and canned and jarred foods. Regarding furan occurrence, 17,056 analytical results were used in the evaluation. No occurrence data were received on methylfurans. The highest exposures to furan were estimated for infants, mainly from ready-to-eat meals. Grains and grain-based products contribute most for toddlers, other children and adolescents. In adults, elderly and very elderly, coffee is the main contributor to dietary exposure. Furan is absorbed from the gastrointestinal tract and is found in highest amounts in the liver. It has a short half-life and is metabolised by cytochrome P450 2E1 (CYP2E1) to the reactive metabolite, cis-but-2-ene-1,4-dialdehyde (BDA). BDA can bind covalently to amino acids, proteins and DNA. Furan is hepatotoxic in rats and mice with cholangiofibrosis in rats and hepatocellular adenomas/carcinomas in mice being the most prominent effects. There is limited evidence of chromosomal damage in vivo and a lack of understanding of the underlying mechanism. Clear evidence for indirect mechanisms involved in carcinogenesis include oxidative stress, gene expression alterations, epigenetic changes, inflammation and increased cell proliferation. The CONTAM Panel used a margin of exposure (MOE) approach for the risk characterisation using as a reference point a benchmark dose lower confidence limit for a benchmark response of 10 percent of 0.064 mg/kg body weight (bw) per day for the incidence of cholangiofibrosis in the rat. The calculated MOEs indicate a health concern. T his conclusion was supported by the calculated MOEs for the neoplastic effects (EFSA 2017).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Galaxolide

CAS No. 1222-05-5. The IUPAC name for galaxolide is 4,6,6,7,8,8-hexamethyl-1,3,4,6,7,8-hexahydrocyclopenta[*g*]isochromene. Also called hexahydrohexamethyl cyclopentabenzopyran or HHCB. Galaxolide is a trade name. It is a mixture of isomers.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for galaxolide and the WHO Guidelines do not have a guideline value.

### Sources to drinking-water

#### 1. To source waters

Galaxolide has become the key synthetic musk ingredient belonging to the polycyclic musk group (see also tonalide datasheet).

HHCB is used as an ingredient in commercial preparations intended to be used to fragrance a wide variety of consumer products such as perfumes, cosmetics, household and laundry cleaning products and air fresheners. The level of HHCB in such preparations is typically at a level of several percent. The principal exposure to galaxolide from household products can be considered to be via the skin. The reasonable maximum use levels in household cleaning products ranges from 0.02 to 0.9 percent. The consumer aggregate exposure to HHCB from the use of household cleaning products has been estimated to be a maximum 0.07 μg/kg bw/day (HERA 2004).

Galaxolide contamination has been detected in the Great Lakes. In a study of lake sediment in Lake Erie found galaxolide levels to be doubling every 8 to 16 years. Galaxolide was detected in 92 percent of water samples from Lake Michigan.

From more than 6000 data points for HHCB in surface water (collected by the USGS from locations in 46 states), the mean calculated value for HHCB concentration in surface water was <1.1 μg/L for all sites; the highest concentrations were measured at sewage outfall sites. The 95th percentile groundwater concentrations at well sites and surface water concentrations at streams and lakes, reservoirs, and impoundment sites were ≤0.35 μg/L (USEPA 2014).

### Forms and fate in the environment

If released to soil, galaxolide is expected to be immobile based on an estimated Koc of 38,600. Volatilisation from moist soil surfaces is expected to be an important fate process based on an estimated Henry’s Law constant of 1.3 x 10-4 atm‑cu m/mole. Adsorption to soil is expected to attenuate volatilisation. A biodegradation half-life in soil of four months indicates that biodegradation is not an important environmental fate process in soil. If released into water, galaxolide is expected to adsorb to suspended solids and sediment based on the estimated Koc. Biodegradation is not an important environmental fate process in water. Volatilisation from water surfaces is expected to be an important fate process based on its estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 18 hours and 10 days, respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. The estimated volatilisation half-life from a model pond is 31 months if adsorption is considered. A BCF of 1,584 suggests bioconcentration in aquatic organisms is very high. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions. NIH.

HHCB is moderately persistent in soil and sediment, has low mobility in soil, suboptimal removal in wastewater treatment plants with removal primarily through sorption to sludge. Published concentrations of HHCB in biosolids ranged from <100 to >100,000 μg/kg dw. The octanol:water partition coefficient = logKow is about 5.5 (USEPA 2014).

Water solubility is 1.7 mg/L at 25°C. Octanol:water partition coefficient = log P, 5.9 (NIH).

### Typical concentrations in drinking-water

Galaxolide was tested for in 24 samples from within a drinking-water treatment facility and from two streams that serve the facility located in a heavily populated, highly urbanised drainage basin in order to assess treatment efficiency. The frequency of detection (detection limit = 0.5 ug/L) of galaxolide was 92 percent of samples, with the highest level of detection in finished water at 0.082 ug/L. NIH.

### Removal methods

Treatment processes that remove particulate matter should reduce the concentration of galaxolide in water.

### Health considerations

HHCB has a low acute toxicity either by the oral or dermal route. In a 90-day study in rats, there were no adverse effects at the highest dose tested, 150 mg/kg bw/day. In a well-conducted 90-day oral study, a NOAEL of 150 mg/kg bw/day for HHCB in rats can be concluded. There were no indications of effects on fertility or the developing foetus at levels as high as 50 mg/kg bw/day. There were no effects on rat pups exposed via the milk during nursing to levels of HHCB over 100 times the maximum level found in human milk samples. HHCB is a non-genotoxic substance. The mutagenicity data and the repeated dose studies with HHCB do not indicate a concern with regard to carcinogenicity nor does HHCB possess any structural features that would raise a concern. HHCB has been reported to have a very weak estrogenic potency *in vitro* but such effects are not seen *in vivo*. HHCB is thus not considered to produce endocrine disruption *in vivo*. A German study of human milk found HHCB up to 1316 μg/kg of fat with a mean of 80 μg/kg. Based on the reported mean fat level of 3.67 percent, this corresponds to a maximum level in the whole milk of 48 μg/kg milk (ppb) with a mean of 2.9 ppb (HERA 2004).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Geosmin

CAS No. 16423-19-1. Geosmin is the common name for trans-1,10-dimethyl-trans-9-decalol, also called [4S-(4α,4aα,8aβ)]-octahydro-4,8a-dimethyl-4a(2H)-naphtalenol. Geosmin exists as the (+) and (-) enantiomers.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for geosmin. The WHO Guidelines do not have a guideline value for geosmin.

### Sources to drinking-water

#### 1. To source waters

Geosmin, described variously as causing a musty, earthy mouldy, odour in water, is a metabolite from biological activity of many micro-organisms such as fungi, actinomycetes, streptomycetes, cyanophytes and phytoplankton. The organisms most frequently linked to these taste and odour problems have been the actinomycetes and several genera of the blue-green algae (cyanobacteria). The most common documented taste and odour producing blue-green algae include *Oscillatoria spp., Aphanizomenon spp., Anabaena spp., and Microcystis spp.* Geosmin odours are usually thought to be related to the death and lysis of the cells.

Geosmin is the distinct smell that soil gives off when it is disturbed or on which it has just rained. Geosmin has been detected by the human nose as low as 10 ng/L. Odour outbreaks are caused by biological production of the naturally occurring (−) enantiomers which are some 10 times more potent than the (+) molecules. Geosmin has frequently been detected in lake and river water samples at concentrations ranging from 5 to 20 ng/L (0.000005 to 0.00002 mg/L), with maxima exceeding 120 ng/L, mainly during summer. Usually these tastes and odours are mildly unpleasant, but on occasion the water can become undrinkable by a segment of the population. Westerhoff (2002) discusses source water monitoring and control measures. Young et al (1996) report the taste and odour thresholds in drinking-water at around 0.0000075 and 0.0000013 mg/L respectively.

#### 2. From treatment processes

None.

#### 3. From the distribution system

None reported.

### Forms and fate in the environment

Geosmin and 2-MIB are relatively stable to chemical and biological degradation and can persist in the open water in the dissolved form for some time.

### Removal methods

Activated carbon can be effective at removing geosmin from water but humic substances can reduce the efficacy (Ng et al 2002; Srinivasan et al 2008). Some carbons may achieve as much as 80 percent removal, others less than 10 percent. Dose rates may need to exceed 20 mg/L, and contact times may need to exceed two hours. Treatment with ozone followed by biological filtration has also proved effective, although ozone on its own is usually not (depending on what else is in the water). Ozone combined with UV may need a high dosage.

A dose of around 10 mg/L of chlorine dioxide may reduce the geosmin concentration by about 50–60 percent, but at that dose, chlorite is likely to be excessive. Chlorine and ozone may reduce the concentration of geosmin but by less than 40 percent, even when dosed at more than 20 mg/L, and potassium permanganate was ineffective (Faust and Aly 1998).

MIB and geosmin can be removed through biologically active sand filters. Experiments were conducted using laboratory sand filter columns using sand taken from South Australian water treatment plants. Sand with a well-established biofilm taken from a 26-year-old filter was capable of removing MIB and geosmin to below detection limit after 11 days of operation at an Empty Bed Contact Time (EBCT) of 15 minutes. Sand without an established biofilm removed 60 percent geosmin and 40 percent MIB after 154 days of operation at 15 minutes EBCT (McDowell et al 2007).

### Analytical methods

#### Referee method

Not needed.

#### Some alternative methods

Various GC/MS methods are used, measuring down to about 1 ng/L, eg, Palmentier and Taguchi (2001).

### Health considerations

Geosmin does not present a health risk in drinking-water.

### Derivation of Maximum Acceptable Value

No MAV.

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Environment Agency. 1998. The assessment of taste, odour and related aesthetic problems in drinking waters 1998. *Methods for the Examination of Waters and Associated Materials*. EA Standing Committee of Analysts, London. Available at: <http://www.environment-agency.co.uk/static/documents/Research/171_taste_odour_in_water.pdf>.

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Srinivasan R, Sorial GA, Ononye G, et al. 2008. Elimination of persistent odorous compounds from drinking water. *Water Science and Technology: Water Supply* 8(2): 121–7. See www.iwaponline.com/ws/00802/0121/008020121.pdf.

Westerhoff, et al. 2002. *Guidance Manual for Reducing 2-Methylisoborneol (MIB) and Geosmin in the Metropolitan-Phoenix Area Water Supply*. Arizona State University, City of Phoenix. www4.eas.asu.edu/pwest/myweb/Taste%20and%20Odor%20Stuff/Guidance%20document%20-%20August%202002.pdf.

Young WF, Horth H, Crane R, et al. 1996. Taste and odor threshold concentrations of potable water contaminants. *Water Research* 30: 331–40.

# Glutaraldehyde

CAS No. 111-30-8. The IUPAC name is pentane-1,5-dial. Also called 1,5-pentanedial, 1,3-diformylpropane, 1,5-pentanedione and glutaric aldehyde; plus a host of trade names.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for glutaraldehyde. The WHO Guidelines do not mention glutaraldehyde.

### Sources to drinking-water

#### 1. To source waters

Glutaraldehyde, a aliphatic dialdehyde, is a disinfectant, sanitiser, biocide (effective at pH 6.5 to 9.0), used as a biocide in sanitary solutions for aircraft and portable toilets, fungicide, microbiocide, tuberculocide, and virucide antimicrobial chemical. It is compatible with chlorine. It is also used in the tanning industry, and is sometimes a component of embalming fluid. Glutaraldehyde is used mainly as an aqueous solution, ranging in concentration from 50 percent w/w to less than 1 percent w/w.

As an antimicrobial agent, glutaraldehyde is applied to various sites, including food handling and food storage establishments such as commercial egg hatcheries, poultry/livestock equipment and processing premises, animal feeding and watering equipment; commercial/industrial buildings and trucks, construction materials, and laundry equipment; oil recovery drilling muds and secondary oil recovery injection water; metalworking fluids; commercial/industrial evaporative condensers and heat exchanger water systems; hospital, dental, veterinary and laboratory premises/equipment; non-critical hospital plastic and rubber items; industrial coatings; and in the manufacture of a variety of materials as a preservative: cleaners, adhesives, paper and paperboard, water based coatings, latex paints, inks and dyes.

In cooling towers and other water recirculating systems glutaraldehyde is used to prevent corrosion and the build-up of microbial growth. The solution is administered in slugs as shock kill doses, either manually or by use of automatic dosing equipment, to give 50–125 mg/L glutaraldehyde in treated water.

Glutaraldehyde tends to polymerise in solution, particularly as the pH increases towards 9, with differing proposals advocated for its chemical composition in solution. It has been reported that commercial glutaraldehyde may contain numerous species, including oligomers, unsaturated derivatives and cyclic aldehydes. Some glutaraldehyde-containing products contain other chemicals, for example, disinfectants activated with sodium bicarbonate and x-ray film developers containing sodium bisulfite (NICNAS 1994).

Glutaraldehyde’s main application, as a cold disinfectant for use in such facilities as hospitals, surgeries and medical clinics, entails discharge of significant quantities to sewer as solutions that are disposed of retain at least 50 percent of their activity. Significant degradation is expected during passage through sewage treatment works. Reaction with proteins present in sewage effluent will also remove significant amounts from aqueous waste streams. Any glutaraldehyde that may enter receiving waters is likely to be rapidly diluted and undergo further biodegradation.

Glutaraldehyde-containing products are also approved for use in aquatic areas such as ponds, flood water, and sewage water. It is not registered for any direct food uses.

### Forms and fate in the environment

When glutaraldehyde is introduced into the environment, it is most likely to remain in the aquatic compartment, given the small air/water partition and soil/water partition coefficients. Aquatic metabolism, under aerobic and anaerobic conditions, and aerobic soil metabolism are major routes of dissipation of glutaraldehyde. The calculated aerobic and anaerobic pseudo first-order half-lifes of glutaraldehyde in flooded river sediment are 10.6 and 7.7 hours , respectively. In abiotic conditions, this can extend to 100 days. Glutaraldehyde meets the OECD criteria for classification as readily biodegradable in freshwater environments and as having the potential to be biodegradable in marine environments. In addition, the metabolism of glutaraldehyde is rapid and proceeds via the formation of glutaric acid as an intermediate to complete mineralisation. Because of its biodegradation, glutaraldehyde is not likely to contaminate surface and ground waters.

Glutaraldehyde is miscible with water, and it is often sold as a 50 percent solution.

### Typical concentrations in drinking-water

There are no antimicrobial uses associated with glutaraldehyde that are expected to significantly impact either surface or groundwater resources (USEPA 2007).

### Analytical methods

#### Referee method

Not needed.

#### Some alternative methods

None needed.

### Health considerations

Glutaraldehyde acts by reacting with the free amino groups of some proteins that are located in the cell walls and membranes of micro-organisms. This reaction leads to cross-linking. Cross-linked microbial cells cannot transport nutrients or perform any critical metabolic functions. Glutaraldehyde also deactivates various membrane-bound enzymes. The kinetics of the cross-linking mechanism is influenced by the pH, the contact time, the glutaraldehyde concentration and the temperature. In viruses, the main targets for glutaraldehyde are nucleic acid, proteins and envelope constituents (ECHA 2014).

For the use of glutaraldehyde in cosmetics, the average daily exposure from extensive use was estimated at 0.037 mg/kg/day. The critical NOAEL (for maternal toxicity and reproductive effects) is 15 mg/kg/day, giving a safety margin of 15/0.037 = 405, so the use of glutaraldehyde in cosmetics is of low concern (OECD 1998).

The USEPA concluded that glutaraldehyde was “not likely to be carcinogenic to humans” by any route of exposure. Glutaraldehyde is considered non mutagenic or genotoxic.

The chronic RfD is 0.16 mg/kg/d, based on a NOAEL of 16.1 mg/kg/d derived from a carcinogenicity study (drinking water) in the rat using an uncertainty factor of 100 (USEPA 2007). A LOAEL was also reported, 61 mg/kg/d, based on increases in non-neoplastic lesions (squamous metaplasia, foreign body granuloma, pirulent inflammation) of the respiratory tract and erosion/ulceration in the mucosa of the glandular stomach.

As at July 2017 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for glutaraldehyde of:

0.1 mg/kg/day for chronic-duration oral exposure.

### Derivation of Maximum Acceptable Value

No MAV.

### References

ECHA. 2014. *Biocidal Products Committee (BPC) Opinion on the application for approval of the active substance: Glutaraldehyde*. Product type: 3. ECHA/BPC/020/2014. 10 pp. <http://echa.europa.eu/documents/10162/fb0dd989-af73-434a-999d-32f6bfe3766e>.

NICNAS. 1994. Glutaraldehyde. *Priority Existing Chemical* 3. Full Public Report. Australian Government. 190 pp. http://nicnas.gov.au/Publications/CAR/PEC/PEC3.asp.

OECD. 1998. *SIDS Initial Assessment Report*: Glutaraldehyde. 83 pp. See: http://www.inchem.org/documents/sids/sids/111308.pdf or <http://www.inchem.org/pages/sids.html>.

OSH. 1992. *The Safe Occupational Use of Glutaraldehyde in the Health Industries*. Department of Labour, Wellington, 22 pp. <http://www.osh.dol.govt.nz/order/catalogue/pdf/glutaral.pdf>.

USEPA. 2007. Reregistration Eligibility Decision for Glutaraldehyde. EPA 739-R-07-006. 79 pp. <http://www.epa.gov/pesticides/reregistration/status.htm>.

# Glycidol

CAS No. 556-52-5. The IUPAC name is 2,3-epoxypropan-1-ol. The CAS name is oxiranemethanol. It can also be called allyl alcohol oxide, 3-hydroxypropylene oxide, epihydrin alcohol, 1,2-epoxy-3-hydroxypropane, 2,3-epoxy-1-propanol and glycidyl alcohol.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for glycidol. The WHO Guidelines do not mention glycidol.

NSF Standard 60 states that the allowable concentration of glycidol in drinking water is 0.001 mg/L.

### Sources to drinking-water

#### 1. To source waters

Glycidol is an [organic compound](http://en.wikipedia.org/wiki/Organic_compound) containing [epoxide](http://en.wikipedia.org/wiki/Epoxide) and [alcohol](http://en.wikipedia.org/wiki/Alcohol) [functional groups](http://en.wikipedia.org/wiki/Functional_group). Glycidol is not known to occur as a natural product. It is a clear, slightly viscous liquid that has a variety of industrial uses, eg, as an intermediate in the production of several other chemicals, as an additive for synthetic hydraulic fluids, as a reactive diluent in some epoxy resin systems, stabiliser in manufacture of vinyl polymers, additive for oil and synthetic hydraulic fluids, and as a sterilant in pharmaceuticals.

A related chemical (glycidaldehyde CAS No. 765-34-4) has been used as a cross-linking agent for the finishing of wool, for the oil tanning and fat liquoring of leather and surgical sutures (IARC 1999).

#### 2. From treatment processes

Glycidol has been reported to be a contaminant in the EPI-DMA polymer (Letterman and Pero 1990). AWWA Standard B452-06 (revised 2014) regulates the use of EPI‑DMA.

3-Monochloropropan-1,2-diol (qv) has a chemical structure which suggests that it may be metabolised to genotoxic intermediates, particularly glycidol.

### Forms and fate in the environment

Fully miscible with water.

### Typical concentrations in drinking-water

NSF (2010) reported the results of testing 112 samples of drinking water for glycidol; the median was <0.0002 mg/L and the range was <0.0002 to 0.0008 mg/L.

### Analytical methods

#### Referee method

Not needed.

#### Some alternative methods

None needed.

### Health considerations

Approximately 87 to 92 percent of 37.5 or 75 mg/kg body weight orally administered glycidol is absorbed from the gastrointestinal tract of male Fischer 344 rats. Seven to eight percent of the dose remained in tissues 72 hours following administration. The highest concentrations were observed in blood cells, thyroid, liver, kidney and spleen.

Glycidol is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity in experimental animals (NTP 1990, IARC 2000) so has been classified in Group 2A: probably carcinogenic to humans. Note that the aldehyde (glycidaldehyde CAS No. 765-34-4) is classed as a possible human carcinogen (Group 2B).

No adequate human studies of the relationship between exposure to glycidol and human cancer have been reported (IARC 2000). This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

The oral RfD for glycidaldehyde was calculated at 0.0004 mg/kg/d (USEPA 1991). USEPA (1991) classified glycidaldehyde as B2: probable human carcinogen.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Letterman RD, Pero RW. 1990. *Contaminants in Polyelectrolytes Used in Water Treatment*. Denver, CO: AWWARF.

IARC. 1999. *IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans* 71. Re-evaluation of some organic chemicals, hydrazine and hydrogen peroxide. http://monographs.iarc.fr/ENG/Monographs/vol71/index.php.

IARC. 2000. *IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans* 77. Some industrial chemicals. Lyon, France: International Agency for Research on Cancer. 529 pp. See also http://www.inchem.org/documents/iarc/vol77/77-14.html.

NSF. 2010. *NSF Fact Sheet: Polyelectrolytes and NSF/ANSI Standard* 60. 12 pp. <http://www.nsf.org/newsroom_pdf/NSFFactSheetPolyelectrolytes.pdf>.

NTP. 1990. Toxicology and carcinogenesis studies of glycidol. In *F344/N Rats and B6C3F1 Mice (Gavage Studies)*. Technical Report Series No 374. NIH Publication No. 90‑2829. Research Triangle Park, NC: National Toxicology Program. 229 pp.

USEPA. 1991. Glycidaldehyde. *Integrated Risk Information System (IRIS)*. http://www.epa.gov/iris/subst/0315.htm.

# Glycolic acid

CAS No. 79-14-1. The IUPAC name is hydroxyethanoic acid. Also called hydroxyacetic acid. Sometimes spelt glycollic acid.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for glycolic acid. The WHO Guidelines do not mention glycolic acid.

### Sources to drinking-water

#### 1. To source waters

Glycolic acid is prepared by chemical synthesis, or extraction from plants where it is formed by photosynthesis. Its main use is in cosmetic products.

Glycolic acid is found in the fruit, leaf, stem and root portions of all plants. Commonly consumed fruits and vegetables are reported to contain from 0.45–7.4 mg glycolic acid per 100 g fresh wet weight. Tea, coffee, fruit juice and other beverages derived from plant sources may contain 5–7 mg glycolic acid per 100 mL. Foods of animal origin are generally low in glycolic acid.

Compared with cosmetic grade glycolic acid, the technical quality has a higher content of process impurities such as formic acid, formaldehyde, diglycolic acid and methoxyacetic acid.

### Forms and fate in the environment

Glycolic acid contains acid as well as primary alcohol functional groups. It undergoes typical oxidation reactions to give glyoxylic acid and oxalic acid and reduction reactions with active metals to form acetic acid. As an acid it forms salts, esters, amides etc. As an alcohol it forms esters, acetals and ethers. It uses both functional groups to form complexes with polyvalent metal ions. In acid environments, glycolic acid can undergo self-esterification to form cyclic and linear polymers known as glycolides. There are no readily hydrolysable groups in glycolic acid.

Highly soluble in water.

### Analytical methods

#### Referee method

Not needed.

### Health considerations

Glycolic acid is absorbed by ingestion, inhalation and through the skin. In humans, it is mainly excreted unchanged in the urine while smaller amounts are metabolised to glyoxylic and oxalic acids, which are also excreted in the urine. The kinetics and metabolism are qualitatively similar in rats and humans; however, rats metabolise a greater proportion to carbon dioxide and eliminate the chemical faster than humans. The no observed adverse effect level (NOAEL) based on a three-month oral rat toxicity test and on maternal and developmental toxicity in pregnant rats is 150 mg/kg/day.

There is no safety concern for the consumer if the substance is used for manufacturing polyglycolic for i) indirect food contact behind polyesters such as polyethylene terephthalate (PET) and polylactic acid (PLA) and ii) direct food contact of a blend of PGA up to 3 percent w/w in PET and PLA (EFSA 2010).

Glycolic acid is not mutagenic. It does not impair fertility or neonatal growth during lactation. There are no animal studies of systemic or developmental toxicity from dermal exposure and no carcinogenicity studies.

### Derivation of Maximum Acceptable Value

No MAV.

### References

EFSA. 2010. Scientific opinion on the safety evaluation of the substance glycolic acid for use in food contact materials. *EFSA Journal* 8(12): 1927. 11 pp. <http://onlinelibrary.wiley.com/doi/10.2903/j.efsa.2010.1927/epdf>.

NICNAS. 2000. Glycolic acid. *Priority Existing Chemical Assessment Report* 12. National Industrial Chemicals Notification and Assessment Scheme, Australia. 145 pp. <https://www.nicnas.gov.au/chemical-information/factsheets>

# Glyoxal

CAS No. 107-22-2. Also called anhydrous glyoxal, oxaldehyde, ethane-1,2-dione, ethanedial, diformyl, ethanedione, biformal, and oxal. Glyoxal can undertake rotational isomerisation between the planar *cis* and *trans* conformations, with *trans*-glyoxal being the more stable isomer.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for glyoxal. The WHO Guidelines do not mention glyoxal.

### Sources to drinking-water

#### 1. To source waters

Glyoxal is a synthetic chemical used for production of paper coatings, textiles and polymers, and as an intermediate for making other chemicals such as glutaraldehyde, quaternary ammonium compounds, pharmaceuticals and dyestuffs, as a cross-linking agent in the production of a range of different polymers. Glyoxal is also used as a disinfectant in health care and dentistry work. Glyoxal is commercialised as a 40 percent aqueous solution.

Glyoxal concentrations up to 0.012 mg/L have been reported in European rivers and groundwater. Due to microbial activity as well as non-enzymatic autoxidation of oil or browning reactions of saccharides, glyoxal is frequently detected in fermented food and beverages. It was found in different brands of beer, wine, and other beverages such as tea at concentrations ranging from about 0.02 mg/L (black tea) up to 1.5 mg/L (sherry wine). In addition, it was detected in a range of fermented products such as soybean paste and yoghurt (0.63–4.2 mg/kg), bakery products such as bread  
(0.07–1.6 mg/kg), different plant materials (3–14 mg/kg), and edible oils (up to 6.5 mg/kg) (WHO 2004).

Methylglyoxal is the substance that is responsible for the antibacterial activity of manuka honey due to its cytotoxicity. Despite that, it is formed by several bacteria of the human intestine; it appears in higher concentrations in the blood of diabetics. It also appears in coffee, beer and soy products. It is also called pyruvaldehyde, acetylformaldehyde, 2-oxopropanal or MGO, with CAS No. 78-98-8, or 1186-47-6 for the hydrate. IARC (1991) found methylglyoxal is not classifiable as to its carcinogenicity to humans (Group 3).

#### 2. From treatment processes

The dialdehyde, glyoxal, a strong mutagen, is formed by ozonation of humic substances. Stockham and Morran detected glyoxal in water treated with polyDADMAC after ozonation.

### Forms and fate in the environment

Although glyoxal can polymerise, in the environment, at low concentrations, it can be assumed that only the monomer is present.

Glyoxal released into the environment is rapidly converted by abiotic processes, such as transformation by photochemically produced hydroxyl radicals. Due to the low soil sorption coefficient (*K*oc) reported for this compound, and its low volatility, it may leach from soil into groundwater. However, it is readily biodegraded and quickly transformed enzymatically by bacteria and fungi. Its low log octanol/water partition coefficient (*K*ow) indicates that glyoxal is unlikely to bioaccumulate (WHO 2004).

Glyoxal is a metabolite of ethylene glycol. It is miscible in water.

### Typical concentrations in drinking-water

Glyoxal has been reported in drinking water at 0.013 mg/L. Dosing ozone at 1.2 to 4.4 mg/L into water containing 2.66 mg/L TOC resulted in glyoxal concentrations of 0.004–0.011 mg/L (Inchem).

Three water utilities in the US reported detecting glyoxal in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.016 mg/L.

### Removal methods

Removed by GAC. Biologically active GAC has been reported to remove 90 – 100 percent of the glyoxal.

### Analytical methods

#### Referee method

Not needed.

#### Some alternative methods

See WHO (2004).

### Health considerations

The main routes of occupational exposure to glyoxal during use as a disinfectant are via inhalation of aerosol and dermal absorption. The general population is exposed mainly through ingestion of glyoxal-containing food, but could be exposed through polluted air in urban regions and through traces of glyoxal in drinking-water. The concentration of glyoxal in human blood plasma has been reported to be  
0.1–1 μmol/litre, with higher levels reported for patients with diabetes or renal failure.

A 28-day study in which glyoxal was administered to rats in drinking-water resulted in a no-observed-adverse-effect level (NOAEL) of 100 mg glyoxal/kg body weight per day. The 90-day feeding of glyoxal to rats resulted in a NOAEL of 125 mg/kg body weight per day (dosage corresponding to 100 percent glyoxal). Effects stated at higher dosages in these two latter studies were reduced water and food intake (first study only) and retardation of body weight gain (both studies). In a study examining more sensitive end-points (serum clinical biochemistry), the lowest tested dosage of 107 mg/kg body weight per day (99 percent glyoxal) corresponded to the lowest-observed adverse-effect level (LOAEL) for a 90-day exposure of rats via drinking-water. A 90-day feeding study in dogs failed to reveal any substance-related changes at the top dose of 115 mg/kg body weight per day (dose corresponding to 100 percent glyoxal).

OECD (2003) states that overall, a NOEL of 100 mg/kg bw/d related to 40 percent glyoxal (40 mg/kg bw/d related to active ingredient) can be retained for repeated dose toxicity.

In a sample risk assessment for the general population, an exposure scenario has been compiled as a hypothesised worst case. Using the daily intake of, maximally, 10 mg glyoxal via food, an estimated intake of 0.16 mg glyoxal/kg body weight per day can be calculated. This is similar to the tolerable intake of about 0.2 mg/kg body weight per day for lifetime oral exposure to glyoxal (WHO 2004).

No studies with long-term exposure to glyoxal by inhalation or oral routes are available; however, glyoxal should be considered to be a potential carcinogen. Chronic LOAEL considered to be <10 mg/kg/day. Glyoxal has a direct mutagenic effect on *Salmonella* strains TA 100, 102 and 104 (AWWARF).

### Derivation of Maximum Acceptable Value

No MAV.

### References

AWWARD. 1991. *Ozone in Water Treatment: Application and Engineering.* Langlais B, Reckhow DA, Brink DR (eds). Cooperative Research Report. Lewis Publishers, CRC Press.

Environmental Working Group (EWG). Accessed 2010. *National Drinking Water Database – Chemical Contaminants*. <http://www.ewg.org/tap-water/chemical-contaminants>.

IARC. 1991. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. Coffee, tea, mate, methylxanthines and methylglyoxal. 513 pp. <https://monographs.iarc.fr/ENG/Monographs/vol51/mono51-13.pdf>.

Inchem. 2000. Disinfectants and disinfectant by-products. *Environmental Health Criteria* 216. International Programme on Chemical Safety. United Nations Environment Programme, International Labour Organisation, World Health Organization. Available at: <http://www.inchem.org/documents/ehc/ehc/ehc216.htm>.

OECD. 2003. *SIDS Initial Assessment Report*: Glyoxal. 56 pp. See: http://www.inchem.org/documents/sids/sids/107222.pdf or <http://www.inchem.org/pages/sids.html>.

Stockham P, Morran J. 2000. Disinfection by-products from a poly(diallyldimethylammonium chloride) based polyelectrolyte used in water treatment. *CRC for Water Quality and Treatment*. Research Report No 4. Available at: <http://www.waterquality.crc.org.au/programs/program2d.htm>.

WHO. 2004. Glyoxal. *Concise International Chemical Assessment Document (CICAD)* 57. International Programme on Chemical Safety (IPCS). 49 pp. http://www.who.int/entity/ipcs/publications/cicad/en/cicad57.pdf.

# Glyoxylic acid

CAS No. 298-12-4. Also called oxoacetic acid, formylformic acid, glyoxalic acid, glyoxalate and oxoethanoic acid.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for glyoxylic acid. The WHO Guidelines do not mention glyoxylic acid.

### Sources to drinking-water

#### 1. To source waters

Glyoxylic acid occurs in unripe fruit and in young green leaves as an intermediate in photorespiration in plants; has also been found in very young sugar beets. Has an obnoxious odour.

Glyoxylic acid has been investigated as an alternative reducing agent for electroless copper plating. Plating rates and bath stability were superior to that of the formaldehyde bath under standard conditions. Glyoxylate ions in the plating bath have no vapour pressure and showed good reducing power in the electroless copper plating. Therefore, glyoxylic acid can replace formaldehyde, and eliminate health and environmental problems resulting from generation of the fumes. It is used in the production of some pharmaceuticals and cosmetics, and glyoxylic derivatives have fungicidal properties.

#### 2. From treatment processes

Glyoxylic acid is one of the DBPs formed when treating water containing natural organic matter with ozone. Stockham and Morran detected glyoxylic acid in water treated with polyDADMAC after ozonation. Glyoxylic acid is the second commonest ketoacid produced during ozonation; ketoacids tend to form higher concentrations than the aldehydes.

### Forms and fate in the environment

Glyoxylic acid is a strong acid. It is described as being soluble in water.

### Analytical methods

#### Referee method

Not needed.

#### Some alternative methods

None needed.

### Health considerations

AWWARF reported that glyoxylic acid has a direct mutagenic effect on *Salmonella* strains TA 97, 100 and 104. Glyoxylic acid appears to be responsible for a portion of the ozone-induced mutagenicity of water containing organic matter.

### Derivation of Maximum Acceptable Value

No MAV.

### References

AWWARD. 1991. *Ozone in Water Treatment: Application and Engineering.* Langlais B, Reckhow DA, Brink DR (eds). Cooperative Research Report. Lewis Publishers, CRC Press.

Inchem. 2000. Disinfectants and disinfectant by-products. *Environmental Health Criteria* 216. International Programme on Chemical Safety. United Nations Environment Programme, International Labour Organisation, World Health Organization. Available at: http://www.inchem.org/documents/ehc/ehc/ehc216.htm.

Kostakis C, Nicholson BC. 2001. *Impact of Ozone on Disinfection By-products: Comparison of three surface waters with differing character*. Cooperative Research Centre for Water Quality and Treatment. Research Report 9. ISBN 1 876616 10 5. www.waterquality.crc.org.au/publications/report09\_impact\_ozone\_DBPs.pdf.

Stockham P, Morran J. 2000. Disinfection by-products from a poly(diallyldimethylammonium chloride) based polyelectrolyte used in water treatment. *CRC for Water Quality and Treatment*. Research Report 4. Available at: http://www.waterquality.crc.org.au/programs/program2d.htm.

# Haloacetaldehydes

Haloacetaldehydes (HALs) include:

|  |  |
| --- | --- |
|  | **CAS no.** |
| **Monohaloacetaldehydes** |  |
| bromoacetaldehyde (BAL) | 17157-48-1 |
| chloroacetaldehyde (CAL) | 107-20-0 |
| iodoacetaldehyde (IAL) | 55782-51-9 |
| **Dihaloacetaldehydes:** |  |
| bromochloroacetaldehyde (BCAL) | 98136-99-3 |
| dibromoacetaldehyde (DBAL) | 3039-13-2 |
| dichloroacetaldehyde (DCAL) | 79-02-7 |
| **Trihaloacetaldehydes** |  |
| bromodichloroacetaldehyde (BDCAL) | 34619-29-9 |
| dibromochloroacetaldehyde (DBCAL) | 64316-11-6 |
| tribromoacetaldehyde (TBA) | 115-17-3 also called bromal |
| trichloroacetaldehyde (TCAL) | 75-87-6 also called chloral |

### Maximum Acceptable Value

The DWSNZ do not have a MAV for haloacetaldehydes, collectively or individually. The WHO Guidelines do not mention haloacetaldehydes other than trichloroacetaldehyde.

There is a separate datasheet for trichloroacetaldehyde because it had a MAV in the 2005 DWSNZ. WHO (2005 and 2011) state that because trichloroacetaldehyde usually occurs in drinking-water at concentrations well below the health-based value of 0.1 mg/L (ie, generally below 0.01 mg/L), it is not considered necessary to derive a guideline value. Trichloroacetaldehyde (chloral) reacts with water to form chloral hydrate.

### Sources to drinking-water

#### 1. To source waters

Dichloroacetaldehyde is a degradation product of dichlorvos.

#### 2. From treatment processes

The haloacetaldehydes (HALs) were the third largest group by weight of identified DBPs formed in drinking water in a US Nationwide DBP Occurrence Study, with the dihaloacetaldehydes being the most common, whether chlorination or chloramination was used. Until recently, the only HAL that has been studied has been trichloroacetaldehyde (chloral) (Postigo et al 2016).

The brominated trihaloacetaldehydes, bromodichloroacetaldehyde, dibromochloroacetaldehyde and tribromoacetaldehyde have also been detected in ozonated waters; as has trichloroacetaldehyde.

From the WRF (2016) study:

**Bromochloroacetaldehyde (BCA)**: patterns were similar to those for DCA. They were, of course, impacted as well by the raw water bromide level as reflected in the bromine substitution factors for the THMs.

**Dichloroacetaldehyde (DCA)** was detected in most, but not all of the utilities. Higher concentrations of dichloroacetaldehyde were observed in chloraminated utilities compared with chlorinated utilities. Utility #10 had the highest levels of dichloroacetaldehyde (up to 33 μg/L). These values are higher than those previously reported by Weinberg and colleagues (2002). In that study, the highest levels were found at a plant using ozone and chloramines. In the case of Utility #10 there seemed to be a trend toward lower levels as chloroform concentrations increased, possibly reflecting a degradation pathway. Aside from Utility #10, the other locations with high DCA values were those using strong oxidants (ie, Utilities #5 (in Jan), 6, 7 and 11). At most utilities, the concentration of dichloroacetaldehyde remained at or below 5 μg/L.

**Bromodichloroacetaldehyde (BDCA)** is formed at levels lower than chloral hydrate, but commensurate with the bromine incorporation expected for these water. Except for Utility #2 (chlorine burn), there appears to be little correlation between BDCA and bromodichloromethane (BDCM) within any given system. For Utility #2, there appears to be a loss of BDCA as BDCM increases suggesting some degradation mechanism. Water from Utility #4e was found to have highest concentration of bromodichloroacetaldehyde (up to 7.5 µg/L), which again suggests that pre-ozonation may increase the formation of this haloacetaldehyde as it has been proposed for the trichloro-derivative (chloral hydrate).

Losses of dihaloacetaldehydes are evident with increasing water age. For the trihaloacetaldehydes the brominated form seems to decrease whereas the trichloro species increases. The latter may be due to further chlorination of the dichloroacetaldehyde in the distribution system (WRF 2016).

In laboratory trials Postigo et al (2016) found (of the mono- and di-HALs) that DCAL was the most common HAL formed, and reached the highest concentrations. Trials using a river water with 6.8 mg/L TOC, 788 µg/L Br and 18 µg/L total I, produced:

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **[µg/L]** | **CAL** | **DCAL** | **BAL** | **DBAL** | **BCAL** | **IAL** |
| chlorination | 0.6 | 4.6 | 0.6 | 2.6 | 0.7 | 0.7 |
| chloramination | 0.2 | 3.1 | 0.8 | 2.1 | 4.1 | 0.5 |

In waters with 2 µg/L I and <10 µg/L Br, HALs were either not detected or were <1 µg/L, except for DCAL which reached 2 µg/L in some waters. Overall, di-HALs, ie, DCAL, DBAL, and BCAL contributed the most to the total concentrations of mono-HALs and di-HALs measured.

#### 3. From the distribution system

No known sources.

### Fate and form in the environment

The HALs are soluble in water.

Dichloroacetaldehyde’s production and use as a chemical intermediate and its formation during the pulp bleaching process may result in its release to the environment through various waste streams. If released to soil, dichloroacetaldehyde is expected to have very high mobility based upon an estimated Koc of 4.3. Volatilisation from moist soil surfaces is expected to be an important fate process based upon an estimated Henry’s Law constant of 8.42 x 10-6 atm‑cu m/mole. Dichloroacetaldehyde may potentially volatilise from dry soil surfaces based upon its estimated vapour pressure of 59 mm Hg at 25°C. If released into [water](https://pubchem.ncbi.nlm.nih.gov/compound/water), dichloroacetaldehyde is not expected to adsorb to suspended solids and sediment in the [water](https://pubchem.ncbi.nlm.nih.gov/compound/water) column based upon its estimated Koc. Volatilisation from [water](https://pubchem.ncbi.nlm.nih.gov/compound/water) surfaces is expected to be an important fate process based upon this compound’s estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 4.7 days and 38 days, respectively (*Pub Chem*, accessed May 2017).

The hydrolysis by-products for bromodichloroacetaldehyde, dibromochloroacetaldehyde, tribromoacetaldehyde and trichloroacetaldehyde are trichloromethane (chloroform), bromodichloromethane, chlorodibromomethane and tribromomethane. At pH 9 the half-life for bromodichloroacetaldehyde is 11 hours, dibromochloroacetaldehyde is 2.5 hours and tribromoacetaldehyde 0.5 hours; the hydrolysis of trichloroacetaldehyde is insignificant. Hydrolysis is slower at pH 7.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See WRF (2016).

### Health considerations

In a study by Jeong et al (2015) the rank order of HAL cytotoxicity is tribromoacetaldehyde (TBAL) ≈ chloroacetaldehyde (CAL) >dibromoacetaldehyde (DBAL) ≈ bromochloroacetaldehyde (BCAL) ≈ dibromochloroacetaldehyde (DBCAL) >IAL >bromoacetaldehyde (BAL) ≈ bromodichloroacetaldehyde (BDCAL) >dichloroacetaldehyde (DCAL) >trichloroacetaldehyde (TCAL). The HALs were highly cytotoxic compared with other DBP chemical classes. The rank order of HAL genotoxicity is DBAL >CAL ≈ DBCAL >TBAL ≈ BAL >BDCAL >BCAL ≈ DCAL >IAL. TCAL was not genotoxic.

USEPA (2016) includes:

2-Chloroacetaldehyde (2-CAA) was examined for carcinogenicity in rats by Daniel et al. (1992). B6C3F1 mice were exposed to 0.1 g/L of 2-CAA (17 mg/kg/day) in a cancer bioassay. There were significant increases for hepatic necrosis and hepatic tumors but not liver weights in the treated rats. Only one dose was evaluated for comparison with the controls. CAL was mutagenic in bacteria and in mammalian cells in vitro but not in mice.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Jeong et al. 2015. Occurrence and comparative toxicity of haloacetaldehyde disinfection by‑products in drinking water. *Environ Sci Technol* 49(23): 13749–59. DOI: 10.1021/es506358x.

Krasner SW, et al. 2012. Formation and control of emerging C- and N-DBPs in drinking water. *J AWWA* 104(11): E582–95. [https://www.jstor.org/stable/jamewatworass.104.11.e582?seq=1#page\_scan\_tab\_contents](https://www.jstor.org/stable/jamewatworass.104.11.e582?seq=1%23page_scan_tab_contents).

Postigo, et al. 2016. Formation of iodo-trihalomethanes, iodo-haloacetic acids, and haloacetaldehydes during chlorination and chloramination of iodine containing waters in laboratory controlled reactions. 28 pp. <http://digital.csic.es/bitstream/10261/147697/1/manuscript_Postigo%20et%20al_033017_repositorio.pdf>.

*Pub Chem*. Accessed May 2017. Dichloroacetaldehyde. *Pub Chem Open Chemistry Database*. National Center for Biotechnology Information. <https://pubchem.ncbi.nlm.nih.gov/compound/6576>.

USEPA. 2016. *Six-Year Review 3 Technical Support Document for Disinfectants/ Disinfection By-products Rules*. 468 pp. <https://www.epa.gov/sites/production/files/2016-12/documents/810r16012.pdf>.

WRF. 2016. *Fate of Non-Regulated Disinfection By-products in Distribution Systems*. Web Report #4242. 240 pp. Water Research Foundation (US) and Drinking Water Inspectorate (UK). <http://www.waterrf.org/PublicReportLibrary/4242.pdf>.

# Haloacetamides

Haloacetamides belong to the loosely defined group of nitrogen containing disinfection by-products (N-DBPs). The main haloacetamides (sometimes referred to collectively as HAcAms) are:

|  |  |  |
| --- | --- | --- |
|  | **CAS no.** |  |
| 2-chloroacetamide | 79-07-2 |  |
| 2-bromoacetamide | 683-57-8 |  |
| 2,2-dichloroacetamide | 683-72-7 | DCAM |
| 2,2-bromochloroacetamide | 62872-34-8 | BCAM |
| 2,2-dibromoacetamide | 598-70-9 |  |
| 2,2-bromoiodoacetamide | 62872-36-0 |  |
| 2,2,2-trichloroacetamide | 594-65-0 | TCAM |
| 2,2,2-bromodichloroacetamide | 98137-00-9 |  |
| 2,2,2-dibromochloroacetamide | 855878-13-6 |  |
| 2,2,2-tribromoacetamide | 594-47-8 |  |

### Maximum Acceptable Value

The DWSNZ do not have a MAV for haloacetamides, collectively or individually. The WHO Guidelines do not mention haloacetamides.

### Sources to drinking-water

#### 1. From treatment processes

DWI (2010) states (re the US Nationwide DBP Occurrence Study): The occurrence of haloacetamides (HAcAms) was reported for the first time, with 2,2-dichloroacetamide being the most prominent species. Haloacetamides were frequently identified in finished water from plants 6, 11 and 12, where chlorine dioxide was applied prior to chlorine/chloramine. In samples taken in February 2002, all five haloacetamide species were identified in plants 11 and 12 (using chloramination). The concentration of individual species ranged from 0.4 to 2.8 μg/L in the plant effluent.

Chloramination was also found to enhance the formation of iodinated DBPs; bromoiodoacetamide was detected in chloraminated drinking waters.

Amino acids as precursors seem to be important. Therefore haloacetamides are more likely to be produced from raw waters containing sewage or animal wastes, or other nitrogen-rich effluents. In a recent study no haloacetamides were produced from humic acid in the absence of protein.

Degradation of haloacetonitriles will produce the corresponding haloacetamides, with subsequent hydrolysis producing haloacetic acids; haloacetamides were most stable at pH 5.

With the development of a new analytical method of high selectivity and sensitivity the WRF (2016) study managed to detect the three most prominent haloacetamides: DCAM, BCAM and TCAM in field samples collected from five drinking water distribution systems, by using solid-phase extraction (SPE) pre-concentration with gas chromatography-mass spectrometry (GC-MS) analysis, with a highest concentration of 13 μg/L for DCAM, followed by BCAM showing a 2.5 μg/L maximum level, and just 1 μg/L for TCAM.

#### 2. From the distribution system

No known sources. Haloacetamides are likely to hydrolyse in the distribution system to form the corresponding haloacetic acid.

### Fate and form in the environment

|  |  |
| --- | --- |
|  | **Water solubility** |
| 2-chloroacetamide | 35 percent |
| 2-bromoacetamide | 24 percent |
| 2,2-dichloroacetamide | 6.4 percent |
| 2,2-bromochloroacetamide | 6.0 percent |
| 2,2-dibromoacetamide | 3.0 percent |
| 2,2-bromoiodoacetamide | 0.7 percent |
| 2,2,2-trichloroacetamide | 0.9 percent |
| 2,2,2-bromodichloroacetamide | ? |
| 2,2,2-dibromochloroacetamide | ? |
| 2,2,2-tribromoacetamide | 0.15 percent (1,500 mg/L) |

### Typical concentrations in drinking-water

Concentration (mg/L) of N-DBPs found in US drinking water (ex DWI 2010):

|  |  |  |  |
| --- | --- | --- | --- |
| **Haloacetamide** | **Minimum** | **Median** | **Maximum** |
| 2-chloroacetamide | ND | ND | 0.0005 |
| 2-bromoacetamide | ND | ND | 0.0011 |
| 2,2-dichloroacetamide | ND | 0.0013 | 0.0056 (5.6 µg/L) |
| 2,2-dibromoacetamide | ND | 0.0006 | 0.0028 |
| 2,2,2-trichloroacetamide | ND | 0.0003 | 0.0011 |

HAcAms found in a UK study averaged 0.0015 mg/L (1.5 µg/L). The lowland water sources that were included in this survey formed more N-DBPs than the upland and groundwater sources. The six treatment works that applied ozone were associated with higher concentrations of HANs and HAcAms than non-ozone treatment works, although this was potentially confounded because all the ozone works were treating lowland source waters which may have had higher N-DBP formation potential. There were no consistent trends between N-DBP concentrations and water age in distribution (distance from the treatment works). Total organic carbon and ultraviolet absorbance at 254 nm were poor predictors of N-DBP concentrations, nor were there clear observed links with other individual measured water quality parameters (DWI 2012).

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See DWI (2010 and 2012).

### Health considerations

Generally there is very little information on the toxicity of haloacetamides. The largest toxicological database is based on chronic cytotoxicity and for the induction of genomic DNA damage in Chinese hamster ovary cells.

The rank order for cytotoxicity of 13 haloacetamides was diiodoacetamide >iodoacetamide >bromoacetamide >tribomoacetamide >bromoiodoacetamide >dibromochloroacetamide >chloroiodoacetamide >bromodichloracetamide >dibromoacetamide >bromochloroacetamide >chloroacetamide >dichloracetamide >trichloroacetamide.

The rank order of their genotoxicity was tribomoacetamide >diiodoacetamide approximately equal to iodoacetamide >bromoacetamide >dibromochloroacetamide >bromoiodoacetamide >bromodichloracetamide >chloroiodoacetamide >bromochloroacetamide >dibromoacetamide >chloroacetamide >trichloroacetamide. Dichloracetamide was not genotoxic.

Cytotoxicity and genotoxicity were primarily determined by the leaving tendency of the halogens and followed the order I >Br >>Cl. With the exception of brominated trihaloacetamides, most of the toxicity rank order was consistent with structure-activity relationship expectations. For di- and trihaloacetamides, the presence of at least one good leaving halogen group (I or Br, but not Cl) appears to be critical for significant toxic activity.

For cytotoxicity the rank order from the most toxic to the least toxic DBP classes was: haloacetaldehydes >haloacetamides >halonitromethanes >haloacetonitriles >>2C haloacids >haloacetic acids >halomethanes.

For induced genomic DNA damage in Chinese hamster ovary cells the rank order from the most genotoxic to the least genotoxic of the DBP classes was: haloacetonitriles >haloacetamides >halonitromethanes >haloacetaldehydes >haloacetic acids >>2C‑haloacids >halomethanes (trihalomethanes only).

N-DBPs, including haloacetonitriles, haloacetamides and halonitromethanes, are far more cytotoxic and genotoxic than DBPs that did not contain nitrogen (eg, haloacetic acids, haloacids and halomethanes).

In a balanced comparison of iodinated, brominated and chlorinated analogues, the cytotoxicity and genotoxicity of the iodinated DBPs was greater than that of their brominated or brominated analogues, with the chlorinated analogues being the least toxic.

While these relative comparisons of cytotoxicity and genotoxicity are informative, these values should be considered only as indicators of *potential* human health risk, rather than translated into *probable* human health risk, which is only appropriately extrapolated from *in vivo* studies.

The European Commission’s Scientific Committee on Consumer Safety (SCCS) reviewed the toxicity of chloroacetamide (SCCS, 2011). Though there were no guideline-compliant developmental or reproductive studies available, the review derived a LOAEL of 24 mg/kg/day based on maternal body weight reduction and skeletal findings in offspring when chloroacetamide was administered from GD 14 to PND 2. The NOAEL for this effect was 3 mg/kg/day. From USEPA (2016).

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2010. *Review of the Current Toxicological and Occurrence Information Available on Nitrogen-containing Disinfection By-products*. DWI 70/2/243. 195 pp. http://dwi.defra.gov.uk/research/completed-research/2000todate.htm.

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# Haloacetic acids

There are nine common haloacetic acids (the HAAs or HAA9):

* monochloroacetic acid, dichloroacetic acid, trichloroacetic acid
* monobromoacetic acid, dibromoacetic acid, tribromoacetic acid
* bromochloroacetic acid, bromodichloroacetic acid, chlorodibromoacetic acid.

**These nine haloacetic acids have separate datasheets.** The first five HAAs shown above are frequently referred to as HAA5, sometimes as total HAAs, or total regulated HAAs. The whole group is often referred to as HAA9.

The haloacetic acids are also classified by their degree of halogenation:

* monohalogented acetic acids (MXAA): monochloroacetic acid and monobromoacetic acid
* dihalogented acetic acids (DXAA): dichloroacetic acid, dibromoacetic acid and bromochloroacetic acid
* trihalogented acetic acids (TXAA): trichloroacetic acid, tribromoacetic acid, bromodichloroacetic acid, chlorodibromoacetic acid.

Iodinated trihaloacetic acids have been found in drinking water; these are discussed in the iodinated DBPs datasheet.

### Maximum Acceptable Value

To account for additive toxicity, the sum of the ratio of the concentration of each haloacetic acid to its respective MAV should not exceed 1.

The DWSNZ state in section 8.2.1.1: “*When more than one determinand that causes similar toxicological effects is present, the sum of the ratios of the concentration of each determinand to its respective MAV does not exceed one for compliance with the DWSNZ. In the DWSNZ, this applies to nitrate/nitrite, trihalomethanes (THMs), the haloacetic acids and haloacetonitriles*.” Note: when a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, section 10.2.5.3).

Haloacetic acids as a group do not have a MAV in the DWSNZ. However, monochloroacetic acid, dichloroacetic acid and trichloroacetic acid have a MAV.

Individual HAAs with regulatory values are (the other four do not have a MAV):

|  |  |  |
| --- | --- | --- |
| monochloroacetic acid | MAV 0.02 mg/L | 0.1 mg/L MAC in Canada |
| dichloroacetic acid | MAV 0.05 mg/L | 0.01 mg/L MAC in Canada |
| trichloroacetic acid | MAV 0.2 mg/L | 0.3 mg/L MAC in Canada |
| monobromoacetic acid | no MAV | no MAC in Canada |
| dibromoacetic acid | no MAV | 0.002\* mg/L in Canada |

\* Health based target.

The maximum contaminant level (MAC) for the HAA5 haloacetic acids (USEPA 2006/2009) is 0.06 mg/L.

In Canada, the maximum acceptable concentration for total haloacetic acids in drinking water is 0.08 mg/L based on a locational running annual average of a minimum of quarterly samples taken in the distribution system. Utilities should make every effort to maintain concentrations as low as reasonably achievable without compromising the effectiveness of disinfection, Health Canada. 2008. The MAC is based on the ability to achieve HAA levels in distribution systems without compromising disinfection. They define total haloacetic acids as the total of monochloroacetic acid, dichloroacetic acid, trichloroacetic acid, monobromoacetic acid and dibromoacetic acid, ie, HAA5.

### Sources to drinking-water

#### 1. To source waters

No known sources.

DWI (2013, Table A.3) reports a study of the haloacetic acids content in sodium hypochlorite feedstocks collected from five different utilities in the US:

Measured concentrations in sodium hypochlorite feedstocks:

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Water utility** | **NaOCl (mg/L)** | **MCAA (μg/L)** | **DCAA (μg/L)** | **TCAA (μg/L)** | **Total HAA (μg/L)** | **HAA/FAC ratio** |
| TN-1 | 20.3 | 73.0 | 402 | 152 | 627 | 30.9 |
| TX | 4.2 | 65.2 | 101 | 35.8 | 203 | 49.3 |
| IL | 6.6 | 92.3 | 91.4 | 27.3 | 211 | 31.6 |
| TN-2 | 7.8 | 13.6 | 40.0 | 8.3 | 61.9 | 8.0 |
| OH | 67.2 | – | 11.6 | 44.6 | 56.1 | 0.8 |

Note: MCAA = monochloroacetic acid, DCAA = dichloroacetic acid, TCAA = trichloracetic acid. The four feedstocks (TN-1, TX, IL, TN-2) giving the highest HAA/FAC ratios were all diluted and/or stored on-site for a period of time. The OH feedstock was undiluted.

#### 2. From treatment processes

Haloacetic acids are disinfection by-products – see individual datasheets.

### Typical concentrations in drinking-water

The Priority 2 Identification Programme found 28 distribution zones supplying drinking-water to a total of 55,184 people where the sum of the ratios of each haloacetic acid was greater than the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.01 mg/L total haloacetic acids in the raw water, and 0.01 to 0.03 mg/L in the treated water.

17,956 water utilities in the US reported detecting total haloacetic acids (HAAs) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.18 mg/L.

Table 3 in Health Canada (2008) shows concentrations of each of the HAA5 in summer and winter, and as the water progresses through the distribution system.

There are three strategies for controlling the levels of haloacetic acids in drinking water: (i) remove the precursors (ii) change the disinfection protocol and (iii) remove the haloacetic acids after they have been generated (DWI 2011).

Trihaloacetic acids tend to occur at about 25 percent of the concentration of the total trihalomethanes (TTHMs). The trihaloacetic acids are fairly stable in water; WRF (2016) reports half-lifes at 23°C:

|  |  |  |
| --- | --- | --- |
| trichloroacetic acid | TCAA | 2,190 |
| bromodichloroacetic acid | BDCAA | 630 |
| dibromochloroacetic acid | DBCAA | 112 |
| tribromoacetic acid | TBAA | 17 |

DWI (2009) summarised data from a UK study in the following table: Table 3 in DWI. 2009. Individual and total HAA concentrations (μg/L) for the three utility systems.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | **Water supply** | **A** | **B** | **C** |
| DCAA | Average (SD)  Minimum  Maximum | <LOD  <LOD  <LOD | 2.0 (0.8)  0.1  4.0 | 6.8 (2.7)  3.12  15.0 |
| TCAA | Average (SD)  Minimum  Maximum | 0.0 (0.0)  0.0  0.1 | 2.8 (2.5)  0.2  8.3 | 9.8 (3.8)  4.1  18.5 |
| BCAA | Average (SD)  Minimum  Maximum | 0.2 (0.3)  0.00  0.2 | 2.3 (0.8)  0.8  3.6 | 1.7 (0.8)  0.6  3.2 |
| BDCAA | Average (SD)  Minimum  Maximum | <LOD  <LOD  <LOD | 3.8 (1.1)  0.0  5.9 | 2.7 (1.7)  0.0  5.3 |
| DBAA | Average (SD)  Minimum  Maximum | 0.3 (0.3)  0.0  0.9 | 1.1 (0.5)  0.0  2.1 | 0.4 (0.2)  0.1  0.7 |
| Total HAA | Average (SD)  Minimum  Maximum | 0.6 (0.6)  0.0  1.9 | 11.9 (3.3)  4.9  20.0 | 21.3 (6.9)  11.1  41.0 |

Water utility system A abstracted water from an aquifer through boreholes and the water was naturally high in soluble iron; the treatment consisted of aeration, filtration and chlorination.

Water utility system B used reservoir water supplied from a lowland river and the treatment process involved coagulation followed by sedimentation or flotation, dual media (anthracite/sand) filtration, granular activated carbon adsorption, and chlorination.

Lastly, the source water for water utility system C came from the combination of two upland reservoirs and the treatment process was direct filtration, followed by second stage filtration with pre-chlorination (for manganese removal and disinfection). A close relationship was found between total HAA yield with algal growth phase and a direct association with biomass was evident.

DWI (2009, 2011a and b, 2012) include a thorough discussion on the analysis of these nine haloacetic acids.

Many factors are known to affect HAA formation, including pH, temperature, disinfection choice and dose as well as water quality parameters such as concentration and character of organic matter and the presence of bromide and iodide. Some relationships are clear, for example HAA formation increases with a decrease in pH although it is important to be specific. There is some agreement in the types of water in terms of character and sources that produce the highest concentrations of HAAs. These ‘high formers’ tend to be surface reservoirs or river waters that had relatively high DOC levels (~5 to 15 mg/L) and were influenced by algal blooms. When using chloramination the majority (90 percent) of the total HAAs would be the dihalogenated species whilst chlorination produces a mixture of mono-, di- and trihalogenated HAAs. Published correlations between THMs and HAAs are generally poor. Brominated haloacetic acids have been found when waters with elevated bromide concentrations (>0.05 mg/L) are chlorinated, although it is typically the mixed chlorinated-brominated HAAs which are the most common species formed. The levels of HAAs were observed to increase in seven of the nine distribution systems tested (DWI 2011a).

In a large UK study, DWI (2011b) found dichloroacetic acid and trichloroacetic acid were the predominant HAAs, generally comprising >50 percent of the HAA9. The recent review of potential revisions to the European Drinking Water Directive suggested that a parameter value of 0.08 mg/L for a total of nine HAAs should be considered if they were identified by a Drinking Water Safety Plan or needed to be controlled by product specification. The 20 water supplies involved in the study were below this level.

### References

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# Halohydrins

### Members of the halohydrin group of compounds and their CAS Numbers

The term halohydrin is a traditional term for alcohols substituted by a halogen atom at a saturated carbon atom otherwise bearing only hydrogen or hydrocarbyl groups (usually used to mean β-halo alcohols). Example: BrCH2CH2OH (ethylene bromohydrin or 2-bromoethanol, CAS 540-51-2), ClCH2CH2CH2OH trimethylene chlorohydrin or 3‑chloropropan-1-ol, CAS 627-30-5), and PhCH(OH)CH2Cl (styrene chlorohydrin or 2‑chloro-1-phenylethanol, CAS 1674-30-2) (IUPAC 1997).

Several halohydrins have been reported to form in water following disinfection with ozone or chlorine. Some halohydrins are suspected human carcinogens. Food presents the main intake for humans, mainly as a result of fumigation, especially with ethylene oxide, with ethylene chlorohydrin (CAS 107-07-3) and ethylene bromohydrin (CAS 540‑51-2) being the main by-products of health concern; drinking-water is not an important route.

Many halohydrins in the list below have synonyms; if information on any of these is required, it may be advantageous to search in Google using the CAS number. The seven halohydrins with a datasheet in the organic chemicals section are indicated, along with the name used.

| **Name in IUPAC Ref** |  | **Name used if in datasheet** |
| --- | --- | --- |
| 2-chloro-1-propanol | 78-89-7 |  |
| glycerol alpha-monochlorohydrin | 96-24-2 | 3-monochloropropane 1,2-diol |
| 2,3-dibromo-1-propanol | 96-13-9 | 2,3-dibromopropan-1-ol |
| 1,3-dibromo-2-propanol | 96-21-9 |  |
| 1,3-dichloro-2-propanol | 96-23-1 | 1,3-dichloro-2-propanol |
| ethylene chlorohydrin | 107-07-3 | see 2-bromoethanol datasheet |
| sodium 3-chloro-2-hydroxypropylsulfonate | 126-83-0 |  |
| 1-chloro-2-propanol | 127-00-4 |  |
| ethylene fluorohydrin | 371-62-0 |  |
| propylene fluorohydrin | 430-50-2 |  |
| 3-fluoro-1,2-propanediol | 453-16-7 |  |
| 2-chloropropane-1,3-diol | 497-04-1 |  |
| chlorocyanohydrin | 513-96-2 |  |
| 1,3-diiodo-2-propanol | 534-08-7 |  |
| ethylene bromohydrin | 540-51-2 | 2-bromoethanol |
| glyceryl iodide | 554-10-9 |  |
| 2,3-dichloro-1-propanol | 616-23-9 | 2,3-dichloro-1-propanol |
| ethylene iodohydrin | 624-76-0 |  |
| 3-chloro-1-propanol | 627-30-5 |  |
| 3-bromo-1-propanol | 627-18-9 |  |
| 4-chloro-1-butanol | 928-51-8 |  |
| butylene chlorohydrin | 1320-66-7 |  |
| 3-bromo-2,2-bis(bromoethyl)-1-propanol | 1522-92-5 |  |
| 11-bromo-1-undecanol | 1611-56-9 |  |
| 2-chloro-1-phenylethanol | 1674-30-2 |  |
| hexamethylene chlorohydrin | 2009-83-8 |  |
| loprodiol | 2209-86-1 |  |
| pentaerythritol dibromide | 3296-90-0 | 2,2-bis(bromomethyl)propane-1,3-diol |
| 6-bromo-1-hexanol | 4286-55-9 |  |
| 1-chloro-3-(2-propenyloxy)-2-propanol | 4638-03-3 |  |
| alpha-bromohydrin | 4704-77-2 | 3-bromopropan-1,2-diol |
| 5-chloropentanol | 5259-98-3 |  |
| 1-butoxy-3-chloro-2-propanol | 16224-33-2 |  |
| (S)-(+)-2-chloropropan-1-ol | 19210-21-0 |  |
| 1-bromo-2-propanol | 19686-73-8 |  |
| alpha-chlorohydrin-1-phosphate | 26807-13-6 |  |
| 4-bromo-1-butanol | 33036-62-3 |  |
| 8-bromo-1-octanol | 50816-19-8 |  |
| 10-bromo-1-decanol | 53463-68-6 |  |
| 9-bromo-1-nonanol | 55362-80-6 |  |
| (R)-(-)-3-chloro-1,2-propanediol | 57090-45-6 | \*\* |
| (S)-(+)-3-chloro-1,2-propanediol | 60827-45-4 | \*\* |

\*\* These are the enantiomers of 3-monochloropropane 1,2-diol (CAS 96-24-2).

### References

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# Halonitromethanes

Halonitromethanes belong to the loosely defined group of nitrogen containing disinfection by-products (N-DBPs). There are nine common halonitromethanes (the HNMs):

|  |  |
| --- | --- |
| chloronitromethane | 1794-84-9 |
| bromonitromethane | 563-70-2 |
| dichloronitromethane | 563-70-2 |
| dibromonitromethane | 598-91-4 |
| bromochloronitromethane | 135531-25-8 |
| trichloronitromethane | 76-06-2 also called chloropicrin or TCNM |
| bromodichloronitromethane | 918-01-4 |
| dibromochloronitromethane | 1184-89-0 |
| tribromonitromethane | 464-10-8 also called bromopicrin |

Only trichloronitromethane (chloropicrin) has a separate datasheet, titled Choropicrin.

### Maximum Acceptable Value

Apart from trichloronitromethane, the DWSNZ do not have a MAV for halonitromethanes, collectively or individually. Apart from trichloronitromethane, the WHO Guidelines do not mention halonitromethanes.

### Sources to drinking-water

#### 1. From treatment processes

DWI (2010) states (re the US Nationwide DBP Occurrence Study): The formation of halonitromethanes (HNMs) was slightly lower than the haloacetonitriles (qv), with a median yield of 1.5 μg/L. A wide range of concentrations for individual halonitromethanes was reported (0.0001 to 0.005 mg/L, ie, 0.1 to 5 μg/L). Bromochloronitromethane, bromodichloronitromethane, trichloronitromethane (or TCNM, also called chloropicrin) and bromopicrin were the dominant species formed, with maximum yields in the range 2.0 to 5.0 μg/L. The predominance of brominated halonitromethanes reflects the high bromide concentrations of the source waters.

In DBP formation potential tests, waters treated with ozone showed an increase in the formation of both dihalonitromethanes (DHNMs) and trihalonitromethanes (TCNM). In contrast, the concentration of HNMs decreased when tests were performed on bio‑filtered water.

Trichloronitromethane is the most frequently reported HNM species, however, several bromonitromethanes were identified recently. Research suggests halonitromethanes are produced from the chlorination of nitrogenous organic species. Trichloronitromethane is thought to be produced after liberation and then chlorination of an existing nitromethane moiety contained within larger molecules. Specifically, trichloronitromethane was found to form from the chlorine substitution reactions of nitrophenols.

Ozone followed by chlorination has been observed to promote trichloronitromethane formation possibly because ozone can convert amino groups to nitro groups, though other groups such as organic amines, amino acids and nitrites have been noted as likely trihalonitromethane precursors too. For the halonitromethanes, pre-ozonation is a key factor in promoting their occurrence.

Trichloronitromethane (and possibly other halonitromethanes) formation increases with increasing pH.

#### 2. From the distribution system

No known sources.

### Fate and form in the environment

|  |  |
| --- | --- |
|  | **Water solubility** |
| chloronitromethane | 3.2% |
| bromonitromethane | 1.9% |
| dichloronitromethane | 1.7% |
| dibromonitromethane | 0.46% |
| bromochloronitromethane | 0.92% |
| trichloronitromethane | 10.6% |
| bromodichloronitromethane | 0.1% |
| dibromochloronitromethane | 0.33% |
| tribromonitromethane | 0.02% |

### Typical concentrations in drinking-water

Concentration (mg/L) of N-DBPs found in US drinking water (ex DWI 2010):

|  |  |  |  |
| --- | --- | --- | --- |
| **Halonitromethane** | **Minimum** | **Median** | **Maximum** |
| chloronitromethane | ND | ND | 0.0008 |
| bromonitromethane | ND | ND | 0.0003 |
| dichloronitromethane | ND | ND | 0.0007 |
| dibromonitromethane | ND | ND | 0.0006 |
| bromochloronitromethane | ND | ND | <0.003 |
| trichloronitromethane | ND | 0.0002 | 0.002 |
| bromodichloronitromethane | ND | 0.0003 | 0.003 |
| dibromochloronitromethane | ND | ND | 0.003 |
| tribromonitromethane | ND | ND | 0.005 (ie, 5 µg/L) |

DWI (2012) reported a UK study. The mean halonitromethane concentration was 0.0004 mg/L (0.4 µg/L). The main halonitromethanes found were chloronitromethane, bromonitromethane, dibromonitromethane and trichloronitromethane.

Chloropicrin (trichloronitromethane) undergoes a slow degradation in the pH range of 6.1 to 8.5. Chloropicrin is rapidly reduced in the presence of pipe corrosion solids, and dissolved oxygen slows the reaction. The reaction rate was most dependent on water-soluble iron content (WRF 2016).

### Analytical methods

Referee method

No MAV.

#### Some alternative methods

See DWI (2010).

### Health considerations

DWI (2010) stated: some DBPs which contain nitrogen, such as halonitromethanes (HNMs) and haloacetonitriles (HANs), were reported to be significantly more toxic than currently regulated DBPs.

The halonitromethanes were generally shown to be more mutagenic and cytotoxic than their halomethane counterparts. It is suggested that this is a function of the electron withdrawing capacity of the nitro group.

Generally there is little information on the toxicity of halonitromethanes, except to some extent for trichloronitromethane (chloropicrin, qv). The largest databases are a few studies on cytotoxicity and genotoxicity.

The rank order of their chronic cytotoxicity (72-hour exposure) to Chinese hamster ovary (CHO) cells was dibromonitromethane >dibromochloronitromethane >bromonitromethane >tribromonitromethane >bromodichloronitromethane >bromochloronitromethane >dichloronitromethane >chloronitromethane >trichloronitromethane.

The rank order to induce genomic DNA damage in CHO cells was dibromonitromethane >bromodichloronitromethane >tribromonitromethane >trichloronitromethane >bromonitromethane >dibromochloronitromethane >bromochloronitromethane >dichloronitromethane >chloronitromethane.

The brominated nitromethanes were more cytotoxic and genotoxic than their chlorinated analogues.

For cytotoxicity the rank order from the most toxic to the least toxic DBP classes was: haloacetaldehydes >haloacetamides >halonitromethanes >haloacetonitriles >>2C haloacids >haloacetic acids >halomethanes.

For induced genomic DNA damage in Chinese hamster ovary cells the rank order from the most genotoxic to the least genotoxic of the DBP classes was: haloacetonitriles >haloacetamides >halonitromethanes >haloacetaldehydes >haloacetic acids >>2C‑haloacids >halomethanes (trihalomethanes only).

While these relative comparisons of cytotoxicity and genotoxicity are informative, these values should be considered only as indicators of *potential* human health risk, rather than translated into *probable* human health risk, which is only appropriately extrapolated from *in vivo* studies.

The HNMs are weak mutagens in *S. typhimurium* TA100, were potent genotoxicants in mammalian cells and induced DNA damage in CHO cells. Dibromonitromethane is the most cytotoxic and mutagenic HNM tested in both *S. typhimurium* and CHO cells (Richardson et al 2007). The HNMs are more cytotoxic than the corresponding HAAs. The brominated nitromethanes and the mixed bromo- and chloro- nitromethanes were more genotoxic than the chlorinated nitromethanes (Richardson et al 2007). From USEPA (2016).

### Derivation of Maximum Acceptable Value

Other than trichloronitromethane (chloropicrin), no MAV.

### References

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WRF. 2016. *Fate of Non-Regulated Disinfection By-products in Distribution Systems*. Web Report #4242. 240 pp. Water Research Foundation (US) and Drinking Water Inspectorate (UK). <http://www.waterrf.org/PublicReportLibrary/4242.pdf>.

# Hexabromocyclododecane

CAS No. 25637-99-4 (and 3194-55-6 for one of the isomers). Also called HBCD, HBCDD, 1,2,5,6,9,10-hexabromocyclododecane and various tradenames.

### Maximum Acceptable Value

Hexabromocyclododecane does not have a MAV and is not mentioned in the WHO Guidelines.

Hexabromocyclododecane was added to the list of Persistent Organic Pollutants (POPs) under the Stockholm Convention, see <http://chm.pops.int/>.

Since August 2010 hexabromocyclododecanes have been included in the US[EPA](https://en.wikipedia.org/wiki/United_States_Environmental_Protection_Agency)‘s List of Chemicals of Concern.

Because HBCD has 16 possible stereo-isomers with different biological activities, the substance poses a difficult problem for manufacture and regulation.

### Sources to drinking-water

#### 1. To source waters

Hexabromocyclododecane is used as an additive flame retardant on its own, or in combination with other flame retardants. HBCDD is used mainly in expanded and extruded polystyrene. Most of this HBCDD-treated polystyrene is used for insulation boards in, eg, buildings and vehicles. Other applications include its use in textile coatings and in high impact polystyrene for electrical and electronic equipment. HBCDD is the third most used brominated flame retardant and the global market demand in 2001 was 16,700 tonnes. Technical HBCDD is a mix of mainly three diastereomers: alpha-, beta-, and gamma-HBCDD, and the final distribution of these diastereomers in technical HBCDD varies with a range of about 70-95 percent γ‑HBCDD and 5-30 percent α- and β-HBCDD.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Fate and form in the environment

Hydrolysis can be assumed to be an insignificant degradation route for HBCDD due to its very low water solubility (0.066 mg/L for the sum of three diastereomers). Theoretically abiotic degradation of HBCDD is possible. In practice abiotic degradation is probably of low significance because of the rather rigid ring-structure of HBCDD.

HBCDD is not readily biodegradable and no biodegradation was observed during 28‑days at a test concentration of 7.7 mg/L. In the degradation simulation tests with sediment, (α+β+γ)-HBCDD was observed to be subject to primary degradation with half-lifes of 66 and 101 days in anaerobic and aerobic sediment at 20°C, respectively. These half-lifes corresponded with 125 and 191 days, after a temperature correction to 12°C was made. Degradation half-lifes in aerobic sediment were calculated at 20°C to be 113, 68 and 104 days for α-, β- and γ-HBCDD, respectively. The temperature corrected values at 12°C were 214, 129 and 197 days. No degradation was observed in the aerobic soil degradation simulation test. The main dehalogenation product, 1,5,9‑cyclododecatriene (CDT; CAS 4903-66-4), is not readily biodegradable, but based on an enhanced ready biodegradation test modified from OECD 301F the substance was observed to be mineralised completely in 63–77 days. The abundance of HBCDD in biota and abiotic samples of remote regions also provides solid evidence of the persistency of HBCDD.

The experimental log octanol-water partition coefficient (logKow) value for technical HBCDD has been determined to 5.62 indicating a high potential for bioaccumulation. HBCDD has a vapour pressure of 6.3×10-5 Pa at 21°C which indicates very low volatility. The substance is slightly volatile from aqueous surfaces based on the calculated Henry’s law constant of 0.75 Pa m3/mol.

### Health considerations

Animal studies indicate that HBCD is rapidly absorbed and excreted (within 72 hours) after oral exposure. Tissue distribution is widespread with the highest concentrations found in fat tissue and muscle followed by the liver, lung, kidney, blood and brain.

According to the available laboratory studies with mammals, HBCDD is not carcinogenic, mutagenic or toxic to reproduction. HBCDD was observed to cause effects in repeated dose studies in liver, thyroid gland and thyroid hormone homeostasis. A NOAEL of 22.9 mg/kg bw/day for liver weight increase was the lowest observed NOAEL in the available repeated dose studies with oral administration in rats.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Hexachlorobutadiene

CAS No. 87-68-3. Also called hexachloro-1,3-butadiene, HCBD, HCB, perchlorobutadiene, 1,1,2,3,4,4-hexachloro-1,3-butadiene, 1,3-hexachlorobutadiene, hexachloro-1,3-butadiene, perchloro-1,3-butadiene, or perchlorobutadiene.

### Maximum Acceptable Value

Based on health considerations, the concentration of hexachlorobutadiene in drinking-water should not exceed 0.0007 mg/L (0.7 g/L).

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of hexachlorobutadiene in drinking water should not exceed 0.0007 mg/L.

Hexachlorobutadiene is one of the “priority pollutants” under the US Clean Water Act.

Hexachlorobutadiene was added to the list of Persistent Organic Pollutants (POPs) under the Stockholm Convention (<http://chm.pops.int/>.)

### Sources to drinking-water

#### 1. To source waters

Hexachlorobutadiene may enter raw water as an industrial and agricultural contaminant. It is used as a solvent in chlorine gas production, an intermediate in the manufacture of rubber compounds, a lubricant, a gyroscopic fluid, a pesticide, and a fumigant in vineyards (although it does not appear to be registered as a pesticide for use in New Zealand). Hexachlorobutadiene is chiefly produced as a by-product in the manufacture of chlorinated hydrocarbons.

In lake and river water in Europe, concentrations up to 0.002 mg/L have been recorded, but the mean level is usually <0.0001 mg/L.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Fate and form in the environment

The highest levels of hexachlorobutadiene have been found in air near factories producing tetrachloroethylene and trichloroethylene. In water it may not volatilise rapidly because of its low vapour pressure. It not very soluble in water, about 2 to 2.2 mg/L. Adsorption to soil particles is an important removal mechanism from water, where it can exceed 0.1 mg/kg in lake sediments, where it is very persistent.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 332 zones, did not find hexachlorobutadiene at detectable concentrations (limit of detection = 0.0005 mg/L) (ESR 2001).

Hexachlorobutadiene has been detected in surface water at concentrations of a few micrograms per litre and in drinking-water at concentrations below 0.0005 mg/L (WHO 2004).

Fifteen water utilities in the US reported detecting hexachlorobutadiene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.0027 mg/L.

### Removal methods

The strong soil adsorption suggests that treatment processes that remove particulate matter should be effective at reducing the concentration of hexachlorobutadiene in water. Trials have shown granular activated carbon to be effective in the removal of hexachlorobutadiene.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

In rats, absorption of hexachlorobutadiene is about 95 percent of the ingested dose and it is found in the blood, liver, brain, spleen, kidney and mesentery. It is metabolised in the gastrointestinal tract and kidney to a number of water soluble metabolites, and excreted in urine.

Long term intermittent human exposure has been reported to cause higher incidences of hypotension, myocardial dystrophy, nervous system and liver disorders, and respiratory tract lesions.

The primary target organ for hexachlorobutadiene toxicity is the kidney. Kidney tumours were observed in a long-term oral study in rats. HCBD has not been shown to be carcinogenic by other routes of exposure.

Tests for mutagenicity with different strains of bacteria have given positive and negative results. Some metabolites have given positive results. The International Agency for Research on Cancer considers that there is limited evidence in experimental animals for the carcinogenicity of hexachlorobutadiene, and has placed hexachlorobutadiene in Group 3 (not classifiable as to its carcinogenicity to humans). USEPA (2009) states that a concentration of 0.09 mg/L hexachlorobutadiene represents a 10-4 cancer risk.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.0002 mg/kg/day for intermediate-duration oral exposure (15–364 days) to hexachlorobutadiene.

The reference dose or RfD (USEPA 2006/2009/2011) for hexachlorobutadiene is 0.0003 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.01 mg/L.

### Derivation of Maximum Acceptable Value

Based on the available metabolic and toxicological information available, a tolerable daily intake approach is considered most appropriate for the derivation of the MAV. A no-observable-adverse-effect level determined for renal toxicity in a two-year feeding study in rats has been used for the basis of the derivation.

The MAV for hexachlorobutadiene in drinking-water was derived as follows:

0.2 mg/kg body weight per day x 70 kg x 0.1 = 0.0007 mg/L (0.7 g/L)

2 L x 1000

where:

* no-observable-adverse-effect level = 0.2 mg/kg body weight per day for renal toxicity in a two-year feeding study in rats
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for limited evidence of carcinogenicity and the genotoxicity of some metabolites).

WHO (2004a) states that a practical quantification level for HCBD is of the order of 0.002 mg/L, which is above the guideline value. However, the most probable source of HCBD in drinking-water is from its use in the manufacture of chlorine, and concentrations in drinking-water can, therefore, be controlled by specifying the HCBD content of such chemicals coming into contact with water.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for hexachlorobutadiene is 0.001 mg/L.

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# Hexachlorocyclopentadiene

CAS No. 77-47-4. The CAS name is 1,2,3,4,5,5’-hexachloro-1,3-cyclopentadiene. Also called hexachloro-1,3-cyclopentadiene, perchlorocyclopentadiene, hexachloropentadiene, percyclopentadiene, HCCP, HCCPD or HEX.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for hexachlorocyclopentadiene. The WHO Guidelines do not have a guideline value for hexachlorocyclopentadiene.

Hexachlorocyclopentadiene is one of the “priority pollutants” under the US Clean Water Act. Their MCL has been set at 0.05 mg/L because the USEPA (confirmed 2009/2011) believes, given present technology and resources, this is the lowest level to which water systems can reasonably be required to remove this contaminant should it occur in drinking-water.

### Sources to drinking-water

#### 1. To source waters

Hexachlorocyclopentadiene’s greatest use is (or was) as a raw material in manufacturing other chemicals, including pesticides (most no longer in use, such as aldrin, chlordane, dieldrin, endrin, heptachlor, and mirex), flame retardants, resins, dyes, pharmaceuticals, plastics, etc. It has no end uses of its own. It was a significant impurity in chlordane and heptachlor.

The main exposure to HCCP in New Zealand would have been through the use of endosulfan (where HCCP was up to 0.1 percent). Approval for the importation, manufacture and use of endosulfan and its formulations was revoked by ERMA, taking effect from 16 January 2009.

Commercial hexachlorocyclopentadiene usually contains significant levels of impurities such as the lower chlorinated cyclopentadienes as well as hexachlorobenzene and octachlorocyclopentene. In some manufacturing processes, the product may be only 75 percent pure. The major contaminants found in an industrial preparation of HCCP from Velsicol were octachlorocyclopentene (0.68 percent), hexachloro-1,3-butadiene (1.11 percent), tetrachloroethane (0.09 percent), hexachlorobenzene (0.04 percent), and pentachlorobenzene (0.02 percent) (EU 2007).

From 1987 to 1993, according to the USEPA’s Toxic Chemical Release Inventory, HEX releases to land and water totalled only 78 pounds, all of which was to water. These releases were primarily from the alkalis and chlorine industries. Hexachlorocyclopentadiene is not commonly found in surface water. In one survey, it was found in less than 0.1 percent of 854 water samples from various sources; the median concentration of hexachlorocyclopentadiene was less than 0.01 mg/L.

#### 2. From treatment processes

Hexachlorocyclopentadiene may be formed during chlorination of water containing humic acid.

### Fate and form in the environment

HEX is not a persistent environmental contaminant. If released to soil, it is likely to adhere to soil particles, where it will be degraded by microbes. EU (2007) states that if released to soil, HCCP will be immobilised by strong adsorption to organic matter . Significant losses on soil surfaces may occur via photolysis. Below the soil surface, photolysis would not be a significant fate process due to light attenuation. Volatilisation from soil surfaces is expected to be of minor importance. In moist soil, HCCP will be subject to chemical hydrolysis (half-life hours to weeks) and biodegradation under aerobic and anaerobic conditions.

In water it evaporates quickly and is attacked by sunlight and other reactive chemicals. Its tendency to accumulate in aquatic life varies greatly from one species to another. EU (2007) states that degradation processes for removal of HCCP from water include photolysis, hydrolysis and biodegradation. In shallow or flowing waters, photolysis is expected to be the predominant fate process. In deeper waters hydrolysis and biodegradation may be more important environmental processes. The photolysis half-life in water and natural sunlight is about four minutes; the reported photodegradation products included three primary products: (2,3,4,4,5-pentachloro-2-cyclopentenone, hexachloro-2-cyclopentenone, and hexachloro-3-cyclopentenone) and three secondary products (pentachloro-cis-2,4-pentadienoic acid, Z- and E-pentachlorobutadiene and tetrachlorobutyne). The hydrolysis half-life is about 5.5 days; sorption to sediments would not significantly affect the rate of hydrolysis.

Water solubility is about 1.0–1.5 mg/L. Vapour pressure = 10 Pa at 25°C. Partition coefficient n-octanol/water =log Pow = 4.0 – 5.5. Henry’s law constant = 2.7 x 10-2 atm.m3/mol at 25°C.

### Typical concentrations in drinking-water

HCCPD is not often found in drinking water, so exposure by this route is unlikely.

137 water utilities in the US reported detecting hexachlorocyclopentadiene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.10 mg/L. This result is something of an outlier – the next highest was 0.010 mg/L.

### Removal methods

Granular activated charcoal combined with packed tower aeration should remove hexachloropentadiene from water (USEPA 2006).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Hexachlorocyclopentadiene affects mainly the liver and kidneys, most commonly via inhalation, by which route it is extremely toxic. The general population is not at risk from exposure to HEX, except in the case of people residing near contaminated areas.

The USEPA has classified hexachlorocyclopentadiene in Group D, not classifiable as to human carcinogenicity.

As at July 2013 ATSDR (http://www.atsdr.cdc.gov/mrls/mrls\_list.html) quotes a minimal risk level (MRL) of 0.1 mg/kg/day for intermediate-duration oral exposure  
(15–364 days) to hexachlorocyclopentadiene.

The reference dose or RfD (USEPA 2001 and 2006/2009/2011) for hexachlorocyclopentadiene is 0.006 mg/kg/d, based on stomach lesions in rats. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.2 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA established an organoleptic effect criterion of 0.001 mg/L for hexachlorocyclopentadiene. Source: [*Quality Criteria for Water* 1986 (“Gold Book”)](Quality%20Criteria%20for%20Water%201986%20(), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

The odour threshold of hexachlorocyclopentadiene in water is 0.0014 mg/L.

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USEPA. 2011. *2011 Edition of the Drinking Water Standards and Health Advisories*. Washington, DC: US Environmental Protection Agency. Available at: [http://water.epa.gov/action/advisories/health\_index.cfm](http://water.epa.gov/action/advisories/drinking/drinking_index.cfm%20) or www.epa.gov/waterscience/.

# Hexachloroethane

CAS No. 67-72-1. Also called perchloroethane, hexachloroethylene, carbon hexachloride, ethane hexachloride, or 1,1,1,2,2,2-hexachloroethane.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for hexachloroethane. The WHO Guidelines do not have a guideline value for hexachloroethane.

The USEPA (2006/2011) established a lifetime health advisory of 0.001 mg/L, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

Hexachloroethane is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Hexachloroethane is used in [aluminium](http://en.wikipedia.org/wiki/Aluminium) [foundries](http://en.wikipedia.org/wiki/Foundry) and by the military for smoke-producing devices. Hexachloroethane may be present as an ingredient in some fungicides, insecticides, lubricants, and plastics. It has also been used to inhibit the explosiveness of methane and combustion of ammonium perchlorate. Hexachloroethane is rarely detected in surface waters or biota and has not been reported in ambient soil, sediments or commercial food products.

#### 2. From treatment processes

Traces of hexachloroethane can be formed when chlorine reacts with carbon compounds in drinking water.

### Fate and form in the environment

Hexachloroethane in lakes or streams and surface soils will evaporate into the air. Microscopic organisms can break it down more easily without oxygen than with oxygen. Hexachloroethane does not appear to build up in plants or animals used for food. Hexachloroethane is relatively persistent in the environment.

Hexachloroethane is fairly soluble in water, more than 12 mg/L, some reports as high as 50 mg/L.

### Typical concentrations in drinking-water

Hexachloroethane has occasionally been reported in drinking-water at concentrations of 0.00003 to 0.0004 mg/L in some locations in the United States.

One water utility in the US reported detecting hexachloroethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.0025 mg/L.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Hexachloroethane is not a very toxic substance. Exposure to large amounts for a long time can affect the liver and kidneys. Animal studies have not shown hexachloroethane to cause birth defects or to affect reproduction.

Liver tumours developed in mice that were orally exposed to hexachloroethane for their whole lifetime. Hexachloroethane will not necessarily have the same effect on people. Male rats that were exposed to hexachloroethane for their lifetime developed kidney tumours. This type of tumour is not found in people.

The International Agency for Research on Cancer considers that there is sufficient evidence in experimental animals for the carcinogenicity of hexachloroethane, and has classified hexachloroethane as possibly carcinogenic to humans (Group 2B). The USEPA has determined that hexachloroethane is a possible human carcinogen. The USEPA (2009/2011) quotes a health advisory of 0.3 mg/L for hexachloroethane, representing a 10-4 cancer risk.

Hexachloroethane appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at July 2013 ATSDR (http://www.atsdr.cdc.gov/mrls/mrls\_list.html) quotes a minimal risk level (MRL) of:

* 1 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.01 mg/kg/day for intermediate-duration oral exposure (15–364 days).

The reference dose or RfD (USEPA 1987/2006/2009/2011) is 0.001 mg/kg/d (rounded from 0.0007 mg/kg/d). This value is based on a NOAEL of 1 mg/kg/day for atrophy and degeneration of the renal tubules in rats exposed for 16 weeks. The NOAEL was divided by an uncertainty factor of 1,000 to account for interspecies extrapolation, human variability, and the use of a subchronic study. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.04 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

The odour threshold of hexachloroethane in water has been reported as low as 0.01 mg/L.

### References

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USEPA. 2011. Toxicological review of hexachloroethane. In *Support of Summary Information on the Integrated Risk Information System (IRIS)*. EPA/635/R-09/007F. 200 pp. <http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showSubstanceList>.

# 2-hexanal

CAS No. 66-25-1. Also called hexaldehyde, n-hexanal and caproaldehyde or caproic aldehyde.

This datasheet also briefly discusses some other aldehydes nei.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2-hexanal. The WHO Guidelines do not have a guideline value for 2-hexanal.

### Sources to drinking-water

#### 1. To source waters

2-Hexanal, a C-6 aldehyde, is one of the causes of grassy odours in water, and generally arises from phytoplankton. It has also been described as “like lettuce”, and occasionally as fishy (due to flagellated algae and diatoms). An offensive cod liver oil type odour caused by *Synura uvella* has been attributed to aldehydes. C-6 aldehydes are found in many other plants as well, including tea and coffee.

Heptanal, a C-7 aldehyde, often occurs as well, and has a more rancid odour.

Octanal has been used in foodstuffs as a flavouring agent in the US and Europe since the 1900s (USEPA 2000).

Nonanal, a C-9 aldehyde, is produced at high volumes and used in the manufacture of personal care products and household products.

Solubility of 2-hexanal in water is 5,000–6,000 mg/L.

#### 2. From treatment processes

Aldehydes, including 2-hexanal, have been reported to increase after oxidative treatment processes, eg, ozone disinfection, probably due to the oxidation of amino acids and double bonds. If subsequently passed through a biologically active filter the concentration may reduce.

Aldehydes in the Los Angeles water treatment plant have been measured before and after dosing with ozone (quoted from Faust and Aly 1998) as follows (micrograms/L):

|  |  |  |
| --- | --- | --- |
| **Aldehyde** | **Raw water** | **Treated water** |
| hexanal | 0.008 | 0.40 |
| heptanal | 0.008 | 2.00 |
| octanal | 0.047 | 0.21 |
| nonanal | 0.139 | 0.46 |
| decanal | 0.615 | 1.08 |
| undecanal | 0.075 | 0.40 |
| dodecanal | 0.014 | 0.11 |
| tridecanal | 0.004 | 0.10 |
| tetradecanal | 0.016 | 0.11 |

#### 3. From the distribution system

Aldehydes have been detected in water leaching from PVC pipes. Only trace amounts of hexanal and octanal were found in the test water from the three successive tests for PVC pipes, whereas nonanal and decanal were present in concentrations from 280 ng/L (0.00028 mg/L) or less (Skjevrak et al 2003).

Fadel et al reported hexanal and heptanal (each at 0.0007 mg/L) to have been the cause of odour problems when oleate-based pipe joint lubricants were used on new mains or repairs.

Being reasonably volatile, 2-hexanal may be more noticeable in water from the hot tap.

### Typical concentrations in drinking-water

See above for levels found in Los Angeles.

One water utility in the US reported detecting propanal in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.001 mg/L.

Five water utilities in the US reported detecting pentanal in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0012 mg/L.

Fourteen water utilities in the US reported detecting butanal in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0037 mg/L.

Two water utilities in the US reported detecting heptanal in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00012 mg/L.

One water utility in the US reported detecting nonanal in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00092 mg/L.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

GC/MS techniques have been used to measure very low concentrations.

### Health considerations

No adverse effects were reported when hexanal was given to rats in drinking-water at concentrations of 1, 10, 100 and 1000 mg/L (calculated to provide doses of about 0.1, 1.2, 12.6 and 124.7 mg/kg bw per day) for four weeks (IPCS 1998).

### Derivation of Maximum Acceptable Value

No MAV.

The odour threshold of 2-hexanal in water has been reported as low as  
0.0001–0.002 mg/L.

### References

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# Hexane

CAS No. 110-54-3 for n-hexane, which is the unbranched 6-carbon alkane (hydrocarbon). Also known as hexyl hydride and normal hexane. The four isomers of C6H14 are methylated derivatives of pentane and butane. They are:

* 2-methylpentane CAS No. 107-83-5
* 3-methylpentane CAS No. 96-14-0
* 2,2-dimethylbutane CAS No. 75-83-2
* 2,3-dimethylbutane CAS No. 79-29-8

### Maximum Acceptable Value

The DWSNZ do not have a MAV for hexane. The WHO Guidelines do not have a guideline value for hexane.

The USEPA concluded on 22 September 2009 that [n-hexane](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane) is known or anticipated to occur in PWSs and may require regulation. Therefore they have added [it](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane) to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

n-Hexane is a very volatile aliphatic hydrocarbon. It is a constituent in the paraffin fraction of crude oil and natural gas and is also used as an industrial chemical and laboratory reagent. Laboratory grade n-hexane contains approximately 99 percent n‑hexane. “Hexane” or “hexanes” is a commercial and industrial product consisting of as little as 52 percent n-hexane, plus a mixture of hydrocarbons with six carbon atoms, including n-hexane and its isomers 2-methylpentane and 3-methylpentane, as well as small amounts of other hydrocarbons such as toluene, 2,3-dimethyl butane, methylcyclopentane or such solvents as acetone or methyl ethyl ketone. Laboratory and industrial solvents such as petroleum ether contain n-hexane from <0.1 percent to as much as 33 percent. In commercial grades of n-hexane, some of the constituents are purposefully added as denaturants, often to discourage the abuse of the chemical to induce “highs” through sniffing or inhalation.

The major use for solvents containing n-hexane is to extract vegetable oils from crops such as soybeans, flaxseed, peanuts, and safflower. They are also used as cleaning agents or solvents in the printing, textile, furniture, and shoemaking industries. Certain kinds of special glues used in the roofing and the shoe and leather industries also contain n-hexane. Several consumer products contain n-hexane. n-Hexane is also present in rubber cement and paint thinners.

n-Hexane is a minor constituent of crude oil and natural gas and, therefore, represents a variable proportion of different petroleum distillates. For example, n-hexane comprises about 11.6 percent of unleaded gasoline and about 2 percent of JP-4 aviation fuel. n-Hexane and the 4 isomers are frequently found in motor vehicle fuel and exhaust.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

If n-hexane is spilled into a lake or river, some will dissolve in the water, but most will float on the surface and then evaporate into the air.

If n-hexane is spilled on the ground, much of it will evaporate into the air before it penetrates the soil. Any n-hexane that penetrated the soil would probably be broken down by bacteria. If n-hexane leaks from an underground storage tank, it will float on the groundwater, rather than mixing with it since n-hexane is lighter than water.

If released to soil, n-hexane is expected to have high mobility based upon an estimated Koc of 130. Volatilisation from moist soil surfaces is expected to be an important fate process based upon an estimated Henry’s Law constant of 1.80 atm‑cu m/mole. n‑Hexane may volatilise from dry soil surfaces based upon its vapour pressure. Utilising the Japanese MITI test, 100 percent of the Theoretical BOD was reached in four weeks indicating that biodegradation is an important environmental fate process in soil and water. If released into water, n-hexane is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is expected to be an important fate process based on its estimated Henry’s Law constant. An estimated BCF of 170 suggests the potential for bioconcentration in aquatic organisms is high, provided the compound is not metabolized by the organism. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (pH 5 to 9) (EAWAG accessed February 2015).

Water solubility of n-hexane is about 9–10 mg/L.

### Typical concentrations in drinking-water

Three water utilities in the US reported detecting n-hexane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.005 mg/L.

### Removal methods

Low concentrations of hexane will be removed by aeration.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

The main route to the body is by inhalation, or absorption through the skin. n-Hexane is metabolised in the body to a number of metabolites. One of these metabolites, 2,5‑hexanedione, is believed to be the ultimate toxic agent in n-hexane-induced neurotoxicity; this metabolite also produced testicular toxicity following oral exposure in rats (IPCS 1991). These metabolites are excreted from the body in the urine within a few days of exposure.

Oral NOAELs for intermediate exposure (15 to 364 days) n-hexane have been reported at about the 500 mg/kg/d level.

The USEPA has established a one-day health advisory for water (child) of 10 mg/L; a 10‑day health advisory for water (child) of 4 mg/L; and a lifetime advisory (adult) of 0.75 mg/L. These are well above the odour threshold in water which is about 0.006 mg/L. USEPA (2011) dropped the adult lifetime advisory.

n-Hexane has not been characterised for carcinogenicity by the Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), or the Environmental Protection Agency (USEPA). USEPA (2005) states that there is *inadequate information to assess the carcinogenic potential of n-hexane*. Specifically, there are no animal carcinogenicity studies available that examine exposure to n‑hexane. Studies indicate that n-hexane is mostly non-genotoxic in short-term testing protocols.

The database for oral exposure to n-hexane is limited to two subchronic gavage studies, both of which were unsuitable for the calculation of an RfD (USEPA 2005).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for hexane is 0.4 mg/L.

A related chemical, 2-hexanone (methyl *n-*butyl ketone), which is an n-hexane metabolite, has also caused peripheral neuropathy in workers.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# 2-hexanone

CAS No. 591-78-6. Also called methyl n-butyl ketone, methyl butyl ketone MBK, MnBK, propylacetone, n-butyl methyl ketone, butyl methyl ketone and 2-oxohexane.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2-hexanone. The WHO Guidelines do not have a guideline value for 2-hexanone.

### Sources to drinking-water

#### 1. To source waters

2-Hexanone is a waste product of wood pulping, coal gasification, and oil shale operations. 2-Hexanone was formerly used in paint and paint thinner, as a solvent for lacquers, ink thinners, nitrocellulose, resins, oils, fats, and waxes, and in various chemical substances. Since it was found to have harmful health effects, it is no longer made in the US, and its uses have been restricted.

2-Hexanone has been found as a natural substance in foods such as cheese, nectarines, nuts, bread, and chicken muscle. 2-Hexanone has been found in milk and cream at levels up to 0.018 mg/L. These levels are far below the levels that have caused harmful effects in animals.

In technical grade 2-hexanone, methyl isobutyl ketone (MIBK, qv) may appear up to 30 percent as an impurity.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

When 2-hexanone is released to rivers or lakes, it dissolves very easily (water solubility has been reported to be about 1.5 to 3.5 percent). It is expected to be quite mobile in water and soils. Biodegradation of 2-hexanone may occur slowly in water and soil, but bioconcentration is not expected. The magnitude of the estimated Henry’s law constant (4.4 to 7.7 x 10-5 atm-m3/mole) indicates that 2-hexanone will volatilise from water, with a half-life in river water of about 10 to 15 days. Volatilisation will be slower from lakes or ponds. 2-Hexanone is a ketone, and ketones are generally not degraded by hydrolysis.

### Typical concentrations in drinking-water

Seventeen water utilities in the US reported detecting 2-hexanone in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0025 mg/L.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

If 2-hexanone enters the body by mouth, about 65 percent of the chemical leaves the body slowly (in about a week), either unchanged or as breakdown products, in the breath and urine. The rest may either stay in the body or may leave the body slowly through the breath or urine. One of the breakdown products, 2,5-hexanedione, may be responsible for the harmful effects on the nervous system. Chronic and subchronic studies conducted with rats, hens, and guinea pigs provide evidence that the nervous system is the target of toxicity following oral exposure to 2-hexanone (USEPA 2009).

USEPA (2009) states that there is “inadequate information to assess the carcinogenic potential” of 2-hexanone. Specifically, there are no animal carcinogenicity studies available that examine exposure to 2-hexanone, and there are no studies available that assert a mutagenic potential of 2-hexanone. The available occupational studies do not present evidence for carcinogenic action of 2-hexanone, although these are limited by frequent co-exposure to other chemicals (eg, MEK).

For an intermediate level (15 to 364 days) of oral exposure, a NOAEL of around 400 mg/kg/d seems likely (ATSDR). A chronic MRL of 0.05 mg/kg/d was as at August 2018.

USEPA (2009) derived a chronic RfD (for axonal swelling of the peripheral nerve as the critical effect) for 2-hexanone of 0.005 mg/kg/d, based on a BMDL10 (ADJ) of 5 mg/kg/d and an uncertainty factor of 1000.

The odour threshold for 2-hexanone in water is about 0.25 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

### References

ATSDR. 1992 and 2009 addendum. *Toxicological Profile for n-Hexanone*. Atlanta, GA: Agency for Toxic Substances & Disease Registry, Department of Health and Human Services. See: [http://www.atsdr.cdc.gov/toxprofiles/index.asp](http://www.atsdr.cdc.gov/toxpro2.html).

Environmental Working Group (EWG). Accessed 2010. *National Drinking Water Database – Chemical Contaminants*. <http://www.ewg.org/tap-water/chemical-contaminants>.

USEPA. 2009. Toxicological review of 2-hexanone. *In Support of Summary Information on the Integrated Risk Information System (IRIS)*. EPA/635/R-09/008F. 136 pp. See: <http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showSubstanceList> see also http://www.epa.gov/iris/subst/1019.htm.

# Cis-3-hexenol

CAS No. 928-96-1. Also called cis-hex-3-en-1-ol, cis-3-hexen-1-ol, (Z)-3-hexen-1-ol and leaf alcohol. A mixture of cis-3-hexen-1-ol and trans-2-hexenal is called green odour.

### Maximum Acceptable Value

Cis-3-hexenol is not mentioned in the WHO Guidelines, and does not have a MAV in the DWSNZ.

### Sources to water

cis-3-Hexenol is derived from leaching of chemicals from grass and vegetation, and from growth of green algae. The compound is produced in plants through the action of the enzyme lipoxygenase. cis-3-Hexenol imparts a pleasant, but nevertheless atypical, grassy flavour to water. The compound often occurs in association with cis‑3‑hexenyl acetate.

cis-3-Hexen-1-ol has been identified as a natural emission from 29 agricultural and natural plant species found in the California’s central valley.

cis-3-Hexen-1-ol is an [alcohol](http://en.wikipedia.org/wiki/Alcohol) and its [esters](http://en.wikipedia.org/wiki/Ester) are also important flavour and fragrance raw materials. The related [aldehyde](http://en.wikipedia.org/wiki/Aldehyde) [cis-3-hexenal](http://en.wikipedia.org/wiki/Cis-3-Hexenal) (leaf aldehyde) has a similar and even stronger smell but is relatively unstable and [isomerises](http://en.wikipedia.org/wiki/Isomerization) into the [conjugated](http://en.wikipedia.org/wiki/Conjugated_system) trans‑2‑hexenal.

cis-3-Hexen-1-ol is a very important [aroma compound](http://en.wikipedia.org/wiki/Aroma_compound) that is used in fruit and vegetable [flavours](http://en.wikipedia.org/wiki/Flavor) and in [perfumes](http://en.wikipedia.org/wiki/Perfume). It is also added to cigarettes. The yearly production is about 30 [tonnes](http://en.wikipedia.org/wiki/Tonne).

Cis-3-Hexen-1-ol is reportedly used in baked goods at 14.5 ppm, fats and oils at 5.80 ppm, frozen dairy at 15.3 ppm, meat products at 58.0 ppm, gelatin pudding at 12.0 ppm, non-alcoholic beverages at 9.10 ppm, hard candy at 10.5 ppm, and chewing gum at 98.5 ppm.

### Forms and fate in the environment

cis-3-Hexenol is unstable at high pH values and does not survive well in alkaline waters.

cis-3-Hexenol is said variously to be “slightly soluble”, “very slightly soluble” or “insoluble” and “floats on water”. One reference (<http://www.ymdb.ca/compounds/YMDB01420>) suggests 0.016 mg/L water solubility.

### Recommended analytical techniques

#### Referee method

No MAV.

### Health considerations

The total estimated per capita intake in Europe is 4.3 mg/person of cis-3-hexen-1-ol, ie, 0.071 mg/kg bw per day, (IPCS, 1999). A NOEL of 120–150 mg/kg bw per day was reported for cis-3-hexen-1-ol in a 98-day study in rats. Therefore a safety margin of >1,000 exists between this NOEL and the daily per capita intake.

### Derivation of Maximum Acceptable Value

No MAV.

The flavour threshold of cis-3-hexenol in water is 0.95 mg/L (Aroxa); the odour threshold is 0.00025 mg/L (Leffingwell).

### References

Aroxa Sales Sheet. *Water Taints Kit*. <http://www.aroxa.com/water/water-flavour-standard-kit/water-taints-kit>.

EA. 1998. The assessment of taste, odour and related aesthetic problems in drinking waters 1998. *Methods for the Examination of Waters and Associated Materials*. Environment Agency (UK). [www.netregs.eu/default.aspx](http://www.netregs.eu/default.aspx) and enter title in search box.

EA. 2004. The microbiology of drinking water (2004) – Part 11 – Taste, odour and related aesthetic problems. *Methods for the Examination of Waters and Associated Materials*. Environment Agency. [www.netregs.eu/default.aspx](http://www.netregs.eu/default.aspx) and enter title in search box.

IPCS. 1999. Safety Evaluation of Certain Food Additives; *WHO Food Additives Series*: 42. Linear and Branched-Chain Aliphatic, Unsaturated, Unconjugated Alcohols, Aldehydes, Acids, and Related Esters. <http://www.inchem.org/documents/jecfa/jecmono/v042je16.htm>.

Leffingwell & Associates. *Odour and Flavour Detection Thresholds in Water (In Parts per Billion)*. http://www.leffingwell.com/odorthre.htm.

Tobacco Information. August 2011. Cis-3-Hexen-1-ol. 10 pp. <http://tobacco-information.bhp.doh.gov.tw/toxicfolder/011.%E5%B8%9D%E5%9C%8B%E8%8F%B8%E8%8D%89/131.pdf>.

# HHCB

CAS No. 1222-05-5. Also called 1,3,4,6,7,8-hexahydro-4,6,6,7,8,8-hexamethylcyclopenta-γ-2-benzopyran (IUPAC and CAS name), 1,3,4,6,7,8-hexahydro-4,6,6,7,8,8-hexamethylindeno(5,6-c)pyran, hexahydrohexamethyl cyclopentabenzopyran, 4,6,6,7,8,8-hexamethyl-1,3,4,6,7,8-hexahydrocyclopenta[g]isochromene, or galaxolide and other trade names. This is the main (74 to 76 percent) isomer. Three other isomers exist – see EU (2008).

Undiluted HHCB is a highly viscous liquid that is impractical to use and handle as a fragrance ingredient. In order to make it manageable for application, it is fluidised with an odour neutral diluent. These dilutions in various solvents are manufactured by blending approximately 65 weight parts of undiluted HHCB with 35 weight parts of diluent. The product name Galaxolide or Galaxolide 50 refers to HHCB diluted with diethyl phthalate (DEP), the most common form of commercial HHCB. This product is at times referred to as Galaxolide 50 DEP. In some cases, the HHCB is diluted with benzyl benzoate (BB) or isopropyl myristate (IPM). When so diluted, it is designated as Galaxolide 50 BB and Galaxolide 50 IPM. The ‘50’ added to the name reflects the main isomer content of ca 50 percent. Taken from EU (2008).

### Maximum Acceptable Value

HHCB does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

Galaxolide has a taste and odour threshold of 5 μg/L (0.005 mg/L).

### Sources to drinking-water

#### 1. To source waters

IEH (2014) selected for consideration all those substances reported as being involved in taste and odour incidents in a developed country, excluding those for which there was no evidence of UK production or import, as well as those already regulated to a limit value either lower than or close to the reported taste and odour threshold. Other prioritised substances were then categorised according to amounts used and their reported taste and odour threshold. This process gave a list of compounds from which substances formed during water treatment were excluded leaving 18 priority compounds.

One, galaxolide (a registered trademark of International Flavours and Fragrances) is considered important to the fragrance industry because it imparts unique odour properties, has the ability to improve fragrance compound fixation and binds fragrances to fabrics. It is used in a very wide range of consumer products such as perfumes, soaps, cosmetics, fabric softener, cleaners and detergents. It is categorised within the group of fragrance materials referred to collectively as ‘polycyclic musks’ and is the main component of this group (IEH 2014).

IEH (2014) reports that in Europe about 15 mg is used per head per day, most ending up in the household wastewater stream.

EU (2008) reports HHCB at 0.01 to 0.03 mg/L in raw sewage in Germany and 0.0012 mg/L in effluent, and 0.0064 mg/L (raw), 0.00014 mg/L (effluent in Holland. The median concentration in several European freshwaters ranged from 0.00001 to 0.0003 mg/L.

#### 2. From treatment processes

No known sources.

### Form and fate in the environment

EU (2008) quotes: vapour pressure = 0.073 Pa at 25°C; Henry’s law constant = 36.9 Pa.m3/mol at 25°C; water solubility = 1.65 mg/L at pH 7; partition coefficient = log Kow = 6.2. The half-life for photodegradation was 109 and 135 h in lake water and distilled water, respectively. The overall biodegradation half-life of the parent in water is 100 hours. In the river die-away test with 0.5 μg/L the parent HHCB disappeared with a half-life of 43 hours to 100 hours (four days) at 5 μg/L.

Galaxolide exhibits a low vapour pressure (5.45 x 10-4 mm Hg) and water solubility is 1.75 mg/L. As such, it is likely to volatilise easily from damp soil although sorption to soil particles may limit this process (IEH 2014).

HHCB is expected to have low mobility in soil, bind strongly to benthic and suspended sediment, and be substantially removed by sorption to sludge in wastewater treatment plants. In river water, the degradation half-life of HHCB has been determined as 33 to 43 hours. The overall half-life for HHCB was concluded to be 100 hours. Volatilisation half-lifes were in the order of days to weeks. It is unlikely to be rapidly removed from soil through biological degradation. Estimated volatilisation half-lifes from modelled stationary and flowing water sources were 10 days and 18 hours respectively. Galaxolide’s propensity to sorb on to suspended particulate material may increase this period in static water sources by a factor of 90 (IEH 2014).

### Removal methods

Galoaxolide is not removed by the drinking water treatments of coagulation, flocculation or clarification. Chlorine may reduce the concentration by about 35 percent. Ozone may halve the concentration. The use of granular activated carbon (GAC) and powdered activated carbon (PAC) are, however, reasonably effective in reducing the concentration (IEH 2014).

### Health considerations

Three studies on galaxolide’s toxicity in humans were identified; all examined toxicity in response to dermal rather than oral or inhalation exposure. Volunteers were exposed to galaxolide though impregnated patches; galaxolide concentrations ranged between 1 percent and 25 percent, the longest tested exposure period was four days. All three studies reported no observable toxicity under any of the test conditions (IEH 2014).

A European Union Summary Risk Assessment Report examining galaxolide toxicity reports the findings of a 13-week oral toxicity study in which rats were exposed to galaxolide (at 5.4, 15.7, 51.8 or 155.8 mg/kg/day). Whilst minor alterations to haematological characteristics were observed between treatment groups, no clear dose-response relationship could be established, indicating that no significant toxicity was elicited. An *in vivo* assessment of reproductive and developmental toxicity in rats established a NOAEL of 50 mg/kg/day. A recent examination of galaxolide toxicity in a peer reviewed journal has suggested a TDI (Tolerable Daily Intake) of 0.5 mg/kg. From IEH (2014).

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2014. *National Assessment of the Risks to Water Supplies Posed by Low Taste and Odour Threshold Compounds*. Final Report Project WT1275. 202 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-281.pdf>.

EU. 2008. *European Union Risk Assessment Report*. 1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta-γ-2-benzopyran. (1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylin-deno[5,6-c]pyran – HHCB. 251 pp. <http://echa.europa.eu/documents/10162/947def3b-bbbf-473b-bc19-3bda7a8da910> or <http://echa.europa.eu/information-on-chemicals/information-from-existing-substances-regulation>.

IEH. 2014. *National Assessment of the Risks to Water Supplies Posed by Low Taste and Odour Threshold Compounds*. Final Report Project WT1275. Institute of Environment and Health, Cranfield University. 202 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-281.pdf>.

# Hydroxybenzoic acids

There are three hydroxybenzoic acids (the IUPAC names):

* 2-hydroxybenzoic acid – CAS No. 69-72-7. The common name is salicylic acid. Also called 2-hydroxybenzenecarboxylic acid, orthohydroxybenzoic acid, 2‑carboxyphenol or 2-HBA
* 3-hydroxybenzoic acid – CAS No. 99-06-9. Also called metahydroxybenzoic acid or 3-HBA
* 4-hydroxybenzoic acid – CAS No. 99-96-7. Also called parahydroxybenzoic acid, 4‑HBA or 4-hydroxybenzenecarboxylic acid.

### Maximum Acceptable Value

Hydroxybenzoic acids are not mentioned in the WHO Guidelines, and do not have a MAV in the DWSNZ.

### Sources to water

2-Hydroxybenzoic acid (salicylic acid) is widely used in [organic synthesis](http://en.wikipedia.org/wiki/Organic_synthesis) (including the production of aspirin) and functions as a [plant hormone](http://en.wikipedia.org/wiki/Plant_hormone)/growth regulator. It is used as a preservative in many products (eg, cosmetics), but is probably best known for its use in anti-acne treatments. 2-Hydroxybenzoic acid is a [phenolic](http://en.wikipedia.org/wiki/Phenol) [phytohormone](http://en.wikipedia.org/wiki/Phytohormone) and is found in plants with roles in plant growth and development, [photosynthesis](http://en.wikipedia.org/wiki/Photosynthesis), [transpiration](http://en.wikipedia.org/wiki/Transpiration), [ion](http://en.wikipedia.org/wiki/Ion) uptake and transport. It also induces specific changes in leaf anatomy and [chloroplast](http://en.wikipedia.org/wiki/Chloroplast) structure. 2-Hydroxybenzoic acid is involved in [endogenous](http://en.wikipedia.org/wiki/Endogenous) signalling, mediating in plant defence against [pathogens](http://en.wikipedia.org/wiki/Pathogens).

3-Hydroxybenzoic acid is used mainly in the production of pesticides, dyestuffs, cosmetics and medical products. 3-Hydroxybenzoic acid and 4-hydroxybenzoic acid have been detected at the several ppm level in some coffees, and some other foods.

4-Hydroxybenzoic acid is used as an antioxidant. 4-Hydroxybenzoic acid (and its derivatives) has antimicrobial properties and has been used as preservatives since the 1920s; the concentration is usually between 0.1–1 percent. It is active against bacteria, yeast and moulds and used to preserve a wide range of different foods. Esters between 4‑hydroxybenzoic acid and methanol, ethanol, propanol, butanols and benzylalcohol are called parabens. p-Hydroxybenzoic acid alkyl esters (parabens) are antimicrobial preservatives allowed for use in foods, drugs, cosmetics and toiletries. They are normally used in combinations containing two or more parabens and/or other preservatives. Under EC Directive 95/2/EC, Annex III, methyl-, ethyl- and propyl parabens and their sodium salts (E214–219) are conditionally permitted for use in a limited number of foods in combination with either sorbates or sorbates and benzoates. This chemical is used as intermediate for pesticide, antiseptics and pharmaceuticals.

No hydroxybenzoic acids appear on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)).

However, 2-hydroxybenzoic acid is listed in Table 1 of ERMA’s Summary of Approvals of Substances Transferred under the Hazardous Substances (Pesticides) Transfer Notice 2004 (as amended), as at 22 May 2008 (<http://www.ermanz.govt.nz/hs/transfer/summaryapprove.html>, then select pesticides …. It is listed as the potassium salt.

And 3-hydroxybenzoic acid and 4-hydroxybenzoic acid are listed in Table 1 of ERMA’s Summary of Approvals of Substances Transferred under the Hazardous Substances (Chemicals) Transfer Notice 2006 (as amended), as at 24 June 2008 (<http://www.ermanz.govt.nz/hs/transfer/summaryapprove.html>, then select chemicals ….).

### Forms and fate in the environment

If released to soil, salicylic acid is expected to have moderate mobility based upon an estimated Koc of 404. The pKa of salicylic acid is 2.98, indicating that it will exist primarily in the anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilisation from moist soil surfaces is not expected to be an important fate process because anions do not volatilise. Salicylic acid is unlikely to volatilise from dry soil surfaces based upon its vapour pressure. If released into water, salicylic acid is expected to adsorb to suspended solids and sediment based upon the estimated Koc; volatilisation from water surfaces is not expected to be an important fate process. Biodegradation in the environment is an important fate process. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

If released to soil, 4-hydroxybenzoic acid is expected to have high to very high mobility based upon Koc values ranging from 142 to 14. The pKa of 4-hydroxybenzoic acid is 4.54, indicating that this compound will primarily exist in anion form and anions generally do not adsorb more strongly to organic carbon and clay than their neutral counterparts. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 7.2 x 10-12 atm‑cu m/mole and because this compound will primarily exist as an anion in the environment. 4-Hydroxybenzoic acid is not expected to volatilise from dry soil surfaces based upon its vapour pressure. 4-Hydroxybenzoic acid is expected to be readily degraded in soil and water environments based on its rapid mineralisation in both aerobic and anaerobic screening studies; it is also fairly rapidly degraded in both aerobic and anaerobic microcosm studies using aquifer sediment or river sediment. If released into water, 4-hydroxybenzoic acid is not expected to adsorb to suspended solids and sediment based upon its measured Koc values. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. In addition, 4-hydroxybenzoic acid’s pKa value indicates that it will exist almost entirely as an anion at pH values of 5 to 9 and therefore volatilisation from water surfaces is not expected to be an important fate process. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

* The water solubility of 2-hydroxybenzoic acid is about 2,200 mg/L.
* The water solubility of 3-hydroxybenzoic acid is about 7,250 mg/L.
* The water solubility of 4-hydroxybenzoic acid is about 5,000–6,000 mg/L.

### Recommended analytical techniques

#### Referee method

No MAV.

### Health considerations

The EC Scientific Committee for Food (SCF) evaluated the parabens in 1994 and established a temporary Acceptable Daily Intake (ADI) of 0–10 mg/kg bw, as the sum of methyl, ethyl and propyl p-hydroxybenzoic acid esters and their sodium salts. The temporary ADI was based on long-term studies in rats with methyl, ethyl and propyl paraben. The common metabolite of parabens, p-hydroxybenzoic acid, was considered to be non-oestrogenic.

4-Hydroxybenzoic acid: Reproductive toxicity was not observed up to the highest test dose of 1000 mg/kg/day, suggesting no reason for concern. This chemical is not genotoxic, based on negative results of bacterial mutation test and chromosomal aberration test *in vitro*. In vaginal cornification and uterotrophic assay of mice, this chemical showed estrogenic response. The NOAEL for systemic toxicity was considered to be 1,000 mg/kg/day (OECD 2000).

### Derivation of Maximum Acceptable Value

No MAV.

### References

EAWAG. Accessed February 2015. *Biocatalysis/Biodegradation Database: Select 1396 compounds*. <http://eawag-bbd.ethz.ch/index.html>.

EFSA. 2004. Opinion of the Scientific Panel on food additives, flavourings, processing aids and materials in contact with food (AFC) related to para hydroxybenzoates  
(E214–219). <http://www.efsa.europa.eu/en/scdocs/scdoc/83.htm>.

OECD. 2000. *SIDS Initial Assessment Report*: 4-Hydroxybenzoic acid. 44 pp. See: http://www.inchem.org/documents/sids/sids/99967.pdf or <http://www.inchem.org/pages/sids.html>.

# 2-hydroxy-5-nitrobenzoic acid

CAS No. 96-97-9. Also called 5-nitrosalicylic acid and 5-nitro-2-hydroxybenzoic acid.

### Maximum Acceptable Value

2-Hydroxy-5-nitrobenzoic acid is not mentioned in the WHO Guidelines, and does not have a MAV in the DWSNZ.

### Sources to water

#### 1. To source waters

2-Hydroxy-5-nitrobenzoic acid is a raw material and intermediate used in organic synthesis of pharmaceuticals, agrochemicals and dyestuffs.

#### 2. From treatment processes

2-Hydroxy-5-nitrobenzoic acid is a potential disinfection by-product (DBP) in WTPs using an advanced oxidation process (AOP).

### Forms and fate in the environment

Solubility in water, about 600 mg/L.

### Typical concentrations in drinking-water

The maximum concentration of 2-hydroxy-5-nitrobenzoic acid was 0.056 µg/L. The daily intake was estimated at 0.0019 µg/kg bw for an adult, 0.0056 for a child and 0.0084 for an infant.

### Recommended analytical techniques

#### Referee method

No MAV.

### Health considerations

DWI (2018) estimates the Threshold of Toxicological Concern (TTC) to be 0.0025 µg/kg bw/d. They did not find a TDI. 2-Hydroxy-5-nitrobenzoic acid is a skin and eye irritant. 2-Hydroxy-5-nitrobenzoic acid was negative in the Ames test and in vitro mammalian chromosome aberration test. It has a potential for DNA binding. It has genotoxic potential but is not mutagenic (Vughs D, et al 2016).

The maximum intake by drinking water for an adult (0.0019 µg/kg bw) is less than the TTC value (0.0025 µg/kg) so adverse effects in adults are not anticipated.

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2018. *Potential for Formation of Disinfection By-products from Advanced Oxidation Processes*. Report DWI 12852.02. 260 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-317.pdf>.

Kolkman A, et al. 2017. *Application of Effect Directed Analysis to Identify Mutagenic Nitrogenous Disinfection By-products after Advanced Oxidation Drinking Water Treatment*. KWR. Watercycle Research Institute. Netherlands. <https://www.mn.uio.no/kjemi/english/research/projects/ICCE2017/wednesday-21.06/helga-eng-auditorium-2/hr.-10:30/1130-kolkman.pdf>.

Vughs D, et al. 2016. *Tracing Genotoxic Disinfection By-products after Medium Pressure UV Water Treatment using Nitrogen Labelling, Mass Spectrometry and Effect Directed Analysis*. KWR. Watercycle Research Institute. Netherlands. <https://www.eawag.ch/fileadmin/Domain1/Abteilungen/uchem/analytik/pdf/Vughs_Oral.pdf>.

# 4-hydroxy-3-nitrobenzoic acid

CAS No. 616-82-0. Also called 3-nitro-4-hydroxybenzoic acid and 3,4‑nitrohydroxybenzoic acid.

### Maximum Acceptable Value

4-Hydroxy-3-nitrobenzoic acid is not mentioned in the WHO Guidelines, and does not have a MAV in the DWSNZ.

### Sources to water

#### 1. From treatment processes

4-Hydroxy-3-nitrobenzoic acid is a potential disinfection by-product (DBP) in WTPs using an advanced oxidation process (AOP).

### Typical concentrations in drinking-water

The maximum concentration of 4-hydroxy-3-nitrobenzoic acid in drinking water was 0.042 µg/L. The daily intake was estimated at 0.0014 µg/kg bw for an adult, 0.0042 for a child and 0.0063 for an infant.

### Recommended analytical techniques

#### Referee method

No MAV.

### Health considerations

Due to structural alerts for genotoxicity DWI (2018) estimates the Threshold of Toxicological Concern (TTC) to be 0.0025 µg/kg bw/d. They did not find a TDI. 4‑Hydroxy-3-nitrobenzoic acid has a potential for DNA binding (Vughs et al 2016, Kolkman et al 2017).

The maximum intake by drinking water for an adult (0.0014 µg/kg bw) is less than the TTC value (0.0025 µg/kg) so adverse effects in adults are not anticipated. The maximum intake in children and infants (0.00422 to 0.00633 μg/kg bw/day) is greater than the TTC value. Therefore, additional research into the occurrence in drinking water and toxicological properties of this DBP may be prudent.

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2018. *Potential for Formation of Disinfection By-products from Advanced Oxidation Processes*. Report DWI 12852.02. 260 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-317.pdf>.

Kolkman A, et al. 2017. *Application of Effect Directed Analysis to Identify Mutagenic Nitrogenous Disinfection By-products after Advanced Oxidation Drinking Water Treatment*. KWR. Watercycle Research Institute. Netherlands. <https://www.mn.uio.no/kjemi/english/research/projects/ICCE2017/wednesday-21.06/helga-eng-auditorium-2/hr.-10:30/1130-kolkman.pdf>.

Vughs D, et al. 2016. *Tracing Genotoxic Disinfection By-products after Medium Pressure UV Water Treatment using Nitrogen Labelling, Mass Spectrometry and Effect Directed Analysis*. KWR. Watercycle Research Institute. Netherlands. <https://www.eawag.ch/fileadmin/Domain1/Abteilungen/uchem/analytik/pdf/Vughs_Oral.pdf>.

# 3-hydroxypropanenitrile

CAS No. 109-78-4. Also called 3-hydroxy propanenitrile, 3-hydroxypropionitrile, 3‑hydroxy-propionitrile, hydracrylonitrile, 2-cyanoethan-1-ol, β-cyanoethanol, ethylene cyanohydrin and glycol cyanohydrin.

### Maximum Acceptable Value

3-Hydroxypropanenitrile is not mentioned in the WHO Guidelines, and does not have a MAV in the DWSNZ.

NSF Standard 60 states that the allowable concentration of 3-hydroxypropanenitrile in drinking water is 0.01 mg/L.

### Sources to water

#### 1. To source waters

3-Hydroxypropanenitrile is used in adhesives, as a chemical intermediate and an industrial solvent.

#### 2. From treatment processes

3-Hydroxypropanenitrile can be a contaminant in polyacrylamide polyelectrolytes and is regulated by NSF/ANSI Standard 60.

### Forms and fate in the environment

If released to soil, ethylene cyanohydrin is expected to have very high mobility based upon an estimated Koc of 1. Volatilisation from moist soil surfaces is not expected to be an important fate process based on an estimated Henry’s Law constant of 4.3 x 10-10 atm‑cu m/mole. Biodegradation in soil may be an important environmental fate process as ethylene cyanohydrin is confirmed to be biodegradable according to the standard test of the Japanese Ministry of Industry and Trade (MITI). If released into [water](http://pubchem.ncbi.nlm.nih.gov/compound/water), ethylene cyanohydrin is not expected to adsorb to suspended solids and sediment in [water](http://pubchem.ncbi.nlm.nih.gov/compound/water) based upon the estimated Koc. Volatilisation from [water](http://pubchem.ncbi.nlm.nih.gov/compound/water) surfaces is not expected to be an important fate process based its estimated Henry’s Law constant. An estimated BCF of 3.2 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to occur under typical environmental conditions due to the lack of readily hydrolysable functional groups. Biodegradation in [water](http://pubchem.ncbi.nlm.nih.gov/compound/water) may be an important environmental fate process as ethylene cyanohydrin is confirmed to be biodegradable. From NIH.

Miscible in water. LogP = log Kow= -0.94.

### Typical concentrations in drinking-water

NSF (2010) reports levels from <0.00001 to 0.0007 mg/L of 3-hydroxypropanenitrile in 113 samples of drinking water that had been treated with polyacrylamide coagulant aids. The median concentration was 0.00003 mg/L.

### Recommended analytical techniques

#### Referee method

No MAV.

### Derivation of Maximum Acceptable Value

No MAV.

### References

NIH. Accessed September 2015. 3-Hydroxypropionitrile. *PubChem Open Chemistry Database*. <http://pubchem.ncbi.nlm.nih.gov/compound/3-Hydroxypropionitrile>.

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# Indeno[1,2,3-c,d]pyrene

Indeno[1,2,3-c,d]pyrene, CAS No. 193-39-5, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet. Also called 1,10-(1,2-phenylene)pyrene.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; benzo[a]pyrene (BaP) levels range from 6 to 35 mg/kg TEQ depending on land use. The equivalent BaP concentration is calculated as the sum of each of the detected concentrations of nine carcinogenic PAHs which are benz(a)anthracene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene and indeno(1,2,3-cd)pyrene, multiplied by their respective potency equivalency factors.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

Indeno(1,2,3-c,d)pyrene concentration ranges in water have been found to be: 0.2–0.5 ng/L in surface water, 0.3–4.8 ng/L in tap water, 0.2–8.7 ng/L in rainfall, 0.2–5.0 ng/L in subterranean water (Irwin 1997).

### Typical concentrations in drinking-water

No data are available on the concentration of indeno[1,2,3-c,d]pyrene in New Zealand drinking-water supplies. Indeno [1,2,3-c,d] pyrene has been detected in some Canadian water supplies (detection limits 0.000001–0.000006 mg/L (1–6 ng/L)). Ten water utilities in the US reported detecting indeno[1,2,3-cd]pyrene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00033 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation. Pyrene was the most rapidly degraded PAH. Benz[a]anthracene, benzo[a]pyrene, and perylene were also highly degraded. Indeno[1,2,3-c,d]pyrene and benzo[g,h,i]pyrene were intermediate with respect to relative degradation. Benzo[k]fluoranthene and fluoranthene were the most slowly degraded of the compounds tested. A variety of complex end-products have been identified (ATSDR 1995).

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons.

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess. The relative carcinogenic potencies of various polynuclear aromatic hydrocarbons, based on bioassays by several routes of administration and related toxicological data, have been ranked in decreasing order as follows: dibenz[a,h]anthracene, benzo[a]pyrene, anthanthrene, indeno[1,2,3-cd]pyrene, benz[a]anthracene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, benzo[j]fluoranthene, cyclopentadieno[c,d]pyrene, benzo[g,h,i]perylene, chrysene and benzo[e]pyrene.

The USEPA (1990) classified indeno[1,2,3-*cd*]pyrene as B2, a probable human carcinogen.

IARC (2010) classified indeno[1,2,3-*cd*]pyrenein Group 2B (possible human carcinogen).

Indeno[1,2,3-c,d]pyrene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

No MAV.

The only time that WHO had a guideline value for other than benzo[a]pyrene was in their 1971 International Standards, which stated that some PAHs are known to be carcinogenic and that the concentrations of six representative PAH compounds (fluoranthene, 3,4-benzofluoranthene, 11,12-benzofluoranthene, 3,4-benzopyrene, 1,12-benzopyrene and indeno[1,2,3-cd]pyrene) should not in general exceed 0.0002 mg/L.

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# Iodinated DBPs

Trihalomethanes (THMs) are traditionally halogen-substituted single-carbon compounds with the general formula CHX3, where X represents a halogen, which may be fluorine, chlorine, bromine, or iodine, or combinations thereof. The datasheet for trihalomethanes covers just chloroform, bromodichloromethane, dibromochloromethane and bromoform, ie, no iodo-THMs. Concern has arisen in more recent years due to there being evidence to suggest that iodinated-DBPs may be of greater toxicological concern than their brominated and chlorinated analogues, despite occurring at much lower concentrations.

There are individual datasheets for iodoacetic acid and iodoform.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for iodinated disinfection by-products. The WHO Guidelines do not have a guideline value for iodinated disinfection by-products.

### Sources to drinking-water

#### 1. From treatment processes

The practice of adding iodine to water is not considered to be common place, however, it can be used in emergency situations, and the military are known to use it as a purification method whilst out in the field, and there has been reference to its use on space missions (eg, Dodd 1997).

Iodo-reactions occur when iodide present in the source water is oxidised, mainly to hypoiodous acid; note however, that ozone can oxidise iodide to iodate which reduces the potential for iodinated-THM formation. It is considered that in general increasing iodide concentrations in the source waters in turn increase the iodinated-DBPs formation. Generally source waters with the highest bromide levels contain the highest concentrations of iodide as well; however, this relationship is not consistent.

The six iodo-THMs have been implicated as DBPs (unregulated) in chlorinated and chloraminated waters, and are:

* bromochloroiodomethane CAS No. 34970-00-8
* chlorodiiodomethane CAS No. 638-73-3
* dichloroiodomethane CAS No. 594-04-7
* bromodiiodomethane CAS No. 557-95-9
* dibromoiodomethane CAS No. 593-94-2
* iodoform CAS No. 75-47-8

It has been suggested that the concentration of iodo-THMs lessens as the chlorine dose increases, and that chloramination (with ammonia addition before chlorine addition) increases the formation of iodo-THMs (Richardson 2005).

There is evidence that the formation of iodinated DBPs is increased by chloramination and reduced by ozonation and that iodinated-THMs may be removed by GAC to some extent. Chloramination is not common in the UK, while ozonation and GAC are widely used. Taking all this information, together with modelling which estimates the formation of iodinated DBPs and limited monitoring data, it appears likely that the levels of iodinated DBPs in England and Wales will be no higher and will probably be lower than the low concentrations detected in the USA. It should be noted that the introduction of a standard for haloacetic acids in England and Wales may lead to an increased use of chloramination and if this occurred, further consideration of the concentration of iodinated DBPs in drinking water would be advisable (DEFRA 2009).

Iodoacetic acid has been detected in chloraminated water in several US drinking waters (Richardson 2006). Richardson (2008) in a 23-city study found the iodo-acids and iodo-THMs were highest at plants with short free chlorine contact times (<1 minute) and lowest at plants with long free chlorine contact times (>45 minutes).

DEFRA (2009) quotes from a 2002 US study by Weinberg that the five iodo-acids that were identified in finished drinking water (treated with chloramine) were: iodoacetic acid; bromoiodacetic acid; (Z)-3-bromo-3-iodopropenoic acid; (E)-3-bromo-3-iodopropenoic acid; and (E)-2-iodo-3-methylbutenedioic acid.

Some of the iodinated THAAs would be expected to rapidly degrade to iodoform, half-lifes (days) in water at 23°C are reported in WRF (2016) to be:

* dichloroiodoacetic acid DCIAA 57
* bromochloroiodoacetic acid BCIAA 9.1
* dibromoiodoacetic acid DBIAA 1.4
* chlorodiiodoacetic acid CDIAA 0.89
* bromodiiodoacetic acid BDIAA 0.14
* triiodoacetic acid TIAA 0.014

In the WRF (2016) study the highest dichloroiodomethane formation (1.75 μg/L) was in a water treated with chloramine. Iodide can be oxidised by a wide range of oxidants into hypoiodous acid (HIO) which can react quickly with NOM to produce I‑THMs. However, had free chlorine or ozone also been used, the HOI would have been oxidised to iodate, stopping the formation of iodinated organic matter.

#### 2. From the distribution system

No known sources.

### Forms and fate in the environment

Environmental occurrence insignificant.

### Typical concentrations in drinking-water

USEPA (2006) and DEFRA (2009) report that dichloroiodomethane and bromochloroiodomethane are the most commonly found iodo-THMs. For example, dichloroiodomethane has been reported to be detected in 85 out of 111 USA water supplies and is reportedly more commonly found than bromoform.

Dichloroiodomethane was detected at the highest concentration (total mean concentration from all 12 plants in the US study, 15.9 μg/L), followed by bromochloroidomethane (11.0 μg/L) and iodoform and dibromoiodomethane (8.85 and 8.29 μg/L, respectively). Plant 12 was observed to have the greatest total iodinated-DBPs concentration (13.23 μg/L), followed by plants 2 and 8 with 5.16 and 4.94 μg/L, respectively; mean raw water bromide concentrations ranged from 0.06 to 0.3 mg/L.

Seven water utilities in the US reported detecting dichloroiodomethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.002 mg/L.

Iodoacetic acid concentrations reported by Richardson (2006 and 2008) ranged from 0.02 to 0.06 micrograms/L, and up to 1.7 micrograms/L. Bromoiodoacetic acid reached 1.4 micrograms/L.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See DEFRA (2009).

### Health considerations

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. Some of their findings were:

* diiodoacetamide was the most cytotoxic agent
* a majority (75.8 percent) induced significant levels of genomic DNA damage. In this group, iodoacetic acid was the most genotoxic.

Iodinated-THMs are associated with causing taste and odour problems at concentration ranges as low as 0.00002–0.0005 mg/L (Richardson et al 2007 – quoted in DEFRA 2009). Therefore, it is considered that the presence or absence of unexplained taste and odour effects can be a good indication of the occurrence of iodinated DBPs, in particular iodinated-THMs at or about the threshold concentrations.

Richardson et al (2008) found the iodinated THMs to be much less cytotoxic than the iodinated acids, with the exception of iodoform. Iodoform was found to be mutagenic in bacteria but did not induce chromosome aberrations in Syrian hamster embryo cells *in vitro*. Of the iodinated THMs studied by Richardson et al (2008), only chlorodiiodomethane was found to be genotoxic. Richardson et al (2008) noted that BrTHMs require glutathione-S-transferase-theta1 (GSTT1) mediated metabolism to form mutagenic intermediates, and it is not known whether this is expressed in the CHO cells used in the Richardson et al (2008) experiment with the iodinated acetic acids. Stated in USEPA (2016).

### Derivation of Maximum Acceptable Value

No MAV.

### References

DEFRA. 2009. *Review of the Current Toxicological and Occurrence Information Available on Iodinated Disinfection By-Products*. WRc Ref: DEFRA 7883.03 DWI Ref: 70/2/233. 104 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70_2_233.pdf>.

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# Iodoacetic acid

CAS No. 64-69-7. Also called monoiodoacetic acid (MIAA). Some other iodoacids (iodinated acids) are discussed briefly in this datasheet too. See also previous datasheet: Iodinated DBPs.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for iodoacetic acid. The WHO Guidelines do not have a guideline value for iodoacetic acid.

### Sources to drinking-water

#### 1. From treatment processes

Iodoacetic acid has been detected in chloraminated water in several US drinking waters (Richardson 2006). Richardson (2008) in a 23-city study found the iodo-acids and iodo-THMs were highest at plants with short free chlorine contact times (<1 minute) and lowest at plants with long free chlorine contact times (>45 minutes).

Other iodoacids (in water treated with chloramine) have been reported by Richardson (2006), including bromoiodoacetic acid (BIAA), (Z)-3-bromo-3-iodopropenoic acid, (E)‑3-bromo-3-iodopropenoic acid, and (E)-2-iodo-3-methylbutenedioic acid. See also the iodinated DBPs datasheet for some data on iodinated trihaloacids.

Iodinated acids identified in drinking water in the United States include MIAA and BIAA. Several longer-chain iodinated acids were also identified as present in treated drinking water: (*Z*)- and (*E*)-3-bromo-3-iodopropenoic acid and (*E*)-2-iodo-3-methylbutenedioic acid. USEPA (2016).

#### 2. From the distribution system

No known sources.

### Forms and fate in the environment

Iodoacetic acid is very soluble in water, about 10 percent.

### Typical concentrations in drinking-water

Iodoacetic acid concentrations reported by Richardson (2006 and 2008) ranged from 0.02 to 0.06 micrograms/L, and up to 1.7 micrograms/L. Bromoiodoacetic acid reached 1.4 micrograms/L.

Iodoacetic and chloroiodoacetic acids were formed when municipal chlorinated tap water was allowed to react with iodised table salt; iodoacetic acid was reported at 1.5 micrograms/L (Becalski et al 2006).

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See Lau and Becalski (2008).

### Health considerations

Iodoacetic acid is a toxic compound, because, like many alkyl halides, it is an [alkylating agent](http://en.wikipedia.org/wiki/Alkylating_agent) which can react with [cysteine](http://en.wikipedia.org/wiki/Cysteine) residues in proteins. In a series of cell cytotoxicity genotoxicity tests conducted by Plewa (of University of Illinois) and reported in Richardson (2006), iodoacetic acid and iodoacetamide were the most toxic DBPs.

Several studies have shown iodoacetate has anti-tumour effects.

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. Some of their findings were:

* diiodoacetamide was the most cytotoxic agent
* a majority (75.8 percent) induced significant levels of genomic DNA damage. In this group, iodoacetic acid was the most genotoxic.

MIAA is the most cytotoxic and genotoxic haloactic acid (HAA) in mammalian cell assays that has been reported in the literature. Similar results are seen when comparing MIAA mutagenicity in *S. typhimurium* and genotoxicity in CHO cells compared to MBAA and MCAA. Wei et al (2013) examined cytotoxicity, genotoxicity and ability to transform NIH3T3 cells to tumorigenic lines and found that prolonged exposure of NIH3T3 cells to MIAA increased the frequencies of transformed cells with anchorage-independent growth and agglutination with concanavalin A. They found that neither MIAA (nor iodoform) increased micronucleus frequency, but that MIAA-transformed cells formed aggressive fibrosarcomas after inoculation into Balb/c nude mice. They concluded that MIAA has a biological activity that is consistent with a carcinogen and that human exposure should be of concern. Taken from USEPA (2016).

### Derivation of Maximum Acceptable Value

No MAV.

### References

Beacalski, et al. 2006. Formation of iodoacetic acids during cooking: Interaction of iodized table salt with chlorinated drinking water. [*Food Additives and Contaminants*](http://www.ingentaconnect.com/content/tandf/tfac;jsessionid=5565s3bsl9pue.alexandra) 23(10): 957–62. Abstract http://www.ingentaconnect.com/content/tandf/tfac/2006/00000023/00000010/art00001.

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USEPA. 2016. *Six-Year Review 3 Technical Support Document for Disinfectants/ Disinfection By-products Rules*. EPA-810-R-16-012. 468 pp. USEPA Office of Water. <https://www.epa.gov/sites/production/files/2016-12/documents/810r16012.pdf>.

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# Iodoform

CAS No. 75-47-8. IUPAC name is triiodomethane. Also called methyl tri-iodide, or methyl triiodide. See also datasheet for Iodinated DBPs.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for iodoform. The WHO Guidelines do not have a guideline value for iodoform.

### Sources to drinking-water

#### 1. To source waters

Iodoform is still sometimes used as a disinfectant on wounds and sores, and in some specialist medical and dental procedures, sometimes mixed with bismuth sub-nitrate.

#### 2. From treatment processes

The six iodo-THMs have been implicated as DBPs (unregulated) in chlorinated and chloraminated waters, and are:

* bromochloroiodomethane CAS No. 34970-00-8
* chlorodiiodomethane CAS No. 638-73-3
* dichloroiodomethane CAS No. 594-04-7
* bromodiiodomethane CAS No. 557-95-9
* dibromoiodomethane CAS No. 593-94-2
* iodoform CAS No. 75-47-8

USEPA (2006) report that dichloroiodomethane and bromochloroiodomethane are the most commonly found iodo-THMs. It has been suggested that the concentration of iodo-THMs lessens as the chlorine dose increases, and that chloramination (with ammonia addition before chlorine addition) increases the formation of iodo-THMs (Richardson 2005).

Hansen et al (1987) reported that the presence of iodoform in concentrations >0.005 mg/L was associated with an objectionable medicinal taste in water being disinfected with chloramine. Production of iodoform was dependant on the order of addition of the chlorine and ammonia.

When iodinated trihalomethanes occur, a medicinal taste or musty odour is imparted to the water. While there are only a few reports of iodoform in treated drinking water in the literature, strong medicinal odours have long been known to occur in such water.

In research for her MS, Dodd (1997) found that under her test conditions, acetone was the only compound identified as an iodinated DBP precursor and it reacted to produce iodoacetone and iodoform (the other organic precursors studied were formaldehyde, 1-propanol, isopropanol, 1-methoxy-2-propanol, methanol, and ethanol). Concentrations of iodoform from 0.34 mg/L to 8.64 mg/L were produced at conditions that included each pH level, iodine concentration, and acetone concentration. The greatest iodoform concentration was produced at pH 8 from 50 mg/L of iodine and acetone. Flavour profile analysis indicated that the odour threshold concentration (OTC) of iodoform was 0.0015 mg/L and the OTC of iodine was 0.60 mg/L. Both iodine and iodoform have medicinal odours, making it difficult to distinguish each compound when present in a mixture.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

If released to soil, iodoform is expected to have very high mobility based upon an estimated Koc of 35. Volatilisation from moist soil surfaces is expected to be an important fate process based upon an estimated Henry’s Law constant of 3.1 *x* 10-5 atm‑cu m/mole. Iodoform’s biodegradation potential in soil or water is unknown. If released into water, iodoform is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is expected to be an important fate process based upon this compound’s estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 40 hours and 25 days, respectively. An estimated BCF of 43 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important fate process because the estimated hydrolysis rate is low.

Water solubility is about 100 mg/L.

### Typical concentrations in drinking-water

Iodoform was found in two French water treatment plants at 80 ng/l and 10–20 ng/l, respectively. This was associated with the presence of chloramines in the water being treated. In the absence of chloramines, chlorine tends to quickly react with organic precursors to preferentially form chlorinated, brominated, and iodated trihalomethanes. One of the French treatment plants used ammonia-rich groundwater (from Toxnet).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

DEFRA (2009) reports: no data have been located on the toxicokinetics of iodoform.

A NOAEL of 178 mg/kg bw/day for male rats and an arbitrary LOAEL of 56 mg/kg bw/day for female rats can be identified from a short term study. NOAELs of 56 and 100 mg/kg bw/day can be identified for male and female mice, respectively, also from a short term study. In rats, an arbitrary LOAEL of 71 mg/kg bw/day for males and a NOAEL of 55 mg/kg bw/day for females have been identified from a long-term study and a NOAEL of 93 mg/kg bw/day has been identified for male and female mice.

Results indicate that iodoform has some genotoxic potential *in vitro*, but the *in vivo* mutagenicity of iodoform cannot be assessed, owing to lack of data. No evidence of a statistically significant increase in tumours in rats and mice due to the administration of iodoform was found in well reported long-term studies.

For a chemical with a threshold level for toxicity, it would be possible to use the NOAELs from the above studies to derive a Tolerable Daily Intake (TDI) for humans using appropriate uncertainty factors. However, iodoform, although it is not carcinogenic in animal tests, is potentially mutagenic as it is positive in the Ames test and there are no data for *in vivo* mutagenicity to confirm or deny this potential. Therefore, until further toxicity data are available, it must be assumed to have no threshold of effect and exposure should be as low as reasonably practicable. Therefore, in the absence of sufficient toxicological data, it is not possible to identify a concentration of iodoform (either as a TDI or a potential concentration in water) that is of no concern for human health.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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Richardson S. 2006. *Overview of Emerging Contaminants of Concern in Drinking Water*. Athens, GA: US Environmental Protection Agency, National Exposure Research Laboratory. [www.usawaterquality.org/conferences/2006/presentations/Richardson.pdf](http://www.usawaterquality.org/conferences/2006/presentations/Richardson.pdf).

Richardson S. 2008. Occurrence and mammalian cell toxicity of iodinated disinfection by‑products in drinking water. *Environ Sci Technol* 42(22): 8330–8. Abstract: <http://www.novoseek.com/DocumentDetailAction.action?docId=19068814&corpus=MEDLINE>.

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USEPA. 2006. Occurrence of iodo-acid and iodo-THM disinfection by-products in drinking water. Science Inventory. Richardson, et al (authors). <http://cfpub.epa.gov/si/si_public_record_report.cfm?dirEntryId=152943>.

# Β-ionone

CAS No. 79-77-6; some references quote 14901-07-6. The IUPAC name for β-ionone is (3*E*)-4-(2,6,6-trimethylcyclohex-1-en-1-yl)but-3-en-2-one.

The ionones are also called the rose ketones, beta-ionone, cyclocitrylideneacetone, irisone, and jonon. Various ionones are used for flavouring, including allyl-α-ionone, β‑isomethylionone, pseudoionone, and *trans*-α-damascone.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for β-ionone. The WHO Guidelines do not have a guideline value for β-ionone.

### Sources to drinking-water

#### 1. To source waters

β-Ionone is a significant contributor to the aroma of roses despite its relatively low concentration, and is an important fragrance chemical used in perfumes. It is also found in violets, freesias and boronia.

In the year 2003 the world production of industrial β-ionone was between 4,000 and 8,000 tonnes pa. β-Ionone occurs also naturally in food (some plants eg, corn, red wine and beef) and plant extracts used for example in perfumes. Typical use concentrations in final products are 0.03 percent (soap), 0.003 percent (detergent), 0.016 percent (creams, lotions) and 0.3 percent (perfume).

The ionones are derived from the degradation of plant [carotenoids](http://en.wikipedia.org/wiki/Carotenoid).

β-Ionone was found in concentrations ranging from 0.002 μg/L up to 1.2 μg/L (0.0012 mg/L) in waters of lakes and rivers mainly due to biotransformation processes in phytoplankton. It is commonly found in taste and odour studies in waters containing cyanobacteria, usually along with geosmin, MIB and β-cyclocitral (qv).

β-Ionone is a certified water flavour standard used to train professional tasters to recognise and scale the intensity of the character of violets; it is marketed by Aroxa. Ionone imparts a pleasant, but nevertheless atypical, sweet, aromatic note to water. This compound is formed by algae or cyanobacteria in water and through the chemical decay of grass and vegetation in lakes and reservoirs.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

According to distribution modelling, water (34 percent), soil (27 percent), sediment (27 percent) and air (12 percent) are the main targets, and β-ionone is readily biodegradable according to OECD criteria (INCHEM 2004).

No data on stability of β-ionone in water are available, but due to the chemical structure a hydrolysis reaction is not expected. The estimated log KOC-values of 2.80 to 3.34 indicate a potential for significant adsorption to soil, sediments and suspended solids. In a biodegradation test on stability in water using spiked river water, only 5 percent of the β-ionone starting concentration (0.006 mg/L) was detected after 20 hours incubation at 22°C under normal light conditions.

β-Ionone has a water solubility of about 170 mg/L.

### Removal methods

Its attraction to solids suggests that processes the remove particulate matter from water should reduce the concentration of β-ionone.

### Analytical methods

#### Referee method

A referee method cannot be selected because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

### Health considerations

β-Ionone has only low acute toxicity after oral ingestion; clinical signs of toxicity were depression and tremors.

The no-observed-effect-level (NOEL) was about 7 and 8 mg/kg bw/day for males and females based on adaptive liver effects in both sexes and minor urine findings in males at 1000 ppm which correspond to a dosage of 72 and 83 mg/kg bw/day for males and females (no-observed-adverse-effect-level, NOAEL). The lowest-observed-adverse-effect-level (LOAEL) was found at 10,000 ppm (720 and 801 mg/kg bw/day for males and females) due to liver, kidney and thyroid findings in both sexes. Based on the results of a GLP and guideline conforming developmental toxicity study (OECD TG 414) with gavage application of β-ionone, the no observed adverse effect level (NOAEL) for maternal toxicity was 100 mg/kg bw/day. The NOAEL for prenatal developmental toxicity could be fixed at the highest tested dose (400 mg/kg bw/day) (INCHEM 2004).

WHO (2009) states “No safety concern at current levels of intake when used as a flavouring agent. The 1984 group ADI of 0–0.1 mg/kg bw for alpha-ionone and beta-ionone, singly or in combination, was maintained at the 51st meeting (1998)”.

WHO (2015) reported that the 28th meeting established a group ADI of 0–0.1 mg/kg body weight. The weight of evidence supports the conclusion that this group of flavouring compounds lacks genotoxic potential.

### Derivation of Maximum Acceptable Value

No MAV.

The odour threshold of β-ionone in water is recorded in INCHEM (2004) at a concentration of 1 μg/L (0.001 mg/L). The flavour threshold in water is reported by Aroxa as 13 µg/L or 0.013 mg/L.

### References

Aroxa Sales Sheet. β-Ionone. <http://www.aroxa.com/water/water-flavour-standard/ionone/>.

INCHEM. 2004. β-Ionone [(E)-4-(2,6,6-trimethyl-1-cyclohexen-1-yl)-3-buten-2-one]. *SIDS Initial Assessment Report*. UNEP Publications. 171 pp. <http://www.inchem.org/documents/sids/sids/79776.pdf>.

WHO. 2009. [*Evaluations of the Joint FAO/WHO Expert Committee on Food Additives (JECFA)*](http://apps.who.int/ipsc/database/evaluations/Default.aspx)*: alpha-IONONE*. <http://apps.who.int/ipsc/database/evaluations/chemical.aspx?chemID=901>.

WHO. 2015. Safety evaluation of certain food additives. *WHO Food Additives Series* 70. Prepared by the 79th meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). 378 pp. <http://www.who.int/foodsafety/publications/monographs/en/>.

# Isobutane nitrile

CAS No. 78-82-0. For synonyms, see <http://www.chemicalbook.com/CASEN_78-82-0.htm>, which includes 2-methylpropanenitrile, 2-methylpropionitrile, 2-methyl-propanenitrile, isobutyronitrile, isopropylcyanide, isopropyl cyanide, 1-cyano-1-methylethane, 2-cyanopropane, alpha-methylpropanenitrile, dimethylacetonitrile, isobutyricacidnitrile and iBN.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for isobutane nitrile. The WHO Guidelines do not have a guideline value for isobutane nitrile.

NSF Standard 60 states that the allowable concentration of isobutane nitrile in drinking water is 0.0003 mg/L.

### Sources to drinking-water

#### 1. To source waters

Isobutyronitrile is used in the synthesis of insecticides and gasoline additives. Nitriles are mostly industrially produced, as intermediates and building blocks in organic synthesis and as organic solvents, but there are also a few examples of naturally occurring nitriles, formed by cyanogenic plants from cyanide. In addition, simple aliphatic nitriles, such as isobutyronitrile (iBN), can be produced during the anaerobic degradation of amino acids.

#### 2. From treatment processes

AWWA (1999) lists isobutyronitrile as a potential contaminant of polyacrylamide polyelectrolytes.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

If released to soil, 2-methylpropanenitrile is expected to have very high mobility based on an estimated Koc of 42. Volatilisation from moist soil surfaces is expected to be an important fate process based on an estimated Henry’s Law constant of 5.39 x 10-5 atm‑cu m/mole. 2-Methylpropanenitrile may volatilise from dry soil surfaces based on its vapour pressure. If released into [water](http://pubchem.ncbi.nlm.nih.gov/compound/water), 2-methylpropanenitrile is not expected to adsorb to suspended solids and sediment based on the estimated Koc. 2‑Methylpropanenitrile has been shown to biodegrade readily using the Japanese MITI screening test with theoretical BOD values of 53.9–66.3 percent; this compound has a high probability of fast biodegradation in the environment. Volatilisation from [water](http://pubchem.ncbi.nlm.nih.gov/compound/water) surfaces is expected to be an important fate process based on its estimated Henry’s Law constant. Estimated volatilisation half-lives for a model river and model lake are 10 hours and seven days, respectively. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low (NIH).

### Typical concentrations in drinking-water

NSF (2010) reports levels from <0.00001 to 0.00003 mg/L isobutane nitrile in drinking water that has been treated with polyacrylamide coagulant aids. The median concentration was 0.000005 mg/L.

LogP = log Kow = 0.46.

### Analytical methods

#### Some alternative methods

By gas chromatography-flame ionisation detector.

### Health considerations

Aliphatic nitriles have been postulated to manifest their toxicity through cyanide liberation.

Some other aliphatic nitriles are discussed in *Acute Exposure Guideline Levels (AEGLS) for Selected Aliphatic Nitriles*. <http://www.epa.gov/oppt/aegl/pubs/aliphaticnitriles_tsd_interimversion_1_07_2007.pdf>.

Rats exposed at 38.6 mg/kg orally showed definite parenchymatous degeneration of the liver, with male rats showing a greater degree of degeneration than females. Increased weight of stomach, liver, and adrenal glands were found at doses of 200 mg/kg orally. It was concluded that isobutyronitrile causes hepatic damage (NIH).

### Derivation of Maximum Acceptable Value

No MAV.

### References

AWWA. 1999. *Analytical Methods for Polymers and their Oxidative By-products*. AWWA/AWWARF.

NIH. Accessed September 2015. Isobutyronitrile. *PubChem Open Chemistry Database*. [http://pubchem.ncbi.nlm.nih.gov/compound/Isobutyronitrile#section=Top](http://pubchem.ncbi.nlm.nih.gov/compound/Isobutyronitrile%23section=Top).

NSF. 2010. *NSF Fact Sheet: Polyelectrolytes and NSF/ANSI Standard* 60. 12 pp. <http://www.nsf.org/newsroom_pdf/NSFFactSheetPolyelectrolytes.pdf>.

WRc. 2013. *Potential Contaminants in Drinking Water Treatment Chemicals: Final Report*. Report No. Defra9033.03. 129 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-272.pdf>.

# 2-isobutyl-3-methoxypyrazine

CAS No. 24683-00-9.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2-isobutyl-3-methoxypyrazine. The WHO Guidelines do not have a guideline value for 2-isobutyl-3-methoxypyrazine.

### Sources to drinking-water

#### 1. To source waters

2-Isobutyl-3-methoxypyrazine, described variously as causing a musty, earthy, odour in water, is a metabolite from biological activity of many micro-organisms such as fungi, actinomycetes and streptomycetes. The threshold odour concentration has been reported to be as low as 0.01 ng/L (0.00000001 mg/L) (Young et al 1996).

2-Isobutyl-3-methoxypyrazine has been described as one of the five most important water odorants (the others being 2-isopropyl-3-methoxypyrazine, 2-methylisoborneol, 2,4,6-trichloroanisole and geosmin, qv).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Removal methods

2-Isobutyl-3-methoxypyrazine can be removed through biologically active sand filters. Ozone can also be effective, moreso than with MIB (Atasi et al 1999).

Powdered activated carbon can be used but the various grades need to be trialled; a dose of up to 20 mg/L may be required.

Chlorine and chlorine dioxide may be able to reduce the concentration of 2-isobutyl-3-methoxypyrazine by at least 80 percent using a dose of up to 10 mg/L; ozone may need a dose greater than 15 mg/L, and potassium permanganate was ineffective (Faust and Aly 1998).

### Analytical methods

#### Referee method

A referee method cannot be selected because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

Various GC/MS methods can measure down to or less than 1 ng/L in water, eg, Palmentier and Taguchi (2001).

### Health considerations

2-Isobutyl-3-methoxypyrazine does not present a health risk in drinking-water.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Atasi KZ, Chen T, Huddleston JI, et al. 1999. Factor screening for ozonating the taste- and odour-causing compounds in source water at Detroit, USA. *Water Science and Technology* 40(6): 115–22.

Environment Agency. 1998. The assessment of taste, odour and related aesthetic problems in drinking waters 1998. *Methods for the Examination of Waters and Associated Materials*. London: EA Standing Committee of Analysts. Available at: [www.environment-agency.gov.uk/static/documents/Research/171\_taste\_\_odour\_in\_water.pdf](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.environment-agency.gov.uk\static\documents\Research\171_taste__odour_in_water.pdf).

Faust SD, Aly OM. 1998. *Chemistry of Water Treatment* (2nd edition). CRC Press, ISBN 1575040115. 581 pp.

Palmentier, Taguchi. 2001. The determination of six taste and odour compounds in water using Ambersorb 572 and high resolution mass spectrometry. *The Analyst* 126: 840–5. See [www.rsc.org/ej/AN/2001/b008013f.pdf](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.rsc.org\ej\AN\2001\b008013f.pdf).

Young WF, Horth H, Crane R, et al. 1996. Taste and odor threshold concentrations of potable water contaminants. *Water Research* 30: 331–40.

# 2-isopropyl-3-methoxypyrazine

CAS No. 25773-40-4.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2-isopropyl-3-methoxypyrazine. The WHO Guidelines do not have a guideline value for 2-isopropyl-3-methoxypyrazine.

### Sources to drinking-water

#### 1. To source waters

2-Isopropyl-3-methoxypyrazine, described variously as causing a musty, earthy, decaying vegetation, or potato odour in water, is a metabolite from biological activity of many micro-organisms such as fungi, actinomycetes and streptomycetes. The threshold odour concentration has been reported to be as low as 0.03 ng/L (0.00000003 mg/L) (Young et al 1996).

2-Isopropyl-3-methoxypyrazine has been described as one of the five most important water odorants (the others being 2-isobutyl-3-methoxypyrazine, 2-methylisoborneol, 2,4,6-trichloroanisole and geosmin).

Some insects use 2-isopropyl-3-methoxypyrazine as part of their communication system; in the wine industry is called the ladybird taint. It can be detected by the human nose at about 1 ng/L in fruit juice.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Removal methods

2-Isopropyl-3-methoxypyrazine can be removed through biologically active sand filters. Ozone can also be effective, moreso than with MIB (Atasi et al 1999).

Powdered activated carbon can be used but the various grades need to be trialled; a dose of up to 20 mg/L may be required.

Chlorine and chlorine dioxide may be able to reduce the concentration of 2-isopropyl-3-methoxypyrazine by at least 80 percent using a dose of up to 10 mg/L; ozone may need a dose greater than 15 mg/L, and potassium permanganate was ineffective (Faust and Aly 1998).

### Analytical methods

#### Referee method

A referee method cannot be selected because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

Various GC/MS methods can measure down to or less than 1 ng/L in water.

### Health considerations

2-Isopropyl-3-methoxypyrazine does not present a health risk in drinking-water, eg, Palmentier and Taguchi (2001).

### Derivation of Maximum Acceptable Value

No MAV.

### References

Atasi KZ, Chen T, Huddleston JI, et al. 1999. Factor screening for ozonating the taste- and odour-causing compounds in source water at Detroit, USA. *Water Science and Technology* 40(6): 115–22.

Environment Agency. 1998. The assessment of taste, odour and related aesthetic problems in drinking waters 1998. *Methods for the Examination of Waters and Associated Materials*. London: EA Standing Committee of Analysts. Available at: [www.environment-agency.gov.uk/static/documents/Research/171\_taste\_\_odour\_in\_water.pdf](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.environment-agency.gov.uk\static\documents\Research\171_taste__odour_in_water.pdf).

Faust SD, Aly OM. 1998. *Chemistry of Water Treatment* (2nd edition). CRC Press, ISBN 1575040115. 581 pp.

Palmentier, Taguchi. 2001. The determination of six taste and odour compounds in water using Ambersorb 572 and high resolution mass spectrometry. *The Analyst* 126: 840–5. See [www.rsc.org/ej/AN/2001/b008013f.pdf](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.rsc.org\ej\AN\2001\b008013f.pdf).

Young WF, Horth H, Crane R, et al. 1996. Taste and odor threshold concentrations of potable water contaminants. *Water Research* 30: 331–40.

# Melamine

CAS No. 108-78-1. The IUPAC name is melamine. The CAS name is 1,3,5-triazine-2,4,6-triamine. Synonyms include cyanuramide, cyanurotriamide, cyanurotriamine, isomelamine, triaminotriazine, 2,4,6-triaminotriazine, triamino-s-triazine, 2,4,6‑triamino-1,3,5-triazine, 2,4,6-s-triazinetriamine, and 1,3,5-triazine-2,4,6(1H,3H,5H)-triimine.

Cyanuric acid (qv) is an analogue of melamine. Trichloromelamine is used as a sanitiser.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for melamine. The WHO Guidelines do not have a guideline value for melamine.

### Sources to drinking-water

#### 1. To source waters

Melamine is used in the manufacture of melamine resins, laminates, surface coating resins, plastic moulding compounds, textile resins, bonding resins, gypsum–melamine resin mixtures, orthopaedic casts, rubber additives and paper products. Melamine is a significant degradate of the pesticide cyromazine (qv).

Melamine, melamine cyanurate, other melamine salts and guanidine compounds are currently the most used group of nitrogen-containing flame retardants. Melamine is used as a flame retardant additive for polypropylene and polyethylene. Melamine cyanurate is employed commercially as a flame retardant for polyamides and terephthalates (PET/PBT) and is being developed for use in epoxy and polyurethane resins. Melamine phosphate is also used as a flame retardant for terephthalates (PET/PBT) and is currently being developed for use in epoxy and polyurethane flame retardant formulations. Also in the development stages for use as flame-retardant additives are melamine salts and melamine formaldehyde for their application in thermoset resins (IPCS 1997).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

The outstanding physical-chemical property concerning the risk assessment is a low n‑octanol/water partition coefficient. Melamine is not readily biodegradable but adapted wastewater treatment plants can degrade it effectively. Water is the most relevant compartment in the environmental fate of the substance. The adsorption to soil is estimated to be low; melamine is slowly degraded in soil with a half life of two to three years, and its high water solubility (about 3,000 mg/L) suggests it is likely to migrate to groundwater (UNEP 1999; OECD 2002).

If released to soil, melamine is expected to have very high mobility based upon an estimated Koc of 5. Volatilisation from moist soil surfaces is not expected to be an important fate process based on an estimated Henry’s Law constant of 1.8 x 10-14 atm‑cu m/mole. No biodegradation using a standard 5-day BOD test was observed, suggesting that biodegradation may not be an important environmental fate process. If released into water, melamine is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant. An estimated BCF of 3 suggests bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

### Health considerations

Melamine has been studied for carcinogenicity in mice and rats of each sex by oral administration. It produced urinary bladder and ureteral carcinomas in male rats but only urinary bladder hyperplasia in male mice. The occurrence of urinary bladder tumours in male rats correlated strictly with calculus formation and exposure to high doses. The dose dependence was confirmed by subsequent studies in male rats in which concomitant administration of sodium chloride to increase urinary output resulted in a decreased tumour yield. No data were available on the reproductive or developmental toxicity of melamine. No data were available on the genetic and related effects of melamine in humans. It was not genotoxic in experimental systems. Melamine is not classifiable as to its carcinogenicity to humans (Group 3) (IARC 1999).

Six studies with rats, oral administration of melamine with the feed and dosing periods of 14 days to three months are available. Depression of body weight gain and elevated water intake were observed at higher doses of about 500 mg/kg/d. The target organ system is the urinary tract. Melamine has a diuretic effect, it produces urinary bladder stones (urolithiasis), hyperplastic epithelial changes of the urinary bladder and calcerous deposits in the proximal kidney tubules. In mice ulceration as well as hyperplasias of the bladder occurred. Changes in the urinary bladder were noted in the studies depending on the dose and the species used. A NOEL of 63 mg/kg/d seems appropriate for long term exposure. Altogether melamine is considered to be not genotoxic and not mutagenic (OECD 2002).

Based on dose–response assessment of subchronic rat studies, modelling of the incidence of bladder stones and application of a safety factor of 200 to account for extrapolation from rats to humans, variation within humans and uncertainties associated with the data, a tolerable daily intake (TDI) of 0.2 mg/kg body weight for melamine was established. The TDI is applicable to the whole population, including infants (WHO 2009a).

The estimated tolerable daily intake (TDI) of melamine is 0.5 mg/kg bodyweight (EFSA 2007).

### Derivation of Maximum Acceptable Value

No MAV.

### References

EAWAG. Accessed February 2015. *Biocatalysis/Biodegradation Database: Select 1396 compounds*. <http://eawag-bbd.ethz.ch/index.html>.

EFSA. 2007. *EFSA’s Provisional Statement on a Request from the European Commission Related to Melamine and Structurally Related Compounds such as Cyanuric Acid in Protein-Rich Ingredients Used for Feed and Food*. Question N° EFSA-Q-2007-093. 11 pp. http://www.efsa.europa.eu/en/scdocs/scdoc/1047.htm

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WHO. 2009a. *Toxicological and Health Aspects of Melamine and Cyanuric Acid*. Report of a WHO Expert Meeting in collaboration with FAO, supported by Health Canada. 74 pp. <http://apps.who.int/iris/bitstream/10665/44106/1/9789241597951_eng.pdf?ua=1>.

# 2-methoxyethanol

CAS No. 109-86-4. Also called methyl cellosolve and ethylene glycol monomethyl ether.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2-methoxyethanol. The WHO Guidelines do not have a guideline value for 2-methoxyethanol.

This chemical is included in the USEPA (2009) third Contaminant Candidate List because its occurrence or anticipated occurrence is likely at levels of concern to human health.

### Sources to drinking-water

#### 1. To source waters

2-Methoxyethanol has not been reported to occur as a natural product. 2‑Methoxyethanol is used mainly as a [solvent](http://en.wikipedia.org/wiki/Solvent) in products such as varnishes, dyes, and resins. It is also used in jet fuels and in the manufacture of printed circuit board laminates. It is a clear, colourless liquid with an [ether](http://en.wikipedia.org/wiki/Diethyl_ether)-like odour. It is in a class of solvents known as [glycol ethers](http://en.wikipedia.org/wiki/Glycol_ethers) which are notable for their ability to dissolve a variety of different types of chemical compounds.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

WHO (2009) reports that owing to its high volatility (vapour pressure 1300 Pa at 25°C), 2-methoxyethanol is expected to be present principally in air. The half-life for 2‑methoxyethanol in the atmosphere has been calculated to be in the range of  
5.7–57 hours, based on its reaction with hydroxyl radicals. 2-Methoxyethanol volatilises rapidly from the water surface, with an estimated half-life of 2.8 h. Biodegradation would also be significant; estimated half-lifes in surface water and groundwater are 1–4 and 2–8 weeks, respectively, based on unacclimated aerobic biodegradation. Physical adsorption to suspended solids and sediments should not be significant. Its high water solubility and low *Kow* would suggest that 2-methoxyethanol would be highly mobile in soil, although it would be expected that much of the substance would volatilise from the soil surface.

Partition coefficient n-octanol/water (log value) = Log Pow = 0.77. 2-Methoxyethanol is miscible with water.

### Typical concentrations in drinking-water

2-Methoxyethanol was listed as a contaminant in drinking-water samples analysed between June 1977 and November 1980 in a survey of 12 cities in the USA. Although no details on concentrations of the substance were provided, they were said to be less than 1 μg/L. Copied from WHO (2009).

### Removal methods

Treatment processes that remove particulate matter will be ineffective. Aeration techniques should remove 2-methoxyethanol from the water.

### Health considerations

2-Methoxyethanol is of low to moderate acute toxicity following oral, inhalation or dermal exposure. Although relevant data are limited, exposure of the general population through environmental media is expected to be low, as a result of reported declining use of the compound in recent years as it is replaced with less hazardous compounds. Testicular degeneration and decreased thymus weights, along with effects on the blood (including anaemia and reduced white blood cell and platelet counts), were also reported in F344/N rats exposed to 2-methoxyethanol in drinking-water for 13 weeks at concentrations equivalent to doses of 71 mg/kg body weight per day or more, which therefore constitutes a lowest-observed-adverse-effect level (LOAEL) for the oral route. A no-observed-adverse-effect level (NOAEL) was not identified in these studies. From WHO (2009).

2-Methoxyethanol is toxic to reproduction (ECHA 2010).

USEPA (IRIS 1991) had no data for oral exposure; ample data exist from inhalation studies.

2-Methoxyethanol is toxic to the bone marrow and testicles. Workers exposed to high levels are at risk for [granulocytopenia](http://en.wikipedia.org/wiki/Granulocytopenia), [macrocytic anemia](http://en.wikipedia.org/wiki/Macrocytic_anemia), [oligospermia](http://en.wikipedia.org/wiki/Oligospermia), and [azoospermia](http://en.wikipedia.org/wiki/Azoospermia). The methoxyethanol is converted by [alcohol dehydrogenase](http://en.wikipedia.org/wiki/Alcohol_dehydrogenase) into [methoxyacetic acid](http://en.wikipedia.org/w/index.php?title=Methoxyacetic_acid&action=edit&redlink=1) which is the substance which causes the harmful effects.

### Derivation of Maximum Acceptable Value

No MAV.

### References

ECHA. 2010. *2-Methoxyethanol, as a Substance of Very High Concern because of its CMR Properties*. European Chemicals Agency. http://echa.europa.eu/documents/10162/13638/supdoc\_2-methoxyethanol\_en.pdf.

USEPA. Updated 1991. 2-Methoxyethanol. *Integrated Risk Information System*. <http://www.epa.gov/iris/subst/0525.htm>.

USEPA. 2009. *Contaminant Information Sheets for the Final CCL 3 Chemicals*. EPA 815‑R-09-012. 216 pp. <http://water.epa.gov/scitech/drinkingwater/dws/ccl/upload/Final-CCL-3-Contaminant-Information-Sheets.pdf>.

WHO. 2009. Selected alkoxyethanols: 2-methoxyethanol. *Concise International Chemical Assessment Document* CICAD 67. 53 pp. <http://www.who.int/ipcs/publications/cicad/methoxyethanol.pdf>.

# 2-methoxy-4,6-dinitrophenol

CAS No. 4097-63-6. Also called 4,6-dinitrogaiacol, 2,4-dinitro-6-methoxyphenol, [2‑hydroxy-3,5-dinitroanisole](https://www.ncbi.nlm.nih.gov/pcsubstance/?term=%222-Hydroxy-3%2C5-dinitroanisole%22%5BCompleteSynonym%5D%20AND%20165182%5BStandardizedCID%5D).

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2-methoxy-4,6-dinitrophenol. The WHO Guidelines do not have a guideline value.

### Sources to drinking-water

#### 1. From treatment processes

2-Methoxy-4,6-dinitrophenol is a potential disinfection by-product (DBP) in WTPs using an advanced oxidation process (AOP).

#### 2. From the distribution system

No known sources.

### Typical concentrations in drinking-water

The maximum concentration of 2-methoxy-4,6-dinitrophenol measured in drinking water was 0.0454 μg/L ITSD eq (Vughs et al 2016).

### Health considerations

DWI (2018) estimates the Threshold of Toxicological Concern (TTC) to be 0.0025 µg/kg bw/d. A NOEL of 97,700 μg/kg bw/day derived from the OECD toolbox. The proposed TDI is 90.6 µg/kg bw/day. 2-Methoxy-4,6-dinitrophenol is a skin irritant.

For 2-methoxy-4,6-dinitrophenol a positive response in the Ames assay, with and without metabolic activation was predicted (no further data available). It was concluded that 2-methoxy-4,6-dinitrophenol is ‘potentially mutagenic’ in the Ames test; however there were ‘insufficient data’ available to further assess genotoxic or carcinogenic potential (Vughs et al 2016). It has a potential for DNA binding (DWI 2018, Kolkman et al 2017, Vughs et al 2016).

The maximum intake of 2-methoxy-4,6-dinitrophenol via drinking water by adults (0.0015 μg/kg/day) is less than the TTC value (0.0025 μg/kg bw/day), and therefore, adverse health effects are not anticipated in adults. The maximum intake by children and infants (0.00454 to 0.00681 μg/kg bw/day) exceeds the TTC value. Therefore, additional research into the occurrence in drinking water and toxicological properties of this DBP may be prudent.

The maximum intake of 2-methoxy-4,6-dinitrophenol via drinking water by adults, children and infants (0.00151 to 0.00681 μg/kg bw/day) is less than the proposed TDI (90.6 μg/kg bw/day). Therefore it is not anticipated that any adverse public health effects will occur following exposure to 2-methoxy-4,6-dinitrophenol via drinking water. The TDI and hence the risk characterisation should be used with caution due to the limitations in the dataset used to derive the LOEL.

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2018. *Potential for Formation of Disinfection By-products from Advanced Oxidation Processes*. Report DWI 12852.02. 260 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-317.pdf>.

Kolkman A, et al. 2017. *Application of Effect Directed Analysis to Identify Mutagenic Nitrogenous Disinfection By-products after Advanced Oxidation Drinking Water Treatment*. KWR. Watercycle Research Institute. Netherlands. <https://www.mn.uio.no/kjemi/english/research/projects/ICCE2017/wednesday-21.06/helga-eng-auditorium-2/hr.-10:30/1130-kolkman.pdf>.

Vughs D, et al. 2016. *Tracing Genotoxic Disinfection By-products after Medium Pressure UV Water Treatment using Nitrogen Labelling, Mass Spectrometry and Effect Directed Analysis*. KWR. Watercycle Research Institute. Netherlands. <https://www.eawag.ch/fileadmin/Domain1/Abteilungen/uchem/analytik/pdf/Vughs_Oral.pdf>.

# Methylcyclopentadienyl manganese tricarbonyl

CAS No. 12108-13-3. Also called MMT, manganese tricarbonyl [(1,2,3,4,5-eta)-1-methyl-2,4-cyclopentadien-1-yl]-, methylcymantrene, and trade names such as Antiknock-33.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for methylcyclopentadienyl manganese tricarbonyl. The WHO Guidelines do not have a guideline value.

### Sources to drinking-water

#### 1. To source waters

MMT is an anti-valve seat recession (AVSR) fuel additive (about 72 mg/L in the fuel), increasingly used internationally as a lead replacement. It is believed that 99.5 percent of the MMT present in the fuel is destroyed during combustion. Use is expected to fall as older cars are taken off the road. As well as the tricarbonyl based product, there are phosphorus, sodium and potassium based products.

In general, releases are of a very diffuse nature; however, spillages, and leakages from underground fuel storage tanks, may provide a potential process for point source releases of MMT into surface water, soils and groundwater.

In New Zealand, as a result of promulgation of the Petroleum Products Specifications Regulations 2002, effective from 1 September 2002, automotive fuel must contain no more than 2.0 mg/L Mn. The limitation on Mn content of fuels is to be reviewed by 2006. This law effectively severely restricts the use of MMT in automotive fuels in New Zealand.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

MMT is highly toxic to aquatic organisms.

In a determination of the ready biodegradation of MMT in a closed bottle test, 46 percent degradation was observed after 15 days, and no further degradation was observed after this time. Photolysis is the main degradation route: a half-life of approximately one minute was recorded when a solution of the compound was exposed to midday sunlight. If open to the air, evaporation would occur. Degradation of MMT in the dark by direct hydrolysis was found to be very slow, if indeed it happens at all, with an estimated minimum half-life (at 25± 2°C) of 500 days.

Henry’s law constant = <10-9 Pa m3/mol or 82 Pa.m3.mol-1; partition coefficient (log Pow) = 3.5. Water solubility is 29 mg/L at 25°C. The relatively large value for Log Kow indicates that MMT would have significant affinity for the organic component of soils and sediments, although the water solubility could bestow some mobility to any MMT that enters the soil/sediment compartment. However, in an investigation of the adsorption of MMT to a variety of soil types as well as to the important soil minerals alumina and silica, it was found that MMT binds to soils and clays. Degradation of MMT spiked into a natural anaerobic aqueous sediment was also studied, providing a degradation half-life of 0.5–1.5 years.

In groundwater, MMT is likely to be relatively persistent and its water solubility indicates it may be mobile in groundwater.

### Analytical methods

#### Some alternative methods

See NICNAS (2003).

### Health considerations

MMT is highly toxic in animals and humans. It is absorbed by all routes of exposure and metabolised predominantly in the liver. Metabolites are excreted in urine and faeces. The critical effects from acute exposure to MMT are neurological and pulmonary dysfunction. Acute lethal exposure to MMT in animals is associated with damage to the lungs by all routes, kidney, liver and spleen effects, tremors, convulsions, dyspnea and weakness. In humans, giddiness, headache, nausea, chest tightness, dyspnea and paresthesia are reported in anecdotal cases of acute occupational exposure.

### Derivation of Maximum Acceptable Value

No MAV.

### References

NICNAS. 2003. Methylcyclopentadienyl manganese tricarbonyl (MMT). *Priority Existing Chemical Assessment Report* 24. 168 pp.

# 2-methyl isoborneol (MIB)

CAS No. 2371-42-8. MIB exists as (+) and (-) enantiomers called (R)‑(‑)‑2‑methylisoborneol and (S)-(+)-2-methylisoborneol.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2-methyl isoborneol. The WHO Guidelines do not have a guideline value for 2-methyl isoborneol.

### Sources to drinking-water

#### 1. To source waters

2-Methyl isoborneol, described variously as causing a musty, earthy, mouldy odour in water, is a metabolite from biological activity of many micro-organisms such as fungi, actinomycetes, streptomycetes, cyanophytes and phytoplankton.

2-Methyl isoborneol has been reported to have a threshold odour of around 5–30 ng/L (ie, 0.00001–0.00003 mg/L). Young et al (1996) reported taste and odour thresholds in drinking-water of about 0.0000025 and 0.0000063 mg/L respectively. Odour outbreaks are caused by biological production of the naturally occurring (−) enantiomers which are some 10 times more potent than the (+) molecules. Usually these tastes and odours are mildly unpleasant, but on occasion the water can become undrinkable by a segment of the population. During these “T&O episodes”, MIB levels can frequently exceed 50 ng/L (Westerhoff 2002). Westerhoff discusses source water monitoring and control measures.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Geosmin and 2-MIB are relatively stable to chemical and biological degradation and can persist in the open water in the dissolved form for some time.

### Removal methods

MIB and geosmin can be removed through biologically active sand filters. Experiments were conducted using laboratory sand filter columns using sand taken from South Australian water treatment plants. Sand with a well-established biofilm taken from a 26-year old filter was capable of removing MIB and geosmin to below detection limit after 11 days of operation at an Empty Bed Contact Time (EBCT) of 15 minutes. Sand without an established biofilm removed 60 percent geosmin and 40 percent MIB after 154 days of operation at 15 minute EBCT (McDowell et al 2007).

Chlorine, chlorine dioxide and ozone may reduce the concentration of MIB but by less than 40 percent, even when dosed at more than 20 mg/L, and potassium permanganate was ineffective (Faust and Aly 1998).

Powdered activated carbon can be used but the various grades need to be trialled; a dose of up to 20 mg/L may be required.

MIB tends to be more difficult to remove than geosmin.

### Analytical methods

#### Referee method

A referee method cannot be selected because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

Various GC/MS methods can measure down to about 1 ng/L in water, eg, Palmentier and Taguchi (2001).

### Health considerations

2-Methyl isoborneol does not present a health risk in drinking-water.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Environment Agency. 1998. The assessment of taste, odour and related aesthetic problems in drinking waters 1998. *Methods for the Examination of Waters and Associated Materials*. London: EA Standing Committee of Analysts. Available at: [www.environment-agency.gov.uk/static/documents/Research/171\_taste\_\_odour\_in\_water.pdf](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.environment-agency.gov.uk\static\documents\Research\171_taste__odour_in_water.pdf).

Environment Agency. 2004. The microbiology of drinking water (2004) – Part 11 – Taste, odour and related aesthetic problems. *Methods for the Examination of Waters and Associated Materials*. UK. [www.environment-agency.gov.uk/static/documents/Research/mdwpart112004\_859972.pdf](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.environment-agency.gov.uk\static\documents\Research\mdwpart112004_859972.pdf).

Faust SD, Aly OM. 1998. *Chemistry of Water Treatment* (2nd edition). CRC Press, ISBN 1575040115. 581 pp.

Jüttner F, Watson SB. 2007. Biochemical and ecological control of geosmin and 2‑methylisoborneol in source waters. *Appl Environ Microbiol* 73(14): 4395–406. See <http://ukpmc.ac.uk/articlerender.cgi?artid=1117132>.

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Palmentier, Taguchi. 2001. The determination of six taste and odour compounds in water using Ambersorb 572 and high resolution mass spectrometry. *The Analyst* 126: 840–5. See [www.rsc.org/ej/AN/2001/b008013f.pdf](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.rsc.org\ej\AN\2001\b008013f.pdf).

Westerhoff, et al. 2002. *Guidance Manual for Reducing 2-Methylisoborneol (MIB) and Geosmin in the Metropolitan-Phoenix Area Water Supply*. City of Phoenix: Arizona State University. www4.eas.asu.edu/pwest/myweb/Taste%20and%20Odor%20Stuff/Guidance%20document%20-%20August%202002.pdf.

Young WF, Horth H, Crane R, et al. 1996. Taste and odor threshold concentrations of potable water contaminants. *Water Research* 30: 331–40.

# Methyl isobutyl ketone

CAS No. 108-10-1. Also called MIBK or MIK, and 4-methyl-2-pentanone, isobutyl methyl ketone, 4-methylpentan-2-one, 2-methyl-4-pentanone, isopropyl acetone, 2‑methyl propyl methyl ketone, isopropyl acetone, 4-methyl-2-oxopentane, hexone and isohexanone.

### Maximum Acceptable Value

There is no MAV for methyl isobutyl ketone in the DWSNZ, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Methyl isobutyl ketone is used widely as a solvent, and in some manufacturing processes, mainly paints, rubbers, resins, pharmaceuticals, resin-based coating systems and other chemicals. It is used in the semiconductor industry and the fragrance and flavour industry. It has been used as a dry cleaning agent and a pesticide. Another increasingly important use of MIBK is in the production of rubber antioxidants.

Methyl isobutyl ketone is found in oranges, grapes, vinegar and in meats. It is also a permitted flavouring agent and is used in food-contact materials.

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

Methyl isobutyl ketone quickly evaporates to a gas if released as a liquid. It dissolves when mixed with water – solubility nearly 2 percent. It evaporates from both water and soil when exposed to air. It will break down quickly in the air into acetone, formaldehyde, and 2-methylpropanal. Estimated volatilisation half-lifes for a model river and model lake are nine hours and six days, respectively (USEPA 2003a).

MIBK is expected to have high mobility in soils based on an estimated Koc value of 11. Bacteria in soil and water will break it down. Since it does not bind to soil well, methyl isobutyl ketone that makes its way into the ground may enter groundwater.

Water solubility about 20,000 mg/L (2 percent).

### Typical concentrations in drinking-water

An Environmental Working Group analysis of methyl isobutyl ketone test results reported by 6,930 US public water suppliers in 12 states shows that between 1998 and 2003, 142 communities drank water containing methyl isobutyl ketone, with concentrations reaching 0.1 mg/L.

Two water utilities in the US reported detecting 4-methyl-2-pentanone in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0025 mg/L. 141 water utilities in the US reported detecting methyl isobutyl ketone in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.10 mg/L. It is not known why EWG reported these two chemicals separately – they are synonyms.

### Analytical methods

#### Referee method

A referee method cannot be selected for MIBK because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

### Health considerations

MIBK is currently approved for use as a component of synthetic flavouring substances and adjuvants (21 CFR 171.515) and as a denaturant in alcohol and rum (27 CFR 21.161) at a maximum concentration of 4 percent (USEPA 2003a).

IPCS (1991) reports a no-observed-effect level (NOEL) of 50 mg/kg per day from a 90-day gavage study on rats.

No oral RfD was developed (USEPA 2003 and 2003a) for MIBK because no critical effect was identified after subchronic exposure. No quantitative estimate of carcinogenic risk from oral exposure was derived because no cancer epidemiology studies in humans and no carcinogenicity assays in animals were located.

USEPA (2003a) states that the data for MIBK are inadequate for an assessment of human carcinogenic potential. This characterisation is based on the absence of both cancer epidemiology studies in humans and carcinogenicity assays in animals. The results of genotoxicity tests in a range of assay systems yielded mostly negative responses.

Massachusetts has a drinking water regulatory limit of 0.35 mg/L based on a RfD of 0.05 mg/kg/d and an uncertainty factor of 1,000, and a 20 percent contribution from drinking water. (Note that the USEPA has withdrawn this RfD). A drinking water notification level of 0.12 mg/L was established by the State of California.

IARC (2012) concluded that MIBK is possibly carcinogenic to humans (Group 2B).

### Derivation of Maximum Acceptable Value

No MAV.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for methyl isobutyl ketone is 0.3 mg/L.

### References

Environmental Working Group (EWG). 2008. Methyl isobutyl ketone. *National Tapwater Quality Database*. <http://www.ewg.org/tapwater/contaminants/contaminant.php?contamcode=2249>.

Environmental Working Group (EWG). Accessed 2010. *National Drinking Water Database – Chemical Contaminants*. <http://www.ewg.org/tap-water/chemical-contaminants>.

IARC. 2012. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 101. Some chemicals present in industrial and consumer products, food and drinking-water. <http://monographs.iarc.fr/>

IPCS. 1990. Methyl isobutyl ketone; *Environmental Health Criteria* 117. INCHEM. International Programme on Chemical Safety. <http://www.inchem.org/documents/ehc/ehc/ehc117.htm>.

IPCS. 1991. *Health and Safety Guides*. Methyl isobutyl ketone (HSG 58). IPCS, INCHEM. <http://www.inchem.org/pages/hsg.html>.

Mass. 1996. *Methyl Isobutyl Ketone*. Massachusetts Department of Environmental Protection. <http://www.mass.gov/dep/water/drinking/standards/mekitone.htm>.

MDH. 2009/2016. *Groundwater Values Table*. Minnesota Department of Health (MDH). See: <http://www.health.state.mn.us/divs/eh/risk/guidance/gw/table.html>.

State of California. 1999. *Proposed Notification Level for Methyl Isobutyl Ketone*. Office of Environmental Health Hazard Assessment. <http://oehha.ca.gov/water/pals/mibk/html>.

USEPA. 2003. Methyl isobutyl ketone. *Integrated Risk Information System (IRIS)*. See: <http://www.epa.gov/iris/subst/0173.htm>.

USEPA. 2003a. Toxicological review of methyl isobutyl ketone. In *Support of Summary Information on the Integrated Risk Information System (IRIS)*. EPA/635/R-03/002. 68 pp. See: <http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showSubstanceList>

WHO. 1990. Methyl isobutyl ketone. *Environmental Health Criteria* 117. World Health Organization.

# Methyl isocyanate

CAS No. 624-83-9. The IUPAC name is methyl isocyanate. Can also be called isocyanatomethane, methyl carbylamine, isocyanic acid methyl ester, and occasionally MIC.

### Maximum Acceptable Value

There is no MAV in the DWSNZ. WHO does not mention methyl isocyanate in their Guidelines.

### Sources to drinking-water

#### 1. To source waters

Methyl isocyanate, a volatile organic chemical, is an intermediate in the production of [carbamate](http://en.wikipedia.org/wiki/Carbamate) [pesticides](http://en.wikipedia.org/wiki/Pesticide) (such as [carbaryl](http://en.wikipedia.org/wiki/Carbaryl), [carbofuran](http://en.wikipedia.org/wiki/Carbofuran), [methomyl](http://en.wikipedia.org/wiki/Methomyl), and [aldicarb](http://en.wikipedia.org/wiki/Aldicarb)). It has also been used in the production of polyurethane foam, plastics, [rubbers](http://en.wikipedia.org/wiki/Rubber) and [adhesives](http://en.wikipedia.org/wiki/Adhesive). It can reappear in the environment as a breakdown product from some of these materials.

As a highly [toxic](http://en.wikipedia.org/wiki/Toxic) and irritating material, it is hazardous to human health, and was involved in the 1984 [Bhopal disaster](http://en.wikipedia.org/wiki/Bhopal_disaster) in India which killed nearly 8,000 people initially and approximately 17,000 people in total (the number of deaths vary considerably in different reports); the methyl isocyanate was released as a gas to the atmosphere.

A related compound, allyl isothiocyanate (CAS No. 57-06-7), is a component of food grade oil of mustard being one of the more important substances responsible for its pungent taste; it has also been used as an insecticide and repellent overseas.

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

Methyl isocyanate is very soluble in water (about 6 to 9 percent), but also reacts with water producing non-toxic ureas such as 1,3-dimethylurea, trimethylbiuret, and methylamine, carbon dioxide and much heat. It also reacts with the water in moist soil, and some will evaporate.

Methyl isocyanate is a methyl isothiocyanate (MITC) generating compound.

### Analytical methods

#### Referee method

A referee method cannot be selected for methyl isocyanate because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

### Health considerations

Due to its reactivity, methyl isocyanate should not be found in water being used as a source of drinking water. Methyl isocyanate has been detected in cigarette smoke (in some brands – about 4 micrograms per cigarette).

Exposure to methyl isocyanate may also occur following applications of the soil fumigant, metam sodium (qv) due to photolysis of the metam sodium breakdown product methyl isothiocyanate (MITC). The yield of methyl isocyanate from MITC has been reported to be about 7 percent in laboratory experiments (in OEHHA 2010). Other fungicides used in New Zealand that release methyl isothiocyanate include mancozeb, metiram, propineb, zineb and ziram; see also IPCS (1988).

OEHHA (2010) discusses health effects, mainly reproductive, and almost entirely via inhalation.

EFSA (2010) derived an ADI of 0.018 mg/kg bw/day for allyl isothiocyanate which was rounded up to 0.02 mg/kg bw/day based on a LOAEL of 9 mg/kg bw/day and applying an uncertainty factor of 500 in order to cover uncertainties resulting from extrapolation from the LOAEL to the NOAEL and from the absence of reproductive toxicity studies.

The International Agency for Research on Cancer (IARC), and the USEPA have not classified methyl isocyanate as to its carcinogenicity.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Methylisothiazolinone

CAS No. 2682-20-4. The IUPAC name is 2-methyl-2,3-dihydro-1,2-thiazol-3-one. Methylisothiazolinone is also called 2-methyl-4-isothiazolin-3-one, 2-methyl-3(2H)-isothiazolin-3-one, 2-methyl-3-isothiazolin-3-one, 2-methyl-2,3-dihydroisothiazol-3-one, 2-methyl-3-isothiazolone, 2-methylisothiazol-3-one, methyl-3(2H)-isothiazolone, or even MIT or MI. This compound is also available as the hydrochloride, CAS No. 26172-54-3. Sometimes mistakenly called methylisothiazoline.

The parent compound is isothiazolinone, CAS No. 1003-07-2. Also called 1,2-thiazol-3-one (IUPAC name).

Because of the large number isothiazolinones that are available, and their wide range of uses which include use as pesticides or biocides, a datasheet has been included in the Pesticides section, under the name of 4,5-dichloro-2-octyl-3(2H)-isothiazolone which can be marketed as Sea-nine 211, with the main application being in marine anti-fouling paints.

### Maximum Acceptable Value

Isothiazolones do not have MAVs in the DWSNZ, and are not mentioned in the WHO Guidelines for Drinking-water Quality.

### Sources to drinking-water

#### 1. To source waters

Isothiazolinones containing sulfur atom, nitrogen, oxygen at 3 position and hydrogen can find application for making broad-spectrum biocides and preservatives such as antiseptic agents, bactericides, slimicides and fungicides. They are also used in adhesives, cutting oils, water systems (eg, cooling towers, pulp and paper), cosmetics (hand-creams, lotions, etc), household goods, liquid soaps and shampoos, wet-wipes and wound protectant for pruning cuts.

Methylisothiazolinone is a component of Kathon 886 (CAS No. 55965-84-9), which is a trade name for a commercial mixture (in the ratio 1:3) of 2-methyl-4-isothiazolin-3-one and 5-chloro-2-methyl-4-isothiazolin-3-one. Amongst its many uses, Kathon has been used to control slime in the manufacture of paper products that contact food.

1,2-Benzisothiazolin-3-one at 0.04 percent is used as a preservative in tralkoxydim (qv).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Degradation of the isothiazolonone biocides is an effective mechanism of detoxification since the metabolites are 4 to 5 orders of magnitude less toxic than the parent. Briefly, the isothiazolinone ring is cleaved between the labile N-S bond yielding N-methyl malonamic acid or N-(n-octyl) malonamic acid. Oxidation continues resulting in the formation of the respective amine and carbon dioxide; taken from ERMANZ HSNO Chemical Classification Information Database.

Water solubility is about 100 percent. Methylisothiazolinone is highly toxic to freshwater, estuarine and marine organisms. Methylisothiazolinone is very volatile: the vapour pressure being 6.2 x 10-4 torr. The Octanol/Water Partition Coefficient, KOW = 0.5 at 24°C which is low so methylisothiazolinone is unlikely to accumulate in fish at a significant level upon exposure (USEPA 1998).

### Health considerations

DEFRA (2000) considered that the main toxicological concerns related to point of contact exposure. Isothiazolinones are moderately to highly toxic by oral administration.

Kathon 886 administrated in the drinking water to rats for three months produced slight gastric irritation at a dose of 20 mg/kg/day; the no effects level (NOEL) was 8 mg/kg/day. Dermal application of Kathon 886 at doses up to 0.4 mg/kg/day for three months produced no systemic toxicity in rabbits. The highest allowed concentration of Kathon in cosmetics is 15 ppm according to the cosmetic directive (Danish EPA 2001).

Developmental and chronic feeding/carcinogenicity studies in rats resulted in no significant effects and the USEPA classified methylisothiazolinone as a Group D chemical, not classifiable as to human carcinogenicity. A RfD was not established for methylisothiazolinone because it is currently registered for non-food use applications only, outside the FDA regulated uses in paper and adhesives which may contact food. Also, chemicals such as methylisothiazolinone, used as disinfectants, microbiocides, microbiostats, and sanitisers have not been reviewed by the FAO/WHO Joint Meeting on Pesticide Residues (JMPR). Results from mutagenicity studies were equivocal (USEPA 1998).

Based on effects on body weight and feed consumption at 1,000 ppm (65.7 and 93.5 mg a.i./kg bw/day in male and female rats respectively) the NOAEL in this study was 250 ppm (equivalent to 19.0 and 24.6 mg a.i./kg bw/day in males and females, respectively) (EU 2003).

A common indication of sensitivity to Kathon CG is [allergic contact dermatitis](http://en.wikipedia.org/wiki/Allergic_contact_dermatitis). Sensitisation to this family of preservatives was observed as early as the late 1980s. Due to increased use of [isothiazolinone](http://en.wikipedia.org/wiki/Isothiazolinone)-based preservatives in recent years, an increase in reported incidences of contact allergy to this product have been reported, and in 2013 it was dubbed the American Contact Dermatitis Society’s 2013 Contact Allergen of the Year.

### Derivation of Maximum Acceptable Value

No MAVs.

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# Methylnaphthalenes

Although not included amongst the 17 PAH “priority pollutants” under the USEPA Clean Water Act, several methylnaphthalenes are found in the environment, three fairly common ones being:

* 1-methylnaphthalene – CAS No. 90-12-0
* 2-methylnaphthalene – CAS No. 91-57-6
* 1,4-dimethylnaphthalene – CAS No. 571-58-4

1-Methylnaphthalene is also called α-methylnaphthalene. 2-Methylnaphthalene is also called β-methylnaphthalene.

Refer also to the polynuclear aromatic hydrocarbons datasheet.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

The most abundant classes of PAH found in diesel-contaminated estuarine sediments were naphthalenes, phenanthrenes, and dibenzothiophenes (DBT). Alkylated PAH made up 93 percent of the total PAH. The high proportions of naphthalenes, phenanthrenes, DBT and alkylated PAH are typical of refined petroleum hydrocarbons (US Department of the Interior 1998).

Methylnaphthalene (CAS No. 1321-94-4) refers to a mixture of approximately two-thirds 2-methylnaphthalene and one-third 1-methylnaphthalene. 2-Methylnaphthalene is a natural component of crude oil and coal, and is found in pyrolysis and combustion products such as cigarette and wood smoke, emissions from combustion engines, asphalt , coal tar residues, and used oils.

Mixtures containing 2-methylnaphthalene are used in the formulation of alkyl-naphthalenesulfonates (used for detergents and textile wetting agents), chlorinated naphthalenes, and hydronaphthalenes (used as solvents). Pure 2-methylnaphthalene is a component used in the manufacture of vitamin K and the insecticide carbaryl (USEPA 2003).

There are 10 isomeric dimethylnaphthalenes, with many of them appearing in crude oil. 1,4-Dimethylnaphthalene normally occurs in potatoes in high enough concentrations to prevent sprouting. Under suitable conditions, the concentration of this substance decreases in the potato, thus allowing buds to develop.

1,4-Dimethylnaphthalene appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). It is a naturally occurring component, endogenous to many plants. As a pesticide active ingredient, it is used as a plant growth regulator to inhibit post-harvest sprouting of potatoes during storage and shipping. No harm is expected to the public, the environment, or applicators if label directions are followed.

2,6-Dimethylnaphthalene is a starting material for high performance engineering plastics and liquid crystallisation polymers.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

Polynuclear aromatic hydrocarbons enter the environment through atmospheric deposition. Because of their low water solubility most polynuclear aromatic hydrocarbons are adsorbed to sediments and suspended solids in aquatic systems. Volatilisation may be important over periods exceeding one month. Most polynuclear aromatic hydrocarbons are susceptible to aqueous photolysis. Polynuclear aromatic hydrocarbons of 3 or fewer fused aromatic rings are biodegraded but for the larger polynuclear aromatic hydrocarbons this is minimal. Polynuclear aromatic hydrocarbons are adsorbed but not greatly accumulated by aquatic biota.

If released to soil, 1-methylnaphthalene is expected to have slight mobility based upon a Koc of 2300. Volatilisation from moist soil surfaces is expected, based upon a Henry’s Law constant of 0.000514 atm‑cu m/mole. However, volatilisation is expected to be attenuated by adsorption to soil. 1-Methylnaphthalene is not expected to volatilise from dry soil based on its vapour pressure. 1-Methylnaphthalene should biodegrade rapidly in the environment where microorganisms have acclimated to PAHs and at a moderate rate in unacclimated soils and aquatic systems. If released into water, 1‑methylnaphthalene is expected to adsorb to suspended solids and sediment based upon the Koc. Volatilisation from water surfaces is expected based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 5.5 hours and 5.3 days, respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. BCF have been reported in the range of 30-680. BCF <30 suggest bioconcentration in aquatic organisms is low and BCF 100-1000 suggest bioconcentration in aquatic organisms is high. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

If released to soil, 2-methylnaphthalene is expected to have slight to no mobility based upon Koc values ranging from 4,400 to 8,500. Volatilisation from moist soil surfaces is expected based upon a Henry’s Law constant of 0.000518 atm‑cu m/mole. However, adsorption to soil is expected to attenuate volatilisation. Based upon aqueous screening test data and die-away tests for ground and marine water, 2‑methylnaphthalene should biodegrade rapidly in soils acclimated to polycyclic aromatic hydrocarbons and at a moderate rate in unacclimated soils. If released into water, 2-methylnaphthalene is expected to adsorb to suspended solids and sediment based upon the Koc values. 2-Methylnaphthalene at a concentration of 0.5 ppm was completely removed within 14 days from acclimated fresh-wellwater grab samples from Tuffenwies and Zurich, Switzerland, with a pH of 8.0, at 10 and 25°C and microbial populations of 300–400 cells/mL. Volatilisation from water surfaces is expected based upon this compound’s Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 5.5 hours and 5.3 days, respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. Measured BCF values range from 30 to 895. BCF <30 suggest bioconcentration in aquatic organisms is low and BCF 100–1,000 suggest bioconcentration in aquatic organisms is high. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

Based on the soil sorption coefficient reported (4,230 L/kg), 1,4-dimethylnaphthalene was considered to have slight mobility in soil (EFSA 2013).

The water solubility of 2-methylnaphthalene is 24.6 mg/L (USEPA 2003). The water solubility of 1-methylnaphthalene is 28.1 mg/L.

### Typical concentrations in drinking-water

Two water utilities in the US reported detecting 2-methyl naphthalene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0002 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

Using the 1996 *Proposed Guidelines for Carcinogenic Risk Assessment*, the human carcinogenic potential of naphthalene via the oral or inhalation routes “cannot be determined” at this time based on human and animal data; however, there is suggestive evidence of potential human carcinogenicity based on increased lung tumours incidence in one species and one sex at the high dose only. Additional support includes increased respiratory tumours associated with 1-methylnaphthalene.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) has developed oral minimal risk levels (MRLs) for some PAHs:

|  |  |  |
| --- | --- | --- |
| **PAH** | **mg/kg/day** | **duration** |
| 1-methylnaphthalene | 0.07 | chronic (>364 days) |
| 2-methylnaphthalene | 0.04 | chronic |

USEPA (2003) states that the data are *inadequate for an assessment of human carcinogenic potential*, based on the absence of data concerning the carcinogenic potential of 2-methylnaphthalene in humans and limited equivocal evidence in. The RfD for 2-methylnaphthalene was calculated to be 0.004 mg/kg-day based on a BMDL05 of 3.5 mg/kg-day and an uncertainty factor of 1,000.

For 1,4 dimethylnaphthalene EFSA (2013 and 2014) and EC (2013) derived an Acceptable Daily Intake (ADI) of 0.1 mg/kg bw per day, based on the two-year study in rats NOAEL with an Uncertainty Factor (UF) of 100; based on the toxicological profile the setting of an Acute Reference Dose (ARfD) was not deemed necessary.

### Derivation of Maximum Acceptable Value

No MAVs.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The chronic health risk limit for 2‑methylnaphthalene is 0.008 mg/L.

The odour in water can be detected as follows:

* 1-methylnaphthalene: 0.0075 mg/L
* 2-methylnaphthalene: 0.01 mg/L.

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# Methyl tertiary-amyl ether

CAS No. 994-05-8. It is also called tert-amyl methyl ether, TAME, 1,1-dimethylpropyl methyl ether, *tert*-pentyl methyl ether, methyl tert-amylether, or 2-methoxy-2-methylbutane.

TAME and MTBE (qv) are close structural analogs (one methyl group difference).

### Maximum Acceptable Value

Methyl tertiary-amyl ether does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines for Drinking-water Quality.

### Sources to drinking-water

#### 1. To source waters

TAME is a gasoline ether oxygenate (GEO) additive produced at some fuel refineries and added to gasoline to improve combustion; this accounts for 97 percent of its use. It has been used as a blend stock since 1992 and can be blended from about 0.9 to 4 percent directly into the gasoline at the refinery; 176,000 tonnes of TAME was consumed within the EU in 2011. Other prominent GEOs are MTBE and ETBE (qv).

The impurity with the highest concentration is 2-methyl 2-butanol (1.23 percent). However TAME is not normally purified to high concentrations but produced and used further as 10–30 percent hydrocarbon mixture (EU 2006).

Concentrations of TAME reported in EU (2006):

US Geological Survey detected volatile organic compounds in 42 surface-water samples collected from streams on Long Island, New York, and in New Jersey, January 27-30, 1997. TAME was detected in 11 samples (26 percent). The median concentration in all detections was 0.02 μg/L and the maximum concentration in all detections was 0.08 μg/L.

In four lakes in Byram Township, Sussex County, NJ, in the summer of 1998, concentrations of TAME in water samples ranged from 0.07 to 0.43 μg/L on June 24 and from 0.2 to 0.69 μg/L on 8 September. Lakes are surrounded by densely populated communities where the use of gasoline-powered watercraft is prevalent.

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

Koc values for GEOs range from 9 (lowest recorded value for ETBE) to 160 (highest recorded value for TAME) indicating very high mobility in soil. In general GEOs exhibit low vapour pressures (<250 mm Hg) and thus may be readily volatilised from dry soils. Henry’s law constants of less than 1 x 10-3 indicate that volatilisation from wet soil is likely to also be a major process in determining environmental fate. No evidence indicating rapid biodegradation of GEOs in soil was found, suggesting that this is not an important fate process (IEH 2014).

Volatilisation of GEOs from water sources is expected to take three to four days from a modelled lake and three to four hours from a modelled river. GEO Koc values suggest that sorption of GEOs to suspended solids in an aqueous environment is unlikely. Microorganism based degradation studies indicated little to no biotransformation of TAME or MTBE after 28 and 232 days incubation respectively, indicating that biodegradation is not an important removal process for GEOs in water sources (IEH 2014).

EU (2006) quotes: vapour pressure = 90 hPa at 20°C; water solubility = 11,000 mg/L at 20°C; partition coefficient n-octanol/water = log Pow = 1.55 at 20°C. Henry’s law constant = 83 Pa.m3/mol at 20°C which indicates lowered (slower) volatility from water to air at environmentally relevant temperatures. Based on physical-chemical properties of TAME and the properties of other structurally related aliphatic ethers TAME is not expected to significantly hydrolyse in natural waters under environmentally relevant pH conditions. TAME is not expected to photolyse directly, or photooxidise significantly via reactions with photochemically produced hydroxyl radicals in water. TAME is not readily biodegradable in the aquatic environment. In an anaerobic, static sediment/water microcosm study TAME was not degraded in 180 days. TAME (3 mg/L) was not degraded in 60 days when incubated with aquifer material, soil, or activated sludge.

### Typical concentrations in drinking-water

EU (2006) reports a Finnish study. Most samples were taken from public water supply groundwater wells/boreholes in service or in reserve. Determination limits were 0.08 μg/L for TAME and 0.1 μg/L for MTBE. Nine samples were taken on 25–28 November 2003. The concentration of TAME was below the determination limit in all of the samples. In two samples, MTBE was detected at 0.48 μg/L and 0.20 μg/L concentrations. These values are well below the odor and taste threshold). Groundwater near petrol stations has been found to contain >10 mg/L.

### Removal methods

IEH (2014) suggests that conventional treatment is not very effective in removing methyl tertiary-amyl ether; activated carbon and ozone should enhance removal. EU (2005) states that it is assumed that water treatment processes do not significantly decrease the concentration of TAME.

### Health considerations

TAME is absorbed efficiently from the rat intestine. A *European Union Risk Assessment Report* on TAME predicted rat oral LD50 values of 2147 and 1602 mg/kg for males and females respectively (combined LD50 of 2152 mg/kg (IEH 2014).

A review by the OECD reported the lowest observed adverse effects level LOAEL to be 125 mg/kg/day based upon increased adrenal weights seen in male rats given TAME orally for 90 days (IEH 2014).

An oral NOAEL of 125 mg/kg was derived. TAME is not considered mutagenic (EU 2006).

### Derivation of Maximum Acceptable Value

No MAV.

TAME is reported to have a taste and odour threshold of 0.008 to 0.44 mg/L (DWI 2014). Modelling showed that very large amounts of fuel would need to be lost from each fuel station within a catchment in order for there to be a risk of exceeding the taste and odour concentration thresholds. TAME is unlikely to exert any toxic effects if consumed in water at its taste and odour threshold (IEH 2014).

EU (2006) reports that the average odour detection in water = 0.19 mg/L and average taste detection threshold = 0.13 mg/L, adding that the lowest odour and taste detection thresholds might be far below these values. However, in general, compounds with odour threshold below 1 mg/l are considered highly odorous. The odour of TAME has been described sweet, rubbery, fruity, ether-like and paint-like, camphor like.

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# Methyl tertiary-butyl ether

CAS No. 1634-04-4. The IUPAC name is 2-methoxy-2-methylpropane. It is also called MTBE, methyl-tert-butyl ether, methyl-t-butyl ether or tert-butyl methyl ether. Can also be called *tert*-butoxymethane, 1,1-dimethylethyl methyl ether, methyl 1,1‑dimethylethyl ether, or 2-methyl-2-methoxypropane.

The commercial product is usually 98 to 99 percent pure.

### Maximum Acceptable Value

WHO (2005/2017) states that a health-based guideline value has not been derived for MTBE, due to the fact that any guideline value that would be derived would be significantly higher than the concentration at which it would be detected by odour (0.015 mg/L is the lowest level eliciting a response in a study using taste and odour sensitive participants).

The USEPA concluded on 22 September 2009 that MTBE is known or anticipated to occur in PWSs and may require regulation. Therefore they added MTBE to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

The major use of methyl tertiary-butyl ether (MTBE) is as a petrol additive with production and consumption for this purpose increasing markedly in the 1990s in most parts of the developed world. MTBE is added to petrol at levels of up to 15 percent by volume as an oxygenate to improve combustion and to lower exhaust emissions, particularly carbon monoxide, and to increase the octane number. Because of the relative scarcity of future low priced, domestically produced methanol for MTBE production and the likely trend toward lower volatility gasoline, ethyl tertiary-butyl ether (ETBE, qv) has emerged as a probable fuel blending candidate for the future. Methyl tert-amylether (qv) has been used for some years.

Petrol sold in New Zealand does not generally contain MTBE although the regulations allow up to 11 percent by volume. It is proposed in New Zealand to restrict the use of MTBE and other ethers. A limit of 1 percent for MTBE will allow for contamination levels; the UK average is 3.4 percent. Two ethers, MTBE and di-isopropyl ether (DIPE), are limited in the Australian petrol standard to a maximum of 1 percent by volume based on concerns about their potential to contaminate surface water and groundwater supplies. Their chemical affinity for water allows these ethers to travel through the ground and pollute aquifers if fuel should leak from underground storage tanks or be lost during transport.

Fugitive emissions from petrol refineries and petrol filling stations are major environmental point sources of MTBE, whereas vehicles themselves emit sufficient MTBE to be a significant source in dense traffic areas. Surface water can be contaminated by petrol spills, and by the use of petrol-powered boats, particularly those using two-stroke engines. Average MTBE concentrations of 0.20–0.25 mg/L have been reported in the lower Rhine and lower Main rivers. Spills and leaking storage tanks can cause more serious problems for groundwater. MTBE is seen as a potentially serious long-term threat to drinking-water supplies if it comes to be widely used at high concentrations in petrol, particularly where there is inadequate control on leakage from underground storage tanks.

MTBE was linked to a number of taste and odour incidents in the Netherlands between 2000 and 2003 and again in 2006. The events are believed to relate to land contamination as a result of release from petrol stations or spillage during shipping. A report by Concawe (2012), notes that MTBE has been observed in ground and surface waters in the UK (IEH 2014).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

MTBE and ETBE have been identified in drinking-water in cross-linked polyethylene (PEX) pipes as a major contributor to taste and odour values. Testing has shown that MTBE levels from new PEX pipes may exceed the California primary maximum contamination level of 0.013 mg/L and the secondary level of 0.005 mg/L (for taste and odour) for at least the first 90 days; however, there have been no reports of consumer complaints (EDAW 2009).

### Forms and fate in the environment

MTBE is expected to volatilise rapidly from soil surfaces. MTBE released in subsoils as a result of leaks from underground storage tanks may be persistent (USEPA 1994) where the half-life may exceed two years (USEPA 2008). API (2007) discusses attenuation techniques.

MTBE is resistant to chemical and microbial decomposition in water. In surface water, MTBE will usually be removed very rapidly due to its high volatility (half-life <12 hours in running water, but many days in lakes). In groundwater, it will be more persistent because its volatilisation to air will be reduced or eliminated. MTBE is very soluble in water (about 4–5 percent), does not adsorb to soil particles to a great degree, and is considered mobile. This volatile organic compound (VOC) has a relatively high vapour pressure (33.5 kPa at 25°C) and a low log octanol/water partition coefficient (1.3).

If released to soil, methyl t-butyl ether is expected to have very high mobility based upon a Koc range of 11-12. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 5.87 x 10-4 atm‑cu m/mole. No biodegradation occurred after 40 days using a top soil and activated sludge inoculum, suggesting that biodegradation is not an important environmental fate process in soil. If released into water, methyl t-butyl ether is not expected to adsorb to suspended solids and sediment based on Koc values. No degradation of methyl t-butyl ether occurred after 60 days using an inoculum of sandy aquifer material, suggesting that biodegradation is not an important environmental fate process in water. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 4 hours and 4 days, respectively. A BCF of 1.5 suggests that bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

MTBE metabolites include tert-butyl alcohol (t-butanol, 2-methyl-2-propanol, 1,1‑dimethylethanol, or TBA; CAS 75-65-0) and formaldehyde. Formaldehyde may be reduced to methanol or oxidised to formic acid, which is further biotransformed to carbon dioxide.

### Typical concentrations in drinking-water

In Canadian drinking-water supplies, MTBE was detected in groundwater at 250 locations and in every Canadian province. Levels ranged from <0.005 to >3.4 mg/L, and 60 percent of samples contained MTBE at concentrations above 0.02 mg/L. Overseas methyl *tert*-butyl ether has been found in stormwater, groundwater, reservoir water and drinking-water, especially in areas where it is used extensively in gasoline; therefore it is not expected to be so common in New Zealand.

936 water utilities in the US reported detecting methyl tertiary-butyl ether (MTBE) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.054 mg/L.

EU (2006) reports a Finnish study. Most samples were taken from public water supply groundwater wells/boreholes in service or in reserve. Determination limits were 0.08 μg/L for TAME and 0.1 μg/L for MTBE. Nine samples were taken on 25–28 November 2003. The concentration of TAME was below the determination limit in all of the samples. In two samples, MTBE was detected at 0.48 μg/L and 0.20 μg/L concentrations. These values are well below the odor and taste threshold).

The maximum concentration of MTBE found in 8,434 samples from 2,616 groundwaters in the UK was 0.067 mg/L, mean 0.0009 mg/L (DWI 2008).

### Removal methods

Municipal water filtration plants that rely on conventional water treatment techniques (coagulation, sedimentation, precipitative softening, filtration and chlorination) have been found to be ineffective in reducing concentrations of VOCs in drinking water. MTBE concentrations in groundwater supplies may be reduced by air stripping, but high air to water flow rates (or long contact times) are needed. Granular activated carbon alone has a limited ability to take up MTBE, but a combination of air stripping and activated carbon in series has been successful in reducing MTBE to levels of 0.015 mg/L or less. Installation of an aeration system reduced by one-third to one-half the cost of operating a GAC adsorption system.

A combination of advanced oxidation using UV and hydrogen peroxide and an activated carbon step can achieve relatively high removal rates, leaving very low residual levels of MTBE in the finished water.

### Analytical methods

#### Referee method

A referee method cannot be selected for MBTE because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

Two USEPA methods (Methods 502.2 and 524.2) are approved for measuring MTBE in drinking-water. Method 502.2 employs purge and trap capillary GC with PIDs and electrolytic conductivity detectors in series. The second (EPA Method 524.2) uses purge and trap capillary GC with MS detectors in series. These methods have a lower detection limit of 0.0005 mg/L, well below the odour threshold. USEPA recognises APHA methods 6210D and 6200B as being equivalent.

### Health considerations

There have been no epidemiological or occupational studies of human health effects following oral ingestion of MTBE (Health Canada 2006).

A recent study specifically designed to set an odour threshold for MTBE in drinking-water was based on the American Society for Testing and Materials (ASTM) method E679-91. Eight concentrations of MTBE in water ranging between 0.002 and 0.100 mg/L were used with a 1.75 step factor. The geometric mean detection threshold for the 57 subjects and the recommended odour threshold was 0.015 mg/L.

The main source of human exposure to MTBE is likely to be from inhalation of air, not oral ingestion. For the 0 to 0.5 year age group, drinking-water ingestion (for non-breast-fed infants) can contribute up to 10 percent of total exposure (when considering water intake relative to body weight); in other age ranges, the contribution is only about 2 percent.

Rats orally exposed to TAME exhibited increased adrenal weights with a LOAEL of 125 mg/kg/day. Due to the structural similarities between GEOs this LOAEL value has been assumed for ETBE and MTBE. A project specific TDI of 0.125 mg/kg/day was calculated through the application of an uncertainty factor of 1000 to this LOAEL. Examination of maximum taste and odour thresholds and TDI values indicated that MTBE is unlikely to exert toxic effects at or below its taste and odour threshold (IEH 2014).

IPCS (1998) concluded that MTBE should be considered a rodent carcinogen but that it is not genotoxic and the carcinogenic response is evident only at high levels of exposure that also induce other adverse effects. The weight of evidence supports a conclusion that MTBE is a rodent carcinogen, but the data are insufficient to reach any conclusions about its potential to cause human cancer.

The available data are therefore considered inconclusive and limited in their use for human carcinogenic risk assessment (IPCS 1998). In 1999 the International Agency for Research on Cancer considered that there is limited evidence in experimental animals for the carcinogenicity of methyl tert-butyl ether, and has classified MTBE in Group 3, not classifiable as to its carcinogenicity to humans, based on limited evidence in experimental animals and inadequate evidence in humans.

As at July 2003 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of:

* 0.4 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.3 mg/kg/day for intermediate-duration oral exposure (15–364 days).

### Derivation of Maximum Acceptable Value

No MAV.

A health-based guideline value has not been derived for MTBE, due to the fact that any guideline value that would be derived would be significantly higher than the concentration at which it would be detected by odour – 0.015 mg/L is the lowest level eliciting a response in the study by Young et al (1996), which used taste- and odour-sensitive participants. This value is below the USEPA drinking-water consumer advisory concentrations of 0.02 to 0.04 mg/L, which is based on taste and odour thresholds (0.04 mg/L for taste and 0.02 mg/L for odour – USEPA 2006/2011).

Health Canada (2006) quotes an aesthetic objective (AO) for methyl tertiary-butyl ether (MTBE) in drinking water of 0.015 mg/L (15 µg/L). This AO is the odour threshold of MTBE. IEH (2014) reports a taste and odour threshold of 0.007–0.21 mg/L.

Overall the odour detection thresholds reported for MTBE in water are  
0.003–0.19 mg/L (variable sources) and taste detection thresholds in water are  
0.003–0.68 mg/L (variable sources); reported in EU (2006).

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The short-term, chronic and subchronic health risk limits are 0.7 mg/L, and a limit of 0.06 mg/L was set for cancer.

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# Monobromoacetic acid

CAS No. 79-08-3. Also called [bromoethanoic acid](http://www.chemindustry.com/chemicals/645109.html). The IUPAC name is [2-bromoacetic acid](http://www.chemindustry.com/chemicals/645113.html). Monobromoacetic acid is one of the USEPA’s 5 haloacids (HAA5); the others are monochloroacetic acid, dichloroacetic acid, trichloroacetic acid and dibromoacetic acid, all of which have datasheets. Refer also to the general haloacetic acids datasheet.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for individual monobromoacetic acid in drinking-water.

WHO (2004 and 2011) states that the available data relating to monobromoacetate was considered inadequate to permit recommendation of a health-based guideline value.

The total maximum contaminant level (MCL) for the five haloacetic acids (USEPA 2006) is 0.06 mg/L.

### Sources to drinking-water

#### 1. To source waters

Brominated acetic acids are formed during disinfection (with ozone) of water which contains bromide ions and organic matter. Bromide ions occur naturally in surface water and groundwater and exhibit seasonal fluctuations in concentrations. Bromide ion concentrations can increase due to saltwater intrusion resulting from drought conditions, or due to pollution. Bromide is introduced into New Zealand surface waters usually by wind blown seaspray.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Water solubility >100 percent. Log octanol/water partition coefficient = 0.41. Health Canada (2008).

No other information available.

### Typical concentrations in drinking-water

Brominated acetates generally are present in surface water and groundwater distribution systems at mean concentrations below 0.005 mg/L.

In the US, from 1998–2003, one water supply exceeded the MCL, reaching 0.10 mg/L (mean 0.02 mg/L). 3,510 water utilities in the US reported detecting monobromoacetic acid in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.072 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found <0.0005 mg/L monobromoacetic acid in the raw water, and <0.0005 to 0.0027 mg/L in the treated water.

### Removal methods

Brominated acetic acids arise in waters as a disinfection by-product, so the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the ozone. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps.

### Analytical methods

#### Referee method

Liquid/liquid extraction, gas chromatography-electron capture detection (APHA 6251B; EPA 552.3). DWI (2011) includes a thorough discussion on the analysis of the nine haloacetic acids.

#### Some alternative methods

See WHO (2004a).

### Health considerations

No studies reporting human health effects from exposure to monobromoacetic acid were reported. Data are limited on the oral toxicity of monobromoacetic acid. Limited mutagenicity and genotoxicity data give mixed results.

Data gaps include subchronic or chronic toxicity studies, multigeneration reproductive toxicity studies, standard developmental toxicity studies and carcinogenicity studies. The available data are considered inadequate to establish guideline values for these chemicals.

In Stage 1 and 2 D/DBPRs, USEPA did not set an RfD or MCLG for MBAA due to lack of data on the dose-response for relevant health effects. Accordingly, there is no MCLG. Copied from USEPA (2016).

Health Canada (2008) states there are insufficient data on the toxicity of monobromoacetic acid identified to establish a health-based target. Acute oral studies have shown monobromoacetic acid to be acutely toxic. However, no subchronic, chronic or carcinogenic studies were conducted or published, nor was a standard developmental or multigeneration study conducted. Mixed results were seen with regards to mutagenicity/genotoxicity studies.

### Derivation of Maximum Acceptable Value

No MAV.

WHO (2004a) states that because monobromoacetic acid has not been tested in subchronic or chronic toxicity studies, the available data are considered inadequate to establish a guideline value. Other data gaps include the absence of multigeneration reproductive toxicity studies, standard developmental toxicity studies and carcinogenicity studies.

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# Monochloroacetic acid

CAS No. 79-11-8. The IUPAC name is chloroethanoic acid. Sometimes called chloroacetic acid, 2-chloroacetic acid, α-chloroacetic acid, monochloroethanoic acid, MCA or MCAA.

Monochloroacetic acid is one of the USEPA’s five haloacids (HAA5); the others are dichloroacetic acid, trichloroacetic acid, monobromoacetic acid and dibromoacetic acid, all of which have datasheets. Refer also to the general haloacetic acids datasheet.

### Maximum Acceptable Value

Based on health considerations, the concentration of monochloroacetic acid in drinking-water should not exceed 0.02 mg/L.

The sum of the ratio of the concentrations of dichloroacetic acid, monochloroacetic acid and trichloroacetic acid to each of their respective MAVs must not exceed one. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, section 10.2.5.3).

The total maximum contaminant level (MCL) for the five haloacetic acids (USEPA 2006/2011) is 0.06 mg/L. The lifetime health advisory for monochloroacetic acid is 0.07 mg/L (USEPA 2006/2009/2011), where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The MAC in Canada is 0.1 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of chloroacetic acid in drinking water should not exceed 0.15 mg/L.

### Sources to drinking-water

#### 1. To source waters

Monochloroacetic acid may be used as an intermediate or reagent in the synthesis of a variety of chemicals, as a pre-emergence herbicide (although apparently not in New Zealand) and in the production of 2,4-D and MCPA, and therefore may enter raw water as an industrial and agricultural contaminant. It may also be a major component in wart removing products. Over 10 percent of the production is converted to the sodium salt.

Monochloroacetic acid has been measured in Antarctic ice at 0.1 to 1.0 µg/L, and usually <0.5 µg/L in European lakes (EU 2005).

#### 2. From treatment processes

Chlorinated acetic acids are formed from natural organic material during water chlorination. Being a disinfection by-product, the USEPA (2007) regulates monochloroacetic acid.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

If released to soil, chloroacetic acid is expected to have very high mobility based upon an estimated Koc of 31. The pKa of chloroacetic acid is 2.87, indicating that this compound will primarily exist in the anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilisation of chloroacetic acid from moist soil surfaces is not expected to be an important fate process based on its pKa. Chloroacetic acid is unlikely to volatilise from dry soil surfaces based upon its vapour pressure. Chloroacetic acid reached 65 percent of its theoretical BOD in three weeks in the Japanese MITI test, suggesting that biodegradation may be an important environmental fate process in soil. However, it was also noted that acclimation would play an important role in biodegradation. If released into water, chloroacetic acid is not expected to adsorb to suspended solids and sediment based on the estimated Koc. Chloroacetic acid incubated in both river water and seawater using the cultivation method resulted in no biodegradation, suggesting that biodegradation is not an important environmental fate process in water. A pKa of 2.87 indicates chloroacetic acid will exist almost entirely in the anion form at pH values of 5 to 9 and therefore volatilisation from water surfaces is not expected to be an important fate process. An estimated BCF of 3.2 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

Volatilisation from water/soil: chloroacetic acid has a pKa of 2.86 and will be completely ionised at environmental pHs. Evaporation from water will therefore not be a significant loss process. Chloroacetic acid does not appreciably absorb UV radiation above 290 nm and is therefore not expected to be directly photolysed. It photodechlorinates very slowly in air-saturated solutions with only <0.4 percent being converted to free chloride when irradiated for 11 hours in a laboratory photoreactor.

Water solubility is >100 percent, ie, miscible. Log octanol/water partition coefficient = 0.22. Health Canada. 2008. EU (2005) states the vapour pressure = 8.7 Pa at 25°C; degradation by photolysis and hydrolysis are very slow; soil half-lifes range from 3 to 33 days (default value 30 days) and 15 days for biodegradation in surface water.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 488 zones, found monochloroacetic acid concentrations in three zones to range from “not detectable” (nd) to 0.010 mg/L, with the median concentration being “nd” (limit of detection = 0.005 mg/L) (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.005 mg/L monochloroacetic acid in the raw water, and <0.005 to 0.023 mg/L in the treated water.

Based on preliminary data, concentrations of chloroacetic acids in Australian drinking waters range from 0.01 mg/L to 0.1 mg/L and concentrations reported overseas range up to 0.16 mg/L, and are typically about half the chloroform concentration (Australian Drinking-water Guidelines). Present in surface water-derived drinking-water at  
<0.002–0.0082 mg/L, mean 0.0021 mg/L (WHO 2004).

7,552 water utilities in the US reported detecting monochloroacetic acid in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.18 mg/L.

### Removal methods

No information is available on methods of removing monochloroacetic acid from contaminated source waters.

As this compound arises predominantly in waters principally as a disinfection by‑product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

### Analytical methods

#### Referee method

Liquid/liquid extraction, gas chromatography with electron capture detection (APHA 6251B; EPA 552.3). DWI (2011) includes a thorough discussion on the analysis of the nine haloacetic acids.

#### Some alternative methods

Chloroacetic acids in water may be determined by solvent extraction with methyl tert-butyl ether, methylation and analysis by gas chromatography with electron capture detection (Method EPA 552.1). Limits of quantification are lower than 0.001 mg/L  
(1 g/L).

### Health considerations

Chloroacetic acids are probably absorbed rapidly after ingestion, but there are no data to confirm this assumption.

Rats given monochloracetate subcutaneously had particularly high levels in liver and kidneys and approximately 50 percent of the dose was excreted in urine within less than one day. Approximately 90 percent of a single oral dose of monochloroacetic acid given to rats was excreted in the urine within 24 hours.

A short-term study of mice exposed to varying concentrations of monochloroacetic acid by gavage report decreased weight gain and increased liver weights at the highest dose, amongst females.

No evidence of carcinogenicity of monochloroacetate was found in two-year gavage bioassays with rats and mice. Monochloroacetate has given mixed results in a limited number of mutagenicity assays and has been negative for clastogenicity in genotoxicity studies. According to National Toxicology Program (USA) MCA is not genotoxic. IARC has not classified the carcinogenicity of monochloroacetic acid.

In the Stage 1 D/DBPR, USEPA did not set an MCLG for MCAA due to the lack of available health data. In the Stage 2 D/DBPR, EPA proposed an MCLG of 0.03 mg/L and finalised an MCLG of 0.07 mg/L. The final MCLG was based on an RfD of 0.01 mg/kg/day, using a NOAEL of 3.5 mg/kg/day for decreased body weight, kidney, liver and spleen weights in rats of 20 percent for drinking water exposure. The USEPA classified MCAA as having inadequate data to support a finding on its carcinogenicity. Copied from USEPA (2016).

The reference dose or RfD (USEPA 2006/2009/2011) for monochloroacetic acid is 0.01 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.35 mg/L.

Health Canada (2008) has adopted a NOAEL of 3.5 mg/kg bw per day for treatment-related changes in body, liver, kidney and testes weights, and derived a TDI of 0.0117 mg/kg/d. Using the TDI derived from the NOAEL, a health-based target of 0.1 mg/Lwas calculated. MCA is considered unlikely to be carcinogenic to humans.

### Derivation of Maximum Acceptable Value

Based on the available metabolic and toxicological information available, a tolerable daily intake approach is considered most appropriate for the derivation of the MAV. A lowest-observable-adverse-effect level found during a two-year feeding study on rat spleens has been used for the basis of the derivation.

The MAV for monochloroacetic acid in drinking-water was derived as follows:

3.5 mg/kg body weight per day x 70 kg x 0.2 = 0.0245 mg/L (rounded to 0.02 mg/L)

2 L x 1000

where:

* lowest-observable-adverse-effect level = 3.5 mg/kg body weight per day from a study in which increased absolute and relative spleen weights were observed in male rats exposed to monochloroacetic acid in drinking-water for two years
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for inter- and intraspecies variation and 10 for use of a minimal LOAEL instead of a NOAEL and database deficiencies, including the lack of a multigeneration reproductive toxicity study).

In the 1995 and 2000 DWSNZ, it had been considered that there was insufficient data to establish a MAV.

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# 3-monochloropropane 1,2-diol

CAS No. 96-24-2. Also called 3-MCPD, 3-chloro-1,2-propanediol or α-chlorohydrin, a member of the chloropropanols. The IUPAC name is 3-chloropropane-1,2-diol. Also called 1-chloro-2,3-dihydroxypropane, 2,3-dihydroxypropyl chloride, 3‑chloropropylene glycol and several others.

(R)-(-)-3-chloro-1,2-propanediol, CAS No. 57090-45-6 and (S)-(+)-3-chloro-1,2-propanediol, CAS No. 60827-45-4 are the enantiomers of 3-monochloropropane 1,2-diol.

3-Monochloropropane 1,2-diol is the most important chloropropanol. Others are (see separate datasheets):

* 1,3 dichloro-2-propanol, CAS 96-23-1. Also called 1,3-DCP.
* 2,3 dichloro-1-propanol CAS No. 616-23-9. Also called 2,3-DCP and  
  1,2 dichloro-3-propanol.

These chemicals belong to the large, loosely defined group of chemicals called halohydrins. Other halohydrins are listed in the halohydrin datasheet.

### Maximum Acceptable Value

There is no MAV in the DWSNZ. WHO does not mention 3-monochloropropane 1,2-diol.

### Sources to drinking-water

#### 1. To source waters

Chloropropanol isomers are by-products of the production of hydrolysed vegetable proteins.

3-Chloro-1,2-propanediol is formed when chloride ions react with lipid components in foods under a variety of conditions, including food processing, cooking, and storage. The compound has been found as a contaminant in various foods and food ingredients, most notably in acid-hydrolysed vegetable protein (acid-HVP) and soy sauces (WHO 2007).

#### 2. From treatment processes

3-Monochloropropane 1,2-diol (3-MCPD) can be present as a contaminant in epichlorohydrin/amine copolymers used as flocculants or coagulant aids in water treatment. These polyamine flocculants have been available for many years as approved products for use in water treatment and thus 3-MCPD may be present in drinking water from their use. DWI, the Scottish Government and BS EN 1409:1998 state that:

i) the average polyamine dose should be 2.5 mg/L and never exceed 5 mg/L of active ingredient

(ii) no batch must contain more than 40 mg of 3-monochloropropane 1,2-diol per kg of active ingredient

(iii) the analytical system used for determining the batch content must have a limit of detection no greater than 4 mg/kg and a maximum total standard deviation no greater than 4 mg/kg at 40 mg/kg. Both estimates must have at least 10 degrees of freedom and have been determined from batches of analyses carried out on not less than five separate days

(iv) the supplier must state for every batch an upper limit for the content of 3‑monochloropropane 1,2-diol.

The UK Drinking Water Inspectorate has set controls on impurity levels of 3-MCPD and on maximum usage rates of flocculant addition to water to achieve a maximum theoretical level in drinking water of 0.0001 mg/L [note that this is identical to the level set in the EC Drinking Water Quality Directive (European Commission, 1998) for potentially genotoxic polymer-derived contaminants of drinking water such as acrylamide and epichlorohydrin], quoted in Robjohns et al (2003).

Several types of polyamine have been used as primary coagulants in water treatment. Each is branched, prepared by condensation polymerisation, and are either unquarternised or partially quarternised. Thus their cationic charge is pH dependent, and they are chlorine-sensitive. Because of this, they are used less frequently than polyDADMAC or epi/DMA polymers.

There have been reports of halohydrins being formed after disinfection with chlorine and with ozone.

#### 3. From the distribution system

No known sources.

### Fate and form in the environment

An estimated Koc value of 1 suggests that 3-chloro-1,2-dihydroxypropane will have very high mobility in soil. Volatilisation from moist soil surfaces is not expected based on an estimated Henry’s Law constant of 6.1 x 10-8 atm‑cu m/mole. Data regarding the aerobic biodegradation of 3-chloro-1,2-dihydroxypropane in soil and water are variable; in screening tests, this compound reached 0 to 1 percent of the theoretical BOD in five days and 68 percent of the theoretical BOD in 14 days. Under anaerobic conditions, 3-chloro-1,2-dihydroxypropane may be resistant to biodegradation. In water, 3-chloro-1,2-dihydroxypropane is not expected to adsorb to sediment or particulate matter based on its Koc value. This compound is not expected to volatilise from water surfaces given its estimated Henry’s Law constant. Bioconcentration in aquatic organisms may be low based on an estimated BCF value of 0.2 (EAWAG accessed February 2015).

IARC (2012) states that α-chlorohydrin is soluble in water.

### Analytical methods

#### Referee method

None needed.

#### Some alternative methods

See DWI (2003); Matthew and Anastasio (2000). See IARC (2012).

### Health considerations

3-Monochloropropane 1,2-diol can arise in certain foodstuffs containing acid-hydrolysed vegetable protein, malts and soy sauces.

It is carcinogenic in the rat, producing tumours in males in the testes, mammary gland and the preputial gland and also kidney tumours in both sexes. It has given positive results in *in vitro* mutagenicity studies, but there have been no satisfactorily conducted *in vivo* studies in somatic cells published in the peer reviewed literature. As a result, and because of the absence of appropriate *in vivo* evidence, several international regulatory agencies had previously judged it prudent to assume that 3‑monochloropropane 1,2-diol possessed mutagenic activity *in vivo* and considered it to be a genotoxic carcinogen.

In a study reported in COC (2000), 3-MCPD was administered via drinking water to groups of 50 male and 50 female F344 rats (aged six weeks at study initiation) for a period of 104 weeks. Concentrations of 0, 20, 100, and 200 mg/L were used. These equated to dose levels of 0, 1.1, 5.2, or 28 mg/kg bw/day in males and 0, 1.4, 7.0, or 35 mg/kg bw/day in females. 3-MCPD was also detected in the drinking water used in this study at 2.7 mg/L and thus control animals were given doses of approximately 0.1 mg/kg bw/day. The high dose group exceeded the Maximum Tolerated Dose as evidenced by a decrease in body weights relative to controls of 33 percent and 35 percent in males and females respectively. There was no evidence of any treatment-related increase in mortality in this study. Survival to termination was acceptable (ie, >50 percent) in all dose groups with the exception of the male high dose group where 21/50 animals survived to termination.

The following has been copied from DWI (2007):

“In the 1990s, 3-MCPD had been regarded as a genotoxic carcinogen. However, in 2000, the Committee on Mutagenicity of Chemicals in Food, Consumer Products and the Environment (COM), after reviewing new data, concluded that 3-MCPD could be regarded as having no significant genotoxic potential in vivo. After considering this advice and available carcinogenicity data, the sister Committee on Carcinogenicity (COC) concluded that 3-MCPD was unlikely to present a carcinogenic risk to humans provided exposure was 1000 fold lower than the NOEL of 1.1 mg/kg bw/d for tumourigenicity in rats.

Also, in light of this new data, the EU Scientific Committee on Food (SCF), during 2001, set a ‘tolerable daily intake’ (TDI) for 3-MCPD of 2 micrograms/kg bw/d. Similarly the Joint FAO/WHO Expert Group on Food Additives (JECFA) set a provisional maximum tolerable daily intake (PMTDI) of 2 micrograms/kg bw/d in 2001.

Present controls, are based on the previous precautionary approach of regarding 3-MCPD as a genotoxic carcinogen. Current controls allow no more than 40 mg/L of 3-MCPD in polyamine flocculants, coupled with a maximum dosing rate of 2.5 mg/L of flocculant. This ensures a theoretical maximum possible concentration of 3-MCPD in drinking water of 0.0001 mg/L. This is the same as the current standard in the Water Supply (Water Quality) Regulations for the genotoxic carcinogen epichlorohydrin.

As 3-MCPD is not considered to be genotoxic and a TDI has been set, these controls could be reviewed. For the time being, however, the DWI has decided to maintain this usage restriction, whilst awaiting further research on other chloropropanols.”

WHO (2007) reports that 3-chloro-1,2-propanediol was re-evaluated by the Committee at its 57th meeting. Short- and long-term studies in rodents showed that 3‑chloro-1,2-propanediol is nephrotoxic in both sexes and also affects the male reproductive tract and male fertility. At that meeting, the Committee considered that the kidney was the main target organ and tubule hyperplasia in the kidney the most sensitive end-point for deriving a tolerable intake. This effect was seen in a long-term study of toxicity and carcinogenicity in male and female Fischer 344 rats given drinking-water containing 3‑chloro-1,2-propanediol. The Committee concluded that 1.1 mg/kg bw per day, the lowest dose, was a lowest-observed-effect level (LOEL) and that this was close to a no‑observed-effect level (NOEL). The Committee established a provisional maximum tolerable daily intake (PMTDI) of 2 μg/kg bw for 3-chloro-1,2-propanediol on the basis of this LOEL, using a safety factor of 500. This factor was considered adequate to allow for the absence of a clear NOEL and to account for the effects on male fertility and for inadequacies in the studies of reproductive toxicity. Data available to the Committee at that time indicated that the estimated mean intake of 3-chloro-1,2-propanediol for consumers of soy sauce would be at or above this PMTDI. The sixty-seventh meeting retained the previously established PMTDI of 2 μg/kg bw for 3-chloro-1,2-propanediol.

A provisional maximum tolerable daily intake (PMTDI) was established of 0.002 mg/kg bw for 3-chloro-1,2-propanediol on the basis of the LOEL of 1.1 mg/kg bw per day and a safety factor of 500, which included a factor of 5 for extrapolation from a LOEL to a NOEL. This factor was considered to be adequate to allow for the absence of a clear NOEL and to account for the effects on male fertility and for inadequacies in the studies of reproductive toxicity. Data available indicated that the estimated mean intake of 3-chloro-1,2-propanediol by consumers of soya sauce would be at or above this PMTDI; IPCS (b).

EFSA (2018) established a TDI of 2 µg/kg per day for 3-MCPD and its fatty acid esters (expressed as MCPD equivalents). Renal tubular cell hyperplasia was reconfirmed as the critical effect in rats during chronic oral exposure to 3-MCPD with a BMDL10–BMDU10 interval of 0.20–1.95 mg/kg bw per day using model averaging. The BMDL10 derived using model averaging was about twofolds higher than the one derived in the previous EFSA opinion (0.08 mg/kg bw per day).

3-MCPD was considered by COM and COC during 2000 and statements from both committees have been issued. 3-MCPD was considered by COC to be non-genotoxic *in vivo.*

The results of a recently-completed long-term toxicity/carcinogenicity study (IPCS a) in rats treated at dose levels of 1.1, 5.2 or 28 mg of 3-chloro-1,2-propanediol/kg bw/day in drinking-water for 104 weeks indicated a carcinogenic effect. Occurrence of treatment-related increased incidences of tumours in the kidneys of both sexes and testis, mammary and preputial gland of male rats were reported.

IARC (2012) considered that 3-monochloro-1,2-propanediol is possibly carcinogenic to humans (Group 2B).

### Derivation of Maximum Acceptable Value

No MAV.

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# MX

CAS No. 77439-76-0. Also called 3-chloro-4-dichloromethyl-5-hydroxy-2(5H)-furanone. Sometimes called mutagen X. Sometimes called chloro(dichloromethyl)-5-hydroxy-2(5*H*)-furanone. Collectively considered one of the chlorinated furanones. Some other halogenated furanones are discussed briefly below.

### Maximum Acceptable Value

MX occurs in drinking-water at concentrations well below those of health concern.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set a guideline value for MX in drinking water.

### Sources to drinking-water

#### 1. To source waters

MX, one of the chlorinated furanones, has no commercial use but may be present in the chlorinated effluents of pulp mills.

#### 2. From treatment processes

MX is formed by the reaction of chlorine with complex organic matter and therefore may be found in treated water as a by-product of the treatment process. It has also been reported to form when using chlorine dioxide and chloramines. Recent studies (Suzuki and Nakanishi 1995) have found brominated analogues in chlorinated water:

* 3-chloro-4-(bromochloromethyl)-5-hydroxy-2(5H)-furanone (BMX-1)
* 3-chloro-4-(dibromomethyl)-5-hydroxy-2(5H)-furanone (BMX-2)
* 3-bromo-4-(dibromomethyl)-5-hydroxy-2(5H)-furanone (BMX-3).

A total of 13 halogenated furanones has been reported in chlorinated drinking-water (Onstad and Weinberg 2004).

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

MX in water gradually undergoes pH-dependent isomerisation (to EMX, 2-chloro-3-(dichloromethyl)-4-oxobutenoic acid) and hydrolytic degradation; at pH 8 and 23°C, the half-life of MX is 6 days. Very soluble in water (50.8 mg/mL at pH 2; 43.7 mg/mL at pH 7), ie, about 5 percent.

### Typical concentrations in drinking-water

No data are available on the concentration of MX in New Zealand drinking-water supplies.

MX has been identified in chlorinated humic acid solutions and drinking-water in Finland, the United Kingdom and the USA, and was found to be present in 37 water sources at levels of 0.000002–0.000067 mg/L (2 to 67 ng/L). The highest reported concentrations were from Finland: up to 500 ng/L (0.0005 mg/L). Most other places report less than 100 ng/L. Five drinking-water samples from different Japanese cities contained MX at concentrations ranging from <3 to 9 ng/L. About a third of drinking-water samples from Massachusetts contained >30 ng/L (Wright et al 2002).

### Removal methods

No information is available on methods for removing MX from contaminated source waters.

As this compound arises in waters as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

MX formation is minimised if the pH of the water is maintained above 7. The stability of MX is dependent on pH. Below pH 7 it is relatively stable but above pH 7 it rapidly breaks down.

### Analytical methods

#### Referee method

A referee method cannot be selected for MX because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods:

No alternative methods can be recommended for MX because a MAV has not been established. However, the following information may be useful:

MX is difficult to detect because of low concentrations and the masking effects of other substances. MX can be analysed in drinking-water using XAD resins, high-pressure liquid chromatography, capillary column gas chromatography with mass spectrometry and selective ion monitoring. No detection limits are cited (Munch et al 1987 and Hemming et al 1986). See IARC (2004) for a review.

### Health considerations

At least 40 percent of a dose of MX administered to rats was absorbed and about 5 percent was recovered in the liver, muscle, skin, kidneys and blood.

MX is a potent mutagen in bacteria and in cells in vitro and has undergone a lifetime study in rats in which some tumorigenic responses were observed. These data indicate that MX induces thyroid and bile duct tumours. IARC has classified MX in Group 2B (possibly carcinogenic to humans)on the basis of rat tumourigenicity and its strong mutagenicity. This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

MX has been reported to be an extremely strong mutagen in one strain of *Salmonella typhimurium*. Responses with other strains were generally positive, although not as strong. MX has been reported to account for between 15 and 60 percent of the mutagenicity in water based on the results of the *Salmonella* mutagenicity assay.

McDonald and Komulainen (2005) derived a combined cancer potency of MX for each gender based on the incidence of all tumors. The drinking water concentration associated with a 1 in 1,000,000 increased cancer risk was calculated to be 7.8 ng/L. From USEPA (2016).

MX has induced significant increases in structural chromosomal aberrations in cultured mammalian cells.

The BMXs were present in chlorinated waters in concentrations comparable to that of MX. The total contribution of MX and the BMXs to total mutagenic activity was between 19.1 to 27.6 percent (Suzuki and Nakanishi 1995). Of 209 DBPs tested, Woo et al (2002) found 20 were of priority concern, which included MX and the three brominated analogues listed above.

#### Some other halogenated furanones (from USEPA 2016)

The Office of Water (USEPA, 2000d) completed a health assessment of chlorohydroxyfuranones (CHFs) in 2000 that summarised the available data at that time. Many of the CHF compounds have data that identify these compounds as genotoxins with differing relative potencies, however studies of their carcinogenic potency were lacking at that time.

Mucochloric acid toxicity was evaluated by OECD SIDS (2003) and a NOAEL for developmental toxicity of 60 mg/kg/day was observed with no LOAEL. Systemic toxicity was found at 30 mg/kg/day shown as reduced food consumption and bodyweight gain. Mucochloric acid is mutagenic in *S. typhimurium* and CHO cells and has induced DNA damage in *E. coli* cells injected into mice.

The brominated halofuranones are generally less mutagenic than MX except for BMX‑2, which caused a 140 percent increase in mutagenicity in *S. typhimurium* compared with MX (Richardson et al 2007).

### Derivation of Maximum Acceptable Value

No MAV.

A health-based value of 0.0018 mg/L (1800 ng/L) can be calculated for MX on the basis of the increase in cholangiomas and cholangiocarcinomas in female rats using the linearised multistage model (without a surface area to body weight correction). However, this is significantly above the concentrations that would be found in drinking-water, and there remains uncertainty over whether MX is genotoxic *in vivo*, particularly at the low doses encountered from drinking-water. In view of the above, and analytical difficulties in measuring this compound at such low concentrations, it is considered unnecessary to propose a formal guideline value for MX in drinking-water (WHO 2004a, 2017).

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# Naphthalene

Naphthalene, CAS No. 91-20-3, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. There are more than 100 different PAHs. Naphthalene is also called naphthene or tar camphor, and even mothballs. Refer to the polynuclear aromatic hydrocarbons datasheet.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

The USEPA (2006) established a lifetime health advisory of 0.1 mg/L for naphthalene, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

### Sources to drinking-water

#### 1. To source waters

The most abundant classes of PAH found in diesel-contaminated estuarine sediments were naphthalenes, phenanthrenes, and dibenzothiophenes (DBT). Alkylated PAH made up 93 percent of the total PAH. The high proportions of naphthalenes, phenanthrenes, DBT and alkylated PAH are typical of refined petroleum hydrocarbons, US Department of the Interior (1998). Naphthalene is an important component of creosote (qv).

Naphthalene has been used as a moth repellent, often in wardrobes; it has been used also as a greenhouse fumigant and against gladiolus thrips. In water, its odour threshold has been reported at about 0.003 mg/L (Young et al 1996). Its principal use is as an intermediate in the production of phthalic anhydride used for the manufacture of phthalate plasticisers, resins, dyes, and insect repellents. Naphthalene is also used in the manufacture of synthetic leather tanning agents and the insecticide carbaryl. Trucks burning diesel have been reported to produce PAHs, naphthalene being the main one, at 0.6 to 5 mg of per km (Environment Australia 2003). EU (2003) states that about 15 tonnes/annum of naphthalene is used for pyrotechnic manufacture in the EU.

The highest concentration reported for surface water in the UK is 6.85 μg/L (the highest measured value for the Tees estuary in the vicinity of an effluent outfall from a steel works). Respective maximum concentrations of 1.3 μg/L and 2.24 μg/L have been reported in the Besós and Adige rivers in Spain and are associated with urban or industrial areas. Values of up to 2.3 μg/L have been measured in urban stormwater run-off. Concentrations of up to 14.1 mg/L have been recorded for heavily contaminated sites. Levels of naphthalene in unpolluted surface water range up to 5 ng/L 0.005 μg/L). Concentrations in groundwaters in the vicinity of contaminated sites range up to 15.3 mg/L. The majority of these were not sites at which production or direct use of naphthalene took place. Levels in uncontaminated groundwater are less than 0.03 μg/L. The mean dissolved rain concentration of naphthalene in Los Angeles and Portland was 100 ±32 ng/L (EU 2003).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

The water solubility of naphthalene is 30 mg/L (USEPA 2003; ICPS 2000).

Naphthalene: Octanol-Water Partition Coefficient (log Kow): 3.3. Henry’s constant: 4.6 x 10-4 atm·m3/mol; 5.1 x 10-13 atm mole/m3. Soil Sorption Coefficient (Koc): values from 200–1,470 have been reported worldwide in a variety of soil types. Naphthalene may be lost from soil via evaporation, volatilisation, and biodegradation. The relative importance of each pathway will vary depending on soil depth and the presence and composition of soil biota, including bacteria, fungi, cyanobacteria, and algae. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 4.4 x 10-4 atm‑cu m/mole. Soil and sediment bind naphthalene to a moderate extent depending on soil type. Naphthalene will move rapidly through sandy soil. However, increasing organic carbon content will increase naphthalene’s sorption to soil. Naphthalene will evaporate from the soil surface, but this process will decrease with increasing soil depth. Biodegradation will remove naphthalene from soil, with an estimated half-life of more than 80 days. If the soil is contaminated with other polycyclic PAHs, biodegradation may be much more rapid, with a half-life of a few hours. Naphthalene is broken down by soil bacteria to naphthalene diol, salicylic acid, and catechol.

NPIC (1994) quotes for naphthalene a soil half-life of 30 days, water solubility of 30 mg/L and a sorption coefficient (soil Koc) of 500. This resulted in a pesticide movement to groundwater rating of low.

EU (2003) reports octanol-water partition coefficient (log Kow) = 3.7; vapour pressure = 10.5 Pa at 25°C. Photolytic half lifes for naphthalene in the three systems ranged from 48.1 to 335 hours. Naphthalene does not contain groups amenable to hydrolysis. The half-life in aerobic water is about 1.5 days, and in soils about 2 days. Naphthalene added to groundwater drawn from a shallow well was found to degrade completely within eight days; there was a lag period of six days followed by complete degradation within the next two days. There was no change in the naphthalene concentration of a sterile control during the experimental period.

Naphthalene will be lost from water by volatilisation, sorption, photolysis, and biodegradation. The relative contributions of these processes will depend in part on the water’s characteristics, including depth, flow rate, and contamination level. Cyanobacteria and microalgae metabolise naphthalene. The primary metabolite was found to be 1-naphthol, with 4-hydroxy-1-tetralone and *cis*-naphthalene dihydrodiol as lesser metabolites. NPIC. If released into water, naphthalene is expected to adsorb to suspended solids and sediment based upon the Koc values. Naphthalene has been shown to biodegrade in water with half-lifes ranging from about 0.8 to 43 days. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are six hours and five days, respectively (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

103 water utilities in the US reported detecting naphthalene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0064 mg/L.

EU (2003) reports that the highest naphthalene level reported in drinking water is 6.7 μg/L from wells near a chemical waste dump in the US. Concentrations up to 1,271 ng/L (1.27 µg/L) have also been reported for supplies in 12 US Great Lakes municipalities. Concentrations measured at other locations range up to about 100 ng/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

There have been few studies on the human health effects of polynuclear aromatic hydrocarbons. Cases of accidental poisoning with naphthalene, resulting in death by acute haemolytic anaemia, have been reported.

USEPA (1998) states that naphthalene is classified as a possible human carcinogen (inadequate human and limited animal data); specifically a classification of Group C using criteria of the 1986 Guidelines for Carcinogenic Risk Assessment. Using the 1996 *Proposed Guidelines for Carcinogenic Risk Assessment*, the human carcinogenic potential of naphthalene via the oral or inhalation routes “cannot be determined” at this time based on human and animal data; however, there is suggestive evidence of potential human carcinogenicity based on increased lung tumours incidence in one species and one sex at the high dose only. Additional support includes increased respiratory tumours associated with 1-methylnaphthalene.

Naphthalene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) has developed oral minimal risk levels (MRLs) for some PAHs:

|  |  |  |
| --- | --- | --- |
| **PAH** | **mg/kg/day** | **duration** |
| naphthalene | 0.6 | acute (1–14 days) and intermediate |
| naphthalene | 0.6 | intermediate (15–364 days) |

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA has a reference dose or RfD of 0.02 mg/L for naphthalene and a Drinking Water Equivalent Level or DWEL of 0.7 mg/L. The USEPA has established a lifetime drinking-water health advisory of 0.1 mg/L for naphthalene.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The short-term, subchronic and chronic health risk limits for naphthalene is 0.07 mg/L.

The odour of naphthalene in water can be detected at 0.021 mg/L.

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# Nitrapyrin

CAS No. 1929-82-4. Nitrapyrin is the trade name for 2-chloro-6-trichloromethyl pyridine. Sometimes written 2-chloro-6-(trichloromethyl) pyridine. A trade name in New Zealand is N-Serve.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for nitrapyrin. The WHO Guidelines do not mention nitrapyrin.

### Sources to drinking-water

#### 1. To source waters

Organic nitrogen is converted from urea to ammonium salts (NH4+) by a variety of bacteria, actinomycetes and fungi in a process known as mineralisation. Autotrophic bacteria (*Nitrosomonas*) convert the ammonium to nitrite (NO2-) and further modification by other bacteria (*Nitrobacter*) converts nitrite to nitrate (NO3-). This process of chemical oxidation is known as nitrification. Nitrogen in these forms is very soluble and is easily removed from the soil by leaching.

Nitrapyrin is a biodegradable nitrogen inhibitor and soil bactericide that slows the rate that soil bacteria, eg, *Nitrosomas/Nitrobacter,* convert ammonia into nitrite/nitrate and nitrous oxide.

Dicyandiamide (DCD) is a more commonly used nitrification inhibitor; dimethylpyrazole-phosphate (DMPP) is a much superior nitrification inhibitor to DCD and is effective at lower concentrations. N-(n-butyl) thiophosphoric triamide is also used – see datasheets.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Available data indicate that nitrapyrin hydrolyses and photodegrades rapidly, hence should not persist in most environments. In soil, nitrapyrin has a half-life of <3 to 35 days, depending on soil type. It is shown to be mobile to moderately mobile in mineral soils, and also prone to volatilise from the application site, so it could leave those sites through leaching or volatilisation. 6-Chlorpicolinic acid (also called chloropicolinic acid or 6-CPA), the primary degradate of nitrapyrin; it is mobile and degrades via hydroxylation (breaking the pyridine ring) and microbial mineralisation.

The drinking water assessment for nitrapyrin and 6-CPA showed that all modelled surface water EECs (which are < 1.71 ppb) and groundwater EECs (which are <278.82 ppb) are less than the chronic DWLOCs (300 or greater) and therefore are not of concern. USEPA (2005).

Water solubility is about 40 mg/L.

NPIC (1994) quotes for nitrapyrin a soil half-life of 10 days, water solubility of 40 mg/L and a sorption coefficient (soil Koc) of 570. This resulted in a pesticide movement to groundwater rating of low.

### Health considerations

Nitrapyrin is considered to have low acute toxicity. USEPA (2005) stated that there is reasonable certainty that no harm to any population subgroup will result from aggregate exposure to nitrapyrin when considering dietary (food and water) exposure.

A chronic dietary assessment was conducted only for the major metabolite, 6‑chlorpicolinic acid, because only 6-CPA residues have been detected in crops, and not residues of nitrapyrin. Chronic dietary exposure is expected to be less than 1 percent of the chronic Population Adjusted Dose (cPAD) for the general US population and all population subgroups, and is therefore below the Agency’s level of concern.

Nitrapyrin is classified as “likely to be human carcinogen”. However, a cancer dietary risk assessment was not conducted for nitrapyrin because exposure to nitrapyrin, per se, in the diet is negligible (zero) and the cancer endpoint is relevant only to nitrapyrin, per se, and not to 6-CPA. Nitrapyrin did not demonstrate an increase in unscheduled rat hepatocyte DNA synthesis and was not genotoxic in mutagenicity tests.

### Derivation of Maximum Acceptable Value

No MAV.

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# Nitrilotriacetic acid

CAS No. 139-13-9. The IUPAC name is 2-(bis(carboxymethyl)amino)acetic acid. Also called NTA, nitrilo-2,2`,2``-triacetic acid, N,N-bis(carboxymethyl)-glycine, nitrilotris(methylenecarboxylic acid), triglycine, triglycollamic acid, aminotriacetic acid and various trade names. It is also available as various salts, chiefly the mono-, di- and trisodium salts. Trisodium nitrilotriacetate CAS No. is 5064-31-3; also called Na3NTA.

### Maximum Acceptable Value

Based on health considerations, the concentration of nitrilotriacetic acid in drinking-water should not exceed 0.2 mg/L.

The maximum acceptable concentration for nitrilotriacetic acid in drinking water in Canada is 0.4 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of nitrilotriacetic acid in drinking water should not exceed 0.2 mg/L.

### Sources to drinking-water

#### 1. To source waters

Nitrilotriacetic acid (NTA), an aminotricarboxylic acid, may be present in raw water that has been contaminated with (mainly untreated) wastewater or industrial discharge. It is a chelating agent and builder used (mainly as the trisodium salt) in laundry detergents as a replacement for phosphate. It is also used in the treatment of boiler water to prevent scale formation, and in the photographic, metal plating, textile manufacturing, and paper and cellulose industries. Its solubility in water at 22.5°C is about 1200 mg/L.

In a Swiss sewage plant, influent concentrations of 0.30–1.5 mg/L (diurnal variation) were detected (EU 2005).

NTA is continuously monitored in German surface waters. In 1997/98, the substance was sampled at 84 locations at 51 rivers and creeks, mostly with 13 samples per year at each location. From a total of 2,283 measurements, the highest detected concentration was 0.10 mg/L H3NTA (= 0.135 mg/L as Na3NTA) (EU 2005).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Fate and form in the environment

In a Modified OECD Screening Test conducted according to OECD guideline 301 E, NTA was found to be readily biodegradable. Meeting the 10 days time window criterion, the substance (initial concentration 50 mg/L) was completely degraded within 14 days; EU. 2005. Biodegradation of NTA is influenced by any metal speciation – eg, mercury complexes are stable. For a Canadian municipal activated sludge plant, a removal of >95 percent in summer and less than 50 percent in winter is reported. EU (2005) uses a biodegradation half-life of 5 days in freshwaters.

Nitrilotriacetic acid mobilises heavy metals from aquatic sediments and is present in water primarily in the form of metal complexes, most of which degrade rapidly. Under certain conditions, nitrilotriacetic acid is broken down by photochemical and chemical reactions. The half-life for biodegradation of 1 to 100 µg/L NTA in groundwater is approximately 31 hours. Complete disappearance from acclimatised river water at concentrations of 5 to 50 mg/L was reported to occur in two to six days, whereas concentrations of NTA less than 5 mg/L are expected to degrade within one day.

If released to soil, nitrilotriacetic acid is expected to have high to moderate mobility based upon an estimated Koc of <286. The pKa1 of nitrilotriacetic acid is 3.03, indicating that this compound will exist almost entirely in anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilisation from moist soil is not expected because the acid exists as an anion and anions do not volatilise. Biodegradation is expected to be the dominant degradation process in soils. In soil degradation studies in five different soils, nitrilotriacetic acid biodegraded under both aerobic and water logged (presumably anaerobic) conditions; after seven days of incubation, degradation was 6–30 percent (average 22.4 percent) in aerobic soil and  
9–34 percent (average 25.4 percent) in water logged soil with no degradation in sterilised soil. If released into water, nitrilotriacetic acid is expected to adsorb to suspended solids and sediment based upon the estimated Koc. Biodegradation of nitrilotriacetic acid in water is expected based on several river die-away studies that measured half-lifes ranging from 0.34 to 15 days. Nitrilotriacetic acid was rapidly biodegraded in stream water (100 percent in 10 days) but poorly degraded in an estuarine water; the degradation was inversely proportional to salinity. Volatilisation from water surfaces is not expected to be an important fate process based on its pKa1. BCFs of <9 to 109 measured in carp (*Cyprinus carpio*) suggest bioconcentration in aquatic organisms is low to moderate. The photolytic half-life of the Fe(III) complex of nitrilotriacetic acid in the top layer of a water body was calculated to be 42.9 minutes at maximum sunlight intensity. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

For Na3NTA EU (2005) quotes: water solubility about 64 percent; partition coefficient = logPow = -2.62.

### Typical concentrations in drinking-water

No data are available on the concentration of nitrilotriacetic acid in New Zealand drinking-water supplies.

The average concentration of nitrilotriacetic acid detectable in the drinking-water of eight cities in the USA was 0.0021 mg/L (2.1 g/L). Surface water concentrations in Europe have been found to be range up to 0.012 mg/L.

In a national survey of 70 Canadian municipalities conducted from November 1976 to February 1977, the mean concentration of NTA in drinking water was 0.003 mg/L (range <0.0002 to 0.030 mg/L). The mean concentration in raw water samples was 0.039 mg/L (range <0.0002 to 0.034 mg/L). Concentrations of NTA in raw and treated water samples exceeded 0.010 mg/L in only 14 percent of the locations. NTA was not detected in approximately 75 percent of 102 wells in three communities in two Canadian provinces (detection limit 0.010 mg/L); NTA concentrations in the remainder of wells for which there was other evidence of pollution by sewage were between 0.015 and 0.25 mg/L.

### Removal methods

Health Canada (1990): Chlorination with 10 to 15 ppm chlorine results in 10 to 90 percent removal of NTA, depending upon pH, metal content, contact time, ammonia level and NTA concentration. Ozonation at ozone concentrations typically used in treatment plants can reduce concentrations of 35 to 350 µg/L NTA by more than 80 percent in five minutes. Activated carbon removes only a small percentage of NTA in water, because most is complexed with metals.

### Analytical methods

#### Referee method

GC-NSD (Malaiyandi et al 1979, *Env Sci & Tech* 13: 59–61; Aue et al 1972 *Jnl of Chromat* 72: 259–67).

#### Some alternative methods

No alternative methods have been recommended for nitrilotriacetic acid because no methods meet the required criteria.

### Health considerations

Studies indicate that daily exposure of consumers to all possible sources of nitrilotriacetic acid is generally <0.001 mg/kg bw. Among the sources specifically considered were drinking-water, showering and bathing, wearing clothes washed with NTA-containing detergents, inhalation of detergents, skin contact with washwater from laundry or dishes and ingestion of residues on hand-washed dishes.

Absorption of nitrilotriacetic acid from the gastrointestinal tract is rapid and it does not appear to be metabolised by mammals. It is excreted in urine and accumulates in bone and possibly the kidneys. NTA accumulates in bone because it forms complexes with divalent cations such as calcium; its turnover time in bone is similar to that of calcium.

Nitrilotriacetic acid does not appear to be have high acute toxicity to mammals. There is little evidence regarding the toxicity of nitrilotriacetic acid in humans. Based on physical examination, blood chemistry analysis, and urinalysis, no adverse health effects were reported in a metabolism study in which volunteers ingested a single dose of 10 mg nitrilotriacetic acid.

Nitrilotriacetic acid has not been shown to be teratogenic or genotoxic in studies conducted to date but has induced urinary tract tumours in rats and mice at high doses. The induction of tumours in rodents is considered to be due to cytotoxicity resulting from the chelation of divalent cations such as zinc and calcium in the urinary tract, leading to the development of hyperplasia and neoplasia.

There is sufficient evidence for the carcinogenicity of nitrilotriacetic acid and its sodium salts in experimental animals; the International Agency for Research on Cancer has placed nitrilotriacetic acid and its salts in Group 2B (possibly carcinogenic to humans). This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

### Derivation of Maximum Acceptable Value

Because nitrilotriacetic acid induces tumours only after prolonged exposure to doses higher than those that produce nephrotoxicity, the MAV for nitrilotriacetic acid in drinking-water was determined using a tolerable daily intake approach. The no-observable-adverse-effect level used in the derivation was determined for nephritis and nephrosis in a two-year study in rats.

The MAV for nitrilotriacetic acid in drinking-water was derived as follows:

10 mg/kg body weight per day x 70 kg x 0.5 = 0.2 mg/L

2 L x 1000

where:

* no-observable-adverse-effect level = 10 mg/kg body weight per day for nephritis and nephrosis in a two-year study in rats
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.5
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for carcinogenic potential at high doses).

The MAV was derived on the basis of a NOAEL for nephrotoxicity effects rather than induced tumours, because this occurred at higher doses. However, a larger uncertainty factor has been used to account for the evidence of urinary tumour induction at high doses.

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# Nitrobenzene

CAS No. 98-95-3. Also called nitrobenzol, mononitrobenzene or essence (or oil) of mirbane.

### Maximum Acceptable Value

The DWSNZ do not include a MAV. Nitrobenzene is included in the [plan of work of the rolling revision](http://www.who.int/entity/water_sanitation_health/dwq/en/index.html) of the WHO Guidelines for Drinking-water Quality. As at 2017, WHO considers nitrobenzene is rarely found in drinking-water at concentrations of health concern.

The USEPA concluded on 22 September 2009 that nitrobenzene is known or anticipated to occur in PWSs and may require regulation. Therefore they added nitrobenzene to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a). Nitrobenzene is on the USEPA List of Priority Pollutants.

### Sources to drinking-water

#### 1. To source waters

There is no natural source of nitrobenzene known. However, nitrobenzene may be formed by OH-initiated photooxidation of benzene which could theoretically be of natural origin. Approximately 95 percent of nitrobenzene is consumed in the production of [aniline](http://en.wikipedia.org/wiki/Aniline) (which is used to manufacture polyurethane). Minor uses include the use as a precursor to [rubber](http://en.wikipedia.org/wiki/Rubber) chemicals, [pesticides](http://en.wikipedia.org/wiki/Pesticide), [dyes](http://en.wikipedia.org/wiki/Dye), and [pharmaceuticals](http://en.wikipedia.org/wiki/Pharmaceutical). Nitrobenzene is also used in shoe and floor polishes, leather dressings, paint [solvents](http://en.wikipedia.org/wiki/Solvent), and other materials to mask unpleasant odours. The use of nitrobenzene in cosmetic products and soap has been banned since the 1980s.

In US surface waters, nitrobenzene was detected in only 0.4 percent of 836 ambient surface water stations, and in 1.8 percent of 1245 reporting stations on industrial waste waters (ATSDR 1990). Nitrobenzene concentrations in rivers in Europe and the US have rarely exceeded 0.01 mg/L.

The USEPA recommends that levels in lakes and streams should be limited to 17 parts of nitrobenzene per million parts of water (17 mg/L) to prevent possible health effects from drinking water or eating fish contaminated with nitrobenzene.

Most of the 90-percentile values in surface water are below 1 μg/L. There are a few measured maximum values that exceed 1 μg/L in the river Rhine which are in the range between 1.2 μg/L and 22.5 μg/L. The monitoring data for the river Elbe show values of about 0.5 μg/L at Zollenspieker (the city Hamburg is situated there) and Semannshöft (also close to Hamburg), respectively. Only in the Czech Republic higher concentrations for the river Elbe of maximum 5.2 μg/L were measured (EU 2007).

DWI (2014) reports that concentrations of nitrobenzene in drinking water are detected but not quantifiable, in groundwater range from 0 to 12 μg/L, in fresh surface water from 0.022 to <100 μg/L, in marine surface water range from 0 to 14.8 μg/L and in effluents from 0 to 91,000 μg/L.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Nitrobenzene will volatilise slowly from soil and surface water (vapour pressure, 0.27 mm Hg at 25°C; EU (2007) states 32.6 Pa) and is subject to slow biodegradation. Adsorption to sediment and bioconcentration are not thought to be significant fate processes in water. Nitrobenzene may leach through the soil and is considered to have intermediate mobility. The half-life of nitrobenzene in aquatic environments has been estimated at 0.3 days. Solubility in water: about 2,000 mg/L. Aromatic nitro compounds are generally resistant to hydrolysis.

If released to soil, nitrobenzene is expected to have very high to moderate mobility based upon Koc values of 30.6 to 370; EU (2007) states logKow = 1.86. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 2.4 x 10-5 atm‑cu m/mole; EU (2007) states 1.3 Pa.m3/mol at 20°C. Nitrobenzene is expected to biodegrade under both aerobic and anaerobic conditions in both soil and water. Nitrobenzene had a half-life of 56 days in an aerobic soil column. Nitrobenzene was rapidly biodegraded after a lag phase of 70 to 85 days in an aerobic aquifer test done with groundwater and sediment from eight locations over a 149-day incubation period. If released into water, nitrobenzene is not expected to adsorb to suspended solids, organic matter and sediment based upon a Koc of 89 measured in river sediment. Nitrobenzene may be degraded slowly in water by photolysis (a half-life of 133 days), by reaction with hydrated electrons in eutrophic lakes (a half-life of 22 days), or by reaction with sunlight and nitrate (a measured half-life of 11 hours). Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 44 hours and 17 days, respectively. BCF values of 1.47 to 28.32 suggest bioconcentration in aquatic organisms is low (EAWAG accessed February 2015). DWI (2014) quotes a log Kow of 1.85 and a log Koc of 1.56.

### Typical concentrations in drinking-water

In a survey of 14 treated drinking-water supplies of varied sources in the United Kingdom, nitrobenzene was detected in one supply, which came from an upland reservoir. In groundwater samples collected from January to March 1987 in Degrémont, France, nitrobenzene was identified as a pollutant at concentrations ranging from 0.003 to 0.012 mg/L.

Three water utilities in the US reported detecting nitrobenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.002 mg/L.

### Removal methods

Pilot plant studies using slow sand filtration (at 0.2 m/h) found the removal of 70 to 80 percent of nitrobenzene from a raw water concentration of 85 ng/L. Nitrobenzene is not oxidised at a significant rate by ozone under water treatment conditions. UV light may be effective with oxidants at long exposures (taken from WHO 2009).

At an equilibrium concentration of 5 mg/L, the isotherm absorption capacity for nitrobenzene on F100 activated carbon is reported to be approximately 60 mg/g. A concentration of 100 mg/L was effectively completely removed by addition of 10 g activated carbon/L. Removals were reported of 42 to 70 percent using 10 to 50 mg/L nitrobenzene solutions with four different nanofiltration membranes. From DWI (2014).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

For members of the general population, both inhalation (ambient air) and ingestion (drinking water) exposures are possible, and are likely to be highest for those individuals living near industrial/manufacturing sources or hazardous waste sites. Potential exposure through the use of consumer products is also possible, but data are lacking to quantify these exposures. Although no studies have been performed on the extent of uptake of nitrobenzene by humans after oral exposure, oral absorption would appear to be rapid and extensive, based on the very large number of clinical reports of poisonings.

In a recent British study, no nitrobenzene was detected (<2 μg/kg) in 49 honey samples collected from hives fumigated with “Frow mixture” containing petroleum-derived substances in addition to nitrobenzene to treat hives against parasitic mites (from USEPA 2009).

Methaemoglobinaemia, with cyanosis, headache, dyspnoea, weakness and ultimately coma and death, is the main characteristic of acute nitrobenzene poisoning. Nitrobenzene may also induce haemolysis, which is, however, usually mild. A major part of the absorbed dose is excreted into the urine: 10 to 20 percent of the dose is excreted as 4-nitrophenol, the concentration of which may be used for biological monitoring. A smaller fraction is excreted as 4-aminophenol.

Nitrobenzene was non-genotoxic in bacteria and mammalian cells *in vitro*. In mammals *in vivo*, it was inactive.

USEPA (2009) derived an oral chronic RfD for nitrobenzene of 0.002 mg/kg/d, based on a BMDL of 1.8 mg/kg/d and an uncertainty factor of 1000 for increased methaemoglobin levels.

For Repeat Oral Dose Toxicity and Carcinogenicity a LOEL of 5 mg/kg bw/day can be identified based on organ weights changes. Based on this study, an oral Tolerable Daily Intake (TDI) of 0.005 mg/kg bw/day (5 μg/kg bw/day) can be derived (DWI 2014).

Based on inhalation studies, the International Agency for Research on Cancer (IARC) has determined that nitrobenzene is possibly carcinogenic to humans (Group 2B. NIEHS (2004) considers nitrobenzene is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity in experimental animals. Nitrobenzene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. The EC (2008) concludes that the human relevance of the positive carcinogenicity studies in animals cannot be assessed due to insufficient data on mechanism of carcinogenicity; and that consumer exposures and indirect exposures are expected to be very low due to the almost exclusive use of nitrobenzene as a chemical intermediate in industry. USEPA (2009) states that nitrobenzene is characterised as “likely to be carcinogenic to humans” for any route of exposure. Nitrobenzene has been shown to be a carcinogen in rats and mice. Adenomas and/or carcinomas with a pronounced dose-response relationship were found in livers of male F344 and male CD rats and in thyroids of male F344 rats.

DWI (2014) states: The available data indicate that nitrobenzene is not genotoxic in bacteria. The results of genotoxicity studies in mammalian *in vitro* systems have been mixed. Overall, these data indicate that nitrobenzene may be very weakly genotoxic in mammalian *in vitro* systems. The results of *in vivo* genotoxicity studies of nitrobenzene are mixed and the data indicate that nitrobenzene may be weakly genotoxic *in vivo*.

ECHA (2016) classifies nitrobenzene in hazard class reproductive toxicity category 1B, ie, ‘May damage fertility’).

### Derivation of Maximum Acceptable Value

No MAV.

Because nitrobenzene occurrence in drinking-water at concentrations above trace levels is infrequent, the WHO (2009/2011) considered it unnecessary to derive a formal guideline value. However, health-based values can be calculated to provide guidance in the event of spills and where there are higher concentrations in industrial areas. Two health-based values, one for short-term exposure and the other for long-term exposure, are derived based on the limited available information.

**Short-term exposure:** A short-term health-based value of 0.03 mg/L can be derived for a 60 kg adult drinking two litres of water per day, using an allocation factor of 20 percent. It should be noted that nitrobenzene is a potent methaemoglobinaemic agent in humans, and this is of particular concern for bottle-fed infants. Currently, data are not adequate to determine a separate value for this end-point.

**Long-term exposure:** A long-term health-based value of 0.008 to 0.063 mg/L can be derived depending on end-point and approach used. It should be emphasised that the derivation of the long-term health-based values includes large uncertainties because of the dose metric conversion from inhalation studies and the possibility of increased metabolism to aniline in the gastrointestinal tract.

The odour threshold of nitrobenzene in water has been reported as 0.03 to 0.11 mg/L (in WHO 2009/2011). The USEPA established an organoleptic effect criterion of 0.03 mg/L for nitrobenzene. Source: [*Quality Criteria for Water*, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

The State of Kansas has applied a drinking-water limit of 0.005 mg/L and Maine 0.0014 mg/L.

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# 4-nitrocatechol

CAS No. 3316-09-4. Also called 4-nitrobenzene-1,2-diol; 4-nitropyrocatechol; 1,2‑dihydroxy-4-nitrobenzene; 4-nitro-1,2-benzenediol.

### Maximum Acceptable Value

The DWSNZ do not include a MAV for 4-nitrocatechol. 4-Nitrocatechol is not mentioned in the WHO Guidelines for Drinking-water Quality.

### Sources to drinking-water

#### 1. To source waters

4-Nitrocatechol can form in the atmosphere when biomass burns.

#### 2. From treatment processes

4-Nitrocatcechol is a potential disinfection by-product (DBP) in WTPs using an advanced oxidation process (AOP).

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Water solubility about 8 percent.

### Typical concentrations in drinking-water

The maximum concentration of 4-nitrocatechol measured in drinking water was 0.027 μg/L ITSD eq. (Vughs et al*.*, 2016).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

A structural alert for genotoxic carcinogenicity (QSA27 nitro aromatic) has been identified, so a Threshold of Toxicological Concern (TTC) of 0.0025 µg/kg bw/d is appropriate (DWI 2018). A NOEL of 736,000 μg/kg bw/day derived from the OECD toolbox, so a TDI of 1,472 µg/kg bw/d was used. A potential for DNA binding was predicted, however details on the structural alerts for this chemical were not available. It was concluded that 4-nitrocatechol is ‘probably not’ mutagenic in an Ames assay; however, there were insufficient data available to assess further genotoxic or carcinogenic potential (Vughs et al 2016). It is also predicted to be mutagenic and both a toxicant and non-toxicant in developmental and reproductive activity models although these predictions were unreliable (DWI 2018).

The maximum intake of 4-nitrocatechol via drinking water by adults (0.00090 μg/kg bw/day) is less than the TTC value (0.0025 μg/kg bw/day) and therefore, adverse health effects following adult exposure to this level of 4-nitrocatechol via drinking water are not anticipated in adults. The maximum intake in children and infants (0.00270 to 0.00405 μg/kg bw/day) exceeds the TTC value. Therefore, additional research into the occurrence in drinking water and toxicological properties of this DBP may be prudent.

4-Nitrocatechol is classed as a skin and eye irritant.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Nitrophenols

CAS No. 88-75-5 for 2-nitrophenol. Also called o-nitrophenol or 2‑hydroxynitrobenzene.

CAS No. 100-02-7 for 4-nitrophenol. Also called p-nitrophenol or 4‑hydroxynitrobenzene.

Note that 3-nitrophenol is not found often and is not discussed in this datasheet, apart from some environmental fate information.

Six dinitrophenols exist as well, the most important being 2,4-dinitrophenol, CAS No. 51-28-5, a USEPA Priority Pollutant. Some information on 2,4-dinitrophenol appears below. 2,4-Dinitrophenol can also be called 1-hydroxy-2,4-dinitrobenzene, α‑dinitrophenol, DNP and various trade names.

### Maximum Acceptable Value

The DWSNZ do not include a MAV. Nitrophenols are not mentioned in the WHO Guidelines.

The USEPA (2006/2011) established a lifetime health advisory of 0.06 mg/L for p‑nitrophenol, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

### Sources to drinking-water

#### 1. To source waters

The two main nitrophenols are very similar in their chemical properties. The manufacture of one almost always produces at least a little of the other. The main sources of the two chemicals are industrial manufacturing and processing; 2‑nitrophenol is used mainly to produce dyes, paint colouring, rubber chemicals, and fungicides, whereas 4-nitrophenol is used mainly to manufacture drugs, fungicides, and dyes, and to darken leather. Nitrophenols can also enter the environment due to hydrolytic and photolytic degradation of some pesticides.

2,4-Dinitrophenol (DNP) is primarily used for making dyes, other organic chemicals, and wood preservatives. It is also used to make photographic developer (diaminophenol dihydrochloride), explosives, and insect control substances. Automobile exhaust releases DNP into the air. Dinitrophenols can be impurities in dinoseb (qv).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Solubility in water: 2-nitrophenol about 1,500–2,000 mg/L; 4-nitrophenol about 16,000 mg/L. Neither is likely to be very volatile. Measured coefficients of soil sorption (Koc) in the range of 44 to 530 indicate a low to moderate potential for soil sorption. Nitrophenols released to soil should be biodecomposed under aerobic conditions. Infiltration into groundwater is expected only under conditions unfavourable to biodegradation. Measured half-lifes for the photochemical decomposition of 4‑nitrophenol in water ranged from 2.8 to 13.7 days. Numerous studies on the biodegradation of 2- and 4-nitrophenol indicate the isomers to be inherently biodegradable in water under aerobic conditions. Mineralisation of nitrophenols under anaerobic conditions requires extended adaptation of microbial communities.

If released to soil, 2-nitrophenol can have very high to moderate mobility based upon Koc values in the range of 13–265. The pKa of 2-nitrophenol is 7.23 indicating that this compound will exist partially in anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilisation of 2-nitrophenol from moist soil surfaces may be an important fate process given a Henry’s Law constant of 1.63 x 10-5 atm‑cu m/mole at 25°C. 2-Nitrophenol was completely biodegraded in an aqueous soil solution inoculum obtained from a waste facility within 7-14 days, but it took more than 64 days to biodegrade in soil, suggesting acclimation may be an important factor in the biodegradability of this compound. If released into water, 2-nitrophenol may have little to moderate adsorption to suspended solids and sediment in the water column based upon the Koc values. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 2.8 and 24 days, respectively. Photolysis in sunlit surface waters may occur. 2-Nitrophenol is not expected to undergo hydrolysis in the environment due to the lack of hydrolysable functional groups. BCF values of <22 measured in carp suggest bioconcentration in aquatic organisms is low. A half-life of 37 days was observed for 2-nitrophenol in a laboratory batch microcosm incubated with groundwater and sediment (EAWAG accessed February 2015).

If released to soil, 3-nitrophenol is expected to have high to moderate mobility based on one measured Koc value of 48 and an estimated Koc of 290. The pKa of 3‑nitrophenol is 8.36, indicating that it will partially exist in anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Experimental Koc data for other nitrophenol isomers indicates 3-nitrophenol may be more mobile in anionic form than neutral form. Volatilisation from moist soil surfaces is not expected to occur based on a Henry’s Law constant of 2 x 10-9 atm‑cu m/mole at 25 deg C. 3-Nitrophenol is not expected to volatilise from dry soil surfaces based upon its vapour pressure. 3‑Nitrophenol was completely biodegraded within 3 to 5 days in one soil inoculum study, but required more than 64 days to biodegrade in another soil study; acclimation may be an important consideration for biodegradation rates in soil and water. 3‑Nitrophenol was degraded 72 percent and 67 percent in 2 flooded soils over a 10‑day incubation period and was readily biodegradable (theoretical BODs of  
48–64 percent) in two-week Japanese MITI tests. If released into water, 3-nitrophenol may have little to moderate adsorption to suspended solids and sediment in the water column based upon the Koc values. Volatilisation from water surfaces is not expected to be an important fate process based on its Henry’s Law constant. 3-Nitrophenol was degraded 90 percent using an activated sludge in 16 days including a 10‑day lag period. 3-Nitrophenol was readily biodegradable in river water die-way tests, suggesting that biodegradation may be an important environmental fate process in water. An estimated BCF value of 10 and reported BCF of 25 in fish suggest the potential for bioconcentration in aquatic organisms is low (EAWAG accessed February 2015).

If released to soil, 4-nitrophenol can have very high to low mobility based upon an observed Koc range of 16 to over 500. A reported median Koc of 234 suggests moderate mobility in soil. The pKa of 4-nitropehnol is 7.15, indicating that it will exist partially in anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. The available experimental data suggests 4-nitrophenol may be more mobile in anionic form. Volatilisation from moist soil surfaces is not expected based upon a Henry’s Law constant of 1.28 x 10-8 atm‑cu m/mole at 20 deg C. 4-Nitrophenol is not expected to volatilise from dry soil surfaces based upon its vapour pressure. The biodegradation half-life of 4-nitrophenol in an acidic soil was reported as 2.5 days and the biodegradation half-life in a basic soil was reported as 10.2 days. However, a large number of biodegradation studies have shown that 4-nitrophenol can degrade over a wide range from very slow to fast; in general, 4-nitrophenol degrades much faster in acclimated soil and water. If released into water, 4-nitrophenol may undergo some adsorption to suspended solids and sediment in water based upon the Koc value. The biodegradation half-life of 4-nitrophenol was reported as 18 hours and 6.8 days in aerobic and anaerobic waters, respectively. Volatilisation from water surfaces is not expected to be an important fate process based on its Henry’s Law constant. Photolysis in surface waters is expected to occur based on photolysis half-lifes of 5.7, 6.7, and 13.7 days at pH 5, 7, and 9, respectively (EAWAG accessed February 2015).

Dinitrophenol (DNP) water solubility is about 5,500 mg/L. Neither photochemical nor other chemical processes have been identified that are significant for the transformation/degradation of dinitrophenols in natural waters. The loss of dinitrophenols from water due to volatilisation is negligible. Moderate amounts of dinitrophenols are removed from water to sediment due to adsorption. Biodegradation may be the most important loss process for dinitrophenols in natural waters. It may take between 4 and 80 days for the level of DNP in soil to decrease by half.

If released to soil, 2,4-dinitrophenol is expected to have very high mobility based upon measured Koc values of 13.5 and 16.6. The pKa of 2,4-dinitrophenol is 4.09, indicating that this compound will exist almost entirely in anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts, as is evident by the measured Koc values. Volatilisation from moist soil is not expected because the acid exists as an anion and anions do not volatilise. Utilising the Japanese MITI test, 0 percent of the theoretical BOD was reached in four weeks indicating that biodegradation is not an important environmental fate process. However, the biodegradation half-life of 2,4-dinitrophenol in an acidic soil was reported as 32.1 days and the biodegradation half-life in a basic soil was reported as 4.6 days. If released into water, 2,4-dinitrophenol is not expected to adsorb to suspended solids and sediment based on the measured Koc values. The biodegradation half-life of 2,4-dinitrophenol was reported as 68 days and 2.8 days in aerobic and anaerobic waters, respectively. Volatilisation from water surfaces is not expected to be an important fate process based upon its anionic state. BCFs of  
<0.4–0.7 and <3.7 were measured in carp (*Cyprinus carpio*) suggesting bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since 2,4-dinitrophenol lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

No information was located regarding the effects of 2-nitrophenol or 4-nitrophenol in humans after inhalation, oral, or dermal exposure. The only toxicological signs of probable relevance are haematological effects observed in animals exposed to 2‑nitrophenol or 4-nitrophenol. These effects were reported in an acute duration inhalation study. Oral lethal doses for the two isomers suggest that 2-nitrophenol is less toxic than 4-nitrophenol. The database for 2-nitrophenol is extremely limited, and the database for 4-nitrophenol is insufficient for deriving reliable NO(A)EL values. Therefore, at present, no tolerable daily intakes (TDIs) or tolerable concentrations (TCs) can be derived for either 2- or 4-nitrophenol (WHO 2000).

The reference dose or RfD for 4-nitrophenol (USEPA 2006/2009/2011) is 0.008 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.3 mg/L.

2,4-Dinitrophenol is sold over the Internet under a variety of names as a slimming aid for dieters (including those who are suffering from eating disorders or body dysmorphia) and body builders. This industrial chemical has caused cases of severe illness and deaths in multiple countries in the last 2–3 years. From <http://www.interpol.int/News-and-media/News/2015/N2015-050>.

The oral RfD for 2,4-dinitrophenol was calculated at 0.002 mg/kg/d (USEPA 1991).

During the 1930s DNP was used extensively as a diet pill, so not surprisingly those most exposed were dieters who used these pills. Because of the harmful effects observed (cataracts in young people) the use of DNP was stopped by the United States government in 1938.

The USEPA recommends that the amount of DNP present in bodies of water, such as lakes and rivers, should not be more than 0.07 mg/L in water used for swimming or where water might possibly be swallowed. No more than 0.765 mg/L should be present in water where people catch fish to eat, but there is no swimming. The USEPA has verified a chronic RfD of 0.002 mg/kg/day for 2,4-DNP based on a LOAEL value for intermediate-duration exposure of 2 mg/kg/day for cataracts (ATSDR 1995).

As at July 2013 ATSDR (see <http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.01 mg/kg/day for acute-duration oral exposure  
(1–14 days) to 2,4-dinitrophenol.

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA (1990) has established a Lifetime Health Advisory (LHA) level of 0.06 mg/L for 4-nitrophenol, a breakdown product of methyl parathion, in drinking water. This means USEPA believes that water containing 4-nitrophenol at or below this level is acceptable for drinking every day over the course of one’s lifetime, and does not pose any health concerns. However, consumption of 4-nitrophenol at high levels well above the LHA level over a long period of time has been shown to cause adverse health effects, including damage to the liver, respiratory stress, and inflammation of the stomach in animal studies.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The short-term, chronic and subchronic health risk limits are 0.007 mg/L, and a limit of 0.01 mg/L was set for cancer.

4-Nitrophenol is also a degradation product of parathion.

### References

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WHO. 2004. *Guidelines for Drinking-water Quality 2004* (3rd edition). Geneva: World Health Organization. Available at: [www.who.int/water\_sanitation\_health/dwq/gdwq3/en/print.html](http://www.who.int/water_sanitation_health/dwq/gdwq3/en/print.html) see also the addenda.

# N-nitrosodi-n-butylamine

CAS No. 924-16-3. Also called dibutylnitrosamine, *N,N-*di*-n-*butylnitrosamine, N‑nitroso-di-n-butylamine, *N-b*utyl*-N-*nitroso-l-butamine or NDBA.

Also, see the datasheet for N-nitrosodimethylamine (NDMA) which includes some information about the other N- nitrosamines.

### Maximum Acceptable Value

The WHO Guidelines do not refer to N-nitrosodi-n-butylamine, and there is no MAV in the DWSNZ.

The 1971 WHO International Standards first mentioned concern over the possibility of nitrosamine formation *in vivo*. As nitrosamines were then considered a possible hazard to human health, the 1971 Standards stated that it may eventually become necessary to reduce the level of nitrates in water if it is found that this source makes a significant contribution to the hazard to human health arising from nitrosamines.

The most important nitrosamines (and their CAS numbers) include:

* N-nitrosodimethylamine (NDMA) 62-75-9 (a USEPA priority pollutant)
* N-nitrosomethylethylamine (NMEA) 10595-95-6
* N-nitrosodiethylamine (NDEA) 55-18-5
* N-nitrosodi-n-propylamine (NDPA) 621-64-7 (a USEPA priority pollutant)
* N-nitrosodi-n-butylamine (NDBA) 924-16-3
* N-nitrosopyrollidine (NPYR) 930-55-2
* N-nitrosopiperidine (NPIP) 100-75-4

### Sources to drinking-water

#### 1. To source waters

*N*-Nitrosodi-*n*-butylamine has been detected in a variety of products as a result of the nitrosation of amines present in these products. *N*-Nitrosodi-*n*-butylamine is present in soybean oil at a concentration of 0.29 mg/kg, in cheese at 0.02 to 0.03 mg/kg, and in smoked or cured meats at 0.0002 to 0.004 mg/kg (IARC 1978). *N*-Nitrosodi-*n*-butylamine has also been detected in tobacco smoke at a concentration of 3 ng/cigarette. It may be present in experimental animal feed at concentrations up to 0.004 mg/kg. *N*-Nitrosodi-*n*-butylamine may be formed from secondary or tertiary n-butylamines and quaternary ammonium salts by reaction with nitrosating agents, such as nitrite, in the stomach or during cooking processes. *N*-Nitrosamines, such as *N‑*nitrosodi-*n*-butylamine, are frequently produced during rubber processing and may be present as contaminants in the final rubber product.

#### 2. From treatment processes

See N-nitrosodimethylamine.

#### 3. From the distribution system

See N-nitrosodimethylamine.

### Forms and fate in the environment

*N*-Nitrosodi-*n*-butylamine is sensitive to light, especially ultraviolet light, and undergoes relatively rapid photolytic degradation. It is soluble in water.

### Typical concentrations in drinking-water

Two water utilities in the US reported detecting N-nitrosodi-n-butylamine (NDBA) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0022 mg/L.

A study in Western Australia (Liew et al 2016) found N-nitrosodi-n-butylamine (NDBA) was detected more frequently and at higher concentrations (up to 9.4 ng/L) than the more commonly studied N-nitrosodimethylamine (NDMA), in the chlorinated systems.

### Removal methods

Probably similar to N-nitrosodimethylamine.

### Recommended analytical techniques

#### Referee method

A referee method cannot be selected for the nitrosamines because MAVs have not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for nitrosamines because MAVs have not been established. However, the following may be useful:

USEPA Method 521 (2004). Determination of nitrosamines in drinking water by solid phase extraction and capillary column gas chromatography with large volume injection and chemical ionisation tandem mass spectrometry (MS/MS).

### Health considerations

Estimates indicate that air, diet, and smoking contribute to potential human exposure at levels of a few μg per day. The degree of this potential exposure is unknown, but is assumed to be sporadic and at relatively low levels. Potential exposure depends on the ability of the nitrosamine to migrate from the product and enter the body. The US Consumer Product Safety Commission (CPSC) and FDA determined that the nitrosamines present in pacifiers and baby bottle nipples can migrate from the pacifier or nipple into saliva, which could result in ingestion of nitrosamines.

*N-*Nitrosodi*-n-*butylamine is carcinogenic in all animal species tested: mice, rats, Syrian golden, Chinese and European hamsters, rabbits and guinea-pigs, after its oral, subcutaneous, intraperitoneal or intravenous administration. It produces benign and malignant tumours in the urinary bladder, oesophagus, liver, respiratory tract, stomach and intestine, and also leukaemia; it is particularly effective as a bladder carcinogen. It is carcinogenic following its administration prenatally and in single doses. The two metabolites, *N-*nitroso*-n-*butyl-*N-*(4-hydroxybutyl)amine and *N-*nitroso*-n-*butyl-*N-*(3-carboxypropyl)amine are also carcinogenic (IARC 1978, last updated 27 March 1998). IARC considers that N-nitrosodi-n-butylamine and N-nitrosomethylethylamine are possible human carcinogens (Group 2B), and USEPA classifies them as B2: a probable human carcinogen.

The USEPA quantitative estimate of carcinogenic risk from oral exposure from drinking-water is 0.06 mg/L at the 10-5 level (1 in 100,000).

The seven chemicals above appear on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.095 mg/kg/day for acute-duration oral exposure  
(1–14 days) for N-nitrosodi-n-propylamine (NDPA).

Nitrosodibutylamine is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# N-nitrosodiethyamine

CAS No. 55-18-5. Also called diethylnitrosamine or N-nitroso-di-ethylamine.

Also, see the datasheet for N-nitrosodimethylamine (NDMA) which includes some information about the other N- nitrosamines.

### Maximum Acceptable Value

There is no MAV in the DWSNZ, and the WHO Guidelines do not refer to N‑nitrosodiethylamine.

The USEPA concluded on 22 September 2009 that N-nitrosodiethylamine is known or anticipated to occur in PWSs and may require regulation. Therefore they added N‑nitrosodiethylamine to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

The 1971 WHO International Standards first mentioned concern over the possibility of nitrosamine formation *in vivo*. As nitrosamines were then considered a possible hazard to human health, the 1971 Standards stated that it may eventually become necessary to reduce the level of nitrates in water if it is found that this source makes a significant contribution to the hazard to human health arising from nitrosamines.

The most important nitrosamines (and their CAS numbers) include:

* N-nitrosodimethylamine (NDMA) 62-75-9 (a USEPA priority pollutant)
* N-nitrosomethylethylamine (NMEA) 10595-95-6
* N-nitrosodiethylamine (NDEA) 55-18-5
* N-nitrosodi-n-propylamine (NDPA) 621-64-7 (a USEPA priority pollutant)
* N-nitrosodi-n-butylamine (NDBA) 924-16-3
* N-nitrosopyrollidine (NPYR) 930-55-2
* N-nitrosopiperidine (NPIP) 100-75-4

### Sources to drinking-water

#### 1. To source waters

N-Nitrosodiethylamine is used primarily as a research chemical. It is used as a gasoline and lubricant additive, antioxidant, stabiliser in plastics, fibre industry solvent, copolymer softener, and starting material for synthesis of 1,1-diethylhydrazine. It is also used to increase dielectric constants in condensers. N-Nitrosamines such as N‑nitrosodiethylamine are frequently produced during rubber processing and may be present as contaminants in the final rubber product.

#### 2. From treatment processes

See N-nitrosodimethylamine.

#### 3. From the distribution system

See N-nitrosodimethylamine.

### Forms and fate in the environment

N-Nitrosodiethylamine is widespread in the environment, but it is rapidly decomposed by sunlight, and thus does not usually persist in ambient air or water illuminated by sunlight.

### Typical concentrations in drinking-water

The compound has been found in high-nitrate well water for drinking at concentrations of 0.00001 mg/L and in deionised water at 0.00033 to 0.00083 mg/L. Wastewater from two chemical plants contained 0.00007 and 0.00024 mg/L.

Nitrosamines originating from wastes have been found in groundwaters at between 0.00003–0.00015 mg/L, California Department of Health Services (2006).

### Removal methods

Probably similar to N-nitrosodimethylamine.

### Recommended analytical techniques

#### Referee method

A referee method cannot be selected for the nitrosamines because MAVs have not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for nitrosamines because MAVs have not been established. However, the following may be useful:

USEPA Method 521 (2004). Determination of nitrosamines in drinking water by solid phase extraction and capillary column gas chromatography with large volume injection and chemical ionisation tandem mass spectrometry (MS/MS).

### Health considerations

The general population may possibly be exposed to unknown quantities of N‑nitrosodiethylamine present in foods, beverages, tobacco smoke, herbicides, pesticides, drinking-water, and industrial pollution.

IARC considers that N-nitrosodiethylamine is reasonably anticipated to be a human carcinogen (Group 2A) based on induction of tumors at multiple sites in both rodent and non-rodent species exposed by various routes. The seven chemicals above appear on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008, and USEPA classifies them as B2: probable human carcinogens.

When administered in the drinking-water, N-nitrosodiethylamine induced liver tumors in guinea pigs, rabbits, dogs, and rats, and nasal cavity tumours in rats.

ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.095 mg/kg/day for acute-duration oral exposure (1–14 days) to N-nitrosodi-n-propylamine.

N-Nitrosodiethylamine is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

### Derivation of Maximum Acceptable Value

No MAV.

The California Environment Protection Agency’s Office of Environmental Health Hazard Assessment (OEHHA) has set a notification level of 10 ng/L for N‑nitrosodimethylamine, N-nitrosodiethyamine and N-nitrosodi-N-propylamine. Notification levels are health-based advisory levels established for chemicals in drinking-water that lack maximum contaminant levels ([MCLs](http://www.dhs.ca.gov/ps/ddwem/chemicals/MCL/mclindex.htm)). When chemicals are found at concentrations greater than their notification levels, certain [requirements and recommendations](http://www.dhs.ca.gov/ps/ddwem/chemicals/AL/notificationlevels.htm#REQUIREMENTS AND RECOMMENDATIONS#REQUIREMENTS AND RECOMMENDATIONS) apply.

Drinking-water concentrations at specified risk levels (USEPA IRIS):

* 1 in 10,000 0.02 micrograms/L (0.00002 mg/L)
* 1 in 100,000 0.002 micrograms/L (0.000002 mg/L)
* 1 in 1,000,000 0.0002 micrograms/L (0.0000002 mg/L)

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# N-nitrosodimethylamine

CAS No. 62-75-9. Also called dimethylnitrosamine, N-nitroso-di-methylamine, *N‑*methyl-*N*-nitrosomethanamine, or NDMA.

This datasheet also includes some information about the other N- nitrosamines.

### Maximum Acceptable Value

No MAV as at DWSNZ 2008. WHO (2006) proposed a health-based value of 0.0001 mg/L. NDMA is included in the [plan of work of the rolling revision](http://www.who.int/entity/water_sanitation_health/gdwqrevision/en/index.html) of the WHO Guidelines for Drinking-water Quality. Arising from that, WHO (2008) derived a GV of 0.0001 mg/L (0.1 µg/L, 100 ng/L); this GV is included in WHO (2017).

The USEPA concluded on 22 September 2009 that N-nitrosodimethylamine is known or anticipated to occur in PWSs and may require regulation. Therefore they added N‑nitrosodimethylamine to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a).

The 1971 WHO International Standards first mentioned concern over the possibility of nitrosamine formation *in vivo*. As nitrosamines were then considered a possible hazard to human health, the 1971 Standards stated that it may eventually become necessary to reduce the level of nitrates in water if it is found that this source makes a significant contribution to the hazard to human health arising from nitrosamines.

The most important nitrosamines (and their CAS numbers) include:

* N-nitrosodimethylamine (NDMA) 62-75-9
* N-nitrosomethylethylamine (NMEA) 10595-95-6
* N-nitrosodiethylamine (NDEA) 55-18-5
* N-nitrosodi-n-propylamine (NDPA) 621-64-7
* N-nitrosodi-n-butylamine (NDBA) 924-16-3
* N-nitrosopyrollidine (NPYR) 930-55-2
* N-nitrosopiperidine (NPIP) 1 100-75-4

Most attention has been directed towards N-nitrosodimethylamine (NDMA). It has also been called:

* dimethylnitrosamine (primary synonym), and
* nitrosodimethylamine
* N-methyl-N-nitrosomethanamine
* N,N-dimethylnitrosamine
* N-methyl N-nitrosomethanamine

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of N-nitrosodimethylamine (NDMA) in drinking water should not exceed 0.0001 mg/L. Action to reduce NDMA is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than NDMA.

The California Department of Public Health (CDPH) has established a notification level for NDMA of 10 ng/L.

Health Canada established a maximum acceptable concentration of 0.00004 mg/L. The Ontario Ministry of the Environment (MOE), Ontario, Canada, has set a Maximum Acceptable Concentration (MAC) for NDMA at 9 ng/L (quoted in DWI 2008a).

N-nitrosodimethylamine and N-nitrosodi-n-propylamine are “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

N-Nitrosodimethylamine is used primarily as a research chemical. Prior to April 1976, the compound was used as an intermediate in the electrolytic production of 1,1‑dimethylhydrazine, a storable liquid rocket fuel containing approximately 0.1 percent N-nitrosodimethylamine as an impurity. Other uses (mostly ceased now) of N-nitrosodimethylamine include the control of nematodes, inhibition of nitrification in soil, plasticiser for rubber and acrylonitrile polymers, in active metal anode-electrolyte systems (high-energy batteries), in the preparation of thiocarbonyl fluoride polymers, as a solvent in the fibre and plastics industry, an antioxidant, a softener of copolymers, and an additive to lubricants (Sittig 1985, Merck 1983). NDMA may be present in discharges from industries such as rubber manufacturing, leather tanning, pesticide manufacturing, food processing, foundries, and dye manufacturing and, in addition, sewage treatment plant effluent. Almost all of the releases are to water (WHO 2006).

NDMA has been detected in a variety of personal care and cosmetic products (eg, shampoos, hair conditioners and toners, bath and shower gels, creams and oils, face tonics, cleansers). This is likely due to the reaction of nitrosating agents such as nitrite or nitrogen oxides, which occur frequently in these products with amine-containing compounds used extensively in ingredients of personal care products; copied from Health Canada (2011).

The following dimethylamine (DMA) formulation pesticides act as a precursor or may contain NDMA as a microcontaminant: bromacil, benazolin, 2,4-D, dicamba, MCPA, and mecoprop (WHO 2002). DWI (2008a) added thiram, dichlofluanid and tolyfluanid to this list. In the 1990s, in testing in Canada (Health Canada 2011) of over 100 samples of formulated pesticidal products (DMA salt of phenoxy acid herbicides) potentially contaminated by NDMA, the compound was detected in 49 percent of the samples with an average concentration of 0.44 μg/g; only six samples contained concentrations above 1.0 μg/g (1.02–2.32 μg/g). Concentrations of NDMA in pesticides have decreased over time. In 1994, approximately 1 million kilograms of DMA formulated phenoxy acid herbicides for commercial use were applied to the terrestrial environment in Canada. Based on the amount of DMA-formulated phenoxy acid herbicides applied in 1994, the average NDMA concentration of 0.44 μg/g and the 49 percent detection rate, it has been estimated that approximately 200 g of NDMA may have been released into the environment through the use of these herbicides.

Sources of some NDMA precursors occur in treated wastewater. WRF (2015) and WRF (2016b) show that the NDMA formation potential correlates with the concentration of sucralose and fluorescence in the raw water, where sucralose and fluorescence are used as surrogate indicators of sewage. The datasheet for artificial sweeteners discusses sucralose.

#### 2. From treatment processes

N-Nitrosodimethylamine (NDMA) was found to be present in the effluent at high levels from an ion exchange treatment facility being used for nitrate removal, California Department of Health Services (2002). Only resins containing trimethyl or dimethyl-ethanol quaternary amine functional groups produced NDMA.

NDMA is formed by the reaction of monochloramine with dimethylamine (DMA, a ubiquitous component of surface waters). NDMA can also be formed by reaction of DMA with hypochlorous acid (HOCl) in the presence of nitrite. NDMA appears not to be formed during ozonation.

Chloramine plants often produced NDMA, whereas chlorine plants typically did not. WRA (2013) states that:

The most significant risk for the formation of NDMA in drinking water treatment is the presence of dichloramine. Therefore any measure that reduces the formation of this compound will aid in management of the disinfection by-product. Three operational measures which can be applied during chloramination to minimise the formation of dichloramine are:

* pH >8.4
* Cl2:NH3–N <5.1
* addition of chlorine prior to ammonia.

Other strategies which can be applied to reduce the risk of NDMA formation in drinking water include:

* optimised organic carbon removal
* reduced use of cationic polymers
* reduced return of waste streams to the head of the plant
* pre-oxidation.

Cationic polyelectrolytes (epi-DMA and polyDADMAC products) also act as precursors for the formation of NDMA by reaction with chloramines or hypochlorite. It is not clear whether it is the polymer itself, impurities, and/or degradation components that are responsible for the NDMA formation potential. WRF (2015) reports the results of investigations, including that the use of polyDADMAC raised NDMA formation potential by 6 and 12 ng/L on a median and maximum basis, respectively.

Overseas, water suppliers have been converting from chlorine to chloramine disinfectant to reduce regulated disinfection by-products. Ironically, this may pose a greater health risk than the currently regulated DBPs; NDMA is found above the minimum reporting level in ~50 percent of samples in treatment systems using chloramination as a primary disinfectant.

NDMA can also occur when the raw water ammonia concentration increases resulting in breakpoint chlorination not being achieved; WRF (2016a).

Zhao et al (2008) discuss the formation of *n*-nitrosamines from eleven disinfection treatments of seven different surface waters.

DWI (2013) refers to NDMA being detected in ferric coagulants, and to a lesser extent, some alum products. All coagulants used in drinking water treatment in England and Wales were analysed and the results showed that NDMA was a contaminant in many of the coagulants, notably in five ferric sulfates (at concentrations up to 19.0 μg/L) produced by two manufacturers using a similar process. The NDMA concentration in one ferric sulfate increased considerably during the study, up to 380 μg/L, increasing NDMA concentrations in treated waters and distribution significantly. The source of the contamination was believed to be a raw material used in the manufacturing process. NDMA concentrations in the coagulant were reduced following a change in the manufacturing process and source of the raw material. NDMA was detected in other ferric and aluminium coagulants at concentrations near the limit of detection of the analytical method indicating concentrations in the coagulants <1.0 μg/L.

Of the other nitrosamines, only N-nitrosomorpholine (no datasheet) was detected in the ferric sulfate analysed (it is often found in sewage too, WRF 2016a). NMOR was detected at concentrations up to 28 μg/L in samples of coagulant taken from a water treatment works but not in any samples of final water from the works.

An investigation in Western Australian drinking water sources compared the production of various N-nitrosamines when chlorinated or chloraminated. Linge et al (2017). In the chloramination experiments, NDMA was the most frequently detected species and was always detected at the highest concentrations compared with the other *N*-nitrosamines: *N*-nitrosoethyl-methylamine (NEMA), *N*-nitrosodibutylamine (NDBA), *N*-nitrosodiethylamine (NDEA), and *N*-nitrosopyrrolidine (NPYR). *N‑*nitrosopiperidine (NPIP) concentrations however, were consistently detected after formation in both chlorination and chloramination. In the chlorination experiments, the most frequently detected species was NEMA, which showed low variability between duplications and concentrations and were at least double the LOD. Waters with the highest concentrations were those with the highest UV254 absorbance. There was no correlation found between total cyanobacterial count and the formation of *N‑*nitrosamines, suggesting that cyanobacteria do not necessarily add to *N‑*nitrosamine precursors in the affected surface waters. Chloraminated waters were found to be between 1.4 and 3.1 times more toxic compared with corresponding chlorinated samples. In all samples where NDMA was detected, NDMA contributed 75 percent or more of the relative toxicity in comparison with the other *N*-nitrosamines species. NDMA was the highest contributor to overall *N*-nitrosamine toxicity, suggesting that other *N*-nitrosamines will possibly only influence toxicity in water sources when they are present at much higher concentrations than NDMA.

#### 3. From the distribution system

The California Department of Health Services (2002) reported that in some instances NDMA is formed slowly, so long detention times in the distribution system may increase the levels of NDMA. Nitrification by nitrifying bacteria may occur in systems that practise chloramination. Similarly, other species of bacteria may cause the formation of NDMA.

NDMA has been found in drinking-water in South Australia, leaching from rubber rings and rubber inserts from gate valves. NDMA was also found to leach into deionised water, suggesting that it came directly from the rubber and not a DBP (Morran et al 2009). WRF (2015) reports results of trials of NDMA leaching and formation from rubber materials.

Where chloramination is used, distribution system samples can have much higher levels of NDMA than the finished water at the treatment plant; levels as high as 0.00016 mg/L have been measured in the distribution system, but concentrations in water at the treatment plant are generally less than 0.00001 mg/L (WHO 2011).

NDMA formation tended to increase in chloraminated distribution systems (the median, 75th percentile, and 90th percentile increase between the plant effluent and distribution system/maximum detention time was 1.7, 6.3, and 18 ng/L, respectively) (WRF 2016a).

### Forms and fate in the environment

N-Nitrosodimethylamine is a yellow, oily liquid of low viscosity, highly soluble (miscible) in water. The compound is sensitive to light, especially ultraviolet light, and undergoes relatively rapid photolytic degradation. N-Nitrosodimethylamine is widespread in the environment, but it is decomposed rapidly by sunlight (photolytic half-life of NDMA vapour exposed to sunlight ranges between 0.5 and 1.0 h), and thus does not usually persist in ambient air or water illuminated by sunlight (HEEP 1980). The log *n*-octanol/water partition coefficient (Kow) is 0.57 and Henry’s law constant (Kaw) is 3.34 Pa·m3/mol at 25°C; Health Canada. 2011. The low vapour pressure and low *n*-octanol/water partition coefficient mean it is not likely to bioaccumulate, adsorb to particulates or volatilise to any significant extent. It will volatilise rapidly from soil (half life a few hours).

Because of its solubility and low partition coefficient, NDMA has the potential to leach into and persist in groundwater. Nitrosamines originating from wastes have been found in groundwaters at between 0.00003–0.00015 mg/L (California Department of Health Services 2006).

If released to soil, observed Koc values of 68–118 indicate N-nitrosodimethylamine is expected to have high mobility. Low adsorption to soils and ready leaching have been observed in several studies. Volatilisation from wet soil surfaces may be an important fate process based on a measured Henry’s Law constant of 1.08 x 10-6 atm‑cu m/mole at 20°C. Under laboratory conditions, greater than 70 percent of N‑nitrosodimethylamine applied to the surface of a moist, warm soil (12 percent moisture content, 22°C) volatilised in 10 hours. N-Nitrosodimethylamine may potentially volatilise from dry soil surfaces based upon its measured vapour pressure. A half-life of about three weeks was reported for N-nitrosodimethylamine in aerobic soil under laboratory conditions; the primary removal processes were volatilisation and biodegradation. At 21°C, observed half-lives in ground cover soil, turfgrass soil and tree soil were 4.1, 5.6 and 22.5 days respectively. The rates and extent of N‑nitrosodimethylamine biodegradation in natural environments, including surface waters, sludges and soils have been observed to be highly variable. If released into water, N-nitrosodimethylamine is not expected to adsorb to suspended solids and sediment in the water column based upon its Koc values. Volatilisation from water surfaces is expected to occur based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 29 and 215 days, respectively. The potential for bioconcentration in aquatic organisms is low based upon an estimated BCF of 3. Hydrolysis is not expected to be an important process. A photodegradation half-life of 79 hours was measured in distilled water exposed to fluorescent light through a pyrex filter (wavelength >280 nm). The direct photolysis rate constant for N-nitrosodimethylamine in aqueous solution exposed to irradiation representing Southern California midsummer, midday sun was observed to be 0.040/minutes with a half-life of 16 minutes (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

Concentrations referred to in California Department of Health Services (2002) were generally <0.000005 mg/L (5 ng/L), with high results around 0.00006 mg/L for disinfection by‑products, and around 0.00015 mg/L for a groundwater. The US Department of Health and Human Services Public Health Service National Toxicology Program reported [0.0008](http://ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s128nitr.pdf%20quotes%20%20%20%20%20%20%20%20%200.0008) mg/L in a drinking-water containing NDMA as a disinfection by‑product.

In 390 samples of raw surface water from 101 water treatment plants sampled for NDMA in Ontario from 1990 to July 1998, concentrations were detectable (>0.000001 mg/L) in the raw water at 37 plants. The average concentration in raw water was 1.27 × 10-6 mg/L. The highest concentration of NDMA in raw water was 0.000008 mg/L (8 × 10-6 mg/L) from two water treatment plants in 1996.

Twenty-six water utilities in the US reported detecting n-nitrosodimethylamine in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00026 mg/L.

Research commissioned by DWI on behalf of DEFRA assessed the occurrence of NDMA in drinking water in England and Wales (available at [www.dwi.gov.uk/research](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.dwi.gov.uk\research)). At over 90 percent of the treatment works, samples of final water were free from detectable concentrations of NDMA (limit of detection 0.9 ng/L). At three treatment works, trace levels of NDMA were found in the final water, all final water concentrations were below 10 ng/L, compared with the proposed WHO guideline value of 100 ng/L (DWI 2008). Concentrations up to 5.8 ng/L were found (DWI 2008a).

### Removal methods

NDMA is not reliably removed by air stripping, activated carbon adsorption, reverse osmosis or biodegradation. It is degraded extremely slowly by ozone. Potential treatment technologies are UV irradiation, UV plus hydrogen peroxide and resin (eg, Ambersorb 572) adsorption. Currently the most common process for NDMA removal is UV irradiation. The principal by‑products of UV photolysis of NDMA are DMA and nitrite. Medium-pressure UV lamps are generally more economic for NDMA destruction than low-pressure lamps. The UV dose required for 90 percent removal of NDMA is about 1000 mJ/cm2, approximately 10 times higher than that required for virus inactivation, or 25 times more than standard water treatment dose rates. NDMA is poorly removed by reverse osmosis. Some nitrosamines are partially removed by NF/RO (Miyashita et al 2009).

Potential methods for reducing the formation of NDMA during disinfection include avoiding the use of chloramination, breakpoint chlorination, and removal of ammonia prior to chlorination (WHO 2011). Pre-oxidation with chlorine or ozone was typically effective at destroying NDMA precursors (WRF 2015, 2016a). Free chlorine (HOCl) applied at low and high exposures reduced subsequent NDMA formation by 53 and 80 percent on a median basis, respectively. Ozone was generally very effective at precursor deactivation. UV irradiation was effective at around 186 mJ/cm2, a dose that is about 4 to 5 times that normally used for disinfection. Permanganate treatment did not significantly reduce NDMA formation.

WRF (2015) is devoted entirely to developing improved strategies for minimising nitrosamine formation during drinking water treatment and provide treatment guidance for utilities. These objectives were achieved through the following research tasks:

* conduct preliminary case studies on nitrosamine formation and control at full-scale drinking water treatment plants
* optimise oxidation strategies for nitrosamine and regulated disinfection by-product control
* optimise polymer usage for nitrosamine and turbidity control
* remove nitrosamine precursors from water
* evaluate and control other sources of nitrosamines and their precursors
* develop a decision document and preliminary cost/benefit analysis of control alternatives.

WRF (2016) recommends:

1 avoid the use of chloramines, or if not possible, minimise nitrification

2 avoid the use of cationic polymers with quaternary amine groups

3 increase contact time with free chlorine if possible

4 avoid raw water sources impaired by wastewater discharges

5 use of strong oxidants like ozone or chlorine dioxide is likely to help in controlling nitrosamine precursors.

WRF (2016b) reports that the NDMA Formation Potential removal by alum clarification was 19 percent, independent of seasons and temporal climatic conditions. PAC addition above 4 mg/L increased the NDMA FP removal. The average removals for reverse osmosis and microfiltration were 81 percent and 7 percent, respectively. Simultaneous application of Cl2 and ClO2 as preoxidants removed about 56 percent of NDMA FPs, while the use of Cl2 or ClO2 as post-oxidants (without pre-oxidation) resulted in 35 percent and 27 percent removals, respectively. Supplemental addition of small dose of Cl2 (0.4–0.8 mg/L) to ClO2 (0.5-1.0 mg/L) during post-oxidation increased the NDMA FP removal to an average of 46 percent at one WTP. The total average NDMA FP removal between raw water and the point of entry (POE) at the WTPs was about 49 percent. The average NDMA FP removals were, in general, independent of seasons and climatic events such as wet/dry periods and high/low river flow conditions.

WRF (2016c) reports results of an Australian study where advanced treatment that used ozone and BAC achieved greater removal of TON compared with conventional treatment (65 percent vs 51 percent, respectively).

### Recommended analytical techniques

#### Referee method

A referee method cannot be selected for the nitrosamines because MAVs have not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for nitrosamines because MAVs have not been established. However, the following may be useful:

USEPA Method 521 (2004). Determination of nitrosamines in drinking water by solid phase extraction and capillary column gas chromatography with large volume injection and chemical ionisation tandem mass spectrometry (MS/MS).

See WHO (2008) for further discussion on analytical techniques. Also see DWI (2008a) and WRF (2015).

### Health considerations

Health Canada (2011) considers that Canadians can be exposed to NDMA through its presence in water, air and food. Drinking water is considered to be only a minor source of exposure to NDMA compared with other sources. Overall, the concentration of NDMA measured in Canadian water supplies is normally well below the MAC.

Individuals who smoke, consume a typical western diet and drink beer, were estimated to have an average daily N-nitrosamine exposure of 24,820 ng, with the majority of (88 percent) coming from tobacco, with food (8 percent), beer (4 percent), and potable water (<1 percent) accounting for the rest. Average intake for non-drinking, non-smoking vegetarians was estimated at 1920 ng/day, with food comprising 94 percent and potable water 6 percent. Personal care products were excluded from the estimates although some have been reported to contain high levels of N-nitrosamines. The authors note however, that endogenous formation of N-nitrosamines in the human body is believed to outweigh endogenous sources by a factor of more than 30 to 1. Therefore avoiding ingestion or inhalation of pre-formed N-nitrosamines is unlikely to have any impact on total exposure (Gushari and Halden 2018).

The National Toxicology Program’s Report on Carcinogens (US Department of Health and Human Services Public Health Service) gives links to profiles of the nitrosamines.

NDMA has consistently shown potent carcinogenicity in all laboratory animal studies. As a result, there has been little attempt to study other toxic end-points, and there are inadequate data available to make a meaningful assessment of end-points other than carcinogenicity.

N-Nitrosodimethylamine is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity in experimental animals, ie, IARC Group 2A and USEPA B2: probable human carcinogen. This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. When administered orally, N-nitrosodimethylamine induced liver hemangiosarcomas, hepatocellular carcinomas, and kidney and lung tumours in mice; kidney and bile duct tumors in rats; hepatocellular carcinomas and bile duct tumors in hamsters; hepatocellular carcinomas and bile duct tumors in rabbits and guinea pigs; liver hemangiosarcomas in ducks; and liver adenomas and adenocarcinomas in fish. No adequate human studies of the relationship between exposure to N‑nitrosodimethylamine and human cancer have been reported.

Estimates indicate that air, diet, and smoking contribute to potential human exposure at levels of a few micrograms per day. N-Nitrosodimethylamine is present in a variety of foods including cheeses, soybean oil, canned fruit, various meat products, bacon, various cured meats, frankfurters, ham (cooked), fish and fish products, spices used for meat curing, apple brandy, other alcoholic beverages, and beer. Concentrations in these foodstuffs have been measured to be between 0 and 85 μg/kg.

The USEPA (2009/2011) quotes a health advisory of 0.00007 mg/L for N‑nitrosodimethylamine, representing a 10-4 cancer risk.

The California Environment Protection Agency’s Office of Environmental Health Hazard Assessment (OEHHA) has set a notification level of 10 ng/L for N‑nitrosodimethylamine, N-nitrosodiethylamine and N-nitrosodi-N-propylamine. Notification levels are health-based advisory levels established for chemicals in drinking-water that lack maximum contaminant levels ([MCLs](http://www.dhs.ca.gov/ps/ddwem/chemicals/MCL/mclindex.htm)). When chemicals are found at concentrations greater than their notification levels, certain [requirements and recommendations](http://www.dhs.ca.gov/ps/ddwem/chemicals/AL/notificationlevels.htm#REQUIREMENTS AND RECOMMENDATIONS#REQUIREMENTS AND RECOMMENDATIONS) apply.

The OEHHA has developed a proposed Public Health Goal (PHG) of 3×10-6 mg/L (0.003 μg/L, or 3 ng/L) for N-nitrosodimethylamine (NDMA) in drinking water. Studies by the California Department of Health Services (DHS) have documented the formation of NDMA during the water disinfection treatment process. The proposed PHG is based on an extra cancer risk of 1 in 1 million for lifetime exposure to NDMA in drinking water. California Department of Health Services OEHHA (2006).

There is also ample evidence that NDMA is genotoxic both *in vivo* and *in vitro* (WHO 2011).

The USEPA recommends that levels (Ambient Water Criterion for humans) in lakes and streams should be limited to 0.0014 ppb (1.4 ng/L) of N-nitrosodimethylamine at a 10-6 risk level, to prevent possible health effects from drinking water or eating fish contaminated with N-nitrosodimethylamine (USEPA 1980).

Ironically, in an attempt to reduce regulated halogenated DBPs when disinfecting with chlorine, some drinking water utilities may be making process changes (such as changing to chloramine disinfection) that could preferentially be forming potentially more toxic unregulated DBPs, such as NDMA.

N-nitrosodimethylamine is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

### Derivation of Maximum Acceptable Value

No MAV as at DWSNZ 2008.

There is conclusive evidence that NDMA is a potent carcinogen in experimental animals by several routes of exposure, including through ingestion of drinking water. NDMA was classified in 1987 by IARC in Group 2A “probably carcinogenic to humans”. As a consequence of the clear evidence of carcinogenicity there have been few studies of other possible toxic endpoints. It has therefore been decided that the existing data is inadequate to quantify health risk for NDMA by any other endpoint than carcinogenicity.

WHO (2006) stated: a guideline value would be based on hepatic biliary cystadenomas in female rats, the most sensitive carcinogenic end-point, observed in a drinking-water study, using a multistage model. Taking a conservative approach to the cancer risk assessment in view of the evidence that humans may be particularly at risk from NDMA the guideline value for NDMA in drinking-water associated with an upper-bound excess lifetime cancer risk of 10-5 can be calculated as follows:

60 kg x 10-5 = 0.1 micrograms/L

2.77 x 10-3 micrograms/kg body weight per day x 2 L

where:

* 60 kg is the average weight of an adult
* 10-5 is the upper-bound limit risk of one additional cancer per 100,000 of the population ingesting drinking water containing NDMA at the guideline value for 70 years
* 2.77 x 10-3 μg/kg body weight per day is the unit risk calculated from the 95 percent lower confidence limit of the TD05 for hepatic biliary cystadenoma in female rats (WHO 2002)
* two litres/day is the daily volume of water consumed by an adult.

A MAV for a 70 kg person would also be 0.1 µg/L (0.0001 mg/L or 100 ng/L).

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# N-nitrosodi-n-propylamine

CAS No. 621-64-7. Also called N,N-dipropylnitrosamine or N-nitroso-di-n-propylamine.

Also, see the datasheet for N-nitrosodimethylamine (NDMA) which includes some information about the other N- nitrosamines.

### Maximum Acceptable Value

There is no MAV in the DWSNZ, and the WHO Guidelines do not refer to N-nitrosodi-n-propylamine.

The USEPA concluded on 22 September 2009 that N-nitrosodi-n-propylamine is known or anticipated to occur in PWSs and may require regulation. Therefore they added N‑nitrosodi-n-propylamine to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

The 1971 WHO International Standards first mentioned concern over the possibility of nitrosamine formation *in vivo*. As nitrosamines were then considered a possible hazard to human health, the 1971 Standards stated that it may eventually become necessary to reduce the level of nitrates in water if it is found that this source makes a significant contribution to the hazard to human health arising from nitrosamines.

The most important nitrosamines (and their CAS numbers) include:

* N-nitrosodimethylamine (NDMA) 62-75-9 (a USEPA priority pollutant)
* N-nitrosomethylethylamine (NMEA) 10595-95-6
* N-nitrosodiethylamine (NDEA) 55-18-5
* N-nitrosodi-n-propylamine (NDPA) 621-64-7 (a USEPA priority pollutant)
* N-nitrosodi-n-butylamine (NDBA) 924-16-3
* N-nitrosopyrollidine (NPYR) 930-55-2
* N-nitrosopiperidine (NPIP) 100-75-4

### Sources to drinking-water

#### 1. To source waters

N-nitrosodi-n-propylamine is a chemical produced by industry in small amounts for research. Small amounts of N-nitrosodi-n-propylamine are produced as a side reaction during some manufacturing processes, as a contaminant in some weedkillers (dinitroaniline-based, eg, trifluralin, isopropalin, and oryzalin), and during the manufacture of some rubber products.

### Forms and fate in the environment

N-nitrosodi-n-propylamine is broken down in water within a few hours.

### Typical concentrations in drinking-water

Nitrosamines originating from wastes have been found in groundwaters at between 0.00003–0.00015 mg/L, California Department of Health Services (2006).

One water utility in the US reported detecting n-nitroso di-n-propylamine in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00000067 mg/L.

### Recommended analytical techniques

#### Referee method

A referee method cannot be selected for the nitrosamines because MAVs have not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for nitrosamines because MAVs have not been established. However, the following may be useful:

USEPA Method 521 (2004). Determination of nitrosamines in drinking water by solid phase extraction and capillary column gas chromatography with large volume injection and chemical ionisation tandem mass spectrometry (MS/MS).

### Health considerations

People may be exposed to low levels of N-nitrosodi-n-propylamine by eating foods treated with sodium nitrite preservatives and by drinking certain alcoholic beverages. Low levels may occur in cigarette smoke.

No studies are available on whether or not N-nitrosodi-n-propylamine causes cancer in people. When administered to rats in the drinking-water, N-nitrosodi-n-propylamine induced carcinomas of the liver and the tongue and papillomas and carcinomas of the esophagus. The Department of Health and Human Services (DHHS) has determined that N-nitrosodi-n-propylamine may reasonably be anticipated to be a human carcinogen. This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008, and the USEPA classified it as B2: a probable human carcinogen.

ATSDR (2010) quotes a minimal risk level (MRL) of 0.095 mg/kg/day for acute-duration oral exposure (1–14 days).

N-nitrosodipropylamine is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA recommends that levels of N-nitrosodi-n-propylamine in lakes and streams should be limited to 0.005 parts N-nitrosodi-n-propylamine per billion parts of water (0.005 ppb or 0.000005 mg/L) to prevent possible health effects from drinking-water or ingesting fish contaminated with N-nitrosodi-n-propylamine.

The California Environment Protection Agency’s Office of Environmental Health Hazard Assessment (OEHHA) has set a notification level of 10 ng/L for N‑nitrosodimethylamine, N-nitrosodiethyamine and N-nitrosodi-N-propylamine. Notification levels are health-based advisory levels established for chemicals in drinking-water that lack maximum contaminant levels ([MCLs](http://www.dhs.ca.gov/ps/ddwem/chemicals/MCL/mclindex.htm)). When chemicals are found at concentrations greater than their notification levels, certain [requirements and recommendations](http://www.dhs.ca.gov/ps/ddwem/chemicals/AL/notificationlevels.htm#REQUIREMENTS AND RECOMMENDATIONS#REQUIREMENTS AND RECOMMENDATIONS) apply.

Drinking-water concentrations at specified risk levels (USEPA IRIS):

* 1 in 10,000 0.5 micrograms/L (0.0002 mg/L)
* 1 in 100,000 0.05 micrograms/L (0.00002 mg/L)
* 1 in 1,000,000 0.005 micrograms/L (0.000002 mg/L)

### References

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California Environmental Protection Agency Department of Health Services OEHHA. 2006. N-Nitrosodimethylamine. *Public Health Goals for Chemicals in Drinking Water*. 60 pp. See: <http://ww2.cdph.ca.gov/certlic/drinkingwater/Pages/NDMAhistory.aspx> and click PHG for NDMA.

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IARC. 1987. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 17. Some n-nitroso compounds. <http://monographs.iarc.fr/ENG/Monographs/allmonos30.php>.

US Department of Health and Human Services Public Health Service National Toxicology Program. *Report on Carcinogens* (11th edition). Regularly updated on: <http://ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s128nitr.pdf>.

USEPA. 1980. *Ambient Water Quality Criteria for Nitrosamines*. Washington, DC: United States Environmental Protection Agency.

USEPA. 2004. EPA Document #: EPA/600/R-05/054 Method 521. *Determination of Nitrosamines in Drinking Water by Solid Phase Extraction and Capillary Column Gas Chromatography with Large Volume Injection and Chemical Ionisation Tandem Mass Spectrometry (MS/MS)*. Cincinnati, Ohio 45268: National Exposure Research Laboratory, Office of Research and Development, US Environmental Protection Agency. See: <http://www.epa.gov/nerlcwww/m_521.pdf>. USEPA Methods can be accessed at <http://water.epa.gov/scitech/drinkingwater/labcert/methods_index.cfm>.

USEPA. 2009. *Contaminant Information Sheets for the Final CCL 3 Chemicals*. EPA 815‑R-09-012. 216 pp. <http://water.epa.gov/scitech/drinkingwater/dws/ccl/upload/Final-CCL-3-Contaminant-Information-Sheets.pdf>.

USEPA IRIS. 1987, revised 1993. N-nitrosodi-n-propylamine. *Integrated Risk Information System*. See <http://www.epa.gov/IRIS/subst/0177.htm>.

# N-nitrosodiphenylamine

CAS No. 86-30-6. Also called N,N-diphenylnitrosamine, *N-*nitroso-*N-*phenyl-benzenamine, N-nitroso-di-phenylamine, and nitrous diphenylamide.

Also, see the datasheet for N-nitrosodimethylamine (NDMA) which includes some information about the other N- nitrosamines.

### Maximum Acceptable Value

There is no MAV in the DWSNZ, and the WHO Guidelines do not refer to N‑nitrosodiphenylamine.

The USEPA concluded on 22 September 2009 that N-nitrosodiphenylamine is known or anticipated to occur in PWSs and may require regulation. Therefore they added N‑nitrosodiphenylamine to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

The 1971 WHO International Standards first mentioned concern over the possibility of nitrosamine formation *in vivo*. As nitrosamines were then considered a possible hazard to human health, the 1971 Standards stated that it may eventually become necessary to reduce the level of nitrates in water if it is found that this source makes a significant contribution to the hazard to human health arising from nitrosamines.

The most important nitrosamines (and their CAS Numbers) include:

* N-nitrosodimethylamine (NDMA) 62-75-9 (a USEPA priority pollutant)
* N-nitrosomethylethylamine (NMEA) 10595-95-6
* N-nitrosodiethylamine (NDEA) 55-18-5
* N-nitrosodi-n-propylamine (NDPA) 621-64-7 (a USEPA priority pollutant)
* N-nitrosodi-n-butylamine (NDBA) 924-16-3
* N-nitrosopyrollidine (NPYR) 930-55-2
* N-nitrosopiperidine (NPIP) 100-75-4

### Sources to drinking-water

#### 1. To source waters

N-nitrosodiphenylamine is a chemical produced by industry in small amounts for research. Small amounts of N-nitrosodiphenylamine are produced as a side reaction during some manufacturing processes, and was used as a retardant during the manufacture of some rubber products.

### Forms and fate in the environment

With a solubility of about 40 mg/L, N-nitrosodiphenylamine can theoretically enter groundwater; however, it tends to sorb to particulate matter. The major environmental fate process for N-nitrosodiphenylamine in water is biodegradation. It is broken down in water within a few weeks.

### Typical concentrations in drinking-water

Nitrosamines originating from wastes have been found in groundwaters at between 0.00003–0.00015 mg/L, California Department of Health Services (2006).

### Recommended analytical techniques

#### Referee method

A referee method cannot be selected for the nitrosamines because MAVs have not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for nitrosamines because MAVs have not been established. However, the following may be useful:

USEPA Method 521 (2004). Determination of nitrosamines in drinking water by solid phase extraction and capillary column gas chromatography with large volume injection and chemical ionisation tandem mass spectrometry (MS/MS).

### Health considerations

IARC (1982) stated that there is limited evidencefor the carcinogenicity of *N‑*nitrosodiphenylamine in experimental animals, but no evaluation of the carcinogenicity of *N-*nitrosodiphenylamine to humans could be made (Group 3).

The USEPA has given N-nitrosodiphenylamine a B2 classification: probable human carcinogen, based on increased incidence of bladder tumours in male and female rats and reticulum cell sarcomas in mice, and structural relationship to carcinogenic nitrosamines. This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. N‑nitrosodiphenylamine is quite likely to be a component of tobacco smoke.

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA recommends that the concentration in drinking-water should not exceed 0.7 mg/L, for a 10-5 risk level.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The cancer health risk limit for N-nitrosodiphenylamine is 0.07 mg/L.

### References

ATSDR. 1993. *Toxicological Profile for N-Nitrosodiphenylamine*. Atlanta, GA: Agency for Toxic Substances & Disease Registry, Department of Health and Human Services. See: <http://www.atsdr.cdc.gov/toxprofiles/index.asp>.

California Environmental Protection Agency Department of Health Services OEHHA. 2006. N-Nitrosodimethylamine. *Public Health Goals for Chemicals in Drinking Water*. 60 pp. See: <http://ww2.cdph.ca.gov/certlic/drinkingwater/Pages/NDMAhistory.aspx> and click PHG for NDMA.

IARC. 1982. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 27. Some aromatic amines, anthraquinones and nitroso compounds, and inorganic fluorides used in drinking water and dental preparations. See: <http://monographs.iarc.fr/ENG/Monographs/allmonos30.php>.

MDH. 2009/2016. *Groundwater Values Table*. Minnesota Department of Health (MDH). See: <http://www.health.state.mn.us/divs/eh/risk/guidance/gw/table.html>.

US Department of Health and Human Services Public Health Service National Toxicology Program. *Report on Carcinogens* (11th edition). Regularly updated on: <http://ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s128nitr.pdf>.

USEPA. 1980. *Ambient Water Quality Criteria for Nitrosamines*. Washington, DC: United States Environmental Protection Agency.

USEPA IRIS. 1987, revised 1993. N-nitrosodiphenylamine. *Integrated Risk Information System*. See <http://www.epa.gov/IRIS/subst/0178.htm>.

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USEPA IRIS. Accessed November 2008. N-nitrosodiphenylamine. *Integrated Risk Information System*. See <http://www.epa.gov/IRIS/subst/0178.htm>.

USEPA. 2009. *Contaminant Information Sheets for the Final CCL 3 Chemicals*. EPA 815‑R-09-012. 216 pp. <http://water.epa.gov/scitech/drinkingwater/dws/ccl/upload/Final-CCL-3-Contaminant-Information-Sheets.pdf>.

# N-nitrosopiperidine

CAS No. 100-75-4. Also called tetrahydro-N-nitroso-pyrrole, 1-nitroso-piperidine, and NPIP

Also, see the datasheet for N-nitrosodimethylamine (NDMA) which includes some information about the other N- nitrosamines.

### Maximum Acceptable Value

There is no MAV in the DWSNZ, and the WHO Guidelines do not refer to N‑nitrosopiperidine.

The 1971 WHO International Standards first mentioned concern over the possibility of nitrosamine formation *in vivo*. As nitrosamines were then considered a possible hazard to human health, the 1971 Standards stated that it may eventually become necessary to reduce the level of nitrates in water if it is found that this source makes a significant contribution to the hazard to human health arising from nitrosamines.

The most important nitrosamines (and their CAS Numbers) include:

* N-nitrosodimethylamine (NDMA) 62-75-9 (a USEPA priority pollutant)
* N-nitrosomethylethylamine (NMEA) 10595-95-6
* N-nitrosodiethylamine (NDEA) 55-18-5
* N-nitrosodi-n-propylamine (NDPA) 621-64-7 (a USEPA priority pollutant)
* N-nitrosodi-n-butylamine (NDBA) 924-16-3
* N-nitrosopyrollidine (NPYR) 930-55-2
* N-nitrosopiperidine (NPIP) 100-75-4

### Sources to drinking-water

#### 1. To source waters

N-Nitrosopiperidine is used primarily as a research chemical and is not produced commercially.

#### 2. From treatment processes

*N*-Nitrosopiperidine has been detected in water disinfected with chlorine and chloramine.

#### 3. From the distribution system

The concentration increases as the water passes through the distribution system.

### Forms and fate in the environment

N-nitrosopiperidine is miscible with water.

### Removal methods

Many nitrosamines, and N-nitrosopiperidine in particular, are broken down by UV light.

### Recommended analytical techniques

#### Referee method

A referee method cannot be selected for the nitrosamines because MAVs have not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for nitrosamines because MAVs have not been established. However, the following may be useful:

USEPA Method 521 (2004). Determination of nitrosamines in drinking water by solid phase extraction and capillary column gas chromatography with large volume injection and chemical ionisation tandem mass spectrometry (MS/MS).

### Health considerations

The general population may possibly be exposed to low concentrations of *N‑*nitrosopiperidine from cigarette smoke and certain foods. Several researchers have reported trace amounts of *N-*nitrosopiperidine in cigarettes, but the chemical has not been found in all brands of cigarettes tested.

Investigators have detected *N-*nitrosopiperidine concentrations as high as 64 μg/kg in meat and fish products such as bacon, bologna, wieners, and smoked cod; the presence of *N*-nitrosopiperidine in meat, cheese, and spices results from the use of sodium nitrite as a preservative (IARC 1978).

*N*-Nitrosopiperidine is reasonably anticipated to be a human carcinogenbased on sufficient evidence of carcinogenicity in experimental animals – Group 2B (IARC 1978).

No adequate human studies of the relationship between exposure to *N‑*nitrosopyrrolidine and human cancer have been reported (IARC 1978, 1987).

### Derivation of Maximum Acceptable Value

No MAV.

### References

IARC. 1978, reviewed 1987. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 17: See: <http://www.inchem.org/documents/iarc/vol17/nitrosopiperidine.html>.

USEPA. 2004. EPA Document #: EPA/600/R-05/054 Method 521. *Determination of Nitrosamines in Drinking Water by Solid Phase Extraction and Capillary Column Gas Chromatography with Large Volume Injection and Chemical Ionisation Tandem Mass Spectrometry (MS/MS)*. Cincinnati, Ohio 45268: National Exposure Research Laboratory, Office of Research and Development, US Environmental Protection Agency. See: <http://www.epa.gov/nerlcwww/m_521.pdf>. USEPA Methods can be accessed at <http://water.epa.gov/scitech/drinkingwater/labcert/methods_index.cfm>.

# N-nitrosopyrrolidine

CAS No. 930-55-2. Also called tetrahydro-N-nitroso-pyrrole, 1-nitroso-pyrrolidine, and NPYR.

Also, see the datasheet for N-nitrosodimethylamine (NDMA) which includes some information about the other N- nitrosamines.

### Maximum Acceptable Value

There is no MAV in the DWSNZ, and the WHO Guidelines do not refer to N‑nitrosopyrrolidine.

The USEPA concluded on 22 September 2009 that N-nitrosopyrrolidine is known or anticipated to occur in PWSs and may require regulation. Therefore they added N‑nitrosopyrrolidine to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

The 1971 WHO International Standards first mentioned concern over the possibility of nitrosamine formation *in vivo*. As nitrosamines were then considered a possible hazard to human health, the 1971 Standards stated that it may eventually become necessary to reduce the level of nitrates in water if it is found that this source makes a significant contribution to the hazard to human health arising from nitrosamines.

The most important nitrosamines (and their CAS Numbers) include:

* N-nitrosodimethylamine (NDMA) 62-75-9 (a USEPA priority pollutant)
* N-nitrosomethylethylamine (NMEA) 10595-95-6
* N-nitrosodiethylamine (NDEA) 55-18-5
* N-nitrosodi-n-propylamine (NDPA) 621-64-7 (a USEPA priority pollutant)
* N-nitrosodi-n-butylamine (NDBA) 924-16-3
* N-nitrosopyrollidine (NPYR) 930-55-2
* N-nitrosopiperidine (NPIP) 100-75-4

### Sources to drinking-water

#### 1. To source waters

*N*-Nitrosopyrrolidine is used primarily as a research chemical and is not produced commercially.

*N*-Nitrosopyrrolidine is produced when nitrite-preserved or -contaminated foods, especially fatty foods, are heat-prepared. Exposure occurs through inhalation of vapours released during cooking or ingestion of food. In recent years, lower concentrations of sodium nitrite in food have resulted in lower concentrations of *N‑*nitrosopyrrolidine in food. For example, the *N*-nitrosopyrrolidine content of bacon decreased from approximately 67 μg/kg in 1971 through 1974 to only 17 μg/kg in 1975 and 1976; when bacon is fried, an average of 50 percent of the *N‑*nitrosopyrrolidine normally present in that meat is detected in the vapour.

Dry premixed cures containing spices and sodium nitrite originally contained *N‑*nitrosopyrrolidine at concentrations of 40 μg/kg; these levels increased to 520 μg/kg after six months of storage. Investigators have also found *N*-nitrosopyrrolidine in tobacco smoke at concentrations up to 0.113 μg/cigarette, and in pipe bowl scrapings at concentrations up to 1.6 mg of *N*-nitrosopyrrolidine/kg of residue (IARC 1978).

#### 2. From treatment processes

*N*-Nitrosopyrrolidine has been detected in water disinfected with chloramine.

### Forms and fate in the environment

N-nitrosopyrrolidine is miscible with water.

### Typical concentrations in drinking-water

Four water utilities in the US reported detecting N-nitrosopyrrolidine (NPYR) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00001 mg/L.

### Recommended analytical techniques

#### Referee method

A referee method cannot be selected for the nitrosamines because MAVs have not been established and therefore the sensitivity required for the Referee method is not known.

### Some alternative methods

No alternative methods can be recommended for nitrosamines because MAVs have not been established. However, the following may be useful:

USEPA Method 521 (2004). Determination of nitrosamines in drinking water by solid phase extraction and capillary column gas chromatography with large volume injection and chemical ionisation tandem mass spectrometry (MS/MS).

### Health considerations

*N*-Nitrosopyrrolidine is reasonably anticipated to be a human carcinogenbased on sufficient evidence of carcinogenicity in experimental animals – Group 2B (IARC 1978). When administered in the drinking-water, *N*-nitrosopyrrolidine induced lung adenomas in mice of both sexes, hepatocellular carcinomas, leukemia, cholangiocarcinomas, and olfactory carcinomas in rats of both sexes, and papillary mesotheliomas of the tunica vaginalis, interstitial cell tumors, and a cavernous hemangioma of the testis in male rats (IARC 1978).

No adequate human studies of the relationship between exposure to *N‑*nitrosopyrrolidine and human cancer have been reported (IARC 1978, 1987).

USEPA Classification – B2: probable human carcinogen, on the basis of tumours at more than one site being observed in two rodent species administered nitrosopyrrolidine orally. Equal numbers (12 to 31) of male and female Sprague-Dawley rats were maintained on water formulated to deliver 0, 0.3, 1, 3, or 10 mg/kg bw/day nitrosopyrrolidine. Animals remained on treatment until they died or became moribund. There was no statistically significant increase in numbers of tumours in the lowest dose group. Dose-related increases in hepatocellular carcinomas and adenomas were observed. Latency periods were also diminished with increasing dose.

The concentration of nitrosopyrrolidine in drinking-water that presents a risk of developing cancer at the 1 in 100,000 (10-5) level is 0.0002 mg/L.

*N*-Nitrosopyrrolidine is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

### Derivation of Maximum Acceptable Value

No MAV.

### References

EFSA. 2016. Identification of the substances from the Carcinogenic Potency Database (CPDB) which are of particular concern even if ingested at doses below 0.0025 μg/kg body weight per day. *EFSA Journal* 14(3): 4407. 11 pp. http://www.efsa.europa.eu/sites/default/files/scientific\_output/files/main\_documents/4407.pdf.

Environmental Working Group (EWG). Accessed 2010. *National Drinking Water Database – Chemical Contaminants*. <http://www.ewg.org/tap-water/chemical-contaminants>.

IARC. 1978. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 17. See: <http://www.inchem.org/documents/iarc/vol17/nitrosopyrrolidine.html>.

USEPA IRIS. 1987, revised 1994. N-nitrosopyrrolidine. *Integrated Risk Information System*. See: <http://www.epa.gov/IRIS/subst/0081.htm>.

USEPA. 2004. EPA Document #: EPA/600/R-05/054 Method 521. *Determination of Nitrosamines in Drinking Water by Solid Phase Extraction and Capillary Column Gas Chromatography with Large Volume Injection and Chemical Ionisation Tandem Mass Spectrometry (MS/MS)*. Cincinnati, Ohio 45268: National Exposure Research Laboratory, Office of Research and Development, US Environmental Protection Agency. See: <http://www.epa.gov/nerlcwww/m_521.pdf>. USEPA Methods can be accessed at <http://water.epa.gov/scitech/drinkingwater/labcert/methods_index.cfm>.

USEPA. 2009. *Contaminant Information Sheets for the Final CCL 3 Chemicals*. EPA 815‑R-09-012. 216 pp. <http://water.epa.gov/scitech/drinkingwater/dws/ccl/upload/Final-CCL-3-Contaminant-Information-Sheets.pdf>.

# Oxirane methyl (propylene oxide) and ethylene oxide

Note: to avoid potential confusion this datasheet also covers ethylene oxide.

The term epoxides represents a subclass of epoxy compounds containing a saturated three-membered cyclic ether; thus oxirane derivatives, eg, 1,2-epoxypropane or 2‑methyloxirane (an epoxide) (IUPAC *Gold Book* <http://goldbook.iupac.org/ET07030.html>).

### Part A: Oxirane methyl

CAS No. 75-56-9. Oxirane methyl is also called methyl oxirane, 2-methyloxirane, propylene oxide, epoxypropane (the IUPAC name), propylene epoxide, 1,2-propylene oxide, 1,2-epoxypropane, propene oxide, propenoxide, methyl ethylene oxide, methylethylene oxide, methyloxacyclopropane, PPO and PO. Note that the isomer 1,3‑propylene oxide is also known as oxetane.

Propylene oxide exists in two optical isomers, and commercial propylene oxide is a racemic mixture.

#### Maximum Acceptable Value

There is no MAV in the DWSNZ, and the WHO Guidelines do not refer to oxirane, oxirane methyl, or ethylene oxide.

Oxirane methyl is one of the 14 VOCs on the USEPA’s 3rd Chemical Contamination List (CCL3).

#### Sources to drinking-water

##### 1. To source waters

Oxirane methyl is used in three main areas: as a monomer in polymer production; as an intermediate in the synthesis of other chemicals; and in direct applications.

The main use of oxirane methyl in polymer production is to make polyether polyols which are mostly used in the manufacture of polyurethane foams. The main use of oxirane methyl as an intermediate is in the production of propylene glycol. Oxirane methyl can also be used in the production of other monomers that are then used in polymer production such as acrylic resins. Oxirane methyl is also used directly as a stabiliser for dichloromethane and other chlorinated hydrocarbons. Oxirane methyl is used in the post-harvest fumigation of dried fruit, cocoa, spices, processed nutmeats, starch and gums in the USA.

Concentrations of oxirane methyl in drinking water are detected but not quantifiable, in fresh surface water not detected and in effluents have a maximum of 47 μg/L. No data are available for groundwater or marine surface water.

#### Forms and fate in the environment

JMPR (2011) reports:

* water solubility is 400,000 mg/L at pH 7
* vapour pressure = 60 kPa
* Henry’s law constant = 12.4 Pa.m3/mole
* partition coefficient n-octanol/water = Kow = 0.03–0.08
* hydrolysis half-life = 11.6 days
* propylene oxide does not absorb solar radiation appreciably at wavelengths greater than 300 nm (it has a maximum absorption at 199.5 nm). Thus direct photolysis does not occur.

DWI (2014) reports:

* water solubility is about 600,000 mg/L (60 percent)
* log Kow = 0.03
* log Koc = 0.37
* Henry’s Law constant = 6.96 x 10-5 atm.m³/mole at 25°C.

EU (2002) reports a half-life in water of 12 to 22 days, at 20–25°C. The product of the hydrolysis reaction is propylene glycol (1,2-propanediol) which biodegrades rapidly in water. Results of biodegradability studies are variable.

#### Removal methods

No data were located on the removal of oxirane methyl during drinking water treatment, however, some predictions on the fate of this chemical in drinking water treatment can be made based on its physico-chemical properties.

A log Koc of 0.37 has been reported for oxirane methyl, which would suggest that it has high mobility in the water column and therefore is unlikely to be amenable to removal by GAC.

Oxirane methyl is volatile; a vapour pressure of 538 mm Hg and a Henry’s Law constant of 6.96 x 10-5 atm.m³/mole have been reported at 25°C. This may indicate that oxirane methyl will be amenable to moderate removal from water by air stripping.

Oxirane methyl is reported to undergo hydrolysis to propylene glycol, with half-lifes of 10.7 to 21.7 days. This process is reported to be accelerated in the presence of chloride ions, which may indicate that chlorination of drinking water may also accelerate the hydrolysis of oxirane methyl.

#### Health considerations

DWI (2014) states:

The weight of evidence indicates that oxirane methyl is genotoxic *in vitro*. When assessing the results of *in vivo* genotoxicity testing, the EU risk assessment concludes that oxirane methyl is a somatic cell mutagen, and that the general toxicological profile for oxirane methyl suggests that perhaps it shows genotoxicity only at sites of initial contact. For Repeat Dose Oral Toxicity and Carcinogenicity no suitable oral toxicity studies were located upon which an oral Tolerable Daily Intake can be derived. However, a number of inhalation studies are available and an oral TDI can be derived by extrapolation from these data. Accordingly an oral Tolerable Daily Intake (TDI) of 0.017 mg/kg bw/day (17 μg/kg bw/day; rounded) can be derived.

The International Agency for Research on Cancer (IARC) has classified oxirane methyl as a group 2B carcinogen (possibly carcinogenic to humans), based on inadequate evidence in humans for the carcinogenicity of oxirane methyl and sufficient evidence in experimental animals for the carcinogenicity of oxirane methyl.

The USEPA maintains a table of Human Health Benchmarks for Pesticides that includes RfDs and ARfDs for (currently) 363 pesticides. These were originally developed in 2012. The table includes a column for “acute or one-day HHBPs”, another for “chronic or lifetime (non-cancer) HHBPs”, and one for “carcinogenic HHBPs”. Details can be accessed at <http://water.epa.gov/drink/standards/hascience.cfm> or <http://iaspub.epa.gov/apex/pesticides/f?p=HHBP:home:10911636297819:::::>. The USEPA acute one day HHBP (Human Health Benchmark for Pesticides) in drinking water for propylene oxide is 6.93 mg/L.

The Acceptable Daily Intake (ADI) adopted in Australia for propylene oxide is 0.006 mg/kg body weight, with a NOEL of 2.9 mg/kg bw. The ARfD is 0.4 mg/kg bw. These values were based on inhalation studies.

The USEPA has classified propylene oxide as a Group B2, probable human carcinogen. They calculated an oral cancer slope factor of 0.24 (mg/kg/d)-1.

Methyloxirane is identified as substance meeting the criteria of Article 57(a) and 57(b) of Regulation (EC) 1907/2006 (REACH) owing to its classification as carcinogen 1B and mutagen category 1B2 which corresponds to classification as carcinogen category 2 and mutagen category 23. ECHA (2012).

The 2017 JMPR Meeting re-affirmed the ADI of 0–0.04 mg/kg bw derived from the NOAEC for systemic effects (reduced body weight gain) in the chronic inhalation studies in rats of 100 ppm (equivalent to approximately 40 mg/kg bw per day orally), supported by the NOAEC of 100 ppm (equivalent to approximately 40 mg/kg bw per day orally) for offspring and parental toxicity (reduced body weight gain) in the reproductive toxicity study in rats. The Meeting re-affirmed the ARfD of 0.04 mg/kg bw on the same basis as the ADI. The Meeting concluded that there was inadequate information to support the derivation of a value based on specific acute effects from dietary exposure.

#### Derivation of Maximum Acceptable Value

No MAV.

#### References

DWI. 2014. *Volatile Organic Compounds – Understanding the Risks to Drinking Water*. Report DWI9611.04. 305 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-292.pdf>.

ECHA. 2012. Methyloxirane [propylene oxide]. *Support Document for Identification of Methyloxirane [Propylene Oxide] as a Substance of Very High Concern because of its CMR Properties*. European Chemicals Agency. 7 pp. <http://echa.europa.eu/documents/10162/5b70a6ed-3e91-45f2-a161-cef5f86ba2a5>.

EU. 2002. Methyloxirane (propylene oxide). *European Union Risk Assessment Report*. 149 pp. <http://echa.europa.eu/documents/10162/5d04d98a-620a-491d-aad1-e6345ef090fd> or <http://echa.europa.eu/information-on-chemicals/information-from-existing-substances-regulation>.

JMPR. 2011. Pesticide residues in food. *Joint FAO/WHO Meeting on Pesticide Residues. Evaluations* 28 pp. <http://www.fao.org/agriculture/crops/thematic-sitemap/theme/pests/jmpr/en/>.

JMPR. 2017. Pesticide residues in food. *Report on Pesticide Residues: FAO Plant Production and Protection Paper* 232. Joint FAO/WHO Meeting. <http://www.fao.org/fileadmin/templates/agphome/documents/Pests_Pesticides/JMPR/Report2017/web_2017_JMPR_Report_Final.pdf>.

USEPA. 1990. Propylene oxide. *Integrated Risk Information System (IRIS)*. <http://www.epa.gov/iris/subst/0403.htm> and see <http://www.epa.gov/ttn/atw/hlthef/prop-oxi.html>.

### Part B: Ethylene oxide

CAS No. 75-21-8. Ethylene oxide is also called oxirane (the IUPAC name), and epoxyethane, 1,2-epoxyethane, diethylene oxide, ethene oxide, oxacyclopropane, oxane, oxidoethane, EtO and EO.

#### Maximum Acceptable Value

There is no MAV in the DWSNZ, and the WHO Guidelines do not refer to oxirane, oxirane methyl, or ethylene oxide.

Ethylene oxide is one of the fourteen VOCs on the USEPA’s 3rd Chemical Contamination List (CCL3).

#### Sources to drinking-water

##### 1. To source waters

The main use of ethylene oxide is reported to be as an intermediate for other chemical products, especially ethylene glycol, polyethylene glycol and to a lesser extent, surfactants. It is used in the gaseous form as a sterilising agent, fumigant, or insecticide. Ethylene oxide is used in the manufacture of choline chloride, glycol ethers, polyglycols, rocket propellant, petroleum demulsifiers, acrylonitrile, and ethanolamines. Ethylene oxide is also used as a formulant or component in many pesticides.

Ethylene oxide has routinely been used as a gas in medical and health care related facilities for sterilising heat-sensitive instruments and equipment that come into contact with biological tissue. Ethylene oxide gas has been used historically to sterilise inexpensive single-use items such as syringes on an industrial scale. Ethylene oxide is also reported to be used to sterilise electronic cardiac pacemakers, blood oxygenators and dialysers in the USA.

Library and museum artefacts are treated with ethylene oxide to control fungi and insects that might cause damage. Ethylene oxide has also been used to sterilise food and food packaging however in 2006 its classification as a food additive was withdrawn by the Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives.

Ethylene oxide can be generated from waterlogged soil, manure, and sewage sludge. Ethylene oxide is also a by-product of fossil fuel combustion and tobacco smoke.

Concentrations of ethylene oxide in groundwater are reported as 28 μg/L, in fresh surface water as 21 μg/L, and in effluents as 2000 μg/L. No data were located for drinking water or marine water.

#### Forms and fate in the environment

DWI (2014) reports:

* ethylene oxide is infinitely soluble, ie, miscible
* log Kow = -0.22
* log Koc = 1.204
* Henry’s Law constant is about 12–19 Pa.m3/mol.

WHO (2003) states that in water, the volatilisation half-life is about one hour, the hydrolysis half-life about 12–4 days, the aerobic biodegradation half-life 20 days to six months, and the anaerobic biodegradation half-life four months to two years. In soil, ethylene oxide is expected to volatilise rapidly. Hydrolysis half-lifes for soil and groundwater are estimated to be between 10.5 and 11.9 days. Ethylene oxide is not expected to bioaccumulate on the basis of its very low log octanol/water partition coefficient (*K*ow).

#### Removal methods

No data on the removal of ethylene oxide via drinking water treatment were located. However, some predictions on the fate of this chemical in drinking water treatment can be made based on its physico-chemical properties and its fate in the environment.

Ethylene oxide is not expected to sorb to activated carbon, based on a log Koc of 1.204. However, volatilisation is expected to be an important fate process; half-lifes in the environment are reported to be 1 hour with no wind, and 0.5 hours at a wind speed of 5 m/s. Therefore, it is likely that significant removal of ethylene oxide will occur by air stripping.

Ethylene oxide will also undergo rapid hydrolysis, although this process is relatively slow compared to volatilisation. Half-lifes of 12 to 14 days have been reported in freshwater at pH 5 to 7. In freshwater, ethylene oxide is hydrolysed to ethylene glycol, while in saltwater, it is hydrolysed to ethylene glycol and ethylene chlorohydrin.

#### Health considerations

DWI (2014) states:

Clear evidence of genotoxicity has been reported in both *in vitro* and *in vivo* tests, indicating that ethylene oxide is genotoxic. Evidence of genotoxicity has also been reported in a number of epidemiological studies in albeit limited numbers of workers exposed to ethylene oxide. For Repeat Oral Dose Toxicity and Carcinogenicity a LOAEL of 7.5 mg/kg bw can be identified based on dose-dependent increases in squamous cell carcinomas, hyperkeratosis and hyperplasia. Using this, an oral Tolerable Daily Intake (TDI) of 0.015 mg/kg bw/day (15 μg/kg bw/day; rounded) can be derived.

The USEPA (2000) considers ethylene oxide to be a probable human carcinogen and has ranked it in Group B1.

In 2012, the International Agency for Research on Cancer (IARC) evaluated ethylene oxide and upgraded the overall classification based on mechanistic and other relevant data to Group 1 (ie, carcinogenic to humans), on the basis that there is sufficient evidence for the carcinogenicity of ethylene oxide in experimental animals, relying heavily on the compelling data in support of the genotoxic mechanism.

#### Derivation of Maximum Acceptable Value

No MAV.

#### References

DWI. 2014. *Volatile Organic Compounds – Understanding the Risks to Drinking Water*. Report DWI9611.04. 305 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-292.pdf>.

USEPA. 2000. Ethylene oxide. *Hazard Summary*. Created in April 1992; revised in January 2000. <http://www.epa.gov/ttnatw01/hlthef/ethylene.html>.

WHO. 2003. Ethylene oxide. *Concise International Chemical Assessment Document (CICAD)* 54: 63 pp. <http://www.who.int/ipcs/publications/cicad/en/cicad54.pdf>.

# PCBs and PBBs

CAS No. 1336-36-3 (for the PCB group). For a full list of the 209 possible polychlorinated biphenyls (PCBs) and their individual CAS numbers, see ATSDR. 2000. For PBBs, see IARC (2014).

There are theoretically 209 different polychlorinated biphenyl (or PCB) congeners, although only about 130 of these were found in commercial PCB mixtures. Commercial PCB preparations were usually mixtures of 50 or more PCB congeners. A common trade name was Aroclor. PCBs are a class of [organic compounds](http://en.wikipedia.org/wiki/Organic_compound) with 1 to 10 [chlorine](http://en.wikipedia.org/wiki/Chlorine) atoms attached to [biphenyl](http://en.wikipedia.org/wiki/Biphenyl) which is a molecule composed of two [benzene rings](http://en.wikipedia.org/wiki/Benzene_ring) each containing six carbon atoms. The [chemical formula](http://en.wikipedia.org/wiki/Chemical_formula) for all PCBs is [C](http://en.wikipedia.org/wiki/Carbon)12[H](http://en.wikipedia.org/wiki/Hydrogen)10-x[Cl](http://en.wikipedia.org/wiki/Chlorine)x.

The term polybrominated biphenyls or polybromobiphenyls (PBBs) refers to a group of halogenated hydrocarbons, formed by substituting hydrogen by bromine in biphenyl. PBBs are not known to occur as natural products. They have a molecular formula of C12H(10-x-y)Br(x+y) where both x and y = 1 to 5. Theoretically 209 of these congeners are possible as well, however, only a few of these have been synthesised individually and characterised. PBBs share many similar properties with the PCBs. The PBB group has a CAS No. 36355-01-8.

Polychlorinated terphenyls (PCTs): CAS No. 61788-33-8.

*IARC Monograph* 107 (2014) deals thoroughly with PCBs (nearly 400 pages), and PBBs (nearly 60 pages).

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polychlorinated biphenyls in drinking-water. The WHO Guidelines do not mention PCBs or PBBs.

The maximum contaminant level for PCBs (USEPA 2006/2009/2011) is 0.0005 mg/L.

Seven PCBs, the Aroclor products 1016, 1221, 1232, 1242, 1248, 1254 and 1260, were “priority pollutants” under the US Clean Water Act.

PCBs appeared in the original list of 12 Persistent Organic Pollutants (POPs) under the Stockholm Convention; see <http://chm.pops.int/>. PCBs and polychlorinated terphenyls appear on the Rotterdam Convention (UNEP) list of chemicals in Appendix III (which effectively bans or severely restricts use of a chemical), see <http://www.pic.int/home.php?type=s&id=77>.

The PBB hexabromobiphenyl was added to the Persistent Organic Pollutants (POP) Stockholm Convention list in May 2009 (ICS 2009 and <http://chm.pops.int/>).

Dioxins and dioxin-like polychlorinated biphenyls (PCBs) are listed as a “priority contaminant” in the Ministry for the Environment’s *Toxicological Intake Values for Priority Contaminants in Soil* (MfE 2011).

### Sources to drinking-water

#### 1. To source waters

PCBs are a class of [organic compounds](http://en.wikipedia.org/wiki/Organic_compound) with 1 to 10 [chlorine](http://en.wikipedia.org/wiki/Chlorine) atoms attached to [biphenyl](http://en.wikipedia.org/wiki/Biphenyl) which is a molecule composed of two [benzene rings](http://en.wikipedia.org/wiki/Benzene_ring) each containing six carbon atoms. There are no known natural sources of PCBs.

PCBs are (or were) used in hundreds of industrial and commercial applications including electrical, heat transfer, and hydraulic equipment; transformer oil, as plasticisers in paints, plastics and rubber products; in pigments, dyes and carbonless copy paper, and many other applications. They are not formed naturally. Their use is being restricted increasingly.

PCB production was banned in the 1970s due to the high [toxicity](http://en.wikipedia.org/wiki/Toxicity) of most PCB [congeners](http://en.wikipedia.org/wiki/Congeners) and mixtures. At present, the major source of PCB exposure in the general environment is redistribution of PCBs previously introduced into the environment.

Similar products, polybrominated biphenyls, were widely used commercially as a [flame retardant](http://en.wikipedia.org/wiki/Flame_retardant) before the 1970s. Because human beings and the environment should not be exposed to PBBs due to their high persistence and bioaccumulation and potential adverse effects at very low levels after long-term exposure, PBBs should no longer be used commercially (IPCS 1994). Several of the common isomers photodegrade with reductive debromination upon exposure to ultraviolet light electronic machines), in coatings and lacquers, and in polyurethane foam. Noting the possible hazards on the environment, PBBs were listed as one of six controlled substances under the [Restriction of Hazardous Substances Directive](http://en.wikipedia.org/wiki/Restriction_of_Hazardous_Substances_Directive) (RoHS), which was enacted into [European Law](http://en.wikipedia.org/wiki/European_Law) in February 2003. RoHS legislation lists PBBs as a “restricted substance” group. Other countries followed suit, resulting most recently in restriction dates instituted in China on 1 March 2007 and South Korea on 1 July 2007. Therefore PBBs are not expected to be found in drinking-water.

The Stockholm Convention meeting stated that: “hexabromobiphenyl (HBB) is an industrial chemical that was used as a flame retardant, mainly in the 1970s. Based on existing data, HBB is no longer produced and is not used in new or existing products”.

The chemical formula for polychlorinated terphenyls can be written as C18H14-nCln, in which n is the number of chlorine atoms, which can range from 1 to 14. The theoretically possible number of different PCTs is several orders of magnitude greater than the number of PCBs, but in practice, as with PCBs, PCTs are sold on the basis of their physical properties, which depend on the degree of chlorination, and not their chemical composition. In the Aroclor series, terphenyls are indicated by 54 in the first two places of the four digit code. PCTs and PCBs have many similar properties and hence had many similar uses.

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; levels range from 0.00009 to 0.0012 mg/kg TEQ for dioxin-like PCBs depending on land use. For dioxin-like PCBs the total toxicity is assessed as a toxic equivalency (TEQ) to 2,3,7,8-TCDD using toxic equivalency factors (TEF). The TEQ is defined as the sum of the products of the concentration of each compound multiplied by the value of its TEF.

PCBs will be banned in New Zealand from the end of December 2016. The EPA is working on a plan to manage any PCBs still in New Zealand after the end of December.

### Forms and fate in the environment

PCBs have low solubilities (generally <0.2 mg/L), and as a result adsorb readily to sediments and suspended matter in the water (IPCS 1992). The strength of adsorption is greater for the more highly chlorinated PCBs, with the volatility of the compounds also being decreased by adsorption. Adsorption can immobilise PCBs for relatively long periods, but desorption does release the compounds back into the bulk water. Sediment thereby acts as a sink for these compounds, and as a possible reservoir.

In water, hydrolysis and oxidation have little effect on PCBs; photolysis appears to be the only abiotic degradation process. The degradation of PCBs is dependent on their degree of chlorination: persistence increases with chlorine content (IPCS 1992).

The stability of PCBs has resulted in their wide dispersion throughout the environment globally. Differences in volatilisation and decomposition rates between different members of the PCB family lead to changes in the composition of PCB mixtures in the environment.

PBBs are nearly insoluble in water; solubility decreases with increasing bromination. PBBs are soluble in fat. Primarily hydrophobic, PBBs adsorb strongly to soils and sediments. Hydrophobic adsorption generally increases with the bromine content of the PBB congener and the organic content of the soil or sediment. In water, PBBs with high bromine content are less soluble and more likely to attach strongly to sediment. PBB congeners with low bromine content are more likely to be soluble in water. Partition ratios between 1-octanol and water (log Kow) increase with the number of bromines. Henry’s law constant for the hexabromobiphenyls ranges from 1.4 x 10-8 to 3.9 x 10-8 atm-m3/mol. PBBs show unusual chemical stability and resistance to breakdown by acids, bases, and reducing and oxidising agents. Several of the common isomers photodegrade with reductive debromination upon exposure to ultraviolet light (IARC 2014).

### Typical concentrations in drinking-water

Globally, PCBs are present in rain and snow in the range 0.001 to 0.25 μg/L, and less than 0.001 μg/L in non-contaminated areas drinking-waters, although levels up to 0.005 μg/L have been reported (IPCS 1992).

PCBs were included routinely on the Department of Health’s surveillance programme during the late 1980s and early 1990s. None were detected (detection limits approximately 0.0005 mg/L) (Nokes 1992).

Twelve water utilities in the US reported detecting total polychlorinated biphenyls (PCBs) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.00056 mg/L.

Arochlors 1016, 1221, 1232, 1242, 1284, 1254, 1260 and 1262 have been have been reported in several US water supplies since 2004, the highest concentration being 0.0003 mg/L.

### Removal methods

There is limited information available about the removal of PCBs from water. Physical data for adsorption to activated carbon show that this medium should be effective in removing PCBs (Faust and Aly 1983). The presence of humic acids in the water can reduce the adsorbance of PCBs by up to 71 percent (Pirbazari et al 1992). The removal of PCBs by coagulation ranges from 10–40 percent depending on the dose and the coagulant; alum is more effective than ferric chloride (Aly and Badawy 1986).

No significant reduction in PCB levels is reported for experiments using ultraviolet light, ozone, and the combination of UV and ozone (Vollmuth and Niessner 1995).

Reverse osmosis has been used to concentrate PCBs (Faust and Aly 1983), so RO should remove them from water, although its efficacy of is unknown.

Chlorination, although capable of reducing the concentrations of some isomers, can react with isomers of lower chlorine content to produce PCBs of higher molecular weight (Aly and Badawy 1986).

### Recommended analytical techniques

#### Referee method

A referee method cannot be selected for the PCBs because MAVs have not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for these PCBs because MAVs have not been established.

### Health considerations

Exposure to PCBs is principally through food, including mother’s milk in the case of babies.

#### Acute poisoning

Occupational exposures have led to skin rashes, and after exposure to high concentrations of PCBs, itching, burning sensations, irritation of the conjunctivae and chloracne have been found, the latter being the most prevalent (IPCS 1992). As PCBs contain contaminants it is difficult to determine the extent to which these signs and symptoms arise from PCBs themselves and those from polychlorinated dibenzofuran contaminants.

#### Chronic exposure

Both IARC (IARC 1987) and the USEPA have concluded that PCBs are probable human carcinogens (Group 2A). IARC (2012) evaluated 3,3′,4,4′,5-pentachlorobiphenyl (CAS No. 57465-28-8) as carcinogenic to humans (Group 1).

The most carcinogenic PCB mixtures are believed to be those bound to sediments. There is evidence that PCBs are linked to cancer in the liver (USEPA), the gall bladder and the gastrointestinal tract (IPCS 1992), and to the production of malignant melanomas. The PCB in question influences which organ is the target. PCBs and PBBs appear on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

PCBs also have effects on the immune, reproductive, nervous, and endocrine systems.

The RfD for arochlor 1016 was calculated at 0.00007 mg/kg/d (USEPA. 1993. The RfD for arochlor 1254 was calculated at 0.00002 mg/kg/d (USEPA (1996).

The USEPA (2009/2011) quotes a health advisory of 0.01 mg/L for PCBs, representing a 10-4 cancer risk.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.01 mg/kg/day for acute-duration oral exposure  
(1–14 days) to polybrominated biphenyls.

As at July 2013 for polychlorinated biphenyls (Arochlor 1254)ATSDR quotes a minimal risk level (MRL) of:

* 0.03 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.02 mg/kg/day for chronic-duration oral exposure (>364 days).

Dioxin-like PCBs have been measured 10-yearly in New Zealand breast milk. The 2008 survey found that over this period, the levels of dioxin-like PCBs have declined by 54 percent to a mean PCB TEQ of 1.29 pg/g lipid (Massey University 2010).

MfE (2011) states:

“Dioxins and dioxin-like polychlorinated-biphenyls (PCBs) are considered to be threshold contaminants, with developmental effects on the reproductive system in male offspring of exposed pregnant females considered the most sensitive toxicity endpoint. These effects are also considered to be protective against carcinogenic effects of dioxins. The maximum monthly intake value of 30 pg TEQ/kg determined by the Ministry of Health is recommended, for consistency between New Zealand agencies. Further it is recommended that toxic equivalency factors (TEFs) developed by WHO[[2]](#footnote-2) for individual dioxins and dioxin-like PCBs are used to calculate toxic equivalent doses (TEQs), as these are based on the latest re-evaluation by WHO, and thus are likely to become the international standard. Inhalation exposure to dioxins and dioxin-like PCBs is likely to be negligible on contaminated sites, due to their low volatility. Dermal absorption of these compounds is dependent on the physicochemical properties of the individual congeners. It is recommended that dermal factors of 0.02, 0.05 and 0.07 are used as conservative estimates of dermal absorption of PCDDs, PCDFs and dioxin-like PCBs, respectively. Dietary intake is the primary source of background exposure to dioxins and dioxin-like PCBs and was estimated to be 0.33 pg/kg bw/day or 10.0 pg I-TEQ/kg bw/month for an adult, and is extended to children.”

All the available data indicate that PBBs have a marked tendency to bioaccumulate and persist. Metabolism is poor and the half-life in man is in the order of 8–12 years, or longer (IPCS 1993).

Polybrominated biphenyls are *probably carcinogenic to humans (Group 2A)* on the basis of mechanistic similarities to polychlorinated biphenyls (IARC 2014).

Several of these substances are on the EC List of 66 Category 1 substances showing evidence of endocrine disrupting activity in at least one species using intact animals; see EC (2015).

### Derivation of Maximum Acceptable Value

No MAVs.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The cancer health risk limit for the PCBs is 0.00004 mg/L.

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# Pentachlorobenzene

CAS No. 608-93-5. Has also been called pentachlorbenzol.

### Maximum Acceptable Value

Pentachlorobenzene is not mentioned in the WHO Guidelines, and does not have a MAV in the DWSNZ.

Pentachlorobenzene was added to the Persistent Organic Pollutants (POP) Stockholm Convention list in May 2009 (ICS 2009; and <http://chm.pops.int/>).

### Sources to drinking-water

#### 1. To source waters

The Stockholm Convention meeting stated that: “pentachlorobenzene (PeCB) was used in PCB products, dyestuff carriers, as a fungicide, a flame retardant and a chemical intermediate such as the production of quintozene and it may still be used for this purpose. PeCB is also produced unintentionally during combustion in thermal and industrial processes. It appears as an impurity in products such as solvents or pesticides”, eg, quintozene (pentachloronitrobenzene) – see datasheet.

Current emissions of pentachlorobenzene to the environment are estimated to be about 85,000 kg/year, based on published information. The largest sources appear to be combustion of solid wastes, 33,000 kg/y, and biomass burning, 44,000 kg/y, with industrial processes less important.

### Forms and fate in the environment

The physical properties of pentachlorobenzene suggest that it will strongly sorb to sediments and soil. Concentrations nowadays in water are usually less than 10 ng/L (<0.00001 mg/L); they were ten times that in the 1970s. The estimated half-life in water of pentachlorobenzene ranges from 194 to 1,250 days and the estimated half-life for anaerobic biodegradation in deeper water ranges from 776 to 1,380 days.

Water solubility about 0.7 mg/L.

Typical concentrations in drinking-water

### Removal methods

Treatment processes that remove particulate matter from water should also remove pentachlorobenzene.

### Analytical methods

#### Referee method

No MAV so not needed.

### Health considerations

Long-term exposure can affect the liver and kidneys and can cause tissue lesions. Animal studies and tests show that pentachlorobenzene can possibly cause toxic effects on human reproduction.

To protect the general public for lifetime exposure, (Reference Dose for Chronic Oral Exposure or RfD), the toxicity of pentachlorobenzene has been studied and an oral reference dose of 0.0008 mg/kg body weight per day has been established by the USEPA (1998), based on a LOAEL of 8.3 mg/kg/day. The corresponding Canadian tolerable daily intake has been set at 0.0005 mg/kg bw/day.

The USEPA has not classified pentachlorobenzene as to human carcinogenicity.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Pentachloroethane

CAS No. 76-01-7 for pentachloroethane. It can also be called 1,1,1,2,2‑pentachloroethane.

### Maximum Acceptable Value

Pentachloroethane is not mentioned in the WHO Guidelines, and does not have a MAV in the DWSNZ.

### Sources to drinking-water

#### 1. To source waters

Datasheets have been prepared for dichloroethane, trichloroethane, tetrachloroethane and hexachloroethane. Pentachloroethane is less common. It was manufactured for use as a solvent, is used as an intermediate in the production of other substances, and may be an impurity in other chlorinated hydrocarbons. It appears as a breakdown product in the reduction of hexachloroethane, particularly in anaerobic conditions; it then usually breaks down to tetrachloroethene. It may be released to the environment as a combustion product of polyvinyl chloride (PVC).

### Forms and fate in the environment

If released to water, volatilisation appears to be an important, if not the dominant, removal mechanism (half-life five hours from a model river). Moderate to slight adsorption of pentachloroethane to suspended solids and sediments may occur. Chemical hydrolysis is not expected to be important.

If released to moist soil, pentachloroethane is expected to have moderate to high mobility. This compound may undergo slow chemical hydrolysis.

### Analytical methods

#### Referee method

No MAV so not needed.

### Health considerations

Technical-grade pentachloroethane (containing 4.2 percent hexachloroethane) was tested for carcinogenicity by oral administration by gavage in one experiment in mice and one experiment in rats. Hepatocellular carcinomas were induced in mice of each sex and hepatocellular adenomas in female mice; a marginally increased incidence of kidney tubular-cell adenomas was observed in male rats but not in female rats.

Pentachloroethane is not mutagenic to *Salmonella typhimurium* (IARC 1999).

The IARC states that pentachloroethane is not classifiable as to its carcinogenicity to humans (Group 3).

### Derivation of Maximum Acceptable Value

No MAV.

### References

IARC. 1986. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans* 41. Some halogenated hydrocarbons and pesticide exposures. <http://monographs.iarc.fr/ENG/Monographs/allmonos49.php>.

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# Perfluoroalkyl substances (PFASs)

### Introduction

Perfluoroalkyl substances (PFASs) comprise a group of fluorinated organic chemicals containing at least one fully fluorinated carbon atom. Concawe (2016) refers to 42 families/sub-families in the group of PFASs. The main families of PFASs that were manufactured are the perfluoroalkyl acids (PFAAs) and the perfluoroalkyl sulfonates (PFAS). The commonest perfluoroalkyl acid was perfluorooctanoic acid (PFOA). The commonest perfluoroalkyl sulfonate was perfluorooctane sulfonate (PFOS) and its isomers.

Perfluoroalkyl acid (PFAA) is a generic term for the various perfluorocarboxylic acids (sometimes referred to as PFCAs), usually from C4: perfluorobutyric acid or perfluorobutanoic acid (PFBA), to C14: perfluorotetradecanoic acid (PFTeDA). The carbon atoms are fully fluorinated.

Likewise, perfluoroalkyl sulfonate (PFAS) is a generic term used to describe any fully fluorinated carbon chain sulfonate, and includes higher and lower homologues. The acids may appear as esters, eg, perfluorohexane sulfonic acid may appear as perfluorohexane sulfonate. PFAS-related substances may be simple salts, or large polymers that contain the PFAS as only a portion of the entire polymer. Several hundred have been synthesised. The 3-carbon chain PFAS is perfluoropropanesulfonic acid (or sulfonate). The 10-carbon chain PFAS is perfluorodecanesulfonic acid (or sulfonate).

Perfluorooctane sulfonate (PFOS) is a fully fluorinated eight carbon length organic sulfonate and is the most prominent PFAS. There are three isomers: perfluorooctane sulfonate (PFOS), perfluorodimethylheptane sulfonate (di-PFOS), and perfluoromethylheptane sulfonate (mono-PFOS). PFOS based substances can be simple salts of PFOS, eg, potassium, lithium, ammonium, diethanolamine or more complex polymer compounds that contain PFOS as a portion of a polymer molecule. The majority of PFOS-related substances are high molecular weight polymers in which PFOS represents a fraction of the total polymer weight.

Perfluorooctanoic acid (PFOA), the most commonly encountered PFCA, is a completely fluorinated organic synthetic acid used to produce fluoropolymers. The IUPAC name is 2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluorooctanoic acid. Some commercial PFOAs are a combination of linear and branched chain isomers. PFOA is also sold as the ammonium salt (CAS No. 3825-26-1); USEPA (2014). See IARC (2016) for isomers of PFOA and its salts.

ATSDR (2009) discusses 13 perfluoroalkyls, being compounds that have been measured in the serum collected from a representative US population 12 years of age and older as follows:

* CAS No. 1763-23-1 perfluorooctane sulfonic acid (PFOS)
* CAS No. 335-67-1 perfluorooctanoic acid (PFOA)
* CAS No. 307-55-1 perfluorododecanoic acid (PFDoA)
* CAS No. 335-76-2 perfluorodecanoic acid (PFDeA)
* CAS No. 357-22-4 perfluorobutyric acid (PFBA)
* CAS No. 375-85-9 perfluoroheptanoic acid (PFHpA)
* CAS No. 375-95-1 perfluorononanoic acid (PFNA)
* CAS No. 2058-94-8 perfluoroundecanoic acid (PFUA)
* CAS No. 355-46-4 perfluorohexane sulfonic acid (PFHxS)
* CAS No. 375-73-5 perfluorobutane sulfonic acid (PFBuS)
* CAS No. 754-91-6 perfluorooctanesulfonamide (PFOSA)
* CAS No. 2355-31-9 2-(N-methyl-perfluorooctane sulfonamide) acetic acid (Me‑PFOSA-AcOH)
* CAS No. 2991-50-6 2-(N-ethylperfluorooctane sulfonamido) acetic acid (Et‑PFOSA-AcOH).

Seven of these substances are perfluoroalkyl carboxylic acids (PFOA, PFDoA, PFDeA, PFBA, PFHpA, PFNA, and PFUA):

* three are perfluoroalkyl sulphonic acids (PFOS, PFHxS, and PFBuS), and
* three are perfluoroalkyl sulfonamides (PFOSA, Me-PFOSA-AcOH, and Et‑PFOSA‑AcOH).

As well as the perfluorinated carboxylic acids (PFCAs) and perfluorinated sulfonic acids (PFSAs), Concawe (2016) discusses perfluorinated phosphonic acids (PFPAs), and other polyfluorinated compounds including fluorotelomer alcohols (FTOHs), fluorotelomer sulfonic acids (FTSs), polyfluorinated alkyl phosphates (PAPs), perfluorooctane sulfonamine (PFOSA), and their derivatives.

CAS No. 307-35-7 represents perfluorooctane sulfonyl fluoride (PFOSF). The IUPAC name is 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-1-octanesulphonyl fluoride. This compound is a common starting point for the production of perfluorooctane sulfonic acid.

Some other perfluoro-compounds that may be encountered include:

* CAS No. 29457-72-5 for lithium perfluorooctane sulfonate (LPOS)
* CAS No. 4151-50-2 for *N*-ethylperfluorooctane-1-sulfonamide (sulfluramid).

Perfluorinated substances are characterised by a fully fluorinated carbon chain with all hydrogens in the chain being replaced by fluorine atoms. The bond between carbon and fluorine is very strong making these substances persistent in the environment. The OECD (2011) lists the perfluorinated chemicals manufactured in 2008 in Annex 3 along with CAS numbers; there are 42.

Concawe (2016) lists 13 PFAS and PFCA precursor compounds. These are generally less stable and may undergo abiotic or biotic transformation to PFAS and PFCA.

Fluorotelomers have an ethyl (CH2-CH2) group between the fully fluorinated carbon chain and the functional group, and are therefore polyfluorinated molecules. Fluorotelomer sulfonates are used in place of PFOS for various applications, including class B fire-fighting foams and industrial surfactants. Fluorotelomers are produced with a variety of different functional groups. The majority of the fluorotelomers are used for manufacturing various fluorotelomer-based products (eg, building blocks for polymers, surfactants and side-chain fluorinated polymers). There is concern that many of these could eventually transform to PFAS and PFCA compounds in the environment. Fluorinated polymers may or may not be a PFAS depending on whether they contain perfluoroalkyl moieties. The fluoropolymer polytetrafluoroethylene (Teflon or PTFE), is a PFAS and is used as a non-stick coating for cookware. Concawe (2016).

### Maximum Acceptable Value

Perfluorooctane compounds are not mentioned in the WHO Guidelines, and do not have a MAV in the DWSNZ.

Until the future DWSNZ are adopted, an “interim guidance levels” for drinking water will be used; these will be the same as the levels used in Australia, see below (Australian Government 2017).

The USEPA concluded on 22 September 2009 that perfluorooctane sulfonic acid and perfluorooctanoic acid are known to, or anticipated to, occur in public water supplies and may require regulation. Therefore they added them to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009a). Meanwhile they have a Provisional Health Advisory value of 0.4 μg/L for PFOA in drinking-water and 0.2 μg/L for PFOS – subsequently changed, see below.

The DWI guidance adopts a multi-tiered approach to the protection of water safety; providing guidance on the levels of PFOS that water companies in England and Wales should act upon in order to fulfil their statutory obligations to ensure the safety of drinking water. Tier 1 has no guideline limit but requires water companies to ensure that PFOS is considered as part of their risk assessment. Tier 2 has a guideline limit of >0.3 μg/L where water companies are required to undertake water sampling and consult local health professionals, whilst the Tier 3 guideline limit is 1 μg/L and requires water companies to put in place measures to reduce concentrations to below 1 μg/L as soon as is practicable. Water is considered to be wholesome if the PFOS levels remain below Tier 3 or 1 μg/L. PFOA was not covered. Taken from HPA (2012).

Thompson et al (2011) state that the currently available provisional guidelines suggested by the USEPA are 500 ng/L and 200 ng/L for PFOA and PFOS respectively (Fulmer 2014 quotes 400 and 200 ng/L respectively); those set by the German Drinking Water Commission are 300 ng/L for combined concentrations of PFOS and PFOA. Note that 500 ng/L = 0.5 µg/L or 0.0005 mg/L.

In May 2016, the USEPA released new Drinking Water Health Advisories for perfluorooctane sulphonate (PFOS) and perfluorooctanoic acid (PFOA) following an assessment of the latest peer-reviewed scientific evidence on the health risks of these chemicals. The Health Advisory levels of 70 parts per trillion (70 ng/L or 0.070 μg/L or 0.00007 mg/L) for both chemicals (alone or combined) are not enforceable regulatory limits, but are expected to guide public water suppliers and state agencies responsible for drinking water safety in their efforts to assess and manage potential health risks. These levels in drinking water are considered to provide a margin of protection against adverse health effects over a lifetime, assuming that drinking water constitutes 20 percent of total exposure. USEPA (2016, 2016a, 2016b). Various countries have been establishing “guidance values” for drinking water:

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Units μg/L** | **Australia 2017** | **USEPA 2016c** | **Germany 2015** | **Denmark 2015** | **Mean** |
| PFOS | 0.07 | 0.07 | 0.23 | 0.1 | 0.12 |
| PFOA | 0.56 | 0.07 | 0.30 | 0.3 | 0.31 |

The values in the column headed Germany are actually “provisional threshold values for groundwater” established by the Bavarian State Office of Environment in 2015 (Concawe 2016). Some other PFASs have regulatory values for groundwater and/or drinking water in a few countries.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **μg/L** |  | **Sweden** | **Minnes** | **NJ** | **Bavaria** | **Baden** |
| PFBS | perfluorobutanesulfonic acid | \* | 7 |  | 3 | 3 |
| PFBA | perfluorobutanoic acid |  | 7 |  | 7 | 7 |
| PFPeA | perfluoropentanoic acid | \* |  |  | 3 | 3 |
| PFHxA | perfluorohexanoic acid | \* |  |  | 1 | 1 |
| PFHpA | perfluoroheptanoic acid | \* |  |  | 0.3 | 0.3 |
| PFNA | perfluorononanoic acid |  |  | 0.02 | 0.3 | 0.3 |
| PFDA | perfluorodecanoic acid |  |  |  | 0.3 | 0.3 |
| 6:2 FTS | 6:2 fluorotelomer sulfonic acid |  |  |  |  | 0.3 |
| PFHpS | perfluoroheptane sulfonic acid |  |  |  |  | 0.3 |
| PFHxS | perfluorohexane sulfonic acid | \* |  |  |  | 0.3 |
| PFPeS | perfluoropentane sulfonic acid |  |  |  |  | 1 |

See Concawe (2016) for details.

\* Maximum tolerable drinking water level is 0.09 μg/L for the sum of PFOS, PFHxS, PFBS, PFOA, PFHpA, PFHxA and PFPeA.

Perfluorooctane sulfonic acid (PFOS), its salts, and perfluorooctane sulfonyl fluoride (PFOSF) were added to the Persistent Organic Pollutants (POP) Stockholm Convention list in May 2009 (ICS 2009; and <http://chm.pops.int/>). Perfluorooctanoic acid (PFOA) and its salts, and PFHxS are proposed to be added in 2017/18.

The pesticides LPOS and sulfluramid will not be used in the US after 2016.

### Sources to drinking-water

#### 1. To source waters

The Stockholm Convention meeting stated that: “PFOS are both intentionally produced and an unintended degradation product of related anthropogenic chemicals. The current intentional use of PFOS is widespread and found in products such as in electric and electronic parts, fire fighting foam (in New Zealand, used on Defence properties), photo-imaging, hydraulic fluids and textiles. PFOS are still produced in several countries today”. High concentrations have been found in groundwater where firefighting training has taken place. The import, manufacture or use in New Zealand of firefighting foams containing PFOS and PFOA were excluded from use as firefighting foams in New Zealand under the HSNO Fire Fighting Chemicals Group Standard in 2006.

Most countries have banned the use of these substances for fire fighting, although there are reports that some are replacing the 8-carbon chain PFASs with 6-carbon chain PFASs.

Perfluorooctane sulfonyl fluoride contains up to 5 percent of perfluoroalkyl  
(C4–C7) sulfonylfluorides as an impurity.

Lithium perfluoro octane sulfonate is listed in ERMA’s Summary of Approvals of Substances Transferred under the Hazardous Substances (Chemicals, and also Pesticides where it said to be in a bait) Transfer Notice 2006 (with amendments), as at 24 June 2008 (<http://www.ermanz.govt.nz/hs/transfer/summaryapprove.html>, then select Summary of Approvals: Chemicals or Pesticides. Perfluorobutyric acid appears in the chemical list with the same CAS number as quoted above for perfluorobutyrate.

Sulfluramid, an acaricide and insecticide, is listed in ERMA’s Summary of Approvals of Substances Transferred under the Hazardous Substances (Pesticides) Transfer Notice 2006 (with amendments), as at 24 June 2008 (<http://www.ermanz.govt.nz/hs/transfer/summaryapprove.html>, then select Summary of Approvals: Pesticides. The CAS name for sulfluramid is *N‑*ethyl‑1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-1-octanesulfonamide. Sulfluramid is not registered for food or crop use.

PFASs have been manufactured since the 1960s. Perfluorooctanoic acid (PFOA) is not only used to manufacture Teflon, but is also a breakdown product of chemicals used to coat food packaging, and stain-resistant coatings for couches, carpets, and clothing (eg, 3M’s “Scotchgard”). IARC (2016) states that PFOA has also been used in cosmetics, greases and lubricants, paints, polishes, adhesives, and fluorinated surfactants, and has found use as a grease and water-repellent coating in food packaging.

Perfluoroalkyls constitute a class of compounds that have been used extensively in surface coating and protectant formulations due to their unique surfactant properties. Major applications have included protectants for paper and cardboard packaging products, carpets, leather products, and textiles (eg, Goretex) that enhance water, grease, and soil repellency. Perfluoroalkyls have also been used as processing aids in the manufacture of fluoropolymers such as non-stick coatings on cookware.

Many countries have restricted production of PFASs since the late 1990s, resulting in significant reductions of these products in human blood and the environment.

### Forms and fate in the environment

PFOSF is transformed to perfluorooctane sulfonate (PFOS) in water through hydrolysis at ambient temperatures. None of the tests for degradation (hydrolysis, photolysis, and biodegradation) showed any indication of degradation of PFOS in aquatic or soil systems, confirming the persistence of PFOS in environmental compartments. PFOS and PFOA are expected to be stable to hydrolysis in the environment based on half-lifes of 41 and 92 years, respectively, calculated from experimental hydrolysis data that were measured over pH 5, 7, and 9 (in ATSDR 2009). Because of their strong carbon-fluorine bonds, PFOA and PFOS are stable to metabolic and environmental degradation and are resistant to biotransformation (USEPA 2014 and 2014a).

Due to their chemical structure, perfluoroalkyls are very stable in the environment and are resistant to biodegradation, photoxidation, direct photolysis, and hydrolysis. The perfluoroalkyl carboxylic acids and sulfonic acids have very low volatility due to their ionic nature. As a group, perfluoroalkyls are persistent in soil and water. Perfluoroalkyls are mobile in soil and leach into groundwater. Perfluoroalkyls have been detected in environmental media and biota in many parts of the world, including oceans and the Arctic, indicating that long-range transport of these substances is taking place; they bioaccumulate in animals and humans. Unlike most other environmentally persistent organic chemicals, PFOS and PFOA bind poorly to soil and organic material, and therefore exist mainly in the dissolved phase in surface waters. At pH values above pH 4 they exist in dissociated anionic form and may bind electrostatically to positively charged particles.

Sulfluramid breaks down to perfluorooctane sulfonic acid (PFOS).

Solubility in water of the perfluorooctane sulfonate anion: 550 mg/L. Solubility in water of lithium perfluorooctane sulfonate: 550 mg/L. Solubility in water of perfluorooctanoic acid (PFOA): 9500 mg/L, and perfluorooctane sulfonic acid (PFOS); 519 mg/L. Solubility in water of sulfluramid: 0.0001 mg/L.

### Typical concentrations in drinking-water

Two water utilities in the US reported detecting perfluorobutane sulfonate (PFBS) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00008 mg/L.

Twelve water utilities in the US reported detecting perfluorobutanoic acid (PFBA) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0023 mg/L.

Three water utilities in the US reported detecting perfluorohexane sulfonate (PFHXS) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00004 mg/L.

Two water utilities in the US reported detecting perfluorohexanoic acid (PFXHA) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00013 mg/L.

One water utility in the US reported detecting perfluorooctane sulfonate (PFOS) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00048 mg/L.

Five water utilities in the US reported detecting perfluorooctanoic acid (PFOA) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00025 mg/L.

Three water utilities in the US reported detecting perfluoropentanoic acid (PFPEA) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00007 mg/L.

DWI (2008) found that where PFOS and PFOA were detected, source water originated primarily from unconfined aquifers as might be expected if resulting from point source contamination events.

PFOS has been monitored in the drinking water in Guernsey and Alderney over the last five years due to its use in fire fighting foams. The levels of PFOS in drinking water in Guernsey have followed a slow downward trend over the last five years from a mean of 0.72 μg/L in 2007/8 to 0.5 μg/L in 2011 whilst those in Alderney have remained relatively static over the last five years with a mean of around 0.45 μg/L. (HPA 2012).

Thompson et al (2011) state that Australia does not have a record of local PFAA manufacturing and has a relatively small inventory of PFAAs. 62 samples were collected directly from drinking water taps in several batches between August and November 2010 at 34 locations including capital cities and regional centres around Australia. PFOS and PFOA were the most commonly detected PFAAs and were quantifiable in 49 percent and 40 percent of samples respectively, and were typically found at the highest concentrations of the PFAAs. PFHxS was also detected in 27 percent of samples and at concentrations generally less than PFOS but higher than PFOA. All samples showed low concentrations of PFAAs, with a greater percentage of non-detects relative to detection. The highest concentration of combined PFAAs found was 36 ng/L. They concluded that drinking water is only a minor contributor to the daily intake of these chemicals in the Australian population, although in some locations it may contribute more.

In Australia, public concerns over PFASs have been heightened in recent years by the discovery of these chemicals in groundwater supplies adjacent to Airforce bases, civilian airports and firefighting training facilities where firefighting foams containing PFASs have been used. In some of these areas, public or private drinking water supplies have been drawn from contaminated groundwater, leading to concerns over possible health effects among consumers (*Health Stream* April 2017).

Fulmer (2014) stated that the USEPA is gathering data on US water supplies. Of 7,411 samples, there were only 44 detections of PFOS, 55 of PFOA and fewer of the other PFASs. Maximum levels to date are 0.93 µg/L of PFOS, 0.14 µg/L of PFOA, 0.44 µg/L of PFHxS and 0.07 µg/L of PFHpA.

Samples from potable water supplies without known point sources of perfluorooctanoate contamination typically contain perfluorooctanoate at <1 ng/L. However, a reported average concentration of perfluorooctanoate of 5.4 ng/L, and a maximum concentration of 33 ng/L, for 15 tap water samples were collected in eight cities in the Republic of Korea (IARC 2016).

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,972 drinking water samples for PFOS between 2013 and 2015, and found 292 samples exceeded the minimum reporting level (MRL) of 0.04 µg/L, and 46 water supplies contained >0.07 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,972 drinking water samples for PFOA between 2013 and 2015, and found 379 samples exceeded the minimum reporting level (MRL) of 0.02 µg/L, and 13 water supplies contained >0.07 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,972 drinking water samples for PFNA between 2013 and 2015, and found 19 samples exceeded the minimum reporting level (MRL) of 0.02 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,972 drinking water samples for PFHxS between 2013 and 2015, and found 207 samples exceeded the minimum reporting level (MRL) of 0.03 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,972 drinking water samples for PFHpA between 2013 and 2015, and found 236 samples exceeded the minimum reporting level (MRL) of 0.01 µg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,972 drinking water samples for PFBS between 2013 and 2015, and found 19 samples exceeded the minimum reporting level (MRL) of 0.09 µg/L.

Some New Zealand groundwaters (usually shallow) sampled in areas where PFASs have been used have been found to contain a variety of PFASs. The concentration of PFOS plus PFHxS in 18 bores near Ohakea ranged from 0.002 to 18.9 µg/L; Pattle Delamore report to Ministry of Defence (2017). The analytical technique used was able to measure the concentration of per- and polyfluoroalkyl substances based on chains of 3  to 14 carbon atoms, plus a range of perfluorooctanesulfonamides, perfluorooctanesulfonamidoacetates, perfluorooctanesulfonamidoethanols, and telomer sulfonic acids.

### Removal methods

Fulmer (2014) reports that NF and RO were effective at removing PFCs. Anion exchange and GAC are capable of removing long chain compounds but less effective for shorter PFASs.

Granular activated carbon has been shown to be effective in removing PFOS and PFOA at parts per billion levels from relatively clean water. GAC consistently removes PFOS at μg/L concentrations with an efficiency of more than 90 percent. However, GAC can be inefficient at removing PFOA and other PFASs. The sorption velocity is faster for longer-chained PFASs and smaller diameter GAC particles; therefore, GAC that is optimised for PFOS removal will not optimally remove other PFAS compounds. Adsorption loadings for GAC are relatively low compared with other contaminants, and competition occurs when other contaminants are present (Concawe 2016).

Ion exchange resins or ion exchange polymers provide a large surface area on to which PFOS can attach. The contaminant removal from water is achieved by the attraction of the negatively charged functional to positively charged functional groups within the resin. The removal is stoichiometric, unlike sorption. A variety of resins containing different functional groups are available. Ion exchange resins are considered suitable for low concentration and high volume water treatment applications. Upon reaching maximum capacity of the resin, regeneration with NaCl solution, ethanol or hot water is possible and would produce a low volume concentrated PFOS waste stream ready for incineration (Concawe 2016). Spent GAC needs to be incinerated. A cost of the process is testing for perfluorooctane compounds to determine treatment rates, efficiency, and when the GAC is loaded.

Conventional methods of wastewater treatment are ineffective at removing PFASs. This means that wastewater treatment plants’ effluents can contain varying quantities of these chemicals and their precursors when the effluents are released into the environment. Drinking water treatment plants downstream will then have contaminated influents. The concentrations of these compounds in the drinking water will vary depending on the source of contamination–industrial or municipal wastewater effluents. The limited benchscale studies that have been done on advanced treatment methods have shown promising results for the removal of these chemicals, especially using GAC, anion exchange for the larger molecules; NF/RO membrane filtration are effective for the smaller molecules as well (WRF 2016).

In 2007, the Minnesota Department of Health arranged for tests to determine if water filtration systems could lower PFAS in water. At that time, there was no NSF standard for reducing PFAS. Fourteen filters were tested, and eleven of these were shown to sufficiently reduce the amount of PFAS in water. Four of these filters were activated carbon devices and seven were reverse osmosis devices. None of the devices were, or are currently, certified for PFAS removal. Since then, NSF developed NSF P473 PFOA/PFOS; water filters or systems are evaluated on their ability to reduce [PFOA and PFOS](http://www.nsf.org/consumer-resources/water-quality/drinking-water/perfluorooctanoic-acid-and-perfluorooctanesulfonic-acid-in-drinking-water) in drinking water and to meet strict material safety and structural requirements as defined in NSF/ANSI 53. The cartridges may be disposed of in household trash; they are not considered hazardous waste; (Michigan DEQ 2017). As at December 2017, three manufacturers (using GAC and RO filters) have met the NSF P473 standard, see <http://info.nsf.org/Certified/DWTU/Listings.asp?ProductFunction=P473%7CPFOA+Reduction&ProductFunction=P473%7CPFOS+Reduction&ProductType=&submit2=Search>.

Advanced oxidation processes probably destroy all PFASs.

### Analytical methods

#### Referee method

No MAV so not needed.

#### Some alternative methods

See DWI (2008); Thompson et al (2011); IARC (2016). Also Concawe (2016) which also discusses sampling difficulties. Care needs to be taken to ensure that laboratory plasticware and components of instruments do not contain PFASs in order to avoid cross-contamination of samples. In addition, there is uncertainty about the potential for contamination from everyday items such as clothing and other items that have been treated with waterproofing agents, clothing laundered with fabric softener, and even cosmetics and sunscreens worn by sampling or laboratory personnel.

### Health considerations

Perfluorooctane sulfonyl fluoride contains one or more organic fluorochemicals that have the potential to be absorbed and remain in the body for long periods of time, either as the parent molecule or as metabolites, and may accumulate with repeated exposures. There are no known human health effects from anticipated exposure to these organic fluorochemicals when used as intended and instructed. There are reports of perfluorooctane compounds having a half-life in the body of more than four years.

Nearly all Canadians carry low levels of perfluorinated chemicals, including PFOS, in their blood as a result of exposure. While exact exposure routes are not well understood, people can be potentially exposed to perfluorinated chemicals from contaminated air, surface and groundwater, contaminated foods, in certain occupational settings, and from the possible release of perfluorinated chemicals during the normal degradation or possible use of commercial products that contain them. Health Canada’s assessment concluded that adequate margins of exposure existed between the amount of PFOS in human blood compared to levels at which effects occurred in animals, including consideration of age differences and differences within and between species. Specific consideration of the risk to children’s health was an integral part of the assessment. Health Canada and Environment Canada have completed ecological and human health screening assessments for PFOS, its salts and precursors. The human health screening concluded that current levels would not have a harmful effect on human health.

The EFSA in 2008 derived an ADI (TDI) of 0.15 μg/kg/d for PFOS and 1.5 for PFOA. Interim guidance values for drinking water were also determined, based on the default assumption for industrial chemicals that drinking water is responsible for 10 percent of human exposure to these chemicals. These guidance values were 0.5 μg/L for PFOS and 5.0 μg/L for PFOA (from *Health Stream* April 2017).

PFOS has shown developmental effects in mammals at low levels (no observed adverse effect level (NOAEL) value of 0.1 mg/kg body weight/day in rats in a two-generation study). There is evidence that PFOS causes chronic and reproductive toxicity in humans and aquatic organisms, and structure activity relationship analysis indicate that these results are applicable to other PFASs as well (stated in NICNAS 2009).

The UK Expert Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) proposed a tolerable daily intake (TDI) for PFOS of 0.3 μg/kg bw/day in 2006. The derivation was based on a study of thyroid function in Cynomolgus monkeys, where a no observed adverse effect level (NOAEL) of 30 μg/kg bw/day was identified. An uncertainty factor of 100 to allow for inter- and intra-species variation to the NOAEL was applied to this figure to provide the TDI. In terms of water that is unfit for human consumption, the DWI recommends the following levels of PFOS in drinking water might lead to acute effects in an individual following consumption HPA (2012):

* 67 μg/L for bottle-fed babies
* 100 μg/L for 1 year old children
* 300 μg/L for adults.

Extensive data in humans and animals demonstrate ready absorption of PFOA and distribution of the chemical throughout the body by noncovalent binding to plasma proteins. The liver is an important binding site with increased liver weight in laboratory animals one of the early, low-dose manifestations of exposure. PFOA is not readily eliminated from humans as evidenced by the half-life of 2.3 years. In contrast, half-life values for the monkey, rat, and mouse are 20.8 days, 11.5 days, and 15.6 days, respectively (USEPA 2014).

Available data on PFAS toxicity is dominated by PFOS, PFOA and also perfluorohexane sulfonate (PFHxS) due to the widespread detection of these compounds in humans and the environment, and concern that these could biomagnify to a level whereby humans consuming fish may be adversely affected. Much less data is available on the toxicology of other PFAS, and this is often inconsistent and fragmentary. For the less investigated polyfluorinated chemicals, toxicology is often estimated based on structure-activity relationships, or structural homologues (Concawe 2016).

The USEPA (2016a) selected 0.00002 mg/kg/day as the RfD for PFOA based on reduced ossification of the proximal phalanges (forelimb and hindlimb) and accelerated puberty in male pups (4 days earlier than controls) as the critical effects. The evidence for the carcinogenicity of PFOA is considered *suggestive* because only one species has been evaluated and studies for gene mutations were negative (USEPA 2014/2016a). USEPA (2016a) estimated a cancer slope factor of 0.07 per milligram per kilogram-day (mg/kg-day)-1 based on testicular tumors, and confirmed that the lifetime HA based on non-cancer effects is protective of the cancer endpoint.

USEPA (2016b) derived a reference dose (RfD) for PFOS of 0.00002 mg/kg/day based on decreased neonatal rat body weight from the two-generation study. PFOS is not readily eliminated from humans as evidenced by the half-life of 5.4 years. The long half-life appears to be the result of resorption from the kidney. USEPA (2014a) has selected 0.00003 mg/kg/day as the RfD for PFOS based on the consistency of the response and with recognition of the use of developmental toxicity and liver weight as the most sensitive endpoints for protection against co-occurring adverse effects. The evidence for the carcinogenicity of PFOS is considered “*suggestive of carcinogenicity”,* but not sufficient to assess human carcinogenicity potential.

New FSANZ TDI levels have been developed from animal study data because the data collected from human epidemiological studies were not considered suitable for derivation of regulatory values. The new TDI for PFOS is 20 ng/kg bw/day (in contrast to the interim level of 150 ng/kg bw/day) and the new TDI for PFOA is 160 ng/kg bw/day for PFOA (in contrast to the interim level of 1,500 ng/kg bw/day). The available data were insufficient to establish a TDI for PFHxS, but FSANZ recommended assumption of the same TDI as PFOS, with the concentrations of these two chemicals to be added together when assessing risks. The resultant guidance values for drinking water (assuming a 10 percent contribution of drinking water to daily intake) are 70 nanograms of PFOS/PFHxS per litre and 560 nanograms of PFOA per litre. Recreational water guidance values are 10-fold higher than drinking water guidance values. The available data indicate that current exposure levels to PFASs for the general population in Australia are very low, and that efforts to reduce exposure should continue to focus on populations at specific contaminated sites (ex *Health Stream* April 2017).

Since manufacturers phased out production of PFOA and PFOS serum levels have fallen in the general population. From 1999-2014, blood PFOA and PFOS levels declined by more than 60 percent and 80 percent respectively (CDC 2017).

Most detailed studies of toxic and adverse health effects have been carried out for PFOS and PFOA. These two compounds, alongside PFHxS, are the compounds which are usually detected at the highest concentrations in human matrices. However, their use is currently being phased out and shorter-chain compounds are increasingly being used as replacements. Concawe (2016) lists half-lifes for some PFASs in various animals.

As at June 2018 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for perfluorooctanoic acid (PFOA) of:

* 0.000003 mg/kg/day for intermediate-duration oral exposure (14–365 days).

As at June 2018 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for perfluorooctane sulfonic acid (PFOS) of:

* 0.000002 mg/kg/day for intermediate-duration oral exposure (14 – 365 days).

As at June 2018 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for perfluorohexane sulfonic acid (PFHxS) of:

* 0.00004 mg/kg/day for intermediate-duration oral exposure (14 – 365 days).

As at June 2018 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for perfluorononanoic acid (PFNA) of:

* 0.000003 mg/kg/day for intermediate-duration oral exposure (14 – 365 days).

IARC (2016) concluded there is *limited evidence* in humans for the carcinogenicity of perfluorooctanoic acid (PFOA) hence it is *possibly carcinogenic to humans (Group 2B)*. A positive association was observed for cancers of the testis and kidney.

### Derivation of Maximum Acceptable Value

No MAV.

DWI (2008) set a “wholesomeness” level (ie, put in place measures to reduce concentrations to below x μg/L as soon as is practicable) as follows:

* perfluorooctane sulphonate (PFOS) x = 0.001 mg/L
* perfluorooctanoic acid (PFOA) x = 0.01 mg/L.

The Minnesota Department of Health (MDH 2009/2016) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limits (HRL, for exposure greater than 10 percent of a lifetime) for some of these compounds are:

* perfluorobutane sulfonate (PFBS) 0.007 mg/L; 0.009 mg/L for subchronic
* perfluorobutyrate (PFBA) 0.007 mg/L; also short-term and subchronic
* perfluorooctane sulfonate and salts (PFOS) 0.0003 mg/L
* perfluorooctanoic acid and salts (PFOA) 0.0003 mg/L

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# Petroleum products

Also called total petroleum hydrocarbons or TPH.

### Maximum Acceptable Value

Other than those chemicals with separate listings, there are insufficient data to derive MAVs for any petroleum products. WHO (2017) adds that taste and odour will in most cases be detectable at concentrations below those of health concern, particularly with short-term exposure.

Note: the following determinands may appear in petroleum products, have a MAV, or have a separate datasheet: benzene, benzo[a]pyrene, ethylbenzene, fluoranthene, methyl t-butyl ether, toluene, xylenes. There may be more!

Acceptable maximum concentrations for drinking water are 50 µg/L (0.05 mg/L) for gasoline, diesel oil, and fuel oil; and 35 µg/L (0.035 mg/L) for methyl tertiary-butyl ether (MtBE – see datasheet). Accessed in October 2013 from University of Maine, <http://www.umaine.edu/WaterResearch/outreach/safe_drinking_water_digest.htm>.

### Introduction: what are petroleum products?

Petroleum products comprise a broad family of several hundred chemical compounds that originally come from crude oil, plus any additives found in commercial products. Common products include gasoline, kerosene, fuel oil, mineral oil, bitumen and asphalt. Approximately 3,000 million tonnes of petroleum fuels, solvents, lubricants, bitumens and other products are produced annually from crude oil. Crude oil, which may be broadly characterised as paraffinic or naphthenic, is a complex mixture of alkanes, cycloalkanes and aromatic hydrocarbons containing low percentages of sulfur, nitrogen and oxygen compounds and trace quantities of many other elements.

**Hydrocarbons** are organic compounds composed of carbon and hydrogen atoms arranged in varying structural configurations. At a simple level, they may be divided into two families: aliphatics and aromatics. The aliphatics may be further subdivided into four groups: alkanes (straight and branched chain), alkenes, alkynes and cyclic alkanes. Alkynes are not generally found in petroleum products and are not considered further. Within each hydrocarbon structural family and subfamily, there are homologous series. Each member of the series differs from adjacent members of the series by a repeating unit, such as a CH2 group. Small amounts (mg/kg quantities) of constituents such as polycyclic aromatic hydrocarbons (PAHs) may also be found in some petroleum products.

**Petroleum products** are derived from crude oil, which undergoes fractionation in order to produce petroleum products for particular uses. Their composition varies according to the type of use and depends on their source and fraction. There are significant compositional differences between petroleum products such as gasoline, diesel oil, aviation fuel and heating oil. Crude oil is distilled, and a variety of petroleum product fractions result, with distinct boiling point ranges. The chemical composition of all these products depends on the sources of crude oil, or on the refinery streams from which they are produced. Petroleum products are not the only source of potential contamination of drinking-water with hydrocarbons. Other sources include petrochemical products such as solvents and coal-derived products.

The approach taken within the petroleum industry is to refer to aliphatic and aromatic fractions on the basis of their boiling point normalised to the boiling point of the *n‑*alkanes or retention time on a boiling point gas chromatographic column. This is characterised by the equivalent carbon (EC) number. For example, the boiling point of hexane, which consists of six carbon atoms, is 69°C, and its EC number is 6. Benzene also consists of six carbon atoms, but its boiling point is 80°C, and its EC number is 6.5. The fractions for the aromatic compounds are presented on the basis of their EC numbers; since many of these relate to fractions of whole values, similar to the situation with benzene, they are usually represented as greater than the lower value. Fractions for aliphatic compounds are also presented on the basis of EC numbers; the EC numbers for aliphatic compounds are the same as the carbon (C) numbers for straight-chain alkanes, but differ for branched and cyclic alkanes. The EC number is used throughout this datasheet.

Petroleum-derived products will often also contain additives, but these are normally present in very low concentrations. The exception is fuel additives such as methyl *tertiary*-butyl ether (MTBE), which is considered separately.

Detailed physical and chemical properties are available for only about 250 petroleum hydrocarbons.

**Diesel fuel** is a complex mixture of normal, branched, and cyclic alkanes (60 to >90 percent by volume; hydrocarbon chain length, usually between C9 and C30); aromatic compounds, especially alkylbenzenes (5 to 40 percent by volume); and small amounts of alkenes (0 to 10 percent by volume) obtained from the middle-distillate, gas-oil fraction during petroleum separation. Benzene, toluene, ethylbenzene, and xylenes and polycyclic aromatic hydrocarbons (PAHs), especially naphthalene and its methyl-substituted derivatives, may be present at levels of parts per million in diesel fuel. Additives are used to influence the flow, storage, and combustion of diesel fuel, to differentiate products, and to meet trademark specifications. Heating fuels and some kerosene jet fuels produced during the refining process may have a composition similar to that of diesel fuel, although with different additives.

**Biodiesel** is usually a fatty acid methyl ester made by reacting vegetable oils or animal fats with methanol. The most common feedstocks are soya bean, rapeseed and palm oils, and various waste animal fats. In general, biodiesel does not significantly change engine efficiency but leads to slightly higher fuel consumption because of its lower energy content. It is usually used in blends with petroleum diesel, where for example B20 refers to a 20 percent biodiesel blend (IARC 2013).

**Bitumens** are engineering materials produced by the distillation of crude oil during petroleum refining and exist in numerous forms and types. Bitumens (sometimes called asphalt in the US) are dark viscous liquids or semi-solids that are non-volatile at ambient temperatures and soften gradually when heated. Asphalt is the term used for a mixture of small stones, sand, filler and bitumen (~5 percent), which is used as a road-paving material. Coal-tar products contain much higher concentrations of polycyclic aromatic hydrocarbons (PAHs) than bitumens, particularly in the three- to seven-ring size range. In contrast, bitumens contain higher concentrations of paraffinic and naphthenic hydrocarbons and their derivatives, whose large size and viscosity result in limited solubility. Similarly, bitumen should not be confused with petroleum pitch, which is the highly aromatic residue produced by thermal cracking (ie, extreme heat treatment) of selected petroleum fractions. The properties and chemical composition of petroleum pitch are therefore quite different from those of refined bitumen. While the term “petroleum pitch” is not used consistently (this term is used to describe different materials in different areas), petroleum pitches are principally used as binders in the manufacture of metallurgical electrodes (IARC 2013).

The following table has been copied from EU (2008). It shows the mean road site concentrations of individual NMVOCs (non-methane volatile organic compounds) in air due to non-isolated benzene in car exhaust at a site in Copenhagen during five days in December 1997. The units are ppbv, parts per billion by volume.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Substance** | **Mean** | **Range** | **SD** | **Median** |
| pentane | 2.4 | 0.4–5.7 | 1.2 | 2.5 |
| trans-2-pentene | 0.2 | 0.01–0.5 | 0.1 | 0.2 |
| 2-methyl-2-butene | 0.4 | 0.02–0.9 | 0.2 | 0.3 |
| cis-2-pentene | 0.1 | 0.01–0.3 | 0.1 | 0.1 |
| 2,2-dimethylbutane | 0.9 | 0.04–2.3 | 0.5 | 0.9 |
| cyclohexane | 0.5 | 0.04–1.1 | 0.3 | 0.5 |
| 2,3-dimethylbutane | 0.4 | 0.03–1.0 | 0.2 | 0.4 |
| 2-methylpentane | 2 0 | 2–5.2.0 | 1.1 | 2.1 |
| 3-methylpentane | 1.1 | 0.1–2.7 | 0.6 | 1.0 |
| n-hexane | 0.8 | 0.1–2.3 | 0.5 | 0.8 |
| isoprene | 0.2 | 0.01–0.6 | 0.1 | 0.2 |
| 2-methyl-1-pentene | 0.04 | 0.01–0.1 | 0.02 | 0.02 |
| cis-2-hexene | 0.03 | 0.01–0.1 | 0.01 | 0.02 |
| 2,4-dimethylpentane | 0.2 | 0.01–0.7 | 0.1 | 0.20 |
| methyl-cyclohexane | 0.3 | 0.02–0.6 | 0.1 | 0.3 |
| 2- and 3-methylhexane | 1.4 | 0.1–3.7 | 0.8 | 1.3 |
| n-heptane | 0.7 | 0.1–1.9 | 0.4 | 0.6 |
| benzene | 3.4 | 0.2–8.0 | 1.7 | 3.3 |
| 2- and 3-methylheptane | 0.4 | 0.01–1.0 | 0.2 | 0.3 |
| toluene | 10.2 | 0.8–21.5 | 5.6 | 8.9 |
| ethylbenzene | 2.0 | 0.1–4.9 | 1.1 | 1.9 |
| o-xylene | 2.7 | 0.1–6.2 | 1.4 | 2.6 |
| m- and p-xylene | 5.5 | 0.3–12.7 | 2.9 | 5.5 |

### Sources to drinking-water

#### 1. To source waters

Petroleum products are used widely in a range of industrial applications. The largest quantities find use as fuels for a range of purposes, including gasoline, diesel oil, aviation fuel and heating oil.

Petroleum products are stored and handled in a range of circumstances, and the primary concern for drinking-water is the potential for spills, particularly to groundwater from underground storage tanks. The actual nature of any contaminants present will largely be a function of their solubility in water, and whether they float, sink or emulsify.

Pleasure craft can also contaminate water. In the absence of New Zealand data, NIWA (2007) conducted a literature search of the effects. The report noted that although outboard exhaust contaminants are not present in concentrations that exceed aquatic protection guidelines, there is a significant risk of boating activities tainting drinking water (odour/taste). BTEX chemicals, low molecular aromatics and MTBE are capable of tainting water at concentrations as low as ca. 20 ng/L (0.02 µg/L). Accordingly, it has been recommended that boating activities not be permitted within 100 m of a water intake structures or on small lakes (with slow flushing times) used for domestic water supply. Despite the potential for negative impacts, it is important to emphasise that four-stroke outboard emissions are at least 10-fold lower than those from the same powered two-stroke engine.

The practice of oiling roads with used oil to reduce dust volumes is expected to have an adverse effect on the quality of water in a stream lying between 5 m and 7 m from the road (5 m and 7 m are the length of the grass verge and the distance from the road with the highest deposition rate, respectively). The oil should not be applied within 10 m of a watercourse to protect both the water and sediment quality. To minimise runoff of free oil product the used oil should only ever be applied to a dry road and when 2–3 days of fine weather is forecasted. Benzo(a)pyrene is the main contributor to the unacceptable risk (MfE 2000).

Bitumen emissions can end up in water through surface runoff from land, and fallout and rainout from the atmosphere. Concentrations of PAHs were determined in water samples collected from water draining from road surfaces and from water upstream and downstream from the point of discharge from road surfaces into stream sites in California, USA. The concentrations of PAHs in all stream and road runoff samples were below the detection limit of 0.5 μg/L. Leaching tests of bitumen-based materials have been conducted in laboratories. Six samples of paving bitumen and four samples of roofing bitumen were leached according to USEPA method SW846–1311. None of the roofing samples tested leached any of the 29 PAHs analysed. Four of the paving samples did not leach any of the 29 PAHs, and the leachates of two paving samples contained detectable amounts of naphthalene and phenanthrene. The levels were below the detection limit of 0.1 μg/L, except for naphthalene with a value of 0.18 μg/L. From IARC (2013).

Sometime between Friday 27 September and Monday 30 September 2013 approximately 19,000 litres of diesel was lost from the storage system on Turoa ski field and flowed into the Makotuku Stream from which Raetihi (population 1,000) draws its water supply. Alternative arrangements were required for about a month. The quantifiable impacts amounted to almost $2 million (Sapere 2014). USEPA (2001) discusses managing storage tanks.

#### 2. From treatment processes

On-site spills.

#### 3. From the distribution system

Petroleum products have been reported passing through plastic pipes, into the water, see Chapter 16, section 16.2.6: Leaching and permation.

### Forms and fate in the environment

The differing chemical and physical properties of petroleum hydrocarbons mean that they will behave differently in the environment. Persistence of petroleum hydrocarbon compounds in the environment is reflected by physical properties such as volatility, so that generally the persistence increases as the boiling point increases. The main processes affecting environmental concentrations are volatilisation, biodegradation and dissolution in water. Only a small proportion of the hydrocarbon constituents will be significantly soluble in water. The hydrocarbons present in contaminated drinking-water will not, therefore, reflect the hydrocarbon composition of the petroleum oil.

### Typical concentrations in drinking-water

Exposure is frequently the result of an accidental spill or short-term incident, in which the main issue for drinking-water is short-term exposure. Such incidents may lead to high concentrations of total petroleum hydrocarbons (TPH), in which case the probability of unacceptable taste and odour being detected by consumers will be significantly increased.

### Removal methods

The first action to be taken if water is contaminated by a spill of petroleum products is containment. In the case of surface waters, floating booms can be used to contain the spill in as small an area as possible, away from drinking-water abstraction points. If a spill occurs and no containment equipment is available, containment booms can be improvised from whatever materials are at hand, such as wood or plastic pipes. Skimming, absorbents, or other methods can be used to remove the petroleum products contained on the surface of the water behind the containment boom. Our regional councils should operate quick response teams.

There is relatively little information on the ability of treatment processes to remove petroleum products from water. Relatively high concentrations of petroleum products are amenable to treatment by coagulation; percentage removal increases as the carbon number increased; WHO (2005):

|  |  |
| --- | --- |
| **Carbon number** | **% removal** |
| 7 | 10 |
| 8 | 20–25 |
| 9 | 75 |
| 10 | 70 |
| >10 | 100 |

Studies have shown that different types of membranes can be effective. Aeration will reduce the concentration of the more volatile components. Activated carbon and some other adsorbents are likely to be reasonably effective for removing low levels of contamination.

### Analytical methods

#### Referee method

A referee method cannot be selected for general petroleum products because MAVs have not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

No alternative methods can be recommended for these products because MAVs have not been established. However, the following information may be useful:

The methods for petroleum hydrocarbons are based largely on gas chromatography and liquid chromatography (TPHCWG 1998a).

The USEPA has published methods for petroleum hydrocarbons in water. These include EPA 418.1, which uses infrared absorbance; however, the detection limit is quite high, at 0.5 mg/L, and the method does not discriminate between different hydrocarbons. The USEPA has also published different methods for TPH in water. These include modified EPA 3510C/8015B, which is based on gas chromatography with flame ionisation detection. This method is for TPH as gasoline, jet fuel and diesel and allows the quantification of the hydrocarbons EC6 through EC20. Other methods, such as EPA 5030B for aromatic volatile aromatics, using gas chromatography with a photoionisation detector, are more specific for the more volatile aromatic components that are most likely to reach drinking-water; the detection limit for EPA 5030B is 0.0005 mg/L in water (USEPA 1997a, 1997b).

Note that following the diesel spill that affected the water supply in Raetihi in 2013 it was considered impractical to test for hydrocarbons because the level of detection for total petroleum hydrocarbons is 0.2 mg/L whereas the odour threshold for diesel in water is 0.0005 mg/L.

### Health considerations

IARC (1989) classified:

* crude oil (CAS No. 8002-05-9) as not classifiable as to its carcinogenicity to humans (Group 3)
* gasoline (CAS No. 8006-61-9) as possibly carcinogenic to humans (Group 2B)
* jet fuel (kerosene is CAS No. 8008-20-6) as not classifiable as to its carcinogenicity to humans (Group 3)
* diesel oil (CAS No. 68334-30-5):
* marine diesel fuel – possibly carcinogenic to humans (Group 2B)
* distillate (light) diesel fuels – not classifiable as to carcinogenicity to humans (Group 3)
* fuel oils (heating oils):
* residual (heavy) fuel oils – possibly carcinogenic to humans (Group 2B),
* distillate (light) fuel oils – not classifiable as to their carcinogenicity to humans (Group 3).

and (IARC 2012):

* untreated and mildly treated mineral oils: carcinogenic to humans (Group 1)
* shale oils (CAS No. 68308-34-9): carcinogenic to humans (Group 1).

In general terms, alkanes have relatively low acute toxicity, but alkanes having carbon numbers in the range of EC5–EC12 have narcotic properties. Alkenes exhibit little toxicity other than weak anaesthetic properties. Most of the smaller aromatic compounds are of relatively low toxicity except for benzene, which is a known human carcinogen.

The fact that petroleum products are complex mixtures of hundreds of individual hydrocarbons is a complicating factor in determining their toxicity in the event of contamination of water. This means that the traditional approach of evaluating individual components is largely inappropriate. In order to overcome this difficulty, it is more practical to consider a series of hydrocarbon fractions and to determine appropriate tolerable concentrations for those fractions. A number of groups have examined such an approach, but the most widely accepted is that developed by the TPHCWG in the USA. This is a multi-agency group, consisting of representatives from industry, government and academia, which has developed and published a series of five monographs detailing the data on petroleum hydrocarbons and, in addition, has developed tolerable intakes for a series of total hydrocarbon fractions.

Of the 250 individual compounds identified in petroleum by the TPHCWG, toxicity data were available only for 95. Of these 95, the TPHCWG concluded that there were sufficient data to develop toxicity criteria for only 25.

The approach used by the TPHCWG to make the problem more manageable was to divide TPH into a series of fractions based on the number of carbon atoms in conjunction with general structure. The toxicity data available on fraction-specific mixtures cover the aromatic fractions (>EC6–EC8, as described above) and the aliphatic fractions of TPH. Data on mixtures containing the higher-molecular-weight substances, >EC8–EC16 and >EC16–EC35 aromatic fractions, refer only to the EC8–EC11 range. There are no toxicity data on the highest-molecular-weight compounds, >EC35. However, compounds above EC20 are neither volatile nor soluble in water. In addition, compounds >EC35 are not likely to be absorbed by the oral or dermal routes of exposure (TPHCWG, 1997a, 1997b, 1998a, 1998b, 1999).

For more detailed discussion, refer to WHO (2005) and the above TPHCWG (excellent) references.

One of the commoner components of petroleum products is n-hexane. This is discussed in ATSDR (1999).

### Derivation of Maximum Acceptable Value

No MAV.

WHO (2005) states that it is not considered appropriate to set formal health-based guideline values for petroleum products in drinking-water, other than those noted specifically at the beginning of this datasheet.

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# Pharmaceuticals

### Maximum Acceptable Value

The concentrations of pharmaceuticals found in drinking-water are typically orders of magnitude less than the lowest therapeutic doses. Therefore, exposure to individual compounds in drinking-water is unlikely to have appreciable adverse impacts on human health. Formal guideline values are therefore not proposed in these Guidelines (WHO 2011).

### Sources to drinking-water

#### 1. To source waters

There are about 3,000 pharmaceuticals registered in the UK and approximately 5,000 substances listed as human pharmaceuticals were sold over the counter in the UK in 2004. Consumption of active pharmaceutical ingredients in industrial countries is estimated to be between 50 and 150 g per person per year, with fewer than 50 compounds making up 95 percent of the total amount of active pharmaceutical ingredient consumption (DWI 2007).

Pharmaceuticals can be introduced into water sources in sewage by excretion from individuals using these chemicals, from uncontrolled drug disposal (eg, discarding drugs into toilets) and from agricultural runoff from livestock manure. They have become chemicals of emerging concern to the public because of their potential to reach drinking-water (WHO 2011).

A study conducted in the US found that less than 2 percent of those surveyed returned unused medication to a pharmacy, 54 percent added them to household solid waste, and 35 percent disposed of them down the toilet or sink. In the UK, 63 percent of those surveyed discarded unused medications in the household solid waste, 22 percent returned them to a pharmacy and 11 percent emptied them into the sink or toilet. Taken from Peake and Braund (2009).

Humans may potentially be exposed to veterinary medicines in the environment by a number of routes including the consumption of: 1) crops that have accumulated substances from soils as a result of exposure to contaminated manure and slurry; 2) livestock that have accumulated veterinary medicines from food material that has accumulated substances from contaminated soils or water; 3) fish exposed to treatments used in aquaculture; and 4) abstracted groundwater and surface waters containing veterinary medicines (DWI 2011).

Over 29 million pounds of antimicrobials were sold for livestock use in 2010 in the US – an estimated three to four times more than the amount used by humans. 60 percent to 80 percent of livestock routinely receive antimicrobials, the majority of which are estimated to be used for animal growth, rather than for medicinal purposes. The WHO has noted that sub-therapeutic antimicrobial use by livestock and poultry is an area of concern because of the selection for antimicrobial resistance. Antimicrobials generally do not biodegrade easily and may be more mobile in aquatic environments, particularly after spreading manures on pasture (USEPA 2013).

### Forms and fate in the environment

Conventional wastewater treatment facilities generally have activated sludge processes or other forms of biological treatment such as biofiltration. These processes have demonstrated varying removal rates for pharmaceuticals, ranging from less than 10 percent to greater than 90 percent (WHO 2011a; Peake and Braund 2009).

### Typical concentrations in drinking-water

Finding pharmaceuticals in drinking-water is a clear indication of the presence of human wastes (USEPA 2006).

Concentrations of pharmaceuticals in treated drinking-water are usually well below 0.05 μg/L (or 50 ng/L). Routine monitoring of pharmaceuticals in water sources and drinking-water at the national level and the installation of specialised drinking-water treatment infrastructure to reduce the very low concentrations of pharmaceuticals in drinking-water are not currently deemed necessary given the limited additional health benefits (WHO 2011a).

A six-year study in the German city of Rastatt examined the potential of a wide range of anthropogenic chemicals to trace and quantify sewer leakage to groundwater. Three compounds were the most widespread; amidotrizoic acid (iodinated x-ray contrast dye, found in 35 percent of groundwater samples), carbemazapine (anticonvulsant drug, in 33 percent of groundwater samples) and acesulfame (artificial sweetener, in 28 percent of groundwater samples).

Concentrations of NSAIDs and statins in drinking-water can be predicted using a modified model for Tier 0 risk assessment of pharmaceuticals in the environment (Box A3.2 from WHO 2017a).

PECdw = A x (100−R) x (100−M) x (100−W)

365 x P x V x D x 100 x 100 x 100

where:

PECdw is the predicted concentration in drinking-water (mg/L)

M is the percentage metabolised in humans

A is the amount of active ingredient used per year in the catchment (mg/y)

R is the removal rate in sewage treatment works – STW (set as a percentage, see below)

P is the population under consideration

V is the volume of wastewater produced per capita per day (assumed to be 200 L)

W is the removal rate in the appropriate WTP scenario

D is the dilution factor in the environment (derived from the 5th percentile flow rate).

To reflect a worst-case situation, conservative assumptions are made for each of the model inputs, as detailed by Watts et al (2007) and summarised below:

* total usage per year [A] is twice that estimated from published statistics (ie, large overestimate)
* there is no metabolism [M = 0 percent] after taking the drug, ie, all of the amount of NSAIDs and statins used are excreted unchanged (large over-estimate)
* there is no loss of NSAIDs or statins in STWs [R = 0 percent] (ie, over-estimate)
* very low river flow rate resulting in low dilution factor [D] (ie, under-estimate)
* no further dilution or loss of NSAIDs or statins during transport between STW discharge point and the WTP intake point (ie, under-estimate)
* removal rate in WTPs is zero [W =0 percent] (ie, over-estimate).

As a worst case, it is assumed that the population of greatest concern are infants <1 year old. Using the predicted worst case assumptions, total predicted drinking-water concentration for NSAIDs is 0.0975 mg/L and 0.00447 mg/L for statins.

WHO (2017a) includes a similar case study for oestrogens.

### Removal methods

Effective treatment of pharmaceuticals depends on the physicochemical properties of the specific compounds. Studies on conventional drinking-water treatment processes have shown that coagulation is largely ineffective in removing pharmaceuticals. Free chlorine is able to remove up to approximately 50 percent of the pharmaceuticals investigated, whereas chloramines have lower removal efficiency. Advanced water treatment processes, such as ozonation, advanced oxidation, activated carbon and membranes (especially nanofiltration and reverse osmosis), are able to achieve higher removal rates (even above 99 percent) for targeted pharmaceutical compounds in various studies in the published literature (WHO 2011a). Dialysis equipment would be expected to reduce the impact of pharmaceutical residues for kidney patients.

WRF (2016) found ozone to be effective for reducing the concentration of four pharmacologically active compounds in the presence of various natural organic matter matrices; details are provided.

### Analytical methods

#### Referee method

A referee method cannot be selected for general pharmaceutical products because MAVs have not been established and therefore the sensitivity required for the referee method is not known.

#### Some alternative methods

See WHO (2011a), DWI (2012).

### Health considerations

Pharmaceuticals are normally governed by stringent regulatory processes and require rigorous preclinical and clinical studies to assess their efficacy and safety before commercialisation. Therefore, pharmaceuticals are generally better characterised than most other environmental contaminants.

Studies to date indicate that appreciable adverse health impacts to humans are very unlikely from exposure to the trace concentrations of pharmaceuticals that could potentially be found in drinking-water. Concentrations of pharmaceuticals in drinking-water are generally more than 1,000-fold below the minimum therapeutic dose (MTD), which is the lowest clinically active dosage. The findings are in line with the evidence published over the past decade, which suggests that appreciable risks to health arising from exposure to trace levels of pharmaceuticals in drinking-water are extremely unlikely (WHO 2011a).

DWI (2011) identified 10 veterinary medicine compounds that might reach UK drinking waters and where intake may be close to or above the acceptable daily intake (ADI). The compounds were altrenogest, apramycin, cephapirin, dicyclanil, florfenicol, lincomycin, luprostiol\*, sulfadiazine, acetylsalicylic acid (aspirin)\* and monensin. There is a datasheet for dicyclanil. \* not listed in NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at Dec 2013 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)). Further studies (DWI 2014) concluded that the 10 compounds investigated are not expected to impact on drinking water quality under realistic worst case conditions in real world catchments; however water companies should continue to review risks on a case by case basis to account for local circumstances and changes.

From the DWI (2007) desk study, results from a simple exposure model were used alongside information on therapeutic doses of pharmaceuticals to identify pharmaceuticals that are likely to be of most concern in UK drinking water sources. The DWI (2012) study was performed to generate actual measurements on the occurrence of pharmaceuticals in source and treated waters in England. The study considered a range of pharmaceutical compounds and their metabolites that have either (a) high predicted exposure concentrations; (b) toxicological concerns; or (c) a low predicted exposure to therapeutic dose ratio. The 17 selected compounds covered a range of chemical classes and varied in terms of their physico-chemical properties. The study was done at four sites where concentrations in source water at the drinking water treatment abstraction point were predicted to be some of the greatest in England. Comparison of measured concentrations of the study compounds in drinking waters with information on therapeutic doses demonstrated that levels of these compounds in drinking water in England are many orders of magnitude lower than levels that are given to patients therapeutically. It would therefore appear that the low or non-detectable levels of pharmaceuticals and illicit drugs present in drinking waters in England and Wales do not pose an appreciable risk to human health. The 17 compounds were:

atenolol β-blocker

benzoylecgonine cocaine metabolite

caffeine stimulant

carbamazepine anti-epileptic

carbamazepine epoxide carbamazepine metabolite

cocaine illicit drug

cyclophosphamide chemotherapy agent

diclofenac non steroidal anti-inflammatory

fluoxetine antidepressant

furosemide diuretic

ibuprofen non steroidal anti-inflammatory

ketoprofen non steroidal anti-inflammatory

naproxen non steroidal anti-inflammatory

norfluoxetine metabolite of fluoxetine

orlistat anti-obesity

simvastatin hypolipidemic

trimethoprim antibiotic

Subsequently, benzoylecogonine, carbamazepine, carbamazepine-10,11-epoxide, ibuprofen and naproxen were detected in UK treated water. Further studies (DWI 2014a) concluded when carrying out a human health risk assessment based on the reported levels of these pharmaceuticals in drinking water, using default exposure parameters for adults, children and infants, and when using toxicological endpoints as the PoD, then the levels of these pharmaceuticals measured in drinking water are not anticipated to pose an appreciable risk to public health.

Tremblay et al (2011) presents in their Table 6 Pharmac’s 2011 ranking by number of prescriptions of the top 20 pharmaceuticals used in New Zealand in 2010. Table 7 presents total antibiotic sales in 2008–09 by antibiotic family and animal group (kg/year) (supplied by Ministry of Agriculture and Forestry 2010). These follow:

|  |  |  |
| --- | --- | --- |
| **Rank** | **Common chemical name** | **Treatment condition** |
| 1 | Paracetamol | Analgesic / Antipyretic |
| 2 | Aspirin | Analgesic / Anti-platelet |
| 3 | Simvastatin | Cholesterol and cardiovascular control |
| 4 | Omeprazole | Dyspepsia, peptic ulcer disease |
| 5 | Amoxycillin | Broad spectrum antibiotic |
| 6 | Metoprolol succinate | β- blocker for blood pressure control |
| 7 | Amoxycillin clauvulanate | Broad spectrum antibiotic |
| 8 | Salbutamol | Asthma (inhaled) |
| 9 | Diclofenac sodium | Analgesic/ Anti-inflammatory |
| 10 | Cilazapril | ACE inhibitor |
| 11 | Zopiclone | Hypnotic |
| 12 | Ibuprofen | Analgesic |
| 13 | Prednisone | Steroid |
| 14 | Flucloxacillin | Antibiotic |
| 15 | Quinapril | ACE inhibitor |
| 16 | Bendrofluazide | Diuretic |
| 17 | Feldopine | Calcium channel blocker |
| 18 | Alendronate sodium | Osteoporosis |
| 19 | Metformin | Type II diabetes |
| 20 | Fluticasone | Asthma (inhaled) |

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Antibiotic family** | **Cattle** | **Pigs/Poultry** | **Companion** | **Multiple** | **Other** | **Total** |
| Aminoglycosides | 172 | 63 | 6.8 | 996 | 0.0 | 1,217 |
| Bacitran | 18,818 | 0.7 | 0.4 | 21,733 |  |  |
| Cephalosporins | 1,019 | 354 | 155 | 1,528 |  |  |
| Clavulanic acid | 20.8 | 164 | 28 | 213 |  |  |
| Fluoroquinolones | 0.1 | 19 | 22 | 41 |  |  |
| Fusidic acid | 1.7 | 2 |  |  |  |  |
| Macrolides/ |  |  |  |  |  |  |
| Lincosamides | 53 | 1,093 | 29 | 4,157 | 5,439 |  |
| Nitrofurans | 17 | 0.5 | 1.5 | 6 |  |  |
| Nitro-imidazoles | 95 | 11 | 1.0 | 49 |  |  |
| Novobiocin | 0.6 | 1 |  |  |  |  |
| Other | 326 | 0.2 | 9.7 | 336 |  |  |
| Penicillins | 7,116 | 66 | 471 | 7,863 | 111 | 15,552 |
| Sulphonamides/ |  |  |  |  |  |  |
| Trimethoprim | 151 | 7 | 0 | 2,680 | 2,059 | 5,187 |
| Tetracyclines | 29 | 440 | 25 | 3,622 | 2.2 | 4,492 |
| Virginiamycin | 0 | 12 | 14 |  |  |  |
| **Total** | **8,561** | **21,070** | **1,082** | **19,533** | **2,189** | **55,809** |

The general population is exposed to a wide range and number of chemicals in all media and there is growing concern around recognition of the potential for a combined adverse effect when chemicals occur together. Assessing the combined risks to human health from exposure to chemical mixtures is much more complex than for single entities. The main challenges faced by regulators are how to determine the degree to which humans are co-exposed to chemicals, what interactions may occur among these, and what specific human health impacts are associated with the chemical mixtures. WHO (2017a) discusses these issues and includes case studies for pharmaceuticals, and oestrogens.

There are no generally accepted procedures for estimating the risk arising for simultaneous exposure to more than one chemical.

IARC (2012) evaluated the carcinogenic risks of several pharmaceuticals.

### Derivation of Maximum Acceptable Value

No MAV.

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# Phenanthrene

Phenanthrene, CAS No. 85-01-8, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. Also called phenanthracene. There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

The principal route of entry to source water is via atmospheric deposition. Based on the literature reviewed, the presence of chrysene and benzo(k)fluoranthene may be indicators for coal combustion emissions. Whereas other PAHs are indicators of other combustion process: benzo(g,h,i)perylene, coronene and phenanthrene are indicators for motor vehicle emissions, pyrene and fluoranthene are associated with incineration and fluorene, fluoranthene and pyrene are associated with oil combustion (Environment Australia 2003).

The most abundant classes of PAH found in diesel-contaminated estuarine sediments were naphthalenes, phenanthrenes, and dibenzothiophenes (DBT). Alkylated PAH made up 93 percent of the total PAH. The high proportions of naphthalenes, phenanthrenes, DBT and alkylated PAH are typical of refined petroleum hydrocarbons (US Department of the Interior 1998).

Phenanthrene is used in the manufacture of dyestuffs and explosives and in biological research.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

Phenanthrene is more stable than its linear isomer [anthracene](http://en.wikipedia.org/wiki/Anthracene). Water solubility is about 1.6 mg/L.

If released to soil, phenanthrene is expected to be immobile based upon Koc values of 9,180–25,300. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 4.23 x 10-5 atm‑cu m/mole. However, adsorption to soil is expected to attenuate volatilisation. Biodegradation of phenanthrene in soil is expected; the half-life of phenanthrene applied to soil at 2.1 ug/g soil and incubated at 15–25°C was 26 days. The half-life when applied at 25,000 ug/g soil and incubated at >25°C was 2.5 days. If released into water, phenanthrene is expected to adsorb to suspended solids and sediment based upon the Koc values. Phenanthrene has biodegradation half-lifes ranging from 19 days (SC water incubated at 27°C) to 11,000 days (RI water incubated at 2°C). Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 1.3 and 13 days, respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. The estimated volatilisation half-life from a model pond is 26–70 months if adsorption is considered. BCF values of 700 to 1,623 suggest bioconcentration in aquatic organisms is high to very high. Phenanthrene had direct aquatic photolysis half-lifes of 6.3 hours and 100 midday hours (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

Thirty-two water utilities in the US reported detecting phenanthrene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00048 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

### Analytical methods

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

IARC (2010) classified phenanthrene in Group 3 (not classifiable as to carcinogenicity).

### Derivation of Maximum Acceptable Value

No MAV.

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# Phenol

CAS No. 108-95-2. Also called carbolic acid, phenylic acid, phenic acid, phenyl alcohol, hydroxybenzene, and benzenol.

### Maximum Acceptable Value

Phenol does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Almost all phenol is used as an intermediate in the chemical industry, eg, manufacturing phenolic resins. Other major uses are in the production of bisphenol-A and caprolactum. Phenol is present in a number of household products.

Phenol can be present in the source water through natural breakdown of organic matter, but possibly also through anthropogenic discharges to catchments. The main environmental source is automobile exhaust (ie, direct emissions as well as photochemical degradation of benzene), from human and animal metabolism and from various other combustion processes. It has also been found from low-temperature carbonisation plants that use hard coal and brown coal, as well as from refineries, pulp manufacture and landfill leachate. Its diffuse nature means that it is extremely unlikely to result in environmental levels sufficient to trigger a specific taste and odour incident. It has been detected in rainwater.

Raw sewage concentrations of phenol in 28 municipal wastewater treatment plants in Ontario (Canada) ranged from 0.016 to 0.276 mg/L (24 hours composites; detected in 55 percent of 221 samples). The highest measured final effluent concentrations of phenol for 7 of those 28 plants ranged from 0.004 to 0.017 mg/L (detected in 22 percent of 55 samples). Animals eliminate phenol as a product of metabolism together with the urine and the faeces. Between 0.5 and 45 mg/L phenol were detected in the liquid manure (12 hours after excretion) produced by pigs. From EU (2006).

The data on the phenol content of the urine varies within the range of 4 to 55 mg/L. With the assumption that humans excrete about 1 to 1.5 litres of urine per day, phenol release rates into the municipal wastewater of 4 to 80 mg per day per person result. Phenol occurs in human sweat between 20 and 80 mg/L. Taken from EU (2006).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

IEH (2014) reports that phenolic taste and odour problems have been traced back to phenol leachates from gaskets and washers. Phenol is able to permeate polyethylene pipes.

### Forms and fate in the environment

In water, neither volatilisation nor sorption to sediments and suspended particulates are expected to be important transport mechanisms. The pKa of phenol is 10, indicating that phenol will exist primarily as the protonated acid at environmental pH values. In alkaline soils and water, phenol will partially exist as an anion, potentially affecting its fate and transport processes. Although it has been shown that plants readily uptake phenol, bioaccumulation does not take place due to a high rate of respiratory decomposition of phenol to CO2. A low Henry’s law constant (3.33 x 10-7 atm cu m/mol) and a vapour pressure of 0.35 mm Hg indicate that phenol is unlikely to be volatilised from wet or dry soil.

Phenol is readily biodegradable in natural water as long as the concentration is not high enough to cause significant inhibition through microbial toxicity.

In soil, phenol biodegrades under both aerobic and anaerobic conditions. The half-life of phenol in soil is generally <5 days (acidic soils and some surface soils may have half-lifes of up to 23 days). Mineralisation in an alkaline, para-brown soil under aerobic conditions was 45.5 percent, 48 percent, and 65 percent after 3, 7 and 70 days, respectively. Half-lifes for degradation of low concentrations of phenol in two silt loam soils were 2.70 and 3.51 hours. EU (2006) reports microbial degradation of phenol in estuarine water samples using phenol concentrations of 0.025 mg/L. Investigations were conducted in summer (24°C) and in winter (10°C). Half-lifes for the mineralisation of phenol were 7 days in summer and 73 days in winter. As the experiments were conducted in sunlight the rate constants are due to both biodegradation and photolysis. The authors showed that biodegradation was the primary removal process for phenol in both winter and summer.

Water solubility of phenol is about 83 g/L (8.3 percent). The partition coefficient = logPow = 1.47. Henry’s law constant = 0.022 Pa m3/mol at 20°C, so it is only slightly volatile from an aqueous solution.

In the UK there is a non-statutory environmental quality standard for surface water of 0.03 mg/L annual average concentrations and 0.30 mg/L maximum allowable concentrations (EU 2006).

### Typical concentrations in drinking-water

IEH (2014) reports that five of the 17 responding UK water companies reported no issues associated with taste and odour. For those companies reporting taste and odour problems (12/17), the majority were associated with biologically (algal) derived compounds (specifically gesomin and/or methylisoborneol, 7/12), or phenols (6/12) and chlorine (4/12).

### Removal methods

Phenol is not removed by conventional treatment. Chlorination produces chlorophenols. Ozone destroys phenol. Activated carbon reduces its concentration.

### Analytical methods

### Health considerations

Oral exposure to phenol at a dose of 14 mg/kg bw has been observed to induce GI (gastrointestinal) effects in humans. Studies of incidents suggest that concentrations of phenol in source water below taste and odour thresholds may not be free from symptoms of gastrointestinal disturbance although the relative importance of phenol and disinfection by-products formed by chlorination during water treatment are uncertain.

Rats given phenol by oral gavage at 4, 12, 40, and 120 mg/kg/day for 14 days showed degeneration of the kidneys, liver or spleen at 12 mg/kg/day and above; a NOAEL of 4 mg/kg/day was established.

The European Food Standards Agency (EFSA) established in 2013 an oral TDI of 0.5 mg/kg/day for phenol.

The International Agency for Research on Cancer (IARC), the USEPA and the US National Toxicology Program (NTP) have determined that phenol is not classifiable as a human carcinogen.

### Derivation of Maximum Acceptable Value

No MAV.

Phenol has a disagreeable sweet and tarry odour. The USEPA established an organoleptic effect criterion of 0.3 mg/L for phenol. Source: [*Quality Criteria for Water* 1986 (“Gold Book”)](Quality%20Criteria%20for%20Water%201986%20(), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

EU (2006) quotes a threshold for taste and odour in water of 0.15 mg/L; as a result Federal Environmental Agency of Germany recommends an aesthetic guide value (*ästhetischer Leitwert*) of 0.001 mg phenol per litre of drinking water in order to guarantee the option to chlorinate water if necessary without deteriorating its aesthetic quality with respect to taste and odour. In Denmark a limit value for drinking water of 0.0005 mg/L of phenol and other phenols has been set to protect against taste from chlorinated phenols generated by chlorination of the water.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. The chronic health risk limit is 4 mg/L.

### References

DWI. 2014. *National Assessment of the Risks to Water Supplies Posed by Low Taste and Odour Threshold Compounds*. Final Report Project WT1275. 202 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-281.pdf>.

EFSA. 2013. Scientific opinion on the toxicological evaluation of phenol. *EFSA Journal* 11(4): 3189. 44 pp. Available at: <http://www.efsa.europa.eu/en/efsajournal/doc/3189.pdf>.

EU. 2006. *European Union Risk Assessment Report*. Phenol. Revised Edition. 240 pp. <http://echa.europa.eu/documents/10162/1ca68f98-878f-4ef6-914a-9f21e9ad2234> or <http://www.baua.de/de/Chemikaliengesetz-Biozidverfahren/Dokumente/RAR_060.pdf?__blob=publicationFile&v=2> or <http://echa.europa.eu/information-on-chemicals/information-from-existing-substances-regulation>.

IEH. 2014. *National Assessment of the Risks to Water Supplies Posed by Low Taste and Odour Threshold Compounds*. Final Report Project WT1275. Institute of Environment and Health, Cranfield University. 202 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-281.pdf>.

MDH. 2016. *Human Health-Based Water Guidance Table*. Minnesota Department of Health. <http://www.health.state.mn.us/divs/eh/risk/guidance/gw/table.html>.

# 2-phenoxyethanol

CAS No. 122-99-6. Also called phenoxyethanol, ethylene glycol monophenyl ether, 1‑hydroxy-2-phenoxyethane, (2-hydroxyethoxy)benzene, EGPhE and phenyl cellosolve.

### Maximum Acceptable Value

2-Phenoxyethanol does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

2-Phenoxyethanol is a chemical preservative, a [glycol ether](http://en.wikipedia.org/wiki/Glycol_ether) often used in [dermatological](http://en.wikipedia.org/wiki/Dermatological) [products](http://en.wikipedia.org/wiki/Product_(business)) such as skin creams and sunscreen. It is a [bactericide](http://en.wikipedia.org/wiki/Bactericide) (usually used in conjunction with [quaternary ammonium](http://en.wikipedia.org/wiki/Quaternary_ammonium) compounds), often used in place of [sodium azide](http://en.wikipedia.org/wiki/Sodium_azide) in biological [buffers](http://en.wikipedia.org/wiki/Buffer_solution) because phenoxyethanol is less [toxic](http://en.wikipedia.org/wiki/Toxic) and non-reactive with [copper](http://en.wikipedia.org/wiki/Copper) and [lead](http://en.wikipedia.org/wiki/Lead). It is used in many applications such as cleaning products, cosmetics (up to 1 percent), vaccines and pharmaceuticals as a [preservative](http://en.wikipedia.org/wiki/Preservative). It is also used as a [fixative](http://en.wikipedia.org/wiki/Fixative_(perfumery)) for [perfumes](http://en.wikipedia.org/wiki/Perfume), an [insect repellent](http://en.wikipedia.org/wiki/Insect_repellent), a topical [antiseptic](http://en.wikipedia.org/wiki/Antiseptic), a [solvent](http://en.wikipedia.org/wiki/Solvent) for [cellulose acetate](http://en.wikipedia.org/wiki/Cellulose_acetate), some [dyes](http://en.wikipedia.org/wiki/Dye), [inks](http://en.wikipedia.org/wiki/Ink), and [resins](http://en.wikipedia.org/wiki/Resins), in [preservatives](http://en.wikipedia.org/wiki/Preservative), [pharmaceuticals](http://en.wikipedia.org/wiki/Pharmaceutical), and in organic synthesis. Phenoxyethanol is an alternative to potentially harmful [formaldehyde-releasing preservatives](http://en.wikipedia.org/wiki/Formaldehyde_releaser). It is used in the synthesis of many plasticisers, germicides and pharmaceuticals.

As at Dec 2013 2-phenoxyethanol is registered as an antifungal in New Zealand; see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm).

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

If released to soil, 2-phenoxyethanol is expected to have very high mobility based upon an estimated Koc of 15. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 4.9 x 10-8 atm‑cu m/mole. The predicted half-life in water and soil is about 360 hours , and 60 days in sediment. If released into water, 2-phenoxyethanol is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is not expected to be an important fate process based upon this compound’s estimated Henry’s Law constant. Phenoxyethanol is not expected to undergo hydrolysis in the environment due to the lack of functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

UNEP (2004) stated that this chemical is currently of low priority for further work because of its low hazard profile.

Water solubility is about 2.8 percent.

### Health considerations

UNEP (2004) reported that EGPhE tested negative for mutagenicity. EGPhE also tested negative in an *in vivo* cytogenicity study in the rat and a mouse micronucleus test. Other *in vitro* chromosomal aberrations and gene mutation assays were also negative. A two-generation continuous breeding, oral feeding study in CD-1 mice with EGPhE resulted in NOAELs of 400 mg/kg bw/day for both parental animals and offspring. NICNAS (2013) reports a later study giving a NOAEL of 400 mg/kg/d.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Dow Chemical Co. 2007. *Product Safety Assessment: Ethylene glycol phenyl ether*. 6 pp. <http://www.dow.com/productsafety/assess/finder.htm>.

EAWAG. Accessed February 2015. *Biocatalysis/Biodegradation Database: Select 1396 compounds*. <http://eawag-bbd.ethz.ch/index.html>.

NICNAS. 2013. *Human Health Tier II Assessment for Ethanol, 2-Phenoxy-*. Inventory Multi-tiered Assessment and Prioritisation (IMAP). <http://www.nicnas.gov.au/>.

UNEP. 2004. Ethylene Glycol Phenyl Ether. *SIDS Initial Assessment Report*. OECD SIDS. 151 pp. <http://www.chem.unep.ch/irptc/sids/oecdsids/122996.pdf>.

# Phosphonates

Phosphonic acids are organic compounds of the form R-PO3H2. Phosphonates comprise a very large group of chemicals incorporating a wide range of uses, as the acid or sodium or other salt. Some commoner or more important members are:

* CAS No. 6419-19-8: amino tris(methylenephosphonic acid) or [ATMP](http://en.wikipedia.org/wiki/ATMP)
* CAS No. 15827-60-8: diethylenetriamine penta(methylene phosphonic acid) or [DTPMP](http://en.wikipedia.org/wiki/DTPMP)
* CAS No.1429-50-1: ethylenediamine tetra(methylene phosphonic acid) or EDTMP
* CAS No. 56744-47-9: hexamethylenediamine tetra(methylene phosphonic acid) or HDTMP
* CAS No. 2809-21-4: 1-hydroxyethylidene-1,1-diphosphonic acid or [HEDP](http://en.wikipedia.org/wiki/Etidronic_acid)

The pesticide glyphosate and its degradation product [aminomethylphosphonic acid](http://en.wikipedia.org/wiki/Aminomethylphosphonic_acid) (AMPA) are also phosphonates, as is ethephon – see pesticide datasheets.

### Maximum Acceptable Value

Phosphonates do not have a MAV in the DWSNZ, and are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Some of the simpler phosphonates occur naturally in some plants and animals, eg, 2‑aminoethylphosphonic acid.

DWI (2014) identified phosphonates as chemicals likely to be a risk to drinking water from personal care products and domestic cleaning products.

Phosphonates are employed as chemical additives to function as threshold antiscalants, corrosion inhibitors, chelants, sludge conditioners, deflocculants, dispersants and crystal growth modifiers in various industrial water treatment processes. They are used predominantly as scale and corrosion preventatives for boiler and cooling tower waters. Phosphonates exist in various formulations as acids or salts and are marketed in the form of concentrated solutions.

In 1998 the consumption of phosphonates was 56,000 tons worldwide. The commonest phosphonates are used a chelating agents in laundry and dishwashing detergents, cleaning agents, cosmetics and pharmaceuticals (up to 2 percent in detergents and cleaning agents). Phosphonates are also used as stabilisers for hydrogen peroxide solutions and formulations.

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

Phosphonates do not significantly degrade in wastewater treatment plants, but are removed from the sewage by adsorption to sludge.

Phosphonates are present mainly as Ca and Mg-complexes in natural waters and therefore do not affect metal speciation or transport. Some phosphonates are adsorbed to particulate matter. Sediment/water adsorption coefficients in the range 250–3,900 were observed at concentration of 0.05 and 0.1 mg/L. At these levels, the concentration of the phosphonates in the water was reduced by two to three orders of magnitude. Although varying somewhat by the structure of the molecules, adsorption is consistently much higher than what can be expected for highly water soluble, low Log Kow chemicals. In addition, adsorption is more pronounced at lower concentrations (HERA 2004).

Research showed that in non-sterile natural water, the ultimate biodegradation of phosphonates ranged from 2.0 to 12.3 percent in the dark, and 13.6 to 17.2 percent in sunlight after 60 days indicating some degradation enhancement by sunlight, at a concentration of 2 mg/L. The corresponding half-life is 395 days. These data show that the phosphonates are slowly degrading under simulated environmental conditions, and that there are several degradation mechanisms, including biodegradation and photodegradation. From HERA (2004).

The phosphonates discussed in this datasheet are highly soluble in water, generally >60 percent.

### Health considerations

The phosphonic acid compounds ATMP, HEDP, DTPMP and their salts can be considered to be of low to moderate acute oral toxicity. ATMP acid was of moderate acute toxicity to mammals. The acute oral LD50 in the rat was determined to be 2,910 mg active acid/kg bw. In comparison, the tetrasodium and pentasodium salt of ATMP were less acutely toxic with LD50 values of 8,610 and 7,120 mg active salt/kg bw, respectively. HEDP acid and its salts are of moderate acute oral toxicity LD50’s in rats and mice ranging from 1,100 to 1878 mg active acid/kg bw. The oral LD50 values of HEDP salts were in a slightly wider range from 581 mg active salt/kg bw to greater than 5,000 mg active salt/kg. DTPMP acid and salts are of low toxicity with oral LD50 values from 3,870 mg active salt/kg bw to less than 8,757 mg active salt/kg bw.

The NOAEL of ATMP acid was established to be 500 mg/kg bw/d on the basis of a two-year rat feeding study. HEDP was investigated in a good quality two-year feeding study. On the basis of this study the NOAEL for HEDP was established to be 19 mg/kg bw/d. In a reliable high quality 90-day oral feeding study the NOAEL for DTPMP was established to be 83–92.3 mg/kg bw/d (HERA 2004).

A substantial amount of toxicological data and information *in vivo* and *in vitro* demonstrates that there is no evidence for ATMP, HEDP and DTPMP being genotoxic, mutagenic or carcinogenic. There is some conflicting data with regard to the mutagenicity of DTPMP, but the overall weight of the evidence suggests it is not mutagenic. There is also no evidence of reproductive toxicity or developmental effects in animals. The long-term toxicity of the acid or salt forms of the phosphonates under review was evaluated in several subacute, subchronic and chronic toxicity studies. In the available chronic and subchronic oral toxicity studies, no adverse effects for ATMP, HEDP and DTPMP were observed at dose level of 500, 24 and 20 mg/kg/day respectively (HERA 2004).

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2014. *Risks to Drinking Water from Personal Care Products and Domestic Cleaning Products*. WRc Ref: DWI9879.03. Report No. DWI9879.03. 121 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-283.pdf>.

HERA. 2004. *Human and Environmental Risk Assessment on Ingredients of European Household Cleaning Products: Phosphonates*. 114 pp. <http://www.heraproject.com/files/30-F-04-%20HERA%20Phosphonates%20Full%20web%20wd.pdf>.

# Phthalates

Phthalates (ie, [esters](http://en.wikipedia.org/wiki/Ester) of phthalic acid) are the di[alkyl](http://en.wikipedia.org/wiki/Alkyl) or alkyl [aryl](http://en.wikipedia.org/wiki/Aryl) esters of [phthalic acid](http://en.wikipedia.org/wiki/Phthalic_acid) (which is also called 1,2-benzenedicarboxylic acid). This datasheet covers some of the commoner phthalates. **Note:** di(2-ethylhexyl)phthalate (CAS No. 117-81-7) has a separate datasheet because it is the only phthalate with a MAV.

* butyl benzyl phthalate (BBP) CAS No. 85-68-7 (previously 58128-78-2)
* dibutyl phthalate (DBP) CAS No. 84-74-2
* diethyl phthalate (DEP) CAS No. 84-66-2
* dimethyl phthalate (DMP) CAS No. 131-11-3
* di-n-octyl phthalate (DnOP) CAS No. 117-84-0.

Some minor phthalates include:

* di-isobutyl phthalate (DIBP) CAS No. 84-69-5
* di-isodecyl phthalate (DIDP) CAS No. 26761-40-0 and 68515-49-1
* di-isononyl phthalate (DINP) CAS No. 28553-12-0 and 68515-48-0
* di-n-hexyl phthalate (DnHP) CAS No. 84-75-3
* di(methoxyethyl) phthalate (DMEP) CAS No. 117-82-8.

OECD (2018) includes a larger list in their Annex 1, where some newer products have a molecular weight above 500.

### Some synonyms

* **Butyl benzyl phthalate:** benzyl n-butyl phthalate; 1,2-benzenedicarboxylic acid; BBP
* **Dibutyl phthalate:** di-n-butyl phthalate; butyl phthalate; 1,2-dibutyl 1,2‑benzenedicarboxylate; DBP
* **Diethyl phthalate:** diethyl o-phthalate; DEP
* **Dimethyl phthalate:** dimethyl 1,2-benzenedicarboxylate; dimethylbenzene-1,2‑dicarboxylate; DMP
* **Di-n-octyl phthalate:** dioctyl phthalate [this is also a synonym for di(2‑ethylhexyl)phthalate]; DNOP
* **Di(methoxyethyl) phthalate:** 1,2-benzenedicarboxylic acid, bis(2-methoxyethyl) ester; DMEP
* **Di-isodecyl phthalate:** 1,2-benzenedicarboxylic acid, diisodecyl ester; DIDP
* **Di-n-octyl phthalate:** 1,2-benzenedicarboxylic acid, dioctyl ester; dnop
* **Di-n-hexyl phthalate:** dihexyl phthalate; 1,2-benzenedicarboxylic acid, dihexyl ester; DHP; dnhp

### Maximum Acceptable Value

Other than di(2-ethylhexyl)phthalate, no other phthalate has a MAV in the DWSNZ, or a Guideline Value in the WHO Guidelines (2011).

As well as bis(2-ethylhexyl) phthalate, butyl benzyl phthalate, di-n-butyl phthalate, diethyl phthalate, dimethyl phthalate, and di-n-octyl phthalate are USEPA Priority Pollutants (see <http://water.epa.gov/scitech/methods/cwa/pollutants.cfm>).

### Sources to drinking-water

#### 1. To source waters

World consumption of phthalates in the early 1990s was estimated to be 3.25 million tonnes, of which di(2-ethylhexyl)phthalate (the dominant PVC plasticiser due to cost) accounted for approximately 2.1 million tonnes. They have a very wide range of uses (see phthalate in Wikipedia; see also NICNAS (2008 and others) and OECD (2018). They are all synthetic, ie, do not occur naturally.

The main use of phthalates is as a plasticiser, with flexible PVC accounting for over 80 percent of world plasticiser consumption. Phthalates were first introduced in the 1920s. Production increased significantly with the introduction of PVC in the 1930s. Unplasticised PVC (uPVC, commonly used in buildings for windows and doors) is a rigid material: the addition of the plasticiser makes the PVC flexible and usable in many other applications including medical equipment. Phthalates can contribute as much as 50 percent of the weight of PVC materials. Other uses include applications as diverse as viscosity control agents, solvents, glues and personal care products. In 2005 phthalates accounted for 88 percent of the plasticiser market, a figure that had fallen to 70 percent by 2014 and is forecast to fall further. There is a trend in the market towards the higher molecular weight phthalates shown in Annex 1, as these are less mobile and hence less likely to disperse out of products and into the environment. In addition, they have a reduced hazard profile in comparison to medium-chain phthalates (carbon backbone length 3 to 7) which have been the focus of regulatory activities (OECD 2018).

The principal phthalates used in cosmetic products are dibutyl phthalate, dimethyl phthalate, and diethyl phthalate. They are used primarily at concentrations of less than 10 percent (although NICAS 2011 states that diethyl phthalate may comprise 10 to 25 percent) as plasticisers in products such as nail polishes (to reduce cracking by making them less brittle), and hair sprays (to help avoid stiffness by allowing them to form a flexible film on the hair), and as solvents and perfume fixatives in various other products.

**Butyl benzyl phthalate** (BBP) is a plasticiser used extensively in vinyl flooring and other flexible polyvinyl chloride (PVC) uses such as food packaging. In children’s toys and childcare articles made from polyvinyl chloride (PVC), BBP is unlikely to be found as the dominant (primary) phthalate plasticiser, as its molecular weight is similar to that of dibutyl phthalate (DBP), a commonly used secondary plasticiser. Therefore, the chemical might be used as a secondary plasticiser (in conjunction with another plasticiser) or occur as a minor contaminant of other phthalates, including diethylhexyl phthalate (DEHP) or diisononyl phthalate (DINP). In the absence of data on the use of BBP in children’s toys, assumptions need to be made in modelling exposures that BBP completely substitutes for DBP in a mixed phthalate plasticiser, at a maximum concentration of 0.5 percent w/w, with a total plasticiser concentration (DINP+BBP) in the PVC of 43 percent. Cosmetic uses of BBP were not reported in Australia. BBP has been reported to have a number of industrial uses, including in the manufacture of adhesives, sealants, coatings, paints and inks. It also serves as a specialty plasticiser in vinyl and acrylic lacquers, nitrocellulose lacquers and polyurethane wheels for forklifts (NICNAS 2015a). EU (2007a) reports that the concentration of BBP in European raw sewage is generally <0.005 mg/L, and generally < 0.002 mg/L in freshwaters.

**Dibutyl phthalate (DBP)** is a commonly used [plasticiser](http://en.wikipedia.org/wiki/Plasticizer) such as resins and PVC. It is used as a softener in paper, an additive to [adhesives](http://en.wikipedia.org/wiki/Adhesive) and printing inks. It is also used as an [ectoparasiticide](http://en.wikipedia.org/wiki/Ectoparasiticide) and insect repellent, but not registered as such in New Zealand. NICNAS (2013) states that DBP is used as a plasticiser in resins and polymers. It is also used as a softener in adhesives, lacquers, varnishes and printing inks. DBP is used in cosmetics as a perfume solvent and fixative; a suspension agent for solids in aerosols; a lubricant for aerosol valves; an anti-foamer; a skin emollient and plasticiser in nail polish (up to 7 percent), and fingernail elongators (extensions). EU (2003) reports concentrations found in European freshwaters, the highest concentration being 0.034 mg/L and most <0.005 mg/L, with a mean of about 0.001 mg/L.

**Diethyl phthalate (DEP)** is used primarily as a solvent and/or vehicle for fragrance in perfumes, cosmetics, personal care products, and nail polishes; an alcohol denaturant in toiletries, detergents and insecticides; and a plasticiser in plastic tools, automotive parts, toothbrushes, food packaging, medical tubing, soft plastic toys and child care articles (NICNAS 2011).

**Di-isodecyl phthalate (DIDP)** is a complex mixture containing mainly C10-branched isomers; eg, the reconstituted chromatogram for a DIDP sample extracted from water shows about 29 different peaks. The main constituents at 70–80 percent content are dimethyl octanols (EU 2003a). DIDP is used industrially as a plasticiser for PVC products such as automotive parts, hoses, gaskets, cable and wire coatings with a maximum concentration of 40 percent (possibly in combination with other phthalates). Internationally, DIDP-plasticised PVC is used in film, sheet and coated products, flooring, roofing and wall coverings, and car undercoating and sealants. Extrusion and injection moulding processes are used to incorporate it in PVC for hoses, wires and cables, footwear and miscellaneous articles. Non-PVC uses include in polymers such as pressure-sensitive adhesives, printing inks, surfactants, anti-corrosion and anti-fouling paints (NICNAS 2015).

**Di-isononyl phthalate (DINP)** is not a pure substance, but a complex mixture containing mainly C9-branched isomers; eg, the reconstituted chromatogram for a DIDP sample extracted from water shows about 29 different peaks. The main constituents are dimethyl heptanols (40–55 percent) and methyl octanols  
(5–40 percent). Up to 95 percent of its use is in PVC applications. EU (2003b). DINP is used in a diverse range of industrial products such as electrical wire and cables, flexible PVC sheeting, coated fabrics, automotive parts, building and construction (waterproofing), vinyl flooring, footwear, sealings, lamination film and in PVC‑containing school supplies (such as scented erasers and pencil case). In imported PVC toys, DINP may be present at a concentration range estimated to be between 0.005 percent and 35 percent, and 40 to 50 percent in some polymers (NICNAS 2012; 2014).

**Di(methoxyethyl) phthalate (DMEP):** NICNAS (2014a) indicates that DMEP may be used as a plasticiser for toys, including inflatable water products, hoppers, play and exercise balls, at a concentration of up to 40 percent (possibly in combination with other phthalates). Based on its physicochemical properties, in a mixed phthalate plasticiser DMEP is assumed to be used at a maximum concentration of 0.5 percent. DMEP has been reported to be used as a plasticiser in the production of nitrocellulose, acetyl cellulose, polyvinyl acetate (PVA), PVC and polyvinylidene chloride intended for contact with food or drink. DMEP is also used as a solvent and in pesticide products. No cosmetic uses were reported.

**Dimethyl phthalate (DMP):** as at 2012 dimethyl phthalate is registered as an antimicrobial in New Zealand; see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm). Dimethyl phthalate is the active ingredient in many insect repellents such as Dimp. Dimethyl phthalate is a plasticiser, and is used as a fragrance ingredient in cosmetics and personal care products. DMP is used as a solvent in the mining industry for mineral recovery, in the manufacture of adhesives, fibreglass, automotive plastics, surface coatings, and in various textile wet processing products, with a small proportion in children’s toys. Concentrations of DMP in domestic detergents, cosmetics, perfumes and personal care products are highly variable and range from 0.00004 percent to 34 percent (in combination with its analogue diethyl phthalate, DEP). In PVC, children’s toys and childcare articles, DMP, a low molecular weight phthalate, is not found as the dominant phthalate plasticiser, but may be used in conjunction with another plasticiser as a secondary plasticiser or occur as a minor contaminant of other phthalates, including diethylhexyl phthalate (DEHP) or di‑isononyl phthalate (DINP).

**Di-n-octyl phthalate (DnOP)** is manufactured in the US, predominantly as a minor component of C6–10 phthalate (approximately 20 percent). Contradictorily, there are reports that there are no known commercial uses of pure DnOP; however, DnOP makes up 20 percent of the component of the commercial phthalate mixture of C6–C10 phthalate. Di-n-octyl phthalate is used to keep plastics soft or more flexible (a plasticiser). DnOP is used in this form in PVC for manufacturing a variety of products including medical tubing and blood storage bags, flooring and carpet tiles, canvas tarpaulins, swimming pool liners, notebook covers, traffic cones, toys and dolls, vinyl furniture upholstery, shower curtains and gloves, wire and cables, garden hoses, leather stripping, cosmetics, flea collars and shoes. DnOP-containing PVC is also used in food applications. Non-PVC applications of DnOP include its use as a dye carrier in plastics production, and for manufacturing adhesives, plastisols and nitrocellulose lacquer coatings (NICNAS 2015).

Phthalate plasticisers are not chemically bound to PVC, so they can migrate, leach and evaporate into water, food or the atmosphere. As plastics age and break down, the release of phthalates accelerates.

The European Union has temporarily banned the use of six phthalates in toys and other articles intended for children aged under three years of age and designed to be put in the mouth. Several countries in Europe also have proposed, or are considering, restrictions on use of phthalates as plasticisers in PVC toys and baby care items.

ECHA (2016) proposed restriction of use of bis(2-ethylhexyl) phthalate (DEHP), benzyl butyl phthalate (BBP), dibutyl phthalate (DBP) and diisobutyl phthalate (DIBP). The proposal seeks to restrict the placing on the market of the following articles containing the four phthalates in a concentration, individually or in combination, above 0.1 percent of the plasticised material by weight:

1. any (indoor or outdoor) articles whose phthalate-containing material may be mouthed or is in prolonged contact with human skin or any contact with mucous membranes, and

2. any phthalate-containing articles that are used (including stored) in an indoor environment where people are present under normal and reasonably foreseeable conditions and potentially exposed via inhalation. This does not apply to articles that are used only in industrial or agricultural workplaces by workers.

#### 2. From the distribution system

It has been estimated that approximately 1 percent of the phthalate ester content of plastic materials in direct contact with water or other liquids may be released (WHO 2003).

### Forms and fate in the environment

**Butyl benzyl phthalate (BBP)** is not persistent in water, sediments, or soil under aerobic conditions, with a half-life of 1 to 7 days. Under anaerobic conditions, BBP is more persistent, with a half-life of a few months. Partition co-efficient octanol/water (log Kow) = 4.8. Henry’s Law constant, atm m3/mol (25°C) = 1.28 x 10-4. EU (2007a) reports a vapour pressure value of 0.00112 Pa (which they say is low), and a water solubility of 2.8 mg/L. Hydrolysis and photolysis are not important processes. Biodegradation is quite rapid, with aerobic half-lifes quoted at <3 days and anaerobic >10 days. The high log Kow and relatively low water solubility of BBP indicates a relatively low mobility in soil. However, binding of BBP to colloidal matter and humic substances may enhance subsurface transport through cracks and macropores in soils.

**Dibutyl phthalate (DBP)** is not expected to volatilise significantly from water to the atmosphere. In soils, migration to groundwater occurs, but is thought to be limited to sites with low organic content. In water and soils, >50 percent of di-n-butyl phthalate is degraded within 1 to 28 days. In a river die-away test using water from three rivers in the Netherlands, it is reported that 90 percent of the initial 0.05 mg/L of di-n-butyl phthalate was removed in three days, while only 10 percent was lost in the sterile controls. If released to soil, dibutyl phthalate is expected to have low mobility based upon log Koc values of 3.05 to 3.14. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 1.81 x 10-6 atm‑cu m/mole. In Davidson clay loam and Lakeland sand, 98 and 66 percent loss occurred in 26 weeks, respectively as a result of biodegradation. Carboxyl-labelled (14)C dibutyl phthalate studied in soil incubation experiments conducted under laboratory conditions had a lag phase of 10 to 20 days, approximately 90 percent of dibutyl phthalate added to soils at rates of 0.1 to 0.4 percent was decomposed within 80 days under aerobic and anaerobic conditions. If released into water, dibutyl phthalate is expected to adsorb to suspended solids and sediment based upon the Koc values. In natural waters, the biodegradation half-life of dibutyl phthalate is estimated as 1 day and anaerobic biodegradation half-life of 2 days. Dibutyl phthalate had an average aerobic and anaerobic biodegradation half-lifes of 2.9 and 14.4 days, respectively, calculated in six river sediment samples taken from Taiwan rivers. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 34 and 250 days, respectively. BCFs of 12, 167 and 172 in fathead minnows, 3.6 in Cyprinus carpio (carp) and 117 in bluegill fish, suggest bioconcentration in aquatic organisms is low to high. However, bioconcentration studies on compounds which are structurally similar suggest that bioconcentration may be lower than that indicated by the regression-derived equations due to the ability of aquatic organisms to metabolise readily this class of compounds. Dibutyl phthalate was found to have a hydrolysis degradation half-life of 22 years at pH 7 and 25°C (EAWAG accessed February 2015). EU (2003) quotes water solubility = 10 mg/L at 20°C, vapour pressure = 9.7 x 10-5 hPa, partition coefficient n-octanol/water = logKow = 4.57 which is high and consequently the equilibrium between water and organic carbon in soil or sediment will be very much in favour of the soil or sediment.

**Diethyl phthalate (DEP)** is likely to undergo biodegradation in the environment. Compared with other phthalates, it has a much lower capacity for binding to aquatic sediments, with between 70 percent and 90 percent of diethyl phthalate estimated to be found in the water column. Diethyl phthalate can be biodegraded either aerobically or anaerobically; abiotic degradation processes are not significant. Diethyl phthalate may leach from soils with low organic matter content into the underlying groundwater (WHO 2003).

**Di-isodecyl phthalate (DIDP):** Partition co-efficient octanol/water (log Kow) = 8.8. Henry’s Law constant, atm m3/mol (25°C) = 1.12 x 10-6. EU (2003a) quotes: vapour pressure = 5.1 x 10-5 Pa at 25°C; water solubility = 0.0002 mg/L; Henry’s law constant = 114 Pa.m3/mol. In Rhine river water di-n-decyl phthalate was totally degraded after 7 days. The initial concentration was 1.1 μg/L related to the test substance. The DT50 was <1 day and the DT90 was <3 days. The test was performed at 25°C. Only the disappearance of the parent compound was determined and the degradation products were unknown.

**Di-isononyl phthalate (DINP):** EU (2003b) quotes: vapour pressure = 6 x 10-5 Pa at 20°C; water solubility = 0.0006 mg/L; Henry’s law constant = 41.4 Pa.m3/mol; partition co-efficient octanol/water (log Kow) = 8.8. In Rhine river water DINP was totally degraded after seven days. The initial concentration was 0.2 μg/L related to the test substance. The DT50 was <1 day and the DT90 was <3 days. The test was performed at 25°C. Only the disappearance of the parent compound was determined and the degradation products were unknown.

**Di(methoxyethyl) phthalate (DMEP):** Partition co-efficient octanol/water (log Kow) = 1.11. Henry’s Law constant, atm m3/mol (25°C) = 2.81 x 10-13.

**Dimethyl phthalate (DMP):** Partition co-efficient octanol/water (log Kow) = 1.60. Henry’s Law constant, atm m3/mol (25°C) = 1.97 x 10-13.

**Di-n-octylphthalate (DnOP)** can be released to water or air during its manufacture, by leaking from plastics in landfills, or from the burning of plastic products. It adheres tightly to soil, sediment, and dust particles. Biodegradation half-lifes of 1 to 4 weeks have been estimated for aerobic surface waters and soils. Partition co-efficient octanol/water (log Kow) = 5.22. Henry’s Law constant, atm m3/mol (25°C) = 6.68 x 10-5.

Water solubilities:

* butyl benzyl phthalate 2.7 mg/L
* dibutyl phthalate 12 mg/L
* diethyl phthalate 1,000 mg/L
* di(methoxyethyl) phthalate 8,500 mg/L
* dimethyl phthalate 4,300 mg/L
* di-n-octyl phthalate 3 mg/L
* di-isobutyl phthalate 1 mg/L
* di-isodecyl phthalate <0.001 mg/L
* di-isononyl phthalate <0.001 mg/L
* di-n-hexyl phthalate 0.05 mg/L

### Typical concentrations in drinking-water

Butyl benzyl phthalate: 19 water utilities in the US reported detecting butyl benzyl phthalate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.008 mg/L.

Di-n-butyl phthalate: 187 water utilities in the US reported detecting di-n-butyl phthalate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.12 mg/L.

Diethyl phthalate: 52 water utilities in the US reported detecting diethyl phthalate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.06 mg/L.

Dimethyl phthalate: three water utilities in the US reported detecting dimethyl phthalate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.009 mg/L.

Di-n-octyl phthalate: four water utilities in the US reported detecting di-n-octyl phthalate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0006 mg/L.

### Removal methods

The strong soil adsorption of the higher molecular weight members (and low water solubility) suggests that treatment processes that remove particulate matter should be effective at reducing the concentration of those phthalates in water. This does not apply to diethyl and dimethyl phthalates.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

Because phthalates are so pervasive in plastics and in the laboratory environment, rigorous control measures are needed to prevent contamination of the sample and to maintain a low background concentration (WHO 2003).

### Health considerations

Diet is believed to be the main source of phthalates in the general population; fatty foods such as milk, butter, and meats are a major source. Most people have metabolites of multiple phthalates in their urine. Generally, more phthalates are taken in from the air than from drinking water. OECD (2018) includes discussion of the various health effects.

**Butyl benzyl phthalate (BBP):** WHO (1999) states that the tolerable intake (TDI) of 1.3 mg/kg body weight was based upon the lower 95 percent confidence limit for the benchmark dose associated with a 5 percent increase in the incidence of pancreatic lesions in male rats in an oral subchronic bioassay divided by an uncertainty factor of 100 (10 for interspecies variation and 10 for intraspecies variation). The NTP concludes that there is minimal concern for developmental effects in foetuses and children. The NTP concurs with the CERHR Phthalates Expert Panel that there is negligible concern for adverse reproductive effects in exposed men (NIH 2003a). A NOAEL of 50 mg/kg/d based on reproductive effects was reported in EU (2007). Butyl benzyl phthalate is not classifiable as to its carcinogenicity to humans (Group 3); IARC. 1999. BBP is rapidly and almost completely absorbed following oral administration. The bioavailability of BBP from oral exposure is assessed as 100 percent for both adults and children. BBP is rapidly metabolised and excreted in the urine, predominantly as metabolites, particularly monobenzyl phthalate (MBzP) and monobutyl phthalate (MBP). Based on the weight of evidence, the available data do not support a mutagenic, genotoxic or carcinogenic potential for BBP in humans. For systemic effects, the no observed adverse effect level (NOAEL) of 151 mg/kg bw/day, derived from a 90-day oral study and based on histopathological changes in the pancreas and gross pathological changes in the liver of Wistar rats, is considered most appropriate for risk characterisation. For fertility-related and developmental effects, the highest NOAEL of 50 mg/kg bw/day is derived from the collective results of the three multi-generation studies, based on reduced birth weight in both sexes at 100 mg/kg bw/day. Butyl benzyl phthalate is listed in the EC Endocrine Disruptors priority list with Category 1 classification (ie, evidence of endocrine disrupting activity in at least one species using intact animals) (NICNAS 2015a).

**Dibutyl phthalate (DBP):** the NTP concurs with the CERHR Phthalates Expert Panel that there is minimal concern for developmental effects when pregnant women are exposed to DBP levels estimated by the Panel (2–10 μg/kg bw/day). Based upon recent estimated DBP exposures among some women of reproductive age, the NTP has some concern for DBP causing adverse effects to human development, particularly development of the male reproductive system. The NTP also concurs with the CERHR Phthalates Expert Panel that there is negligible concern for reproductive toxicity in exposed adults (NIH 2003b). A NOAEL of 50 mg/kg/d based on reproductive effects was reported in EU (2007). See also datasheet for Endocrine Disrupting Compounds; DWI (2012) found that further studies on the endocrine disrupting effects of dibutyl phthalate were justified. NICNAS (2013) states that DBP has been reported to be non-genotoxic in most *in vitro* and all *in vivo* animal tests performed to standard testing guidelines. Based on the information available for genotoxicity, DBP is not likely to be a genotoxic carcinogen. However, the NICNAS assessment found that DBP alone and/or with the simultaneous use of multiple cosmetic products containing DBP by children and the general population can result in high risk of reproductive toxicity. Therefore, a recommendation to restrict the use of DBP in cosmetics is warranted.

**Diethyl phthalate (DEP):** WHO (2003) states that a tolerable intake (TDI) of 5 mg/kg body weight was estimated from a NOAEL of 1,600 mg/kg body weight per day for developmental effects to which an uncertainty factor of 300 was applied. Available data do not support a genotoxic or carcinogenic potential for DEP and DEP does not appear to be a potent testicular toxin in animal studies. However, DEP alters certain fertility-related parameters and induces developmental effects in newborn rodent pups. While human studies are limited, the adverse effects on fertility parameters and development are considered relevant to humans, where the exposure level of DEP is high and within a critical window of development (NICNAS 2011).

**Di-isodecyl phthalate (DIDP):** the NTP concurs with the CERHR Phthalates Expert Panel that there is minimal concern for developmental effects in fetuses and children. The NTP also concurs with the CERHR Expert Panel that there is negligible concern for reproductive toxicity in exposed adults. These conclusions are based on the assumption that the general US population is exposed to DIDP at less than 30 μg/kg bw/day (NIH 2003d). DIDP absorption through the gastrointestinal tract is incomplete. As the saturation point is reached, the absorption decreases as the dose increases following oral administration. The bioavailability of DIDP from oral exposure is assessed as 100 percent for both adults and children. For the systemic and developmental effects, no observed adverse effect levels (NOAELs) of 60 and 100 mg/kg bw/day were determined, respectively. Based on the weight of evidence, the available data do not support a mutagenic, genotoxic or carcinogenic potential for DIDP in humans (NICNAS 2015).

**Di-isononyl phthalate (DINP):** the NTP concurs with the conclusions of the CERHR Phthalates Expert Panel and has minimal concern for DINP causing adverse effects to human reproduction or fetal development. The NTP has minimal concern for developmental effects in children; NIH (2003e). NICNAS (2012) stated that overall, the risk estimates indicate low concern for children at the current reported levels of DINP in toys and child care articles.

**Di(methoxyethyl) phthalate (DMEP):** is rapidly and almost completely absorbed following oral administration. The bioavailability of DMEP by the oral route is assessed as 100 percent for both adults and children. Following absorption, distribution of DMEP is widespread into tissues, including the placenta, but there is no evidence of accumulation in the body. DMEP is rapidly metabolised and excreted in the urine, predominantly as metabolites such as monomethoxyethyl phthalate (MMEP), 2‑methoxyethanol (2-ME) (also known as ethylene glycol monomethyl ether (EGME)), and methoxyacetic acid (MAA). Based on the weight of evidence, the available data do not support a mutagenic, genotoxic or carcinogenic potential for DMEP in humans. For the systemic and developmental effects, the no observed adverse effect levels (NOAELs) of 33 and 20 mg/kg bw/d are derived for DMEP by applying a factor of three for the lowest observed adverse effect level (LOAEL) to NOAEL extrapolation, respectively. The NOAEL of 10 mg/kg bw/d derived for the testicular toxicity effects of DBP is used for filling a data gap in this assessment (NICNAS 2014a).

**Dimethyl phthalate:** DMP is rapidly and almost completely absorbed following oral administration. The bioavailability via the oral route is assessed to be 100 percent for both adults and children. DMP is also rapidly metabolised and excreted, predominantly as a metabolite, monomethyl phthalate (MMP), via the urine. For systemic organ effects, by applying the low MW phthalate category approach and read-across from DEP data, a no observed adverse effect level (NOAEL) of 150 mg/kg bw/d is derived for DMP based on increased liver weight at 750 mg/kg bw/d. Based on the weight of evidence, the available data do not support a mutagenic, genotoxic or carcinogenic potential for DMP. A conservative NOAEL of 40 mg/kg bw/d was derived for reproductive effects of DMP (NICNAS 2014).

**Di-n-hexyl phthalate (DnHP):** the NTP concluded that animal studies did not determine exposure levels at which no adverse effects occur and no human exposure information was available. The NTP concludes that there is insufficient hazard and exposure information to reach a conclusion regarding the potential for DnHP to adversely affect human development or reproduction (NIH 2003f). ECHA (2013) lists di‑n-hexyl phthalate as “May impair fertility. May cause harm to the unborn child”.

**Di-n-octylphthalate (DnOP):** exposure occurs mainly from eating food or drinking water that is stored in plastic containers. Di-n-octylphthalate has not been classified as to its carcinogenicity by the Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), or the USEPA. The NTP concurs with the CERHR Phthalates Expert Panel’s conclusion that there is negligible concern for effects on adult reproductive systems. This conclusion is based on the assumption that humans are exposed to levels of DnOP less than 30 μg/kg bw/day. Although DnOP is reported to be lethal to mouse fetuses at high doses, data are not available to determine exposure levels at which no adverse effects occur. Therefore, the NTP concludes that there is insufficient information on developmental toxicity to reach a conclusion regarding the potential for DnOP to adversely affect human development (NIH 2003c). DnOP is rapidly absorbed from the gastrointestinal tract following oral administration. It is metabolised predominantly to mono-n-octylphthalate (MnOP) and eliminated in urine. The bioavailability of DnOP from oral exposure is assessed as 100 percent for both adults and children. For systemic toxicity, a NOAEL of 37 mg/kg bw/day has been established based on histological changes in the liver and thyroid observed at 350 mg/kg bw/day (lowest observed adverse effect level: LOAEL). A NOAEL of 83 mg/kg bw/day for developmental toxicity is derived for DnOP by applying a factor of three for the LOAEL to NOAEL extrapolation. DnOP is non-genotoxic and non-mutagenic. Limited data on its carcinogenic potential suggest that DnOP could act as a promoter of pre-neoplastic hepatic (liver) lesions in rats through a non-peroxisome proliferative mechanism. Based on the weight of evidence, the available data do not a support carcinogenic potential for DnOP in humans (NICNAS 2015).

In general, the lower MW phthalates (methyl and ethyl) appear not to induce developmental effects; the high MW phthalates induce slight developmental effects at high dose (NICNAS 2008).

|  |  |  |  |
| --- | --- | --- | --- |
|  | **RfD1** | **DWEL2** | **USEPA Cancer Group** |
| butyl benzyl phthalate | 0.2 | 7 | C: Possible human carcinogen |
| dibutyl phthalate | 0.1 | 4 | D: Not classifiable as to human carcinogenicity |
| diethyl phthalate | 0.8 | 30 | D: Not classifiable as to human carcinogenicity |
| dimethyl phthalate | – | – | D: Not classifiable as to human carcinogenicity |

1 mg/kg/d

2 mg/L

The following phthalates are on the EC List of 66 Category 1 substances (EC 2015) showing evidence of endocrine disrupting activity in at least one species using intact animals:

* butylbenzylphthalate (BBP), di-(2-ethylhexyl)phthalate (DEHP) = dioctylphthalate (DOP), and di-n-butylphthalate (DBP).

**Oral Minimal Risk Levels (MRLs)**, ATSDR as at July 2013 see: <http://www.atsdr.cdc.gov/mrls/mrls_list.html>.

Dibutyl phthalate:

* An MRL of 0.5 mg/kg/day has been derived for acute-duration oral exposure (14 days or less) to di-n-butyl phthalate, based on a NOAEL of 50 mg/kg/day in rats.
* An intermediate-duration oral MRL was not derived for di-n-butyl phthalate.
* No chronic-duration oral studies for humans or animals were identified; thus, a chronic oral MRL was not derived.

Diethyl phthalate:

* An MRL of 7 mg/kg/day has been derived for acute-duration oral exposure (14 days or less) to diethyl phthalate, based on a NOAEL of 50 mg/kg/day in rats.
* An MRL of 6 mg/kg/d for intermediate-duration oral exposure (15–364 days).

Di-n-octylphthalate:

* An MRL of 3 mg/kg/day has been derived for acute-duration oral exposure (14 days or less) to di-n-octyl phthalate, based on a LOAEL of 1,000 mg/kg/day in rats.
* An MRL of 0.4 mg/kg/day has been derived for intermediate-duration oral exposure (15 to 364 days) to di-n-octyl phthalate, based on a NOAEL of 40.8 mg/kg/day in rats.
* No chronic oral MRLs were derived for di-n-octylphthalate because no reliable data exist on adverse effects of chronic-duration oral exposure to di-n-octylphthalate.

### Derivation of Maximum Acceptable Value

No MAV.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. For butyl benzyl phthalate, the acute, short-term, chronic and subchronic health risk limits are 0.1 mg/L.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. For dibutyl phthalate, the acute, short-term, chronic and subchronic health risk limits are 0.02 mg/L.

The Minnesota Department of Health (MDH) has adopted permanent rules defining health risk limits for contaminants in drinking water. For diethyl phthalate, the chronic health risk limit is 6 mg/L.

For dimethyl phthalate, the chronic health risk limit is 70 mg/L.

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# Polyacrylamide

The IUPAC name is poly(2-propenamide). Also called poly(1-carbamoylethylene).

Polyacrylamide products include cationic, non-ionic, and anionic polymers. They include both dry powders, and emulsions that typically range in concentration from 10 percent to 40 percent active polymer. These polyelectrolytes are differentiated by the wide range of molecular weights, and by their degree of cationicity (or anionicity).

Polyacrylamide polyelectrolytes are the most commonly used coagulant aids in New Zealand.

Refer to the datasheets for information about the following impurities or by-products which are regulated by NSF Standard 60:

* acrylamide
* acrylic acid
* 3-hydroxypropane nitrile
* isobutane nitrile
* acrylonitrile.

Polyacrylamide polyelectrolyte usage is also regulated in the UK by the DWI, for example:

**A.1.2 Products based on polyacrylamide**

For the products listed below the following conditions apply:

(i) no batch must contain more than 0.020 percent of free acrylamide monomer based on the active polymer content

(ii) the dose must average no more than 0.25 mg/l and never exceed 0.50 mg/l of the active polymer

(iii) an upper limit for the content of free acrylamide monomer must be stated by the supplier for every batch

(iv) the method used for the analysis for free acrylamide monomer is that published in the series “Methods for the Examination of Waters and Associated Materials” entitled “Determination of Acrylamide Monomer in Waters and Polymers 1987” (HMSO 1988 Method number 115. ISBN 01175 2039X.

NSF (2010) reports the following after testing 113 samples:

|  |  |  |  |
| --- | --- | --- | --- |
| **Contaminant** | **Allowable concentration in drinking water, mg/L** | **Median contribution to drinking water, mg/L** | **Range  mg/L** |
| acrylamide | 0.0005 | 0.00006 | <0.00001–0.0004 |
| acrylic acid | 0.40 | 0.0015 | <0.00002–0.01 |
| 3-hydroxypropane nitrile | 0.01 | 0.00003 | <0.00001–0.0007 |
| isobutane nitrile | 0.0003 | 0.000005 | <0.00001–0.00003 |
| acrylonitrile | 0.00006 | 0.00002 | <0.00001–0.00005 |

Stockham and Morran (2000) summarised some earlier studies on the disinfection of water treated with polyacrylamides. The major DBPs from the chlorination of acrylamide monomer were found to be 2,3-dichloropropionic acid (qv) and monochloroacrylic acid.

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# Polyamine polyelectrolytes

Polyamine polyelectrolytes are solutions that typically range in concentration from 10 percent to 65 percent active polymer by weight. They are polyquaternary polymers produced from the reaction of dimethylamine and epichlorohydrin. These polymers have a low to medium molecular weight and a high charge density. They are cationic.

Refer to the datasheets for information about the following impurities or by-products which are regulated by NSF Standard 60:

* epichlorhydrin
* dimethylamine
* 1,3-dichloro-2-propanol
* 1,2-dichloro-3-propanol
* ethylenediamine
* glycidol.

NSF (2010) reports the following after testing 112 samples:

|  |  |  |  |
| --- | --- | --- | --- |
| **Contaminant** | **Allowable concentration in drinking water, mg/L** | **Median contribution to drinking water, mg/L** | **Range mg/L** |
| epichlorhydrin | 0.002 | <0.0004 | <0.0004–0.0009 |
| dimethylamine | 0.12 | 0.021 | <0.004–0.12 |
| 1,3-dichloro-2-propanol | 0.009 | 0.0006 | <0.001–0.008 |
| 1,2-dichloro-3-propanol | 0.009 | <0.001 | <0.001–0.008 |
| ethylenediamine | 2.0 | <0.001 | <0.001–0.002 |
| glycidol | 0.001 | <0.0002 | <0.0002–0.0008 |

Polyamine polyelectrolyte usage is also regulated in the UK by the DWI, for example:

**A.1.3 Products based on polyamine**

For the products listed below the following conditions apply:-

(i) the dose must average no more than 2.5 mg/l and never exceed 5.0 mg/l as the active polymer

(ii) no batch must contain more than 40 mg/kg 3-monochloropropane-1,2-diol

(iii) the analytical system used for determining the batch content must have a limit of detection no greater than 4 mg/kg. Both estimates must have at least 10 degrees of freedom and have been determined from batches of analyses carried out on not less than five separate days

(iv) the supplier must state for every batch an upper limit for the content of 3‑monochloropropane-1,2-diol.

NZWWA (1999) states that epichlorohydrin levels shall not exceed 5 mg/kg of active polymer (5 ppm or 5 x 10-4 percent by weight).

Some studies have indicated that poly(epichlorohydrin dimethylamine) (polyamine) and poly(diallyldimethylammonium chloride) (polyDADMAC) may form N‑nitrosodimethylamine (NDMA – see datasheet) when in contact with chloramine.

### References

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# Polybromodiphenyl ethers

Polybrominated diphenyl ethers (the PBDEs or PBDPEs) are a class of structurally similar brominated hydrocarbons in which 2 to 10 bromine atoms are attached to the molecular structure (ie, diphenyl ether), and are commonly referred to as a sub-group of the (poly)brominated flame retardants (BFRs). The flame retardancy of polybrominated diphenyl ethers (PBDEs) increases with the number of bromine atoms in the molecule. Therefore only the higher brominated BDEs like the penta, octa or deca form are of commercial interest. Monobrominated structures (ie, one bromine atom attached to the molecule) are often included when describing PBDEs. There are 209 different molecular combinations, or congeners, that are possible for PBDEs, although only a limited number exist in commercial mixtures. Based on the number of bromine substituents, there are 10 homologous groups of PBDE congeners (monobrominated through to decabrominated), with each homologous group containing one or more isomers. Note however, that individual commercial products are usually mixtures of several congeners.

Polybromodiphenyl ethers (PBDEs) are also called polybromobiphenyl ethers (PBBEs). See EU (2002) for discussion on nomenclature.

The PBDE group as a whole has the CAS No. 32536-52-0. One of the core compounds used in their manufacture, p-bromodiphenyl ether, has CAS No. 101-55-3. The main PBDEs are:

* 4 bromines: tetrabromodiphenyl ether CAS No. 40088-47-9
* 5 bromines: pentabromodiphenyl ether CAS No. 32534-81-9
* 6 bromines: hexabromodiphenyl ether CAS No. 36483-60-0
* 7 bromines: heptabromodiphenyl ether CAS No. 68928-80-3
* 8 bromines: octabromodiphenyl ether CAS No. is 32536-52-0
* 9 bromines: nonabromodiphenyl ether CAS No. is 63936-56-1
* 10 bromines: decabromodiphenyl ether which is also called bis(pentabromophenyl) ether CAS No. is 1163-19-5.

Some simpler brominated phenols are discussed in Appendix 2.5: Aesthetic Determinands. The main ones include dibromophenols and 2,4,6-tribromophenol.

Polybrominated biphenyls (PBBs) are also used in flame retardants; these are discussed with the PCBs.

Note that hexabromodiphenyl ether (CAS No. 36483-60-0) refers to the hexabromo homolog group, whereas 2,2’,4,4’,5,5’-hexabromodiphenyl ether (BDE-153) (CAS No. 68631-49-2) is a congener within that group.

Likewise, pentabromodiphenyl ether (CAS No. 32534-81-9) refers to the pentabromo homolog group, whereas 2,2’,4,4’,5-pentabromodiphenyl ether (BDE-99) (CAS No. 60348-60-9) is a congener within that group.

Likewise, tetrabromodiphenyl ether (CAS No. 40088-47-9) refers to the tetrabromo homolog group, whereas 2,2’,4,4’-tetrabromodiphenyl ether (BDE-47) (CAS No. 5436‑43-1) is a congener within that group.

### Maximum Acceptable Value

Polybromodiphenyl ethers do not have a MAV in the DWSNZ, and are not mentioned in the WHO Guidelines.

The PBDE pentabromodiphenyl ether was added to the Persistent Organic Pollutants (POP) Stockholm Convention list in May 2009 (UNEP Decision POPRC-1/3, and <http://chm.pops.int/>). The commercial product is a mixture of:

* pentabromodiphenyl ether 50–62 percent w/w
* tetrabromodiphenyl ether 24–38 percent w/w
* tribromodiphenyl ether 0–1 percent w/w
* hexabromodiphenyl ether 4–12 percent w/w
* heptabromodiphenyl ether trace.

Tetrabromodiphenyl ether, hexabromodiphenyl ether, heptabromodiphenyl ether and octabromodiphenyl ether were also added to the Persistent Organic Pollutants (POP) Stockholm Convention list; <http://chm.pops.int/>. Decabromodiphenyl ether is proposed to be added (as at March 2016).

Environment Canada (2013) developed Federal Environmental Quality Guidelines for PBDEs in water:

* triBDE (total) 46 ng/L
* tetraBDE (total) 24 ng/L
* pentaBDE (total) 0.2 ng/L
* pentaBDE-99 4 ng/L
* pentaBDE-100 0.2 ng/L
* hexaBDE (total) 120 ng/L
* heptaBDE (total) 17 ng/L
* octaBDE (total) 17 ng/L

### Sources to drinking-water

#### 1. To source waters

The three main commercial PBDE products are pentabromodiphenyl ether, octabromodiphenyl ether and decabromodiphenyl ether. They are not pure substances, for example, pentabromodiphenyl ether denotes the main component of the mixture which can vary between manufacturers; generally it is 50–62 percent w/w, with 24–38 percent tetrabromodiphenyl ether and about 10 percent hexabromodiphenyl ether.

Pentabromodiphenyl ether (pentaBDE) product is used in foam mainly polyurethane) for cushioning in upholstery. This product accounts for up to about 10 percent of the PBDE usage.

Octabromodiphenyl ether (octaBDE) product is used in plastics for business equipment. The actual composition specification may vary depending on the manufacturer and so only general ranges of compositions are generally reported. Recent testing of different supplies of octaBDE gave an average of 5.5 percent hexabromodiphenyl ether, 42.3 percent heptabromodiphenyl ether, 36.1 percent octabromodiphenyl ether, 13.9 percent nonabromodiphenyl ether and 2.1 percent decabromodiphenyl ether. It is no longer made in Europe. Octabromodiphenyl ether is always used in conjunction with antimony trioxide. In Europe, it is primarily used in acrylonitrile-butadiene-styrene (ABS) polymers at 12–18 percent weight loadings in the final product. Minor uses include high impact polystyrene (HIPS), polybutylene terephthalate (PBT) and polyamide polymers, at typical loadings of 12–15 percent weight in the final product (EU 2003).

Decabromodiphenyl ether (decaBDE) makes up 82 percent of these products manufactured globally. Its main use is as flame retardants the textile industry and for electronic enclosures, such as television cabinets.

A number of brominated compounds that are structurally similar to the brominated diphenyl ethers have been found to be present in some marine species, especially marine sponges, and some green algae (eg, *Cladophora*) (EU 2002).

The Stockholm Convention meeting stated that: “bromodiphenyl ether congeners are a group of brominated organic substances that inhibit or suppress combustion in organic material, which are used as additive flame retardants. Brominated diphenyl ethers are mainly manufactured as commercial mixtures where several isomers, congeners and small amounts of other substances occur”.

The European Union has banned the sale of products containing more than 0.1 percent pentaBDE and octaBDE effective from 15 August 2004. The first commercial productions of PBDEs had begun in the 1970s in Germany.

No levels of PeBDPE in water have been reported. Hexabromodiphenyl ether (a minor component) has been tested frequently in and around Japan and no samples exceeded the detection limit of 0.00004 mg/L. (EU 2001).

Toms et al (2006) report results of testing for 26 polybrominated diphenyl ether (PBDEs) congeners in 39 locations from all states and territories of Australia. PBDEs were detected in samples from 35 of 46 sites and the total PBDE concentration (excluding the LOD (limit of detection)) ranged from non-detect to 60,900 pg/g dry weight (dw) with an overall mean (± standard deviation) and median of 4,707 ± 12,580 and 305 pg/g dw, respectively. As expected, the sites with the highest concentrations were the estuaries with the highest degree of urbanisation and industrialisation. Marine and freshwater locations on the whole had lower PBDE concentrations than estuarine locations. In 86 percent of sediment samples the congener profile was dominated by BDE-209 (a decabromodiphenyl ether). Overall, the concentrations of PBDEs in Australian sediments were relatively low compared with studies on sediments in industrialised countries from the northern hemisphere.

MfE (2010) believes that at least 12 tonnes of BDEs are currently being imported into New Zealand in finished consumer goods annually, 280 tonnes are contained in articles “in use” and 60 tonnes are being deposited in landfills annually. All three volumes are likely to be decreasing. Risk analysis indicates that the range of the current estimate of “in use” BDEs is from 163 to 440 tonnes. Using the approach outlined in the report, there is an estimated 740 tonnes of BDEs in articles currently deposited in landfills. Using stated assumptions on the rate of disposal to landfills and article usable lifetime, it is estimated that total deposited tonnes will reach 1,200 tonnes within 10 years, although the rate is slowing with a reduced level of prevalence in both imported and New Zealand made goods.

NICNAS (2011) states that PBFRs are not manufactured in Australia, but are imported as pure chemicals or mixtures, or in polymer resins or extruded polystyrene foam boards. These chemicals are used exclusively as flame retardants, typically in concentrations ranging from 3 to 12 percent depending on the product, although concentrations above and below this range are also used.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

As additives, PBDEs are physically mixed into product applications, rather than chemically bound. Therefore, they have the potential to migrate from their matrix into the environment when conditions are ideal. Their use in products found in the home and office results in them being found in dust.

Under certain combustion/pyrolysis conditions, decabromodiphenyl ether, and polybrominated diphenyl ethers in general, can form brominated dibenzofurans and brominated dibenzo-p-dioxins (EU 2002).

PBDEs have not been associated with actual health-related effects. Concerns have increased, however, because some of these chemicals (particularly the pentaBDEs) have been found persisting in the environment at varying concentrations.

PBDEs do not dissolve easily in water, and therefore, high levels of PBDEs are not found in water. The very small amounts of PBDEs that do occur in water stick to particles and eventually settle to the bottom. Sediments at the bottom of bodies of water, such as lakes and rivers, generally act as reservoirs for decaBDEs, which can remain there for years. Some lower brominated PBDEs (eg, tetra- and penta-congeners of PBDE) in water may build up in fish to low concentrations (about 10 billionths of a gram to one-millionth of a gram of PDBE per gram of fresh fish.

PBDEs bind strongly to soil particles. Rainwater is not expected to spread PBDEs much below the soil surface; thus, it is unlikely that PBDEs will enter groundwater. They have low vapour pressures so are not likely to volatilise.

The estimated half-life in water for two polybrominated diphenyl ether (PBDE) congeners (PBDE-47 and PBDE-99) is 150 days. PBDE-47 and PBDE-99 are two congeners within the family of polybrominated diphenyl ethers (ie, 2,2’,4,4’-tetrabromodiphenyl ether and 2,2’,4,4’,5-pentabromodiphenyl ether respectively). The higher brominated compounds slowly degrade in the environment into the lower brominated compounds.

EU (2001) quotes some physico-chemical properties of pentabromodiphenyl ethers: vapour pressure = 4.69 x 10-6 Pa; water solubility = <0.0024 mg/L; the partition coefficient = Kow = 6.57 (this high value suggests strong adsorption to soil).

EU (2002) quotes some physico-chemical properties of decabromodiphenyl ethers: vapour pressure = 4.63 x 10-6 Pa at 21°C (= 3.47.10-8 mmHg); water solubility = <0.0001 mg/L; the partition coefficient = Kow = 6.27 (this high value suggests strong adsorption to soil); Henry’s law constant = 44 Pa.m3/mol; bromine content about 83 percent; decabromodiphenyl ether can be considered to be stable to hydrolysis. Decabromodiphenyl ether is unlikely to biodegrade rapidly in the environment under aerobic conditions. The low vapour pressure for commercial decabromodiphenyl ether indicates that it is unlikely to volatilise from spillage to land. However, given the very low water solubility of this substance, volatilisation from surface water may still occur to a small extent, although adsorption onto sediment is likely to dominate and further reduce this tendency.

EU (2003) quotes some physico-chemical properties of octabromodiphenyl ethers: vapour pressure = 6.59 x 10-6 Pa at 21°C; water solubility = <0.0005 mg/L; the partition coefficient = Kow = 6.29 at 25°C (this high value suggests strong adsorption to soil). The rate of biodegradation will be assumed to be effectively zero for environmental modelling purposes.

In a limited survey, MfE (2010) found the total concentration of the BDEs analysed in New Zealand landfill leachate was only up to 0.4 µg/L (0.0004 mg/L) so concluded that properly designed and managed landfills are a secure final depository of BDE containing plastics.

### Typical concentrations in drinking-water

Because PBDEs are hydrophobic in nature, this class of compounds has not been detected in water to any significant extent.

### Removal methods

Because PBDEs bind strongly to soil particles, they will be removed from water by treatment processes that remove particulate matter.

### Analytical methods

#### Referee method

No MAV, so not needed.

#### Some alternative methods

See MfE (2010).

### Health considerations

Concern for the possible health effects of PBDEs has heightened recently due to evidence that components of pentabromodiphenyl ether (pentaBDE) commercial mixtures are ubiquitously distributed at very low levels in the environment, biota, human tissues, and breast milk.

Brominated flame retardants (BFRs) are not allowed to be used in polymers which may have contact with food. FSANZ (2007) reported thirty of the thirty five types of food analysed were found to contain PBDE at very low levels (parts per billion range). Eggs, pork chops, bacon, cream, hamburger, lamb chops, sheep liver, beef sausage, pizza, chocolate, potato crisps had relatively higher levels, while foods such as vegetables had relatively lower levels. Full fat milk, reduced fat milk, canola oil, tap water and table salt did not have quantifiable amounts of PBDE. While dietary exposure to PBDE was low, the more commonly consumed foods (bread, vegetables, dairy and meats) contributed more to dietary exposure. Overall, they found that the general population had a low exposure to PBDE through food and that current intakes of PBDE through diet are unlikely to be a significant health concern. They concluded that food is a minor contributor to human exposure to PBDE compared to other sources such as indoor air and dust.

The toxicity of decaBDE is generally much less pronounced than for octa- and pentaBDE commercial products following acute and repeated-dose exposures. This dissimilar toxicity is likely related to the preferential accumulation of lower brominated congeners in the body, due to their greater partitioning and retention in lipid-rich tissues and lower rates of metabolism and elimination relative to decaBDE. In particular, in comparison with the lower brominated mixtures, oral studies in rats found that decaBDE is minimally absorbed (0.3 to 2 percent), has a relatively short half-life (<24 hours), and is rapidly eliminated via faecal excretion (>99 percent in 72 hours). These toxicokinetic differences appear to be related to the number and location of bromines on the diphenyl oxide molecule, and correlate with environmental monitoring data indicating that decaBDE has low bioaccumulation potential.

A chronic-duration oral MRL was not derived for lower brominated BDEs due to insufficient data. No acute- or chronic-duration oral MRLs were derived for decaBDE due to insufficient data.

The USEPA derived reference doses (RfDs) for octaBDE and pentaBDE of 3x10-3 and 2x10-3 mg/kg/day, respectively.

The oral RfD for 2,2’,4,4’,5-pentabromodiphenyl ether (BDE-99) was calculated at 0.0001 mg/kg/d (USEPA. 2008. The oral RfD for 2,2’,4,4’,5,5’-hexabromodiphenyl ether (BDE-153) was calculated at 0.0002 mg/kg/d (USEPA. 2008. The oral RfD for 2,2’,3,3’,4,4’,5,5’,6,6’-decabromodiphenyl ether (BDE-209) was calculated at 0.007 mg/kg/d (USEPA. 2008. The oral RfD for tetrabromodiphenyl ether (BDE-47) was calculated at 0.0001 mg/kg/d (USEPA 2008).

The USEPA has classified decabromodiphenyl ether in Group C, as a possible human carcinogen, and nonabromodiphenyl ether, octabromodiphenyl ether, hexabromodiphenyl ether, pentabromodiphenyl ether, tetrabromodiphenyl ether, tribromodiphenyl ether, *p,p’*-dibromodiphenyl ether, and *p*-bromodiphenyl ether in Group D, not classifiable as to human carcinogenicity.

As at October 2015 and March 2017 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for PBDEs (lower brominated) of:

* 0.00006 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.000003 mg/kg/d for intermediate-duration (15–364 days).

As at October 2015 and March 2017 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for PBDEs (deca-brominated) of:

* 0.01 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.0002 mg/kg/d for intermediate-duration (15–364 days).

The acute oral MRL is based on a NOAEL of 1 mg/kg/day for reduced serum levels of thyroid T4 hormone in fetal rats that were exposed to pentaBDE on days 4 to 20 of gestation. The MRL was estimated by dividing the NOAEL by an uncertainty factor of 30 (component factors of 10 for animal to human extrapolation and 3 for human variability). A component factor of 10 was not used for human variability because the MRL is based on effects observed in a sensitive subgroup.

The intermediate oral MRL is based on a LOAEL of 2 mg/kg/day for minimal liver effects in rats that were exposed to pentaBDE for 90 days). The MRL was estimated by dividing the LOAEL by an uncertainty factor of 300 (component factors of 3 for use of a minimal LOAEL, 10 for animal to human extrapolation, and 10 for human variability).

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for PBDEs (deca-brominated) of 10 mg/kg/d for intermediate-duration (15–364 days). The MRL was derived based on a NOAEL of 1,000 mg/kg/day for developmental toxicity in rats exposed to decaBDE for 19 days during gestation. The MRL was estimated by dividing the NOAEL by an uncertainty factor of 100 (10 for animal to human extrapolation and 10 for human variability).

BDEs have been measured in New Zealand breast milk. The 2008 survey found that of the 36 BDEs determined, the highest levels were measured for BDE47 (2,542 pg/g), followed by BDE153 (717 pg/g), BDE100 (536 pg/g) and BDE99 (532 pg/g) (Massey University 2010). The baseline data for BFRs show that the BDEs that are most abundantly present in the New Zealand breast milk samples are similar to those reported for other countries, and that the levels are comparable to or higher than those measured in Europe, while being substantially lower than those reported for the United States and Australia.

The monitoring results for human serum indicated that the mean level of the five major PBDE congeners, BDE-47, 99, 100, 153 and 154, in adult Australians, was 11.0 ng/g lipid weight (lw) for men, 8.2 ng/g lw for women, and 9.5 ng/g lw in women of childbearing age. Little temporal or regional variation was seen. However, higher concentrations were seen in younger age groups compared with adults. For toddlers in the 0–4 age group, the mean results were 48.3 ng/g lw for females and 44.6 ng/g lw for males. The higher levels in younger age groups were consistent with limited international data showing similar trends (NICNAS 2007).

The primary health concerns revolve around the potential of some PBFRs to act as carcinogens, endocrine disruptors and neurodevelopmental toxicants and the lack of adequate toxicological data for others to fully assess their hazards (NICNAS 2001).

### Derivation of Maximum Acceptable Value

No MAV.

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# Polychlorinated diphenyl ethers (PCDEs)

The polychlorinated diphenyl ethers (PCDEs) are a group of 209 halogenated aromatic compounds that are structurally related to the polybrominated flame retardants.

### Maximum Acceptable Value

Polychlorinated diphenyl ethers do not have a MAV in the DWSNZ, and are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

The 10 main groups are monochloro-, dichloro-, trichloro-, tetrachloro-, pentachloro-, hexachloro-, heptachloro-, octachloro-, nonochloro-, and decachloro-diphenyl ether.

PCDEs have similar properties to the polychlorinated biphenyls (PCBs), the dibenzo-p-dioxins and the dibenzofurans.

As the degree of chlorination increases, the vapour pressure decreases, resulting in the mono- to octachlorodiphenyl ethers tending to accumulate in water systems, and the more chlorinated species being found more randomly.

PCDEs are thought to originate in the production of herbicides and other chlorinated phenolic compounds. They are not used much in industry, being found mainly as accidental by-products, such as in fly ash and incinerator waste. Commercial grade trichlorophenols have been found to contain from 100 to 1,000 mg tetra- to octa-PCDEs per kg of trichlorophenols. Octa- and nona-PCDEs comprised up to 8 percent of some PCP formulations. PCBs were found to contain from near zero to 51 percent PCDEs (NICNAS 2002).

The concentrations of the total PCDEs in Whitby Harbour (Ontario) sediments were as high as 2 pg/g (dry weight basis) and total PCDE concentrations in the biota ranged from 6 ng/g to 2775 ng/g wet weight (592 ng/g to 123 000 ng/g lipid normalised). The mean estimated water concentration of total PCDEs was 29 ng/L. The total PCDE concentration was greater in the benthic food web than the pelagic food web, indicating that the contaminants are boundto the sediments and readily transferred to the benthic feeding biota (Villeneuve 1998).

Appropriate waste disposal procedures for PCBs, PCP etc, and soil contaminated with these, should limit the effects of the PCDEs.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Water solubility decreases with increasing chlorine content. The range is from about 4 to 12 mg/L for the monochlorodiphenyls, down to 0.06 mg/L for the decachlorodiphenyls. Likewise for the octanol/water partition coefficient, log Kow ranging from about 4.5 up to 8.2; see NICNAS (2002) for details. Their persistence has led to their wide environmental distribution, albeit it at very low concentrations.

Surveys have shown that PCDE residues are present in sediments, fish, wildlife, and human tissues (Becker et al 2008).

### Typical concentrations in drinking-water

Because PCDEs are hydrophobic in nature, this class of compound is not likely to be detected in water to any significant extent.

### Removal methods

Because PCDEs bind strongly to soil particles, they should be removed from water by treatment processes that remove particulate matter.

### Analytical methods

#### Referee method

No MAV, so not needed.

### Health considerations

Because of the widespread use of substances containing PCDEs, food is considered to be the most common route of exposure for humans.

PCDEs may contribute significantly to the toxicity of halogenated aromatics associated with chlorinated phenol-derived contaminated areas such as toxic chemical waste dumpsites (Becker et al 2008).

Their toxicity is low compared with the polychlorinated dibenzo-p-dioxins and the dibenzofurans. The liver and thyroid are the target organs.

### Derivation of Maximum Acceptable Value

No MAV.

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# polyDADMAC

CAS No. 26062-79-3. The CAS number for diallyldimethylammonium chloride (the monomer DADMAC) is 7398-69-8.

Polydiallyldimethylammonium chloride (polyDADMAC) is a polyquaternary amine, prepared from diallyldimethylammonium chloride (DADMAC). It can be used as a polymer to assist coagulation in water treatment, often as a primary coagulant. PolyDADMAC products are typically sold as 10 percent to 40 percent active polymer solutions, certified to a maximum use level for water treatment of 25 to 100 mg/L of product, which equates to a dose of 10 mg/L of active polymer in the treated water. PolyDADMAC generally contains <1 percent of the monomer.

Refer to the datasheets for information about the following impurities or by-products:

* allyl chloride
* diallyldimethylammonium chloride
* dimethylamine
* N-nitrosodimethylamine

NSF Standard 60 regulates the monomer and dimethylamine content. The allowable concentration of the monomer in drinking water is 0.05 mg/L, and 0.12 mg/L for dimethylamine.

NSF (2010) reports results of testing 97 samples of polyDADMAC. The median contribution of the monomer to drinking water was 0.013 mg/L (range <0.0025 to 0.05 mg/L) and 0.012 mg/L (range <0.0004 to 0.10 mg/L) for dimethylamine.

In the UK, DWI regulates the use of chemicals added to drinking water. For products based on polyDADMAC (polydiallyldimethyl ammonium chloride):

For the products listed below the following conditions apply:

(i) the dose must not exceed 10 mg/L of active polymer.

Stockham and Morran (2000) reported an Australian water treatment study using polyDADMAC in conjunction with chlorine and ozone. The results indicated that chlorination of the polymer produced very few DBPs, with minimal AOX and THM formation, a result supported by other workers. Trichloroacetic acid and chloral hydrate were the main specific DBPs detected. However, ozonation of the polymer resulted in formation of significant levels of aldehydes and ketoacids, as well as a series of disinfection by‑products, tentatively identified as di-keto acids. Glyoxal, methyl glyoxal formaldehyde, glyoxylic acid, pyruvic acid and formaldehyde were the main individual DBPs detected.

Ozonation of polyDADMAC in the presence of bromide resulted in the formation of significant AOX, brominated THMs and HAAs. Traces of a compound tentatively identified as dibromoethene was also identified.

Chlorination of pre-ozonated solutions of polyDADMAC resulted in a dramatic increase in THMs, HAAs and AOX levels. Of particular concern was the unusually high level of the potential carcinogen trichloronitromethane produced under these conditions. Dichloroethene was also detected.

A report published for USEPA in 2004 states there is virtually no exposure to monomer during manufacture and emissions to air, water and soil are very low. The product is used almost exclusively as a monomer in the manufacture of cationic, water-soluble polymers. The report quotes a NOAEL of 50 mg/kg/d based on decreased body weight gain in rats in a 13-week study. A NOAEL of 6 mg/kg/d was quoted for teratology.

WRF (2015) reports that N-nitrosodimethylamine (NDMA) formation was higher for polyDADMAC polymers reacted with chloramines in reagent water than for other polymers tested, ranging from 32 to 162 ng of NDMA per mg of polymer. Other nitrosamines were detected but below the method reporting level. The amount of NDMA formed by a particular polyDADMAC polymer is dependent on the specific source water and treatment system. Their research found that the same polymer will form different amounts of NDMA when used at different sites. In addition, the NDMA formation from polyDADMAC polymers is much less when the water is chloraminated after the polymer has been removed in the sedimentation tank.

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# Polynuclear aromatic hydrocarbons

Sometimes called polyaromatic hydrocarbons or PAHs.

The following list comprises the 17 PAH “priority pollutants” under the USEPA Clean Water Act. There is a separate datasheet for each of these.

* acenaphthene CAS No. 83-32-9
* acenaphthylene CAS No. 208-96-8
* anthracene CAS No. 120-12-7
* benzo 1,12-perylene (or benzo[g,h,i]perylene) CAS No. 191-24-2
* benzo 11,12-fluoranthene (or benzo[k]fluoranthene) CAS No. 207-08-9
* benzo 3,4-fluoranthene (or benzo[b]fluoranthene) CAS No. 205-99-2
* benzo 3,4-pyrene (or benzo[a]pyrene) CAS No. 50-32-8
* benzo[a]anthracene CAS No. 56-55-3
* 2-chloronaphthalene CAS No. 91-58-7
* chrysene CAS No. 218-01-9
* dibenz[a,h]anthracene CAS No. 53-70-3
* fluoranthene CAS No. 206-44-0
* fluorene CAS No. 86-73-7
* indeno[1,2,3-c,d]pyrene CAS No. 193-39-5
* naphthalene CAS No. 91-20-3
* phenanthrene CAS No. 85-01-8
* pyrene CAS No. 129-00-0

There are more than 100 different PAHs. WHO (2001) discusses properties of 77 different chloronaphthalenes.

As well the 17 PAHs referred to above, a datasheet has been prepared for methylnaphthalenes, and for creosotes, coal tars and coal tar pitches. This datasheet discusses PAHs in a collective or comparative sense, and covers some PAHs that do not have a datasheet.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene and fluoranthene (based on WHO, fluoranthene was removed from the MAV table in the 2008 revision of the 2005 DWSNZ). Those two PAHs have their own datasheets.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

The Prescribed Concentration or Value (PCV) for the sum of 4 PAHs in England and Wales is 0.0001 mg/L (0.1 µg/L). See Notes. The 4 PAHs are benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene and indeno[1,2,3-c,d]pyrene. Benzo[a]pyrene has a PCV of 0.01 µg/L.

### Sources to drinking-water

#### 1. To source waters

Polycyclic aromatic hydrocarbons are a large group of organic compounds formed from the incomplete combustion of organic matter, and usually appear as a mixture. They have very little industrial use but are formed naturally in forest fires, and volcanic activity or from anthropogenic activities such as domestic fires, vehicle emissions, coke ovens, coal gas manufacture (including related contaminated soils), and aluminium smelters.

The principal route of entry to source water is via atmospheric deposition. Based on the literature reviewed, the presence of chrysene and benzo(k)fluoranthene may be indicators for coal combustion emissions. Whereas other PAHs are indicators of other combustion process: benzo(g,h,i)perylene, coronene and phenanthrene are indicators for motor vehicle emissions, pyrene and fluoranthene are associated with incineration and fluorene, fluoranthene and pyrene are associated with oil combustion (Environment Australia 2003).

PAHs also occur prominently in diesel but are not currently regulated in New Zealand (annual consumption around 2,400 million litres in 2001). The Ministry of Economic Development proposes that the PAH content of diesel be reduced to a maximum of 11 percent by mass, thereby aligning with current European standards and 2006 Australian specifications.

The most abundant classes of PAH found in diesel-contaminated estuarine sediments were naphthalenes, phenanthrenes, and dibenzothiophenes (DBT). Alkylated PAH made up 93 percent of the total PAH. The high proportions of naphthalenes, phenanthrenes, DBT and alkylated PAH are typical of refined petroleum hydrocarbons (US Department of the Interior 1998).

Laboratories can purchase diesel fuel containing certified levels of PAHs, for example, one has the following composition:

|  |  |
| --- | --- |
| **PAH** | **Typical certified concentration (ppm)** |
| acenaphthene | 20 |
| acenaphthylene | 10 |
| fluorene | 30 |
| 1-methylnaphthalene | 250 |
| 2-methylnaphthalene | 170 |
| naphthalene | 80 |
| phenanthrene | 40 |

One of the main characteristics of diesel exhaust is the release of particles. The particles are mainly aggregates of spherical carbon particles coated with inorganic and organic substances. The organic fraction consists of soluble organic compounds such as aldehydes, alkanes and alkenes, and high molecular weight PAH and PAH‑derivatives, such as nitro-PAHs. Many of these PAHs and PAH-derivatives, especially nitro-PAHs, have been found to be potent mutagens and carcinogens, CARB (1998); IARC. 1989. As nitroPAHs have been detected in the emissions of kerosene heaters, fuel gas and LPG burners used for heating and cooking at home, as well as in the fumes of cooking oils, there is therefore a potential indoor exposure to nitroPAHs in poorly ventilated conditions (IPCS 2003).

Naphthylacetic acid is used as a plant growth regulator (a synthetic hormone in the auxin family), see datasheet in the pesticides section.

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; benzo[a]pyrene (BaP) levels range from 6 to 35 mg/kg TEQ depending on land use. The equivalent BaP concentration is calculated as the sum of each of the detected concentrations of nine carcinogenic PAHs which are benz(a)anthracene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene and indeno(1,2,3-cd) pyrene, multiplied by their respective potency equivalency factors.

IARC (2013) discussed the PAHs found in bitumen. Refer also to the datasheet for petroleum products.

IARC (2013a) evaluated nine agents divided into two broad (nitrogen and sulphur) categories: N-heterocyclic PAHs, also known as azaarenes, including five acridines and two carbazoles; and S-heterocyclic PAHs, also known as thiaarenes, including two thiophenes [S-substituted cyclopentadiene moiety]. *N*-heterocyclic PAHs and *S‑*heterocyclic PAHs generally occur as products of incomplete combustion of nitrogen- and sulfur-containing organic matter. Thermal degradation of nitrogen containing polymers may produce *N*-heterocyclic PAHs. Concentrations in water are low, often measured in the ng/L range, and usually result from industrial wastes.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

PAHs are reactive with chlorine at high chlorine doses and at high PAH concentrations. PAHs are less reactive with chlorine at conditions found in water treatment. Additionally reactions at pH<6 tend to produce oxygenated products and reactions at pH>6 produce both oxygenated and chlorinated by‑products. The conditions presented in the literature do not allow conclusions to be made on which species are more amenable to oxidation (DWI 2015). This study referred to earlier work on the leaching of PAHs from coal tar. Phenanthrene was found to be the dominant PAH but an additional 38 PAH compounds were found. When the leachate was chlorinated the distribution of PAHs was significantly modified with fluorene becoming the most prominent PAH with concentrations of fluoranthene and phenanthrene considerably diminished. Oxygen substituted PAHs such as dibenzofuran became more abundant and several new oxygenated compounds were found. Intermixed with the oxygenated compounds were low concentrations of chlorine- and bromine-substituted PAHs. Chlorination by‑products measured in real distribution systems included fluorenone, anthraquinone, cyclopenta[d,e,f]phenanthrenone, 3-chlorofluoranthene and 1‑chloropyrene. This shows that PAHs are readily halogenated by chlorine but not as susceptible to oxidation.

### Forms and fate in the environment

Polynuclear aromatic hydrocarbons enter the environment through atmospheric deposition. Because of their low water solubility most polynuclear aromatic hydrocarbons are adsorbed to sediments and suspended solids in aquatic systems. Volatilisation may be important over periods exceeding one month. Most polynuclear aromatic hydrocarbons are susceptible to aqueous photolysis. Polynuclear aromatic hydrocarbons of three or fewer fused aromatic rings are biodegraded but for the larger polynuclear aromatic hydrocarbons this is minimal. Polynuclear aromatic hydrocarbons are adsorbed but not greatly accumulated by aquatic biota.

### Typical concentrations in drinking-water

The review of organic contaminants in New Zealand drinking-water supplies between 1987 and 1992 contained polynuclear aromatic hydrocarbons results from 217 samples, representing 204 supplies. The concentrations of the individual polynuclear aromatic hydrocarbons are summarised below. Some overseas data has been included.

A number of PAHs have been assessed in Phase 1 of the P2 Programme. With the exception of fluoranthene, none have been detected. The limits of detection range from 0.0001 to 0.0002 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation. Pyrene was the most rapidly degraded PAH. Benz[a]anthracene, benzo[a]pyrene, and perylene were also highly degraded. Indeno[1,2,3-c,d]pyrene and benzo[g,h,i]pyrene were intermediate with respect to relative degradation. Benzo[k]fluoranthene and fluoranthene were the most slowly degraded of the compounds tested. A variety of complex end-products have been identified (ATSDR 1995).

### Analytical methods

See benzo[a]pyrene datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

Occupations associated with exposures to polynuclear aromatic hydrocarbons, of which benzo[a]pyrene is a component, have been associated clearly with human cancer.

Benzo[a]pyrene is absorbed principally through the gastrointestinal tract and the lungs. The rate of absorption of different polynuclear aromatic hydrocarbons is influenced by their lipid solubilities and the content of polyunsaturated fatty acids in the diet. Most of the toxicological literature deals with benzo[a]pyrene. Few studies are available for the other polynuclear aromatic hydrocarbons – see individual datasheets.

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess. The relative carcinogenic potencies of various polynuclear aromatic hydrocarbons, based on bioassays by several routes of administration and related toxicological data, have been ranked in decreasing order as follows: dibenz[a,h]anthracene, benzo[a]pyrene, anthanthrene, indeno[1,2,3-cd]pyrene, benz[a]anthracene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, benzo[j]fluoranthene, cyclopentadieno[c,d]pyrene, benzo[g,h,i]perylene, chrysene and benzo[e]pyrene.

IARC (2010) classified:

* cyclopenta[*cd*]pyrene, dibenz[*a*,*h*]anthracene, dibenzo[*a*,*l*]pyrene in Group 2A (probable human carcinogen)
* benz[*j*]aceanthrylene, benz[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, benzo[*c*]phenanthrene, chrysene, dibenzo[*a*,*h*]pyrene, dibenzo-[*a*,*i*]pyrene, indeno[1,2,3-*cd*]pyrene, 5-methylchrysene, in Group 2B (possible human carcinogen);
* acenaphthene, acepyrene (3,4-dihydrocyclopenta[*cd*]pyrene), anthanthrene, anthracene, 11*H*-benz[*bc*]aceanthrylene, benz[*l*]aceanthrylene, benzo[*b*]chrysene, benzo[*g*]chrysene, benzo[*a*]fluoranthene, benzo[*ghi*]fluoranthene, benzo[*a*]fluorene, benzo[*b*]fluorene, benzo[*c*]fluorene, benzo[*ghi*]perylene, benzo[*e*]pyrene, coronene, 4*H*-cyclopenta[*def*]chrysene, 5,6-cyclopenteno-1,2-benzanthracene, dibenz[*a*,*c*]anthracene, dibenz[*a*,*j*]anthracene, dibenzo[*a*,*e*]fluoranthene, 13*H‑*dibenzo[*a*,*g*]fluorene, dibenzo[*h*,*rst*]pentaphene, dibenzo[*a*,*e*]pyrene, dibenzo[*e*,*l*]pyrene, 1,2-dihydroaceanthrylene, 1,4-dimethylphenanthrene, fluoranthene, fluorene, 1-methylchrysene, 2-methylchrysene, 3-methylchrysene, 4‑methylchrysene, 6-methylchrysene, 2-methylfluoranthene, 3-methylfluoranthene, 1‑methylphenanthrene, naphtho[1,2-*b*]fluoranthene, naphtho[2,1-*a*]fluoranthene, naphtho[2,3-*e*]pyrene, perylene, phenanthrene, picene, pyrene, triphenylene in Group 3 (not classifiable as to carcinogenicity).

The following PAHs appear on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008: benzo[k]fluoranthene, benzo[b]fluoranthene, benzo[a]pyrene, benzo[a]anthracene, chrysene, dibenz[a,h]anthracene, indeno[1,2,3-c,d]pyrene and naphthalene. Also on the list (not otherwise mentioned in this datasheet) such as: benzo[j]fluoranthene, dibenzo[a,e]pyrene, dibenzo[a,h]pyrene, dibenzo[a,i]pyrene, dibenzo[a,l]pyrene, 7,12‑dimethyl benz[a]anthracene and 5-methylchrysene.

IARC considers that diesel engine exhaust is probably carcinogenic to humans (Group 2A), and gasoline engine exhaust is possibly carcinogenic to humans (Group 2B). See IARC (1989) for information on 15 nitroarenes (PAHs with one or more nitro-groups.

### Derivation of Maximum Acceptable Value

There is a MAV for benzo[a]pyrene – see datasheet. There are insufficient data to derive MAVs for any of the other polynuclear aromatic hydrocarbons in drinking-water.

The only time that WHO had a guideline value for other than benzo[a]pyrene was in their 1971 International Standards, which stated that some PAHs are known to be carcinogenic and that the concentrations of six representative PAH compounds (fluoranthene, 3,4-benzofluoranthene, 11,12-benzofluoranthene, 3,4-benzopyrene, 1,12-benzopyrene and indeno[1,2,3-cd]pyrene) should not in general exceed 0.0002 mg/L.

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# Polyhexamethylene biguanide

Polyhexamethylene biguanide occurs as the hydrochloride compound: CAS No. 32289‑58-0 (27083-27-8 also used). The CAS name is poly(iminoimidocarbonyliminoimidocarbonyl-iminohexamethylene) hydrochloride. A common trade name is baquacil. Can also be called polyhexamethylene biguanidine hydrochloride, polihexanide or PHMB.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for polyhexamethylene biguanide. The WHO Guidelines do not refer to for polyhexamethylene biguanide.

### Sources to drinking-water

#### 1. To source waters

Polyhexamethylene biguanide hydrochloride, a polymer of chlorhexidine, is occasionally used (at 25–50 mg/L) with didecyl dimethyl ammonium chloride and hydrogen peroxide in lightly loaded swimming pools as a disinfectant or more correctly a sanitiser or biocide (NZS 5826:2010). It also has some antiseptic and fungicidal properties. Didecyl dimethyl ammonium chloride has a datasheet in the pesticides section.

An antimicrobial product/skin and coat conditioner described as “chlorhexidine diacetate” appears on the NZFSA’s complete database of *Agricultural Compounds and Veterinary Medicines* (ACVM) as at 2012 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)).

### Form and fate in the environment

PHMB is stable hydrolytically in the environment and has a half-life of more than 30 days. This may be of environmental concern for surface water contamination, in the event of exposure to surface water. Studies for other fate processes are not required by and have not been submitted to the Agency (USEPA 2005).

### Analytical methods

#### Referee method

No MAV so no need.

### Health considerations

Polyhexamethylene biguanide hydrochloride has very low toxicity and has been used in eye drops, as a mouth rinse and for treating skin infections.

The USEPA concludes on the basis of conservative calculations that acute and chronic non-cancer dietary and OH&S risks are below the Agency’s level of concern for adults but may indicate a risk of concern for children. Aggregate risk assessments including all sources of exposure indicated a possible concern for both adult’s and children’s exposures.

In relation to cancer risks, the USEPA classified polihexanide into the category “Suggestive Evidence of Carcinogenicity, but Not Sufficient to Assess Human Carcinogenic Potential” (copied from APVMA 2005). The chronic RfD = 0.2 mg/kg/day (USEPA 2005).

### Derivation of Maximum Acceptable Value

No MAV.

### References

APVMA. 2005. The reconsideration of approvals and registrations relating to polihexanide. *Polihexanide Review Scope Document* 12 pp. See: <http://www.apvma.gov.au/products/review/docs/polihexanide_scope.pdf>.

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# 2-Propen-1-ol

CAS No. 107-18-6. The IUPAC name is 2-propenol. 2-Propen-1-ol is also known as allyl alcohol, 2-propenyl alcohol, propene-1-ol-3, vinyl carbinol and 3-hydroxypropene.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for 2-propen-1-ol. The WHO Guidelines do not refer to 2-propen-1-ol.

The USEPA concluded on 22 September 2009 that 2-propen-1-ol is known or anticipated to occur in PWSs and may require regulation. Therefore they added 2‑propen-1-ol to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

2-Propen-1-ol is not found naturally, and has no uses except in the production in a wide range of other compounds.

The production volume of 2-propen-1-ol was estimated at 136,100 t/year worldwide in 2003. 2-Propen-1-ol is an important starting material, and is used in the manufacture of 1,4-butanediol, 2-methyl-1,3-propandiol, allyl diglycol carbonate, diallyl phthalate, diallyl isophthalate, allyl glycidyl ether, epichlorohydrin, allyl methacrylate, styrene 2‑propen-1-ol and resins for coating applications, flavourings such as allyl hexanoate, contact herbicide, as an intermediate for manufacturing pharmaceuticals, fire retardants and herbicides.

### Form and fate in the environment

2-Propen-1-ol is expected to be stable in water because it contains no functional groups that are susceptible to hydrolysis. 2-Propen-1-ol is readily biodegradable under aerobic conditions within 14 days. 2-Propen-1-ol will stay exclusively in the water compartment (99.7 percent) if released to water. If released to soil, 2-propen-1-ol will be distributed mainly to the water (19.4 percent) and soil (80.4 percent) compartment.

An estimated Koc value of 1.32 indicates that 2-propen-1-ol is not expected to adsorb to suspended solids and sediments. It is miscible with water.

### Analytical methods

#### Referee method

No MAV so no need.

### Health considerations

Consumers may be potentially exposed to 2-propen-1-ol from ingestion of foods. 2‑Propen-1-ol has been detected in crab meat, mussels and garlic. 2-Propen-1-ol is rapidly formed in the body from the hydrolysis of allyl esters used as flavour agents in food. The estimated intake of 2-propen-1-ol from this route is 18 μg/kg bw/day in Europe and 5.8 μg/kg bw/day in the USA.

Animal studies demonstrate that 2-propen-1-ol appears to be oxidised readily in the liver, giving a variety of metabolic products, such as acrolein, acrylic acid, glycidaldehyde, and glyceraldehyde. Among these metabolites, the most reactive metabolite, acrolein may cause hepatotoxicity in the liver.

In a repeated dose oral toxicity study, 2-propen-1-ol had adverse effects on kidney tissues in rats, administered in the drinking water continuously for 15 weeks at or above a level of 100 ppm (8.3 mg/kg bw/day in males and 6.9 mg/kg bw/day in females). The NOAEL was 50 ppm of 2-propen-1-ol in drinking water (equivalent to 4.8 mg/kg bw/day in male rats and 6.2 mg/kg bw/day in female rats) based on adverse effects on kidney tissues (increases in absolute kidney weight and relative kidney weight) for females and on an increase in relative stomach weight for male and females at 100 ppm.

A study gave no clear evidence of carcinogenicity in male rats, but there was equivocal evidence of carcinogenicity in the liver of female rats.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Ptaqiloside

CAS No. 87625-62-5. Ptaquiloside is (2R,7S,7aR)-7-hydroxy-2,5,7-trimethyl-3a-[(3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)oxan-2-yl]oxyspiro[3,7a-dihydro-2H-indene-6,1’-cyclopropane]-1-one. Also known as Pta. It has been described as a norsesquiterpene [glucoside](http://en.wikipedia.org/wiki/Glucoside) of illudane.

DWI (2010) states that:

Other potentially harmful substances include other illudanes and protoilludanes, indanones (including various pterosins and pterosides), quercetin and its glycoside rutin, kaempferol, shikimate, thiaminases, prunasin, and various ecdysteroids.

Other substances that have been identified in bracken include the dihydrocinnamic acids, tannins, phloretic acid, braxins, astragalin, isoquercetin, tiliroside (kaempferol-3-*p*-coumaroylglucoside), p-hydroxystyrene glycocides, dihydroferulic acid, 2,3-butanediol, 3-methylbutan-2-ol, monomethylsuccinate, 2-hydroxymethyl ester of propanoic acid, 4-methoxymethyl ester of butanoic acid, monomethyl ester of butanedioic acid, methyl-5-oxoproline, 2(3H)‑dihydrofuranone, and *t-*2-methylcyclohexanol.

However, ptaquiloside is the major toxin, believed to cause 50 percent of the carcinogenic activity of bracken.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for ptaquiloside. The WHO Guidelines do not refer to ptaquiloside.

### Sources to drinking-water

#### 1. To source waters

Ptaquiloside is a toxin found (first isolated in 1983) in many species of bracken, including the New Zealand and Australian species *Pteridium esculentum*. Bracken quickly colonises disturbed areas and can outcompete other plants to form a dense and widespread understorey. Maori treated the rhizomes as a source of edible starch. Ptaquiloside has also been found in some other ferns (Rasmussen 2003).

Ptaquiloside is found in the fronds. A New Zealand study found concentrations of ptaquiloside in bracken varied greatly, and in many stands ptaquiloside was not found. A higher incidence of ptaquiloside, and some very high concentrations, were found in areas where [bovine enzootic haematuria](http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/210400.htm) and/or acute haemorrhagic syndrome is known to occur. The study collected frond samples from 275 sites across New Zealand; 63 percent contained ptaquiloside, with a mean concentration of 3.8 mg per gram (dry weight), and a maximum of 13.3 mg per gram dry weight (1.33 percent dw) (Rasmussen LH, et al 2008).

In 2004, Danish scientist Lars Holm Rasmussen released a study showing ptaquiloside can leach from the plant into the [water supply](http://en.wikipedia.org/wiki/Water_supply), which may explain an increase in the incidence of [gastric](http://en.wikipedia.org/wiki/Gastric_cancer) and [oesophageal cancers](http://en.wikipedia.org/wiki/Oesophageal_cancer) in humans in bracken-rich areas. In addition, livestock have been known to eat the young shoots and fronds, which can then cause [cancer](http://en.wikipedia.org/wiki/Cancer) in the animals, especially when untreated. Ptaquiloside has been identified in the milk of cows, and in groundwater. Humans can be exposed by direct consumption of the plant, contaminated water or milk, and spore inhalation (DWI 2010).

A study in the UK (DWI 2015) detected ptaquiloside in the feeder stream to a reservoir that was deemed to be high risk based on bracken coverage and proximity of the abstraction point to bracken, with concentrations ranging from <0.05 μg/L to 0.35 μg/L.

### Form and fate in the environment

Ptaquiloside is very soluble in water; it has a low hydrophobicity and low affinity for binding to organic matter. Therefore the chemical is expected to be very mobile in soils with short residence times in the profile. Adsorption may be higher on clay minerals, however this process is poorly understood. Degradation of the chemical in soil is expected to be rapid. DWI. 2010. PTA is at its most stable at pH 6 to 7; above pH 8 it is less stable. Pterosin B (PTB) is the main degradation product, and is more stable (DWI 2015).

### Typical concentrations in drinking-water

The DWI study concluded a worst case situation of 0.012 mg/L ptaquiloside in raw water. Drinking water wells in Denmark and Sweden have been found to contain  
0.004–0.006 mg/L and 0.045 mg/L respectively.

There were no positive detections of ptaquiloside or its degradation product, pterosin B, in any of the samples collected from the public water supplies in both England and Wales. For reference, the limit of quantification for all water samples was ordinarily 0.05 μg/L (DWI 2015).

### Removal methods

DWI (2010) calculated the ptaquiloside content after the following treatment processes, assuming a raw water content of 0.012 mg/L:

* 0.0015 mg/L using chlorination
* 0.0086 mg/L using filtration + ultraviolet disinfection
* 0.00086 mg/L using coagulation-flocculation, clarification, filtration and chlorination
* 0.00009 mg/L using coagulation-flocculation, clarification, ozonation and granular activated carbon + chlorination.

The removal of ptaquiloside by each of the processes was estimated based on knowledge of the structure of ptaquiloside and its sorption properties, and the underlying processes of each treatment.

### Analytical methods

#### Referee method

No MAV so no need.

#### Some alternative methods

See Table 4.3 in Rasmussen (2003). Also see DWI (2015).

### Health considerations

The toxicity of bracken and its constituent, ptaquiloside has been investigated in studies of farm and laboratory animals and in human epidemiological studies. The focus of these studies has primarily been on exposure via the oral route. Bracken causes a range of well-defined syndromes in farm animals. These include thiamine deficiency of monogastric animals, acute haemorrhagic syndrome (AHS) associated with bone marrow aplasia and upper alimentary ulceration, a progressive retinal degeneration (PRD) called “bright blindness” and two neoplastic disease syndromes: bovine enzootic haematuria and upper alimentary carcinoma.

The mutagenic and carcinogenic potential of bracken, extracts of bracken components, and ptaquiloside have been studied extensively in laboratory animals. In oral carcinogenicity studies in mice, rats, guinea-pigs and cows, bracken induced benign and malignant intestinal tumours in all species, except cows. Bracken also induced bladder carcinomas in rats, guinea-pigs and cows, lymphocytic leukaemias in mice and mammary carcinomas in rats. Oral administration of bracken processed for human consumption produced intestinal cancers, but at a lower rate of incidence than unprocessed bracken. Whereas starch made from bracken rhizomes did not produce tumours in rats, oral administration of boiling water extracts of bracken to rats induced intestinal and bladder tumours. Oral administration of bracken in mice included maternal toxicity, some embryotoxicity and some minor abnormalities in offspring. Oral administration of ptaquiloside isolated from bracken in rats produced mammary, intestinal and bladder tumours and unscheduled DNA synthesis in primary liver hepatocytes.

There are several epidemiological studies of human populations in Japan, Brazil, Venezuela, Costa Rica and Wales that show an association between exposure to bracken toxins and the development of cancers of the stomach and oesophagus. Although a strong association between eating bracken and cancer in cattlehas been established, the strength of association is less in human studies, and no study has identified a definitive link. Human studies have shown an association between eating bracken and human stomach cancers, but the studies lack the statistical power to demonstrate a clear dose-response relationship. A Japanese study has shown a higher risk of oesophageal cancer in people who ate bracken regularly than in those who ate it only rarely. Another Japanese study has suggested an association between eating wild plants (mainly bracken) and pancreatic cancer in men.

Following an evaluation of the carcinogenic potential of bracken, IARC concluded that there was sufficient evidence for the carcinogenicity of bracken in experimental animals and limited evidence for the carcinogenicity of ptaquiloside. They concluded, however, that there was inadequate evidence for the carcinogenicity of bracken in humans, and they classified bracken in Group 2B: possibly carcinogenic to humans.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Pyrene

Pyrene, CAS No. 129-00-0, is one of the 17 polyaromatic hydrocarbons (polynuclear aromatic hydrocarbons or PAH) “priority pollutants” under the USEPA Clean Water Act. Also called benzo[def]phenanthrene and pyrene[def]phenanthrene.

There are more than 100 different PAHs. Refer to the polynuclear aromatic hydrocarbons datasheet.

### Maximum Acceptable Value

There are insufficient data to derive MAVs for any of the polynuclear aromatic hydrocarbons in drinking-water other than benzo[a]pyrene.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for PAHs other than benzo[a]pyrene. However comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

### Sources to drinking-water

#### 1. To source waters

The principal route of entry to source water is via atmospheric deposition. Based on the literature reviewed, the presence of chrysene and benzo(k)fluoranthene may be indicators for coal combustion emissions. Whereas other PAHs are indicators of other combustion process: benzo(g,h,i)perylene, coronene and phenanthrene are indicators for motor vehicle emissions, pyrene and fluoranthene are associated with incineration and fluorene, fluoranthene and pyrene are associated with oil combustion; Environment Australia. 2003. Automobiles produce about 1 μg of pyrene per km.

Pyrene can comprise up to 2 percent of coal tar.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Treated water may be contaminated by leaching from coal-tar liners in water distribution systems. A large range of PAH compounds can be associated with carbon black which is used in rubber hose and fittings and some types of plastic pipes (IARC 2010). Coal-tar lining is not found very often in New Zealand today.

### Forms and fate in the environment

If released to soil, pyrene is expected to have no mobility based upon Koc values of 61,936-90,000. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 1.19 x 10-5 atm‑cu m/mole. However, adsorption to soil is expected to attenuate volatilisation. Direct photolysis by natural sunlight is also expected to be an important environmental fate process for pyrene based on photolysis studies performed on a variety of substrates. Biodegradation is expected to occur slowly, with estimated half-lifes ranging from several weeks to years. If released into water, pyrene is expected to adsorb to suspended solids and sediment based upon the Koc values. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 4.5 and 37 days, respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. The estimated volatilisation half-life from a model pond is 29 years if adsorption is considered. BCFs of 72–970 in trout suggest bioconcentration in aquatic organisms is moderate to high. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

Water solubility is about 0.14 mg/L.

### Typical concentrations in drinking-water

Twenty-one water utilities in the US reported detecting pyrene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.00055 mg/L.

### Removal methods

Polynuclear aromatic hydrocarbons are generally very insoluble in water and hence adsorb readily to available surfaces. As a result, conventional coagulation/flocculation is able to achieve high levels of removal by removing particles to which the polynuclear aromatic hydrocarbons are adsorbed, and by providing floc surfaces on to which polynuclear aromatic hydrocarbons in the bulk water may adsorb. Good removal can also be achieved by granular activated carbon.

PAHs in water can be oxidised by chlorination and ozonation. Pyrene was the most rapidly degraded PAH. Benz[a]anthracene, benzo[a]pyrene, and perylene were also highly degraded. Indeno[1,2,3-c,d]pyrene and benzo[g,h,i]pyrene were intermediate with respect to relative degradation. Benzo[k]fluoranthene and fluoranthene were the most slowly degraded of the compounds tested. A variety of complex end-products have been identified (ATSDR 1995).

### Analytical methods

#### Referee method

Refer to the polynuclear aromatic hydrocarbons datasheet.

### Health considerations

For non-smokers, food is regarded as the primary route of exposure. Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains, fats and oils, and in fish from contaminated waters. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition. PAHs are formed during some methods of food preparation, such as charbroiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient, tobacco smoke and indoor air. The use of open fires for heating and cooking may increase PAH exposure.

The health effect of primary concern is carcinogenicity. Many polynuclear aromatic hydrocarbon-containing mixtures have been associated with increased incidence of cancer, but the contribution of each of the individual components to the overall carcinogenic potency is difficult to assess. The relative carcinogenic potencies of various polynuclear aromatic hydrocarbons, based on bioassays by several routes of administration and related toxicological data, have been ranked in decreasing order as follows: dibenz[a,h]anthracene, benzo[a]pyrene, anthanthrene, indeno[1,2,3-cd]pyrene, benz[a]anthracene, benzo[b]fluoranthene, pyrene, benzo[k]fluoranthene, benzo[j]fluoranthene, cyclopentadieno[c,d]pyrene, benzo[g,h,i]perylene, chrysene and benzo[e]pyrene.

The US Environmental Protection Agency has determined that pyrene is not classifiable as to human carcinogenicity based on no human data and inadequate data from animal bioassays.

IARC (2010) classified pyrene in Group 3 (not classifiable as to carcinogenicity).

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA has a reference dose or RfD of 0.03 mg/L for pyrene.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The subchronic limit is 0.09 mg/L. The chronic health risk limits (exposure greater than 10 percent of a lifetime) for pyrene is 0.05 mg/L.

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# Pyruvic acid

CAS No. 127-17-3. The IUPAC name is 2-oxopropanoic acid. Also called acetylformic acid, α-ketopropionic acid, and pyroracemic acid.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for pyruvic acid. The WHO Guidelines do not refer to pyruvic acid.

### Sources to drinking-water

#### 1. To source waters

Pyruvic acid (CH3COCO2H) is an [alpha-keto acid](http://en.wikipedia.org/wiki/Keto_acids). The [carboxylate](http://en.wikipedia.org/wiki/Carboxylate) [anion](http://en.wikipedia.org/wiki/Anion) of pyruvic acid is known as pyruvate. Pyruvate plays an important role in biochemical processes. Pyruvic acid has been detected in some coffees (<0.2 percent dry weight).

Pyruvaldehyde (or methylglyoxal) is the cytotoxic agent in manuka honey – see glyoxal datasheet.

#### 2. From treatment processes

Stockham and Morran found pyruvic acid in water treated with polyDADMAC after ozonation. Pyruvic acid is the commonest ketoacid produced during ozonation; ketoacids tend to form higher concentrations than the aldehydes (Kostakis and Nicholson).

### Analytical methods

#### Referee method

No MAV so no need.

### Health considerations

Pyruvate (also known as pyruvic acid) occurs naturally in the body and is an end product of the metabolism of sugar or starch. It is formed from the sugar glucose (blood sugar) during the process known as glycolysis. Glycolysis is one of the energy generating pathways that our bodies use every second of every day to make ATP (adenosine triphosphate) – our “ultimate” energy molecule.

Pyruvate is a relatively new supplement to the athletic and sports nutrition industry. Pyruvate is the compound which starts the Krebs cycle. The Krebs cycle is an energy cycle which is directly responsible for the production and precursors of ATP (Energy). Pyruvate is currently thought to have numerous benefits which include: reduction of fatty mass, the lowering of blood lipids, improved endurance during aerobic activity, increased ATP/energy production, and a decrease in the chance of regaining previously lost fat mass.

### Derivation of Maximum Acceptable Value

No MAV.

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# Quinoline

CAS No. 91-22-5. Also called quinolin, 1-azanaphthalene, 1-benzazine, benzopyridine, leucoline, benzo[b]pyridine, 1-benzine, chinoleine, chinoline and leucol.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for quinoline. The WHO Guidelines do not refer to quinoline.

The USEPA concluded on 22 September 2009 that quinoline is known or anticipated to occur in PWSs and may require regulation. Therefore they added quinoline to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

### Sources to drinking-water

#### 1. To source waters

Coal tar is the principal source of commercial quinoline. Quinoline is used mainly as an intermediate in the manufacture of other products such as nicotinic acid, 8‑hydroxyquinoline sulphate and niacin. Quinoline is also used as a catalyst, a corrosion inhibitor, in metallurgical processes, in the manufacture of dyes, as a preservative for anatomical specimens, in polymers and agricultural chemicals, and as a solvent for resins and terpenes. It is also used as an antimalarial medicine. Quinoline has a strong disagreeable odour.

Quinoline may be formed as a trace pollutant during incomplete combustion of nitrogen-containing substances.

Potential sources of quinoline release to water include discharges of creosote (qv), coal tar and associated contaminated groundwater from contaminated sites at former coal gasification plants and former and existing steel plants equipped with coke ovens, coal tar distillation facilities, wood impregnation plants, aluminium smelters and leaking storage tanks. Much of the quinoline monitoring information is related to past industrial activities. Underground coal gasification has been a source of quinoline contamination of groundwater.

No data were located for drinking water. Concentrations of quinoline in groundwater range from <0.006 to 11,200 μg/L, in fresh surface water of 20 μg/L, in marine surface water of 0.0017 μg/L and in effluents from 0.02 to 10,000 μg/L (DWI 2014).

### Form and fate in the environment

In the case of abandoned gasworks, treatment that successfully deals with PAHs and BTEX should also address quinoline contamination.

If released to surface water, quinoline will remain for the most part in that compartment. Similarly, if released to soil, the molecule will remain mainly in soil. Quinoline breaks down quickly in surface water but is more persistent in groundwater.

Water solubility at 25°C is about 6,000 mg/L. The vapour pressure of quinoline is 0.009 mm Hg at 25°C and its log octanol/water partition coefficient (log Kow) is 2.0. DWI (2014) reports the log Kow = 1.88 to 2.06, Koc = 2.84 to 10.9, Henry’s Law constant = 1.67 x10-6 atm.m³/mol at 25°C.

### Removal methods

DWI (2014) reports:

Quinoline is expected to volatilise very slowly from water surfaces, based on a Henry’s Law constant of 1.67 x 10-6 atm.m3/mol at 25°C. Therefore, it is not expected to be amenable to air stripping.

Quinoline is expected to undergo some removal by absorption on to activated carbon. In a recent study using bamboo charcoal, a maximum absorption capacity of 91.7 mg/g was calculated using an initial concentration of 200 mg/L.

### Analytical methods

#### Referee method

No MAV so no need.

### Health considerations

Quinoline is considered *likely to be carcinogenic in humans* in accordance with proposed USEPA carcinogen risk assessment guidelines on the basis of observations of exposure-related increased incidence of an unusual malignant tumour in multiple strains of rats and mice, multiple experiments using oral, i.p. and s.c. dosing at an early age. This determination is supported by studies that demonstrate that quinoline is genotoxic. The USEPA previously classified quinoline as a Group C *possible human carcinogen* under the existing EPA cancer guidelines. However, recent evidence from mitogenicity and mutagenicity studies and two dietary studies in rats indicate that “sufficient” animal evidence exists, and that quinoline would now be classified as a Group B2 *probable human carcinogen* under the 1986 guidelines.

A potential exposure to quinoline may occur from the inhalation of cigarette smoke  
(1–20 µg/cigarette).

The USEPA has not established a Reference Dose ([RfD](http://www.epa.gov/ttn/atw/hlthef/hapglossaryrev.html#rfd)) for quinoline.

DWI (2014) states that the results of *in vitro* and *in vivo* assays are generally positive for mutagenicity. A LOAEL of 0.05 percent (500 mg/kg diet; approximately 25 mg/kg bw/day) was identified based on haemagioendothelioma and increased aspartate aminotransferase (AST) and alkaline phosphatase. Hence an oral Tolerable Daily Intake (TDI) of 0.025 mg/kg bw/day (25 μg/kg bw/day) is derived.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Rhodamine

Rhodamine B: CAS No. 81-88-9. The CAS name is *N*-[9-(2-carboxyphenyl)-6-(diethylamino)-3*H*-xanthen-3-ylidene]-*N*-ethylethanaminium chloride.

Rhodamine 6G: CAS No. 989-38-8. The CAS name is 2-[6-(ethylamino)-3-(ethylimino)-2,7-dimethyl-3*H*- xanthen-9-yl]benzoic acid ethyl ester, monohydrochloride.

These dyes are sold under a variety of trade names (see IARC 1978).

### Maximum Acceptable Value

The DWSNZ do not have a MAV for Rhodamine dyes. The WHO Guidelines do not refer to Rhodamine dyes.

### Sources to drinking-water

#### 1. To source waters

Rhodamine dyes can be used as a tracer in water to detect leaks, directions of flow, dispersion rates, and to calibrate flow meters. This is because they [fluoresce](http://en.wikipedia.org/wiki/Fluorescence), so can be detected easily at very low concentrations and inexpensively with instruments called [fluorometers](http://en.wikipedia.org/wiki/Fluorometer) (spectrofluorimeters).

A common water tracer grade is called Rhodamine WT (CAS No. 37299-86-8), called xanthylium, 9-(2,4-dicarboxyphenyl)-3,6-bis(diethylamino)-, chloride, disodium salt, by USEPA.

They have also been use to colour foods, and in fluorescence microscopy.

### Form and fate in the environment

Rhodamine dyes are highly soluble in water.

### Analytical methods

#### Referee method

No MAV so no need.

#### Some alternative methods

Can be measured at or below 0.0001 mg/L when using fluorometry or an appropriate custom-made sensor.

### Health considerations

Rhodamine B has been tested in mice and rats by subcutaneous injection and, in inadequate studies, by oral administration. It was carcinogenic in rats when injected subcutaneously, producing local sarcomas. Rhodamine 6G is carcinogenic in rats after its subcutaneous injection: it produces sarcomas. It has been inadequately tested in mice by oral administration.

Despite that, IARC has classified Rhodamine B and 6G in Group 3: Not classifiable as to carcinogenicity to humans.

Rhodamines are not classified as Hazardous Substances, Priority Pollutants or Toxic Pollutants under the CWA.

The toxicology of Rhodamine B is relatively well studied. The acute LD50 for Rhodamine B in mice is approximately 900 mg/kg/day (IARC 1987). Similarly, the i.p. LD50 of Rhodamine B in rats ranges from approximately 60 to 140 mg/kg. The subchronic toxicity of Rhodamine B was investigated in several studies (IARC 1987). The lowest exposure level reported to cause adverse effects is from a multigeneration study using Charles River rats (Bio/Dynamics 1981a). A dietary concentration of 0.02 percent (200 mg/kg chow) caused early mortality in F1 rats, an effect that was not observed at a dietary concentration of 0.002 percent (20 mg/kg chow). Assuming that rats consume food amounts equivalent to 5 percent of their body weight/day (USEPA 1986), the NOAEL in this study would be 1 mg/kg/day with a corresponding LOAEL of 10 mg/kg/day (USEPA 1997).

### Derivation of Maximum Acceptable Value

No MAV.

The standards established by USEPA in the *Federal Register* 63(40) state the maximum Rhodamine WT concentrations to be 10 micrograms per litre (0.01 mg/L) for water entering a drinking water plant (prior to treatment and distribution) and 0.1 micrograms per litre (0.0001 mg/L) in drinking water. This information was obtained in August 2004. See [www.smig.usgs.gov/SMIG/rhodamine\_reader.doc](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.smig.usgs.gov\SMIG\rhodamine_reader.doc).

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# Styrene

CAS No. 100-42-5. The IUPAC name is phenylethene. Also called ethenylbenzene, vinyl benzene, phenethylene, phenylethene, phenylethylene, styrol, styrolene and cinnamene, and others.

### Maximum Acceptable Value

Based on health considerations, the concentration of styrene in drinking-water should not exceed 0.03 mg/L.

The maximum contaminant level or MCL (USEPA 2006/2009/2011) is 0.1 mg/L. The USEPA (2006/2009/2011) established a lifetime health advisory of 0.1 mg/L, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on aesthetic considerations (odour), the concentration of styrene in drinking water should not exceed 0.004 mg/L. Styrene would not be a health concern unless the concentration exceeded 0.03 mg/L.

### Sources to drinking-water

#### 1. To source waters

Styrene is used predominantly (65 percent of total product) in the production of polystyrene plastics and resins. Styrene is also used as an intermediate in the synthesis of copolymers such as styrene-acrylonitrile (SAN) and acrylonitrile-butadiene-styrene (ABS), both representing approximately 9 percent of styrene use, and styrene-butadiene rubber (SBR), representing approximately 6 percent of styrene use. A related polymer, styrene-butadiene latex (approximately 7 percent), is used in making carpet, coatings for paper, and as part of latex paints. An additional 7 percent of styrene is formulated with unsaturated polyester resins in such things as boat hulls (fibreglass reinforcement materials). The remaining amounts of styrene produced are used for several types of applications, including less common thermoplastics, for laboratory and water purification uses (ion-exchange resins) and glues and adhesives. Styrene copolymers are also frequently used in liquid toner for photocopiers and printers (ATSDR 2007). The commercial product normally contains a very small amount (12 to 15 ppm) of tertiary butyl catechol (4-tert-butylpyrocatechol or 4-tert-butylbenzene-1,2-diol) as a polymerisation inhibitor.

Styrene may occur in source waters as a result of industrial contamination or the breakdown of discarded polystyrene products in landfills. Styrene is used extensively in New Zealand in the production of resins for the fibreglass industry. Polystyrene is not manufactured here but is moulded into many products. Forest fires may contribute to atmospheric concentrations of styrene. It is also a component of diesel exhaust.

Small amounts of chlorinated styrenes (mainly ortho- and para-styrene) are manufactured, mainly to produce polychlorostyrene (NICNAS 2002).

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

There is a possibility of leaching small amounts of [styrene monomer](http://www.shellchemicals.com/styrene_monomer/1,1098,347,00.html) from [ABS (Acrylonitrile Butadiene Styrene)](http://en.wikipedia.org/wiki/Acrylonitrile_butadiene_styrene) when in contact with water for a long time. To get the least amount of leaching try to get ABS pipe that meets [ANSI/NSF Standard 61](http://www.curaflo.com/CuraFlo/AboutEpoxyPipeLining/WaterSafetyCertified/ansinsf61faq.htm) or equivalent.

Glass reinforced polyester (GRP) tanks are made from a resin solution. This is usually by dissolving an unsaturated polyester resin into styrene which is then used to make a polyester moulding compound by mixing together the resin solution with fillers, thickening agents and other manufacturing additives. Styrene analysis should routinely be carried out for this type of product (DWI 2001).

### Form and fate in the environment

Styrene is expected to be volatilised readily to the atmosphere where it will react with hydroxyl radicals and ozone. Biodegradation is the major route of removal of styrene from soils; microbes isolated from landfill soil degraded 95 percent of the styrene present in 16 weeks (USEPA 1994).

Styrene rapidly volatilises from surface water with estimated half-lifes from a river or pond of 0.6 days and 13 days respectively (USEPA 1994). In surface water styrene will oxidise and form peroxides or aldehydes with a penetrating odour. It is biodegraded and not bioaccumulated. Its solubility in water is about 250–400 mg/L.

EU (2002) states: vapour pressure = 5 mm Hg (667 Pa) at 20°C; octanol-water partition coefficient = log Kow = 3.02. Styrene does not adsorb solar radiation appreciably at wavelengths greater than 300 nm, therefore degradation of styrene by direct photolysis is unlikely, and styrene contains no hydrolysable groups. Styrene can be biodegraded quite readily in water under aerobic conditions. The biodegradation half-life in water has been estimated to be 2–4 weeks and 300 days in sediment. Styrene degrades more slowly in groundwater than in surface waters – half-life estimated between 4 and 30 weeks.

If released to soil, styrene is expected to have low to moderate mobility based upon an estimated Koc of 960. Volatilisation from moist soil surfaces is expected to be an important fate process. For example, in 1.5 cm deep samples of a loamy soil, 26 percent of 2 mg/kg styrene that was added volatilised in 31 days. Styrene may volatilise from dry soil surfaces based upon its vapour pressure. Biodegradation by aerobic microorganisms may lead to extensive or complete destruction of styrene in soil. It was found that 97 and 87 percent of 8–14C-styrene added to soil at levels of 2.0 g/kg was converted to 14C-CO2 in 16 weeks in a landfill soil and sandy loam soil, respectively. If released into water, styrene is expected to adsorb to suspended solids and sediment based upon the estimated Koc. In lake water, 10 to 20 percent mineralisation was observed in three weeks with samples containing 2.5 ug to 1.0 mg/L styrene. Degradation of styrene is rapid in sewage under aerobic conditions. Volatilisation from water surfaces is expected to be rapid. Under laboratory conditions, 50 percent of 2 to 10 mg styrene per litre (depth not specified) was lost by volatilisation in one to three hours in lake water samples and in six to seven hours in distilled water. A BCF of 13.5 for goldfish suggests bioconcentration in aquatic organisms is low. Styrene is not expected to undergo hydrolysis in the environment due to the lack of hydrolysable functional groups (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 301 zones, did not find styrene at detectable concentrations (limit of detection = 0.0005 mg/L) (ESR 2001).

Styrene has been detected in drinking-water and surface water at concentrations below 0.001 mg/L (WHO 2004). It is only very rarely found in groundwater.

203 water utilities in the US reported detecting styrene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.02 mg/L.

The maximum concentration found in 7326 samples from 2540 groundwaters in the UK was 0.002 mg/L (DWI 2008).

### Removal methods

Granular activated carbon can be used to remove styrene from water. It can be oxidised also by ozone. The oxidation products (aldehydes, ketones and benzoic acid) may themselves need to be removed if they are present in sufficient quantities.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

Low levels of styrene occur naturally in plants as well as a variety of foods such as fruits, vegetables, nuts, eggs, beverages, and meats. The daily exposure to styrene has been estimated to be 0.04 mg per person, with smokers receiving a higher dose. Exposure to styrene is most likely to occur from breathing indoor air that is contaminated with styrene vapours from building materials, tobacco smoke, and use of copying machines. Exposure may also occur by breathing automobile exhaust.

Uptake of styrene is rapid and it is distributed widely in the whole body, with a preference for lipids. It is metabolised by a number of tissues and organs to styrene‑7,8-oxide. Elimination of styrene from lipid depots is less rapid (half-life two to four days) than from other tissues and no tendency for long-term accumulation exists. More than 90 percent of the oral dose is excreted rapidly as metabolites in urine.

Styrene has a low acute toxicity.

Short-term controlled studies in volunteers exposed to styrene by inhalation at concentrations above 210 mg/m3 in air showed that it can cause irritation of mucous membranes of eyes, nose and/or respiratory tract and depression of the central nervous system.

No chromosomal aberrations in peripheral lymphocytes could be detected in workers occupationally exposed to low concentrations of styrene, but significantly elevated frequencies of chromosomal aberrations were observed in peripheral lymphocytes of workers occupationally exposed to much higher styrene concentrations.

Based on the available data, the International Agency for Research on Cancer (IARC 2002) has classified styrene in Group 2B (possibly carcinogenic to humans). It possesses mutagenic properties in *in vitro* systems with metabolic activation only. *In vivo* studies showed positive effects only at high doses. The available data suggest that the carcinogenic effects of styrene are due to the formation of the carcinogenic metabolite styrene-7,8-oxide as a consequence of overloading the detoxication mechanisms after exposure to high styrene levels.

Styrene oxide appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.1 mg/kg/day for acute-duration oral exposure (1–14 days) to styrene.

The reference dose or RfD (USEPA 1990/2006/2009/2011) for styrene is 0.2 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 7 mg/L.

Styrene is on the EC List of 66 Category 1 substances showing evidence of endocrine disrupting activity in at least one species using intact animals (EC 2015).

### Derivation of Maximum Acceptable Value

A tolerable daily intake approach has been used for the derivation of a MAV for styrene in drinking-water. A no-observable-adverse-effect level determined for reduced body weight in a two-year drinking-water study in rats has been used as the basis of the derivation.

The MAV for styrene in drinking-water was derived as follows:

7.7 mg/kg body weight per day x 70 kg x 0.1 = 0.027 mg/L (rounded to 0.03 mg/L)

2 L x 1000

where:

* no-observable-adverse-effect level = 7.7 mg/kg body weight per day for reduced body weight from a 2-year drinking-water study in rats
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for carcinogenicity and genotoxicity of the reactive intermediate styrene-7,8-oxide).

The odour threshold for styrene has been reported at a concentration as low as 0.004 mg/L. The taste threshold of styrene in water at 40°C ranges from 0.02 mg/L to 2.6 mg/L, depending on individual sensitivities.

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# Tetrachlorobenzenes

Tetrachlorobenzene may refer to any of three [isomeric](http://en.wikipedia.org/wiki/Isomer) chemical compounds (congeners):

* [1,2,3,4-tetrachloro](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane)benzene (CAS No. 634-66-2; also called 1,2,3,4-benzene tetrachloride
* [1,2,3,5-tetrachloro](http://en.wikipedia.org/wiki/1,1,2,2-Tetrachloroethane)benzene (CAS No. 634-90-2)
* 1,2,4,5-tetrachlorobenzene (CAS No. 95-94-3); also called s-tetrachlorobenzene.

### Maximum Acceptable Value

Tetrachlorobenzenes are not mentioned in the WHO Guidelines, and there are no MAVs in the DWSNZ.

### Sources to drinking-water

#### 1. To source waters

1,2,3,4-Tetrachlorobenzene has been used as a component in dielectric fluids and in the synthesis of fungicides. 1,2,4,5-Tetrachlorobenzene has been used as an intermediate for herbicides and defoliants, insecticide moisture-resistant impregnant, in electric insulation, and in packing protection.

Commercial 1,2,4-trichlorobenzene may contain up to 0.5 percent tetrachlorobenzenes.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Retention of chlorobenzenes in soil increases with the organic content of the soil; there is a positive correlation between the degree of chlorination of the compound and its adsorption on organic matter.

Tetrachlorobenzenes are resistant to microbial degradation in anaerobic soils and groundwater.

1,2,3,4-Tetrachlorobenzene is expected to have low mobility in soils based upon log Koc values in the range of 3.5–4.7 measured in soils and sediment. Volatilisation of 1,2,3,4-tetrachlorobenzene from dry soil surfaces is not expected to be important based upon the vapour pressure of this compound. Volatilisation from moist soil surfaces is expected based on the Henry’s Law constant of 6.9 x 10-4 atm‑cu m/mole at 25°C, but adsorption may attenuate this process. Biodegradation of 1,2,3,4‑tetrachlorobenzene is expected to occur slowly based on a half-life of 34.5 days in a sewage sludge amended soil and a half-life of 18 days in anaerobic river sediment. In water, 1,2,3,4-tetrachlorobenzene is expected to adsorb to sediment or particulate matter based on its measured Koc values. This compound is expected to volatilise from water surfaces given its Henry’s Law constant, but adsorption may attenuate this process. Estimated volatilisation half-lifes for a model river and model lake are 6 and 150 hours , respectively when neglecting adsorption. The volatilisation half-life from a model pond is about 128 days when adsorption is considered. When irradiated with light greater than 285 nm, this compound was 46 percent degraded in a water solution within 40 hours, suggesting that photolysis in surface waters may be important. The potential for bioconcentration in aquatic organisms is considered high based on BCF values in the range of 490 to 1,700 measured in carp and log BCF values of 3.7-4.1 measured in trout (EAWAG accessed February 2015).

The solubility of tetrachlorobenzenes in water is:

* [1,2,3,4-tetrachloro](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane)benzene: 12 mg/L
* [1,2,3,5-tetrachloro](http://en.wikipedia.org/wiki/1,1,2,2-Tetrachloroethane)benzene: 2.8 mg/L
* 1,2,4,5-tetrachlorobenzene: 2.2 mg/L.

### Typical concentrations in drinking-water

In general, the lower chlorinated benzenes are detected more frequently in drinking-water and are present in higher concentrations; 1,2,3,4-tetrachlorobenzene ranged from 0.1 to 0.4 ng/L in three cities beside Lake Ontario. 1,2,4,5-tetrachlorobenzene ranged from 0.0 to 0.3 ng/L in three cities beside Lake Ontario. 1,2,3,5-tetrachlorobenzene was <0.1 ng/L. Reported in IPCS (1991).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Intake from food compared with that from other sources increases with increasing degree of chlorination; food contributes a greater percentage of the total daily intake of tetrachlorobenzenes and pentachlorobenzene than air. However, exposure levels for such congeners are likely to be less than 0.05 µg/kg body weight. A limited number of studies have shown that, on a body weight basis, breast-fed infants may receive a higher dose of chlorobenzenes than members of the adult population.

Long-term exposure studies (up to 6 months) on several species of experimental animals indicated a trend for the toxicity of chlorobenzenes to increase with increased ring chlorination. However, there was considerable variation in the long-term toxicities of different isomers of the same congener. Major target organs were the liver and kidney; at higher doses, effects on the haematopoietic system were reported and thyroid toxicity was noted in studies on 1,2,4,5-tetrachlorobenzene and pentachlorobenzene.

The lowest reported NOELs (in rats, by ingestion) reported in IPCS (1991) were:

* 1,2,3,4-tetrachlorobenzene: 3.4 mg/kg/day corresponding to a TDI of 0.01
* 1,2,3,5-tetrachlorobenzene: 0.42 mg/kg/day corresponding to a TDI of 0.001
* 1,2,4,5-tetrachlorobenzene: 0.034 mg/kg/day corresponding to a TDI of 0.0001.

These tolerable daily intakes were based on an uncertainty factor of 500. The TDIs of the tetrachlorobenzenes are lower than the other chlorobenzenes (other than hexachlorobenzene).

The USEPA (1991) established a NOAEL of 0.34 mg/kg/d for oral exposure to 1,2,4,5‑tetrachlorobenzene, and a RfD of 0.0003 mg/kg/d based on an uncertainty factor of 1000, resulting from the appearance of kidney lesions in a subchronic rat study. The RfD is an estimate of the highest daily oral exposure humans can be exposed to without resulting in harmful effects.

### Derivation of Maximum Acceptable Value

No MAV.

The thresholds for smell and taste for 1,2,4,5-tetrachlorobenzene were reported in IPCS (1991) at 0.006 mg/L and 0.0064 mg/L, respectively.

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# Tetrachloroethane

Tetrachloroethane may refer to either of two [isomeric](http://en.wikipedia.org/wiki/Isomer) chemical compounds:

* [1,1,1,2-tetrachloroethane](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane) (CAS No. 630-20-6). Also called (chloromethyl)trichloromethane
* [1,1,2,2-tetrachloroethane](http://en.wikipedia.org/wiki/1,1,2,2-Tetrachloroethane) (CAS No. 79-34-5). Can also be called sym-tetrachloroethane, or s-tetrachloroethane, or acetylene tetrachloride.

### Maximum Acceptable Value

Tetrachloroethane is not mentioned in the WHO Guidelines, and there is no MAV in the DWSNZ.

The USEPA concluded on 22 September 2009 that [1,1,1,2-tetrachloroethane](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane) is known or anticipated to occur in PWSs and may require regulation. Therefore they added [1,1,1,2-tetrachloroethane](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane) to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009).

The USEPA (2006/2011) established a lifetime health advisory of 0.07 mg/L for [1,1,1,2‑tetrachloroethane](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane), where the lifetime health advisory is the concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The USEPA (2006) established a lifetime health advisory of 0.0003 mg/L for [1,1,2,2‑tetrachloroethane](http://en.wikipedia.org/wiki/1,1,1,2-Tetrachloroethane), where the lifetime health advisory is the concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity. This lifetime health advisory did not reappear in USEPA (2009 or 2011).

1,1,2,2-Tetrachloroethane is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

1,1,1,2-Tetrachloroethane has a sweet [chloroform](http://en.wikipedia.org/wiki/Chloroform)-like odour. It is (was) used as a [solvent](http://en.wikipedia.org/wiki/Solvent), eg, in the manufacture of pesticides, and in the production of [wood stains](http://en.wikipedia.org/wiki/Wood_stain) and [varnishes](http://en.wikipedia.org/wiki/Varnish). As a Freon [refrigerant](http://en.wikipedia.org/wiki/Refrigerant), it was used under the name R-130a. This material is listed in Schedule 2 of the Control of Substances Hazardous to Health Regulations (COSHH) 1999. Its use in the UK is banned for diffusive applications such as surface or fabric cleaning except for R&D and analysis. 1,1,1,2-Tetrachloroethane may be formed incidentally during the manufacture of other chlorinated ethanes and released into the environment as air emissions or in wastewater. 1,1,1,2-Tetrachloroethane is found as an impurity in technical-grade 1,1,2,2-tetrachloroethane.

1,1,2,2-Tetrachloroethane has the highest solvent power of any [chlorinated hydrocarbon](http://en.wikipedia.org/wiki/Chlorinated_hydrocarbon). As a Freon [refrigerant](http://en.wikipedia.org/wiki/Refrigerant), it was used under the name R-130. It was once widely used as a [solvent](http://en.wikipedia.org/wiki/Solvent) and as an intermediate in the industrial production of [trichloroethylene](http://en.wikipedia.org/wiki/Trichloroethylene), [tetrachloroethylene](http://en.wikipedia.org/wiki/Tetrachloroethylene), and [1,2-dichloroethylene](http://en.wikipedia.org/wiki/1,2-dichloroethylene), and in cleaning and degreasing metals, in paint removers, varnishes and lacquers, in photographic films, as an extractant for oils and fats, and in pesticides. However, 1,1,2,2-tetrachloroethane is no longer used much due to concerns about its toxicity; hence it is rarely found in water now.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Most of the two tetrachloroethanes released to the environment eventually move to the air or groundwater. 1,1,2,2-Tetrachloroethane released to the aquatic environment is rapidly removed via volatilisation, with an estimated half-life of 6.2 hours from running water and 3.5 days from still water. Hydrolysis and biodegradation are the principal routes of removal from groundwater. The hydrolysis half-life in subsurface sediment at 25°C was determined to be 29 days. Neutral and base-catalysed hydrolyses of 1,1,2,2-tetrachloroethane in pure water yielded trichloroethene as essentially the sole degradation product (WHO 1998). 1,1,2,2-Tetrachloroethane is not readily biodegradable. It is expected to undergo dehydrochlorination under hydrolytic alkaline conditions to trichloroethylene, and to biodegrade under anaerobic conditions. The tetrachloroethanes are not likely to adsorb to soil or sediments.

The solubility of 1,1,1,2-tetrachloroethane in water is about 1,100 mg/L, and the solubility of 1,1,2,2-tetrachloroethane in water is about 2,900 mg/L.

The octanol/water partition coefficient (P) for 1,1,1,2-tetrachloroethane: log P, 2.93. The octanol/water partition coefficient (P) for 1,1,2,2-tetrachloroethane: log P, 2.39 (IARC 2013).

### Typical concentrations in drinking-water

Eighteen water utilities in the US reported detecting 1,1,1,2-tetrachloroethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.014 mg/L.

Ten water utilities in the US reported detecting 1,1,2,2-tetrachloroethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.17 mg/L.

### Removal methods

Potential treatment technologies for removing 1,1,2,2-tetrachloroethane from water include air-stripping and activated carbon. Contaminants with an adsorption capacity expressed as Freundlich isotherm constant (K) >200 µg/g are considered to be amenable to GAC treatment; the Freundlich isotherm constant (K) value for 1,1,2,2‑tetrachloroethane is 823 µg/g (from USEPA 2008).

WRF (2014) reports that 1,1,1,2-tetrachloroethane is characterised with a low Henry’s law constant (0.077 dimensionless air/water). However, the results showed that low profile air stripping is effective for 1,1,1,2-tetrachloroethane removal. 1,1,1,2‑Tetrachloroethane was almost completely removed at the three temperatures and air to water ratio of about 150-167. A 98.9 percent removal was observed at 12°C and air to water ratio of about 70 and lower removal efficiency (94.5 percent) was observed at 4°C. The removal efficiency remained high at the lowest air to water ratio, 97.4 percent, 94 percent, and 77.3 percent removal efficiencies were achieved at temperatures of 20°C, 12°C and 4°C respectively. The temperature effect also became noticeable at the lowest air to water ratio. All experiments succeeded to produce sub ug/L effluents except the experiment of 53 air to water ratio at 4°C.

WRF (2014) reports that 1,1,2,2-tetrachloroethane is characterised with a low Henry’s law constant (0.0139 dimensionless air/water), which is the second lowest among the 13 focus VOCs trialled. Low profile air stripping is effective for 1,1,2,2-tetrachloroethane removal at high air to water ratios, and at high temperatures at moderate air to water ratios. 1,1,2,2-Tetrachloroethane was almost completely removed at the three temperatures and air to water ratio of above 500. Lower removal efficiencies (97.6 percent, 87.3 percent and 65 percent) were observed at lower air to water ratios (150, 167 and 162), sub ug/L effluent was not achieved at 4°C, and the temperature change effect was significant at this range of air to water ratios. At air to water ratio of 72 the removal efficiency at 12°C dropped to 66 percent and at the air to water ratio of 70 the removal efficiency at 4°C dropped to 30.6 percent, both did not achieve sub ug/L effluent. At the low air to water ratio of about 53, the removal efficiency dropped to between 58.3 percent and 19.7 percent, at temperatures between 20°C and 4°C, and no sub ug/L effluent was achieved.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

See IARC (2013).

### Health considerations

Most of the two tetrachloroethanes, when ingested, will enter the bloodstream. The inhalation route is more important than oral; 1,1,2,2-tetrachloroethane has been found in cigarette smoke at 3–6 µg per cigarette (USEPA 2008).

The toxicological profile of 1,1,2,2-tetrachloroethane has also not been well characterised; because of the chemical’s declining use, available data are confined primarily to early limited studies. Based on the results of available *in vivo* and *in vitro* assays, 1,1,2,2-tetrachloroethane has, at most, weak genotoxic potential.

OECD (2005) states that 1,1,2,2-tetrachloroethane can be considered as very toxic to humans exposed acutely. In an oral long term bioassay, 1,1,2,2-tetrachloroethane has been shown to induce hepatocellular carcinoma in mice but it was not carcinogenic in rats. The lowest oral threshold dose (LOAEL) in rats was found around 3 mg/kg bw/day indicating large margins of safety when comparing with the trace levels of 1,1,2,2‑tetrachloroethane when it is detected in food or drinking water in northern America.

The International Agency for Research on Cancer (IARC) has determined that 1,1,1,2‑tetrachloroethane and 1,1,2,2-tetrachloroethane cannot be classified as to their ability to cause cancer in humans (ie, Group 3). However, IARC (2013) evaluated them both as *possibly carcinogenic to humans (Group 2B).*

The USEPA has classified 1,1,2,2-tetrachloroethane as a Group C possible human [carcinogen](http://en.wikipedia.org/wiki/Carcinogen) based on increased incidence of hepatocellular carcinomas in mice (although negative results were obtained on rats); similar findings were obtained for 1,1,1,2-tetrachloroethane. The profile for tumour induction by 1,1,2,2‑tetrachloroethane is similar to that of dichloroacetic acid, its primary metabolite. The USEPA (2009/2011) quotes a health advisory of 0.1 mg/L for 1,1,1,2‑tetrachloroethane, representing a 10-4 cancer risk.

USEPA (2008) reports health advisories for 1,1,2,2-tetrachloroethane in drinking water:

* 1- and 10-day, 10 kg child: 3 mg/L
* longer-term, 10 kg child: 0.4 mg/L
* longer-term, adult: 1 mg/L
* DWEL: 0.4 mg/L.

1,1,2,2-Tetrachloroethane has been classified as *likely to be carcinogenic to humans* so a lifetime health advisory is not recommended; the cancer risk at the DWEL is 1 x 10-3. The concentration in drinking water corresponding to a 10-6 risk is 4 x 10-4 mg/L; to a 10-5 risk is 4 x 10-3 mg/L; and that for a 10-4 risk is 4 x 10-2 mg/L (ie, 0.04 mg/L) (USEPA 2008).

1,1,2,2-Tetrachloroethane appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.5 mg/kg/day for intermediate-duration oral exposure  
(15–364 days) to 1,1,2,2-tetrachloroethane, based on a BMDL10 of 53.88 mg/kg/d for hepatic necrosis in rats.

The oral reference dose or RfD (USEPA 1996/2006/2009/2011) for 1,1,1,2‑tetrachloroethane is 0.03 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 1 mg/L.

The reference dose or RfD (USEPA 2006) for 1,1,2,2-tetrachloroethane was 0.00005 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006) was 0.002 mg/L. USEPA (2009/2010/2011) changed these to 0.02 and 0.4 mg/L respectively. USEPA (2010) quotes a subchronic oral RfD of 0.05 mg/kg/d.

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA (1987) derived a drinking-water risk estimate (10-5 or 1 in 10,000) of 0.002 mg/L for 1,1,2,2-tetrachloroethane. The USEPA has determined that exposure to 1,1,2,2-tetrachloroethane in drinking-water at a concentration of 0.04 mg/L for up to 10 days is not expected to cause any adverse effects in a child, and that lifetime exposure to 0.0003 mg/L of 1,1,2,2 tetrachloroethane in drinking-water is not expected to cause any adverse effects. The odour threshold of 1,1,2,2-tetrachloroethane in water is 0.5 mg/L (USEPA 2008).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for 1,1,1,2-tetrachloroethane is 0.07 mg/L, and the cancer health risk for 1,1,2,2-tetrachloroethane is 0.002 mg/L.

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# Tetrachloroethene

CAS No. 127-18-4. Also known as tetrachloroethylene, 1,1,2,2-tetrachloroethylene, ethylene tetrachloride, perchloroethylene, perchlorethylene, carbon dichloride, PCE or perc, and many trade names.

### Maximum Acceptable Value

Based on health considerations, the concentration of tetrachloroethene in drinking-water should not exceed 0.05 mg/L.

The maximum contaminant level or MCL (USEPA 2006/2009/2011) is 0.005 mg/L. The USEPA (2006/2011) established a lifetime health advisory of 0.01 mg/L, where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The maximum acceptable concentration in Canada is 0.03 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of tetrachloroethene in drinking water should not exceed 0.05 mg/L.

The Prescribed Concentration or Value (PCV) for tetrachloroethene in England and Wales is 0.01 mg/L. See Notes.

Tetrachloroethene is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Tetrachloroethene can be released to the aquatic environment as an industrial contaminant. It is used as a solvent, metal degreaser, and in the dry-cleaning industry; in 1990, at least 53 percent of world demand for tetrachloroethene was for dry cleaning. It is still the main dry-cleaning solvent used in New Zealand. It typically has a purity of 95 percent or more for dry-cleaning and industrial grades, 99 percent or more for more refined grades, and 99.995 percent for isomerisation and fluorocarbon grades. The commercial product may contain stabilisers, the main one being 2,3‑epoxypropyl isopropylether (EU 2005).

Water may enter the dry cleaning cycle for several reasons. A small amount of water is sometimes added to the solvent to remove water soluble impurities. Water may be contained in the clothes; the amount contained varies depending upon the fabrics. Water is also added at the end of the distillation stage in order to dry the sludge. With open-circuit equipment the waste air stream in the final drying stage may be passed through an activated carbon bed and water may be used to regenerate the activated carbon. The water and solvent in the contact water are separated in a separator where the heavy solvent forms a bottom layer and water rises to the top and may be run off. The separated water may contain tetrachloroethylene up to its solubility limit in water (approximately 150 mg/L) (EU 2005).

Production in the EU and USA has approximately halved over the last 10–20 years (WHO 2006). It is also used as a degreasing solvent in metal industries, as a heat transfer medium and in the manufacture of fluorohydrocarbons. The dry cleaning and technical grades often include amines or mixtures of epoxides and esters which are added as stabilisers to prevent decomposition, up to 0.35 percent. It has also been used as a veterinary anthelmintic.

Tetrachloroethene is reported to be produced by several temperate and sub-tropical marine macro-algae; this natural production may be an important part of the global atmospheric chlorine budget (ATSDR 1997).

EU (2005) states that in surface (river) waters the measured concentrations range from 0.00001 mg/L to 0.17 mg/L, with typical concentrations below 0.005 mg/L. Rainwater generally contains <0.1 µg/L. Groundwater concentrations of tetrachloroethylene vary widely, but generally they appear to be higher than surface water concentrations. This could be because measurements in groundwater are often taken where a problem (eg, spill) is thought to exist. The majority of groundwater levels are below 0.01 mg/L, though concentrations as high as 1.30 mg/L have been reported for a contaminated site.

#### 2. From treatment processes

Tetrachloroethene may be formed from naturally occurring organic precursors during the chlorination of drinking-water.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Most tetrachloroethene released to the environment is found in the atmosphere. In water, tetrachloroethene does not readily undergo hydrolysis or photolysis. Water solubility is about 150 mg/L (ATSDR 1997), or 206 mg/L at 25°C (IARC 2013). In water, it is resistant to abiotic and aerobic degradation, but it is biodegraded under anaerobic conditions to yield trichloroethene, dichloroethene, vinyl chloride, ethane and ethylene. Tetrachloroethene can persist in waters where volatilisation cannot occur (half-life from 3 hours to 14 days, or even >30 days in still water). It volatilises less readily from soil than from water. It is expected to be fairly mobile in soils (particularly sandy soils) and hence is likely to leach into groundwater, where it is quite stable. Tetrachloroethene does not appear to bioaccumulate in animals or food chains.

EU (2005) quotes: vapour pressure = 1.9 kPa at 20°C; octanol-water partition coefficient (log Kow) = 2.53; Henry’s law constant = 2,114 Pa m3/mole at 20ºC. Degradation of tetrachloroethylene in water by hydrolysis is very slow, with half lifes in the order of years reported. Tetrachloroethylene does not appear to undergo aerobic biodegradation. Test results indicate that tetrachloroethylene can be adsorbed onto soils of varying organic carbon contents; the amounts adsorbed though are negligible, hence tetrachloroethylene is relatively mobile in groundwater in the absence of any removal processes.

If released to soil, tetrachloroethylene is expected to have moderate mobility based upon Koc values in the range of 200–237; tetrachloroethylene has often been detected in groundwater. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 0.0177 atm‑cu m/mole. Tetrachloroethylene may volatilise from dry soil surfaces based upon its vapour pressure. Volatilisation half-lifes in the range of 1.2–5.4 and 1.9–5.2 hours were measured from a sandy loam soil surface and an organic topsoil, respectively. Using soil microcosms (60 percent municipal solid, 40 percent bulk, industrial, and sewage treatment sludge), tetrachloroethylene, present at 5 ug/L, exhibited no degradation when incubated at 20°C, indicating that biodegradation is not a fast environmental fate process in soil. There is evidence that slow biodegradation of tetrachloroethylene occurs under anaerobic conditions when the microorganisms have been accli7mated, yielding trichloroethylene as a product. If released into water, tetrachloroethylene is not expected to adsorb to suspended solids and sediment in water based upon the Koc data. The biodegradation half-lifes of tetrachloroethylene in aerobic and anaerobic waters were reported as 180 and 98 days, respectively, suggesting that biodegradation is not a fast environmental fate process in water. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are four hours and five days, respectively. Measured BCF values of 26–115 in fish indicate that bioconcentration in aquatic organisms is low to high. Hydrolysis is not expected to be an important environmental fate process based on a hydrolysis half-life of nine months. Tetrachloroethylene may undergo indirect photolysis in natural waters when photosensitisers such as humic material are present (EAWAG accessed February 2015).

Its octanol-water partition coefficient is reported to be in the range 758–2512 (log Kow= 2.9 – 3.4), and the vapour pressure is 2.5 kPa at 25°C. Dichloroacetic acid and trichloroacetic acid are metabolites (these have separate datasheets) (IARC 2013).

### Typical concentrations in drinking-water

The 1992 review of organic contaminants in New Zealand drinking-water supplies from 1987 to 1992 contained tetrachloroethene results from 52 samples. It was found in only one of these samples, at a concentration of 0.0017 mg/L.

The P2 Chemical Determinand Identification Programme, sampled from 505 zones, found tetrachloroethene in 7 zones at concentrations ranging from 0.001 to 0.004 mg/L, with the median concentration being “nd” (limit of detection = 0.001 mg/L) (ESR 2001).

Concentrations in drinking-water are generally below 0.003 mg/L, although much higher levels have been detected in groundwater (0.023 mg/L), and 1 mg/L in contaminated groundwater (WHO 2004).

In a national survey of 481 municipal and 215 private domestic groundwater supplies, Health Canada (1995) reports less than 4 percent of supplies contained tetrachloroethylene concentrations above 1 µg/L. The average of their maximum concentrations was 22 µg/L for municipal supplies and 13,380 µg/L for private supplies (the latter was heavily influenced by two or three contamination incidents). It was estimated that about 1.7 million of the 7.1 million Canadians who rely on groundwater for household use were covered by this survey and that tetrachloroethylene was detected in water supplying about 663 000 (40 percent) of that population.

803 water utilities in the US reported detecting tetrachloroethylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.015 mg/L. See Table 1.3 in IARC (2013) for examples of water contamination.

### Removal methods

Tetrachloroethene present in contaminated source waters can be removed by adsorption on to granular activated carbon, or more cost-effectively by air stripping. See Health Canada (1995) for further details. At the household level, boiling for  
3–5 minutes may remove up to 99 percent of tetrachloroethylene from water.

Although considered to arise predominantly from industrial contamination in overseas waters, there is evidence that tetrachloroethene is also formed as a disinfection by-product.

When its appearance in a water results from chlorination, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

Tetrachloroethene, once present as a disinfection by-product, can be removed by adsorption on to granular activated carbon, or by air stripping.

WRF (2014) reports that tetrachloroethylene is characterised with a high Henry’s law constant (0.53 dimensionless air/water). However, the results showed that low profile air stripping is effective for tetrachloroethylene removal. Tetrachloroethylene was almost completely removed at the three temperatures and air to water ratio of about 150. A complete removal was observed at 12°C and air to water ratio of about 70 slightly lower removal efficiency (99.8 percent) was observed at 4°C. The removal efficiency remained high at the lowest air to water ratio, 99.9 percent, 99.7 percent, and 98.3 percent removal efficiencies were achieved at temperatures of 20°C, 12°C and 4°C respectively. The temperature effect also became noticeable at the lowest air to water ratio.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551).

### Health considerations

Results from animal studies indicate that tetrachloroethene is absorbed rapidly and completely from the gastrointestinal tract. It is eliminated primarily by the lungs. In the body it is metabolised to trichloroacetic acid.

The most notable acute effect of short-term exposure is depression of the central nervous system. Longer-term studies of up to 3 months, using mice and rats, reported weight loss, and some evidence of liver and kidney toxicity at high doses. Inhalation exposures have resulted in maternal and foetal toxicity in mice, rats and rabbits.

In humans, oral doses of 4.2 to 6 g tetrachloroethene administered to patients to control worm infections caused central nervous system effects, such as inebriation, perceptual distortion and exhilaration. Several developmental effects, such as eye, ear, central nervous system, chromosomal, and oral cleft anomalies, were associated with exposure to tetrachloroethene and other solvents in contaminated drinking-water supplies. Inhalation exposures have been associated with reproductive effects in female dry cleaners, including menstrual disorders and spontaneous abortions.

IARC has classified tetrachloroethene in Group 2A (probably carcinogenic to humans); this was confirmed in IARC. 2013. It reportedly produces liver tumours in mice, with some evidence of mononuclear cell leukaemia in rats and kidney tumours in male rats. The overall evidence from studies conducted to assess the genotoxicity of tetrachloroethene, including induction of single-strand DNA breaks, mutation in germ cells, and chromosomal aberrations *in vitro* and *in vivo*, indicates that tetrachloroethene is not genotoxic. This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at October 2015 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for tetrachloroethene:

* 0.008 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.008 mg/kg/day for intermediate-duration oral exposure (14–365 days)
* 0.008 mg/kg/day for acute-duration oral exposure (one year or more).

The reference dose or RfD (USEPA 1988/2006/2009/2011) is 0.01 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.5 mg/L.

USEPA (2012) states that the RfD of 0.006 mg/kg-day replaces the previous RfD of 0.01 mg/kg-day entered on the IRIS database on 03/01/1988. The previous RfD had been based on a NOAEL of 14 mg/kg-day.

### Derivation of Maximum Acceptable Value

In view of the overall evidence for non-genotoxicity and evidence for a saturable metabolic pathway leading to kidney tumours in rats, it is considered appropriate to use a tolerable daily intake approach for the derivation of a MAV for tetrachloroethene in drinking-water. The no-observable-adverse-effect level used as the basis of the derivation was indicated by a six-week gavage study in male mice and a 90-day drinking-water study in male and female rats.

14 mg/kg body weight per day x 70 kg x 0.1 = 0.049 mg/L (rounded to 0.05 mg/L)

2 L x 1000

where:

* no-observable-adverse-effect level = 14 mg/kg body weight per day indicated by a six-week gavage study of hepatotoxic effects in male mice and a 90-day drinking-water study in male and female rats
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1,000 (100 for intra- and interspecies variation and 10 for carcinogenic potential).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime), and the subchronic limit, for 1,1,2,2-tetrachloroethylene is 0.007 mg/L.

The odour threshold in water is 0.3 mg/L.

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# Tetrachlorophenols

There are three tetrachlorophenols:

* 2,3,4,5-tetrachlorophenol: CAS No. 4901-51-3. Also called 2,3,4,5-TeCP.
* 2,3,4,6-tetrachlorophenol: CAS No. 58-90-2. Also called 2,3,4,6-TeCP.
* 2,3,5,6-tetrachlorophenol: CAS No. 935-95-5. Also called 2,3,5,6-TeCP.

CAS No. 25167-83-3 seems to have been allocated to tetrachlorophenols as a group.

### Maximum Acceptable Value

The DWSNZ do not include a MAV for tetrachlorophenols. The WHO Guidelines do not mention tetrachlorophenols.

The maximum acceptable concentration in Canada for 2,3,4,6-tetrachlorophenol is 0.1 mg/L.

### Sources to drinking-water

#### 1. To source waters

The tetrachlorophenols have been used as a wood and leather preservative. All the chlorophenols have been used as biocides. However, they do not appear on ERMA’s Full List of ACVM approved veterinary medicines and pesticides, as at 2009.

#### 2. From treatment processes

Chlorophenols are most likely to occur in drinking-water as disinfection by-products through the reaction of naturally-occurring organic matter with chlorine.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Because the tetrachlorophenols are slightly water-soluble (100–200 mg/L), weakly acidic, and have low vapour pressures, it is anticipated that volatilisation does not play a significant role in removing them from water. These tetrachlorophenols are present predominantly in the ionised form so will be less inclined to sorb to particulate matter or organics. Biodegradation has been reported. Aquatic biota may bioconcentrate chlorinated phenols with bioconcentration factors increasing with increasing chlorine substitution.

### Removal methods

Chlorophenols can be removed from contaminated source water by adsorption on to activated carbon. The effectiveness of the processes is pH dependent. Greater adsorption occurs as the pH is lowered.

However, as the tetrachlorophenols are most likely to arise in New Zealand waters principally as disinfection by-products, the preferred method for minimising their formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine.

Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products. The formation of chlorophenols can be reduced largely by the use of chlorine dioxide in place of chlorine.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Chlorophenols can be removed by adsorption on to activated carbon. The effectiveness of the processes is pH dependent. Greater adsorption occurs as the pH is lowered.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

### Analytical methods

#### Referee method

Chlorophenols: Micro Liquid/Liquid Extraction Gas Chromatographic Method (APHA 6251B).

#### Some alternative methods

1. Liquid/Liquid Extraction Gas Chromatographic Method (APHA 6420).

2. Liquid/Solid Extraction Gas Chromatographic/Mass Spectrometric Method (EPA 526).

3. Liquid/Liquid Extraction Gas Chromatographic/Mass Spectrometric Method (APHA 6410B).

4. Acetylation Liquid/Liquid Extraction Gas Chromatographic/Mass Spectrometric Method (EPA 1653).

### Health considerations

Chlorophenols are well-absorbed after oral administration and they readily penetrate the skin. Chlorophenols do not appear to accumulate in body tissues in rats but are metabolised rapidly and eliminated from the body, principally in urine.

The oral RfD for 2,3,4,6-tetrachlorophenol was calculated at 0.03 mg/kg/d (USEPA 1992) based on a NOAEL of 25 mg/kg/d for increased liver weights and centrilobular hypertrophy in an oral subchronic rat study. 2,3,5,6-Tetrachlorophenol is metabolised to a more toxic substance, tetrachloro-p-hydroquinone.

IARC (1999) considers combined exposures to polychlorophenols or to their sodium salts are possibly carcinogenic to humans (Group 2B).

The hexa, hepta, and octa congeners are the major PCDD contaminants in technical tetrachlorophenols.

### Derivation of Maximum Acceptable Value

No MAV.

Health Canada established an aesthetic objective for 2,3,4,6-tetrachlorophenol of 0.001 mg/L based on odour; levels above the AO would render drinking water unpalatable.

The USEPA established an organoleptic effect criterion of 0.001 mg/L for 2,3,4,6-tetrachlorophenol. Source: [*Quality Criteria for Water*, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

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# Toluene

CAS No. 108-88-3. Also called methylbenzene, monomethylbenzene, toluol, methylbenzol or phenylmethane.

### Maximum Acceptable Value

Based on health considerations, the concentration of toluene in drinking-water should not exceed 0.8 mg/L.

The maximum contaminant level (USEPA 2006/2009/2011) is 1 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on aesthetic considerations (taste and odour), the concentration of toluene in drinking water should not exceed 0.025 mg/L. Toluene would not be a health concern unless the concentration exceeded 0.8 mg/L.

Toluene is one of the “priority pollutants” under the US Clean Water Act.

EPA established an environmental exposure limit of 0.33 mg/L (330 µg/L) for toluene in fresh water (<http://www.epa.govt.nz/search-databases/Pages/substance-exposure-limit-register.aspx>).

### Sources to drinking-water

#### 1. To source waters

Toluene may be found in source waters due to industrial discharges as a result of human activity or from natural sources. It occurs naturally as a component of crude oil, and is present in petrol (5 to 11 percent by weight), sometimes as an additive to increase octane ratings. Trucks burning diesel have been reported to produce 1 to 7 mg of toluene per km (Environment Australia 2003). It is produced during petroleum refining and also occurs in natural gas, emissions from volcanoes, forest fires and cigarettes. Commercially it is used as a solvent, especially for paints, coatings, gums, oils, and resins, and is used in the production of nylon, plastics, and polyurethanes. Toluene was once used as a medicinal anthelmintic agent against roundworms and hookworms. It is also used overseas to produce benzene and toluene diisocyanate.

The average concentration of toluene in the Rhine River in the Netherlands is approximately 0.002 mg/L. EU (2003) reports that toluene has been measured above the detection limit in seven out of 146 measurements (14 percent) during 1985 to 1996 in German surface waters (the rivers Rhine and Elbe). The mean concentration was 0.10 mg/L and the median concentration 0.0005 mg/L. In US groundwater, 85 percent of 39 wells analysed contained toluene; the concentrations were <0.01 mg/L.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

Toluene may leach from some compounds used to seal water reservoirs. This possible source may need to be considered in the event of unacceptable concentrations of toluene appearing in a supply.

### Forms and fate in the environment

In surface soil, volatilisation to air is an important fate process for toluene. In the environment, biodegradation of toluene to carbon dioxide occurs with a typical half life of 1 to 7 days.

In surface water, toluene volatilises rapidly to the air, the rate of which depends on the amount of turbulence. Solubility in water is about 500 to 600 mg/L. Biodegradation and sorption are less important for the removal of toluene from surface waters. Any toluene that finds its way to groundwater will be quite stable. Its log octanol-water partition coefficient (Kow) is about 2.74, and it has a vapour pressure of 3.8 kPa at 25°C.

If released to soil, toluene is expected to have high to moderate mobility based upon Koc values in the range of 37–178. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 6.64 x 10-3 atm‑cu m/mole. Toluene may volatilise from dry soil surfaces based upon its vapour pressure. Biodegradation is expected to occur rapidly in soil surfaces, with half-lifes in the range of several hours to 71 days. If released into water, toluene is not expected to adsorb to suspended solids and sediment based upon a Koc of 166 measured in lake sediment. Biodegradation is expected to occur rapidly in water, with reported half-lifes of four and 56 days in aerobic and anaerobic water, respectively. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are one hour and four days, respectively. Measured BCF values of 13 and 90 in fish suggest bioconcentration in aquatic organisms is low to moderate. Hydrolysis is not expected to be an important environmental fate process for toluene due to lack of hydrolysable functional groups (EAWAG accessed February 2015).

EU (2003) quotes: vapour pressure = 3000 Pa at 20°C; water solubility = 515 mg/L at 20°C; octanol/water partition coefficient = logKow = 2.65; Henry’s law constant is estimated to be 537 Pa m3/mole at 20°C; toluene is not expected to hydrolyse under normal environmental conditions; direct photolytical degradation of toluene is estimated to be negligible; biodegradation half-life in surface water is 30 days; toluene is readily degraded in sewage plants; 83 to 92 days in a range of aerobic soils and 900 days anaerobic soil. The leaching potential is confirmed by groundwater monitoring results. Contaminated wells have exceeded 0.3 mg/L, one reaching 6.4 mg/L.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 301 zones, found toluene in four zones at concentrations ranging from 0.0011 to 0.020 mg/L, with the median concentration being “nd” (limit of detection = 0.001 mg/L) (ESR 2001).

Approximately 1 percent of all groundwater supplies in the United States have concentrations greater than 0.002 mg/L. 1,489 water utilities reported detecting toluene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.076 mg/L.

Concentrations of a few micrograms per litre have been found in surface water, groundwater and drinking-water; point emissions can lead to higher concentrations in groundwater (up to 1 mg/litre). It may also penetrate plastic pipes from contaminated soil.

The maximum concentration found in 10,979 samples from 2,728 groundwaters in the UK was 0.058 mg/L, mean 0.0006 mg/L (DWI 2008).

### Removal methods

Toluene can be removed from water by adsorption on to granular activated carbon, or by air stripping. Significant oxidation can also be expected by ozonation, although oxidation products may themselves be a concern if present in high enough quantities.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

In humans, toluene is absorbed readily from the gastrointestinal tract after oral intake, and is distributed preferentially in adipose tissue, successively followed by adrenals, kidneys, liver, and brain. It is metabolised rapidly in the liver to benzyl alcohol, benzoic acid, and to a lesser extent, phenols.

Toluene has a low acute toxicity via the oral route.

Virtually all data available for humans refer to exposure to toluene via the inhalational route. Exposure is increased by smoking and in heavy traffic. Upon acute exposure, the predominant effects were impairment of the central nervous system and irritation of mucous membranes. Fatigue and drowsiness were the most sensitive effects. The toxic effects of toluene after long-term exposure are basically the same.

Controlled long-term studies via the oral or inhalational route are lacking. The acute oral toxicity is low. Toluene exerts embryotoxic and fetotoxic effects, but there is no clear evidence of teratogenic activity in laboratory animals and humans.

Toluene generally did not exhibit mutagenic activity in tests on bacteria, yeast cells, and mammalian cells *in vitro*.

Available evidence suggests that toluene should not be regarded as an initiating carcinogen and the International Agency for Research on Cancer has classified it in Group 3 (not classifiable as to its carcinogenicity in humans, and there is evidence suggesting lack of carcinogenicity of toluene in experimental animals). Epidemiological studies on the occurrence of cancer as a consequence of exposure of human populations to toluene alone are lacking. This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. USEPA (2005) states that there is inadequate information to assess the carcinogenic potential of toluene because studies of humans chronically exposed to toluene and mixtures containing toluene are inconclusive, and toluene was not carcinogenic in adequate inhalation cancer bioassays of rats and mice exposed for life.

As at October 2015 and June 2017 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for toluene of:

* 0.8 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.2 mg/kg/day for intermediate-duration oral exposure (15–364 days).

The oral chronic reference dose or RfD (USEPA 2005, 2006/2009/2011) is 0.08 mg/kg/d, based on a BMDL of 238 mg/kg/d and an uncertainty factor of 3000. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 3 mg/L.

### Derivation of Maximum Acceptable Value

A tolerable daily intake approach has been used to derive the MAV for toluene in drinking-water. The lowest-observable-adverse-effect level used in the derivation is for hepatotoxicity effects in mice determined from a 13-week gavage study.

The MAV for toluene in drinking-water was derived as follows:

312 x (5/7) mg/kg body weight per day x 70 kg x 0.1 = 0.78 mg/L (rounded to 0.8 mg/L)

2 L x 1000

where:

* lowest-observable-adverse-effect level = 312 mg/kg body weight per day for hepatotoxicity in mice (normalised for 5 days/week dosing in derivation)
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for the short duration of the study and use of a LOAEL instead of a NOAEL).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for toluene is 0.2 mg/L, as are the short-term and subchronic limits.

The taste threshold for toluene has been reported at concentrations between 0.04 and 0.16 mg/L (mean 0.14 mg/L), and the odour threshold at about 0.024 to 0.17 mg/L. The aesthetic objective in Canada is not greater than 0.024 mg/L.

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# o-Toluidine

CAS No. 95-53-4. The IUPAC name is 2-methylaniline. The CAS name is 2‑methylbenzenamine. Also called orthotoluidine. All three ortho, meta and para isomers of toluidine exist. Toluidine can also be called methylaniline, or amino-1-methylbenzene. o-Toluidine can also be called o-methylaniline, 2-methylaniline, and 2‑amino-1-methylbenzene.

m-Toluidine is CAS No. 108-44-1. p-Toluidine is CAS No. 106-49-0.

Not to be confused with o-tolidine (CAS No. 119-93-7), a one-time common reagent for laboratory analysis of chlorine and manganese in water.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for o-toluidine, and it is not mentioned in the WHO Guidelines.

o-Toluidine is one of the 14 VOCs on the USEPA’s 3rd *Chemical Contamination List* (CCL3).

### Sources to drinking-water

#### 1. To source waters

o-Toluidine is used as a chemical intermediate in chemical synthesis of herbicides, rubber chemicals, dye and pigment intermediates, resin hardeners, fungicide intermediates, corrosion inhibitor in paints and pharmaceutical intermediates. The largest use of o-toluidine is for the preparation of methyl ethyl aniline (MEA, 6-ethyl-o-toluidine) which is used as an intermediate in the manufacture of herbicides such as acetochlor, metochlor and propisochlor. It is also used in the synthesis of rubber chemicals such as di-tolyl-phenyl-p-phenylenediamine (DTPD), which is a rubber antioxidant. o-Toluidine is also used for the photometric determination of glucose in the blood (DWI 2014).

o-Toluidine has been detected in a variety of foods including fresh kale, celery and carrots, and in shelled peas, red cabbage, and black tea aroma. USEPA (2000).

Concentrations of o-toluidine in groundwater range from 0.06 to 9.2 μg/L (combined o and p isomers), in fresh surface water from 0.03 to 20 μg/L and in effluents from 42.5 to 18,800 μg/L.

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

With regard to its chemical structure o-toluidine is not expected to hydrolyse under environmental conditions. o-Toluidine is readily biodegradable (MITI, comparable to OECD TG 301 C: biodegradation 65 percent after 28 days, OECD TG 301 A:  
88–90 percent after 28 days, OECD TG 301 E: ca. 90 percent after 28 days). From the measured coefficient of distribution between the sediment and water (Kd = 0.013) the log Koc was calculated to 0.87. These results indicate a low sorption potential of o‑toluidine on to the organic phase of soil or sediments. However, adsorption of o‑toluidine in soil can be enhanced by ion-ion interactions as the results with clay minerals show (INCHEM 2004).

Data from DWI (2014) includes:

* water solubility about 16,000 mg/L (1.6 percent)
* log Kow = 1.4 at 24.5°C
* Koc = 52
* Henry’s Law constant = 1.98 x 10-6 atm.m³/mol at 25°C.

### Removal methods

DWI (2014) offer the following:

o-Toluidine may be readily removed from drinking water by Fenton reactions. In a study using a fluidised-bed process (1.35 litre fluidised-bed reactor), 1 mM Fe2+ and 17 mM H2O2 at pH 3±0.5, removal of o-toluidine was reported to be 99.8 percent (note – not very practical for public water supply).

No other data on the removal of o-toluidine during drinking water treatment were located, however, some inference on its likely fate can be made based on its physico-chemical properties and fate in the environment.

o-Toluidine is not expected to adsorb to carbon, based on Kocs 40 to 250. Hydrolysis is also not expected to be an important fate process. However, it is expected to volatilise very slowly from water surfaces, based on a Henry’s Law constant of 1.98 x10-6 atm.m³/mol at 25°C, therefore it is not expected to be amenable to air stripping. Volatilisation half-lives of 19 and 140 days have been estimated for a model river and model lake, respectively.

### Health considerations

o-Toluidine has low acute toxicity. o-Toluidine is rapidly absorbed via gastrointestinal tract and is rapidly distributed, metabolised and excreted mainly via urine. o-Toluidine has been found in tobacco smoke.

Overall o-toluidine shows some genotoxic potential and clastogenic activity *in vitro*, however, the *in vivo* genotoxic potential of o-toluidine remains uncertain.

For Repeat Dose Oral Toxicity and Carcinogenicity a LOAEL of 1,000 mg/kg diet (reported to be approximately 150 mg/kg bw/day) was identified reductions in bodyweight. From this data an oral Tolerable Daily Intake (TDI) of 0.15 mg/kg bw/day (150 μg/kg bw/day) is derived. However, o-toluidine is an International Agency for Research on Cancer (IARC) Group 1 carcinogen and therefore its concentration in water should be as low as reasonably practicable. IARC state that there is moderate mechanistic evidence indicating that the carcinogenicity of o-toluidine involves metabolic activation, formation of DNA adducts, and induction of DNA-damaging effects.

### Derivation of Maximum Acceptable Value

No MAV.

### References

DWI. 2014. *Volatile Organic Compounds – Understanding the Risks to Drinking Water*. Report DWI9611.04. 305 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70-2-292.pdf>.

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USEPA. 2000. 2-Methylaniline (o-Toluidine). *Hazard Summary*. 2 pp. <http://www.epa.gov/ttnatw01/hlthef/methylan.html>.

# Tonalide

CAS No. 1506-02-1 and 21145-77-7. The IUPAC name for tonalide is 1-(3,5,5,6,8,8-hexamethyl-6,7-dihydronaphthalen-2-yl)ethanone. Tonalide can also be called tonalid, acetyl methyl tetramethyl tetralin, acetyl-1,1,2,4,4,7-hexamethyltetraline, AHTN, fixolide and musk fragrance. Tonalide is a commonly used trade name. See also galaxolide datasheet.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for tonalide, and it is not mentioned in the WHO Guidelines.

The Minnesota Department of Health developed a guidance value of 20 ppb for AHTN in drinking water. MDH (2011) considers the liver the organ most sensitive to AHTN exposure.

### Sources to drinking-water

#### 1. To source waters

Tonalide’s production and use as a fragrance used in cosmetics, body lotions, detergents, fabric softeners, household cleaning products and air fresheners may result in its release to the environment through various waste streams.

#### 2. From treatment processes

No known sources.

### Forms and fate in the environment

If released to soil, tonalide is expected to be immobile based on Koc values of 6,309 to 63,000. Volatilisation from moist soil surfaces is expected to be an important fate process based on its Henry’s Law constant of 1.4 x 10-4 atm‑cu m/mole. Adsorption to soil is expected to attenuate volatilisation. Biodegradation is not an important environmental fate process in soil or water. If released into water, tonalide is expected to adsorb to suspended solids and sediment based on its Koc. Volatilisation from water surfaces is expected to be an important fate process based on its estimated Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 15 hours and 9 days, respectively. However, volatilisation from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. The estimated volatilisation half-life from a model pond is 230 days if adsorption is considered. BCFs of 597 to 1,069 suggest bioconcentration in aquatic organisms is high to very high. Tonalide is not expected to undergo hydrolysis in the environment due to the lack of functional groups that hydrolyse under environmental conditions. Water solubility 1.25 mg/L. Tonalide is frequently found in sediments and sewage sludge (NIH accessed July 2016).

### Typical concentrations in drinking-water

Tonalide was detected in 12 raw water samples leading up to a drinking water treatment facility and found at a maximum concentration of 0.5 µg/L in finished water. Taken from NIH.

### Removal methods

Low levels of AHTN were detected in a drinking water treatment plant in Iowa. The average removal efficiency ranged from 79 percent in cold weather to 89 percent under warm weather conditions (Biomonitoring California 2013).

Treatment processes that remove particulate matter should reduce the concentration of tonalide in water. Ozone can break down the tonalide molecule.

### Health considerations

AHTN has frequently been found in human milk. In a 28-day oral gavage study, no effects of AHTN were seen at doses up to and including 10 mg/kg bw/day (EU 2008).

Some reports suggest tonalide has some endocrine effects (Biomonitoring California 2013).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The short term value is 0.1 mg/L, subchronic 0.03 mg/L, chronic 0.02 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

### References

Biomonitoring California. 2013. *Synthetic Polycyclic Musks*. 14 November SGP Meeting. 29 pp. <http://biomonitoring.ca.gov/sites/default/files/downloads/110813PolycyclicMusksDesig3.pdf>.

EU. 2008. 1-(5,6,7,8-tetrahydro-3,5,5,6,8,8-hexamethyl-2-napthyl)ethan-1-one (AHTN). *European Union Risk Assessment Report*. 259 pp. <http://echa.europa.eu/documents/10162/26e223a9-eda9-4e79-8c4d-650d2a3c1124>.

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NIH. Accessed July 2016. Tonalid. Compound Summary for CID 89440. *PubChem Open University Database*. [https://pubchem.ncbi.nlm.nih.gov/compound/Tonalid#section=Top](https://pubchem.ncbi.nlm.nih.gov/compound/Tonalid%23section=Top).

# Tribromoacetic acid

CAS No. 75-96-7. Also called 1,1,1-tribromoacetic acid, tribromoethanoic acid or TBA. Refer also to the haloacetic acids datasheet.

### Maximum Acceptable Value

The WHO Guidelines do not have a guideline value for tribromoacetic acid, nor does it have a MAV in the DWSNZ.

### Sources to drinking-water

#### 1. To source waters

Tribromoacetic acid has few uses.

There are nine common haloacetic acids (HAAs): monochloracetic acid (MCAA), dichloroacetic acid (DCAA), trichloroacetic acid (TCAA), monobromoacetic acid (MBAA), dibromoacetic acid (DBAA), tribromoacetic acid (TBAA), bromochloroacetic acid (BCAA), bromodichloroacetic acid (BDCAA), and dibromochloroacetic acid (DBCAA). These are referred to as the total haloacetic acids, or sometimes HAA9.

Of this chemical family only MCAA, DCAA, TCAA, MBAA and DBAA are currently regulated as drinking water contaminants in the US. These five regulated HAAs are often referred to as HAA5.

#### 2. From the treatment process

Tribromoacetic acid can form in water treated with chlorine and with ozone.

### Forms and fate in the environment

Haloacetic acids are very soluble in water, and are quite stable chemically.

### Typical concentrations in drinking-water

Sixty-nine water utilities reported detecting tribromoacetic acid in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.065 mg/L. This result was somewhat atypical, the next highest was 0.008 mg/L.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

DWI (2011) includes a thorough discussion on the analysis of the nine haloacetic acids.

### Health considerations

The results of a rat study (NTP 1998) indicate that TBA at up to 39 mg/kg/d marginally reduced water consumption and did not affect reproductive function or produce general toxicity. From these data, TBA is not a reproductive toxicant in males or females at doses up to 39 mg/kg/d.

Tribromoacetic acid was not mutagenic in tests with *Salmonella typhimurium* (USEPA 2001).

Of 19 individual halogenated organic compounds tested, trichloroacetic acid and tribromoacetic acid (both previously detected in swimming pool water) exhibited the greatest irritant effect.

NTP conducted a short-term study on the reproductive and developmental effects of TBAA (NTP 1998b). Doses up to 400 ppm were administered in drinking water to male and female rats (peri-conception and gestational exposure) for two weeks. No reproductive effects were observed in males or females and evaluation of the newborn heart and brain did not reveal any treatment-related effects. However, not data from a study that used a standard one or two generation protocal. Thus, additional research is needed. Taken from USEPA (2016).

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA has established some drinking water limits for HAAs. The sum of the concentrations of the five regulated HAAs (MCAA, DCAA, TCAA, MBAA, DBAA) may not exceed the MCL of 0.06 mg/L. Individual MCL goals have also been established by the USEPA for dichloroacetic acid and trichloroacetic acid. No limit has been established for tribromoacetic acid.

### References

DWI. 2011a. *Evaluation of Haloacetic Acid Concentrations in Treated Drinking Water*. Report No. WT1236. 111 pp. <http://dwi.defra.gov.uk/research/completed-research/reports/DWI70_2_242.pdf>.

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Environmental Working Group (EWG). Accessed 2010. *National Drinking Water Database – Chemical Contaminants*. <http://www.ewg.org/tap-water/chemical-contaminants>.

IPCS. 1999. Disinfectants and disinfection by‑products. *Environmental Health Criteria* 216. INCHEM. International Programme on Chemical Safety. <http://www.inchem.org/documents/ehc/ehc/ehc216.htm>.

NTP. 1998. *Short Term Reproductive and Developmental Toxicity of Tribromoacetic Acid Administered in Drinking Water to Sprague-Dawley Rats*. NTP Study Number: RDGT94009. See abstract at: <http://ntp.niehs.nih.gov/index.cfm?objectid=070EBAD8-C65A-5914-B30C76894774E340>.

USEPA. 2001. *Final Report: Genotoxicity and occurrence assessment of disinfection by‑product mixtures in drinking water*. National Center for Environmental Research. <http://cfpub.epa.gov/ncer_abstracts/index.cfm/fuseaction/display.abstractDetail/abstract/840/report/F>.

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# Tributyltin oxide

CAS No. 56-35-9. Also called TBTO and bis(tributyltin)oxide or bis(tri-n-butyltin) oxide, or hexabutyldistannoxane.

Other tributyltin compounds include:

* tributyltin hydride: CAS No. 688-73-3
* tributyltin naphthenate is discussed in a datasheet in the Pesticides section.

The commoner triphenyltin compounds are:

* triphenyltin hydroxide CAS No. 76-87-9
* triphenyltin acetate CAS No. 900-95-8.

### Maximum Acceptable Value

The WHO (2004 and 2011) did not establish a guideline value for tributyl tin because it is considered unlikely to occur in drinking-water.

In DWSNZ 2005, the provisional MAV for tributyltin oxide had been 0.002 mg/L  
(2 g/L).

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of tributyltin oxide in drinking water should not exceed 0.001 mg/L, and there are insufficient data to set a health-based guideline value for dialkyltins (qv).

Tributyl tin is classified as a Persistent Organic Pollutant. Tributyltin compounds appear on the Rotterdam Convention (UNEP) list of chemicals in Appendix III (which effectively bans or severely restricts use of a chemical), see <http://www.pic.int/home.php?type=s&id=77>.

The USEPA concluded on 22 September 2009 that triphenyltin hydroxide is known or anticipated to occur in wastewater plants and may require regulation; they added triphenyltin hydroxide to their CCL 3 (Drinking Water Contaminant Candidate List 3). Therefore information on triphenyltin has been added to this datasheet.

### Sources to drinking-water

#### 1. To source waters

The group of compounds known as the organotins comprises a large number of compounds with different properties and applications. Of these the dialkyl and tributyltin compounds are the ones most likely to be found in raw water. The trisubstituted compounds are used in the preservation of materials (wood, stone, textiles), as biocides, and as disinfectants. Tributyltins and triphenyltins may be found in raw water and sediment as a result of their use as antifouling agents. Tributyltin is slowly released from the painted surface as the polymer is hydrolysed in seawater, providing protection against encrustations for as long as 4–5 years.

The use of tributyl-organotin compounds, particularly tributyltin oxide, in antifouling paints has now been banned in a number of countries because it is extremely toxic to aquatic life, particularly harmful to the oyster industry. Tributyltin is also used as a biocide in boiler waters and cooling towers. The main tri-organotin compounds that were used were bis(tributyltin) oxide, triphenyltin hydroxide and tributyltin fluoride. Although this datasheet is headed tributyltin oxide, TBT compounds are expected to exist mainly as TBT hydroxide, TBT chloride, and TBT carbonate. Triphenyltin is also used as a non-systemic fungicide with mainly protective action, but apparently not in New Zealand.

#### 2. From the treatment process

No known sources.

#### 3. From the distribution system

No known sources. Note that tributyltin products are not used as stabilisers in PVC or CPVC pipes (see dialkyltin datasheet).

### Forms and fate in the environment

Under aerobic conditions, tributyltin takes one to three months to degrade but in anaerobic soils, this compound may persist for more than two years. All of the breakdown products are less toxic than TBT itself.

There is little information available on the fate of organotins in the aquatic environment. Tributyltin is thought to accumulate in aquatic food chains. Because of the low water solubility of TBT (4 mg/L) and other properties, it will bind strongly to suspended material such as organic material or inorganic sediments and precipitate to the bottom sediment. Rates of sedimentation vary with location, organic content, particle size, and type of material. Reported half-lifes of the compound in freshwater are six to 25 days, depending on the initial concentration. Because of the low levels of UV light beyond the topmost few centimetres, it is unlikely photolysis plays a major role in degradation of tributyltin compounds. In the Great Lakes, concentrations from 0.00002 to 0.0008 mg/L have been recorded.

Triphenyltin is strongly adsorbed to sediment and soil, and little desorption occurs. Its half-life in water has been estimated to be several days in summer and 2–3 weeks in winter.

### Typical concentrations in drinking-water

No data are available on the concentration of tributyltin in New Zealand drinking-water supplies. No overseas data are available for tributyltin concentrations in drinking-water.

There are no data on levels of triphenyltin in drinking-water (WHO 1999a).

### Removal methods

No information is available on processes that can be used to remove tributyltin oxide from water. However, the tendency of tributyltin to bind to particulate matter suggests that treatment processes that remove turbidity should reduce the concentration of tributyltin.

### Analytical methods

#### Referee method

Liquid/Liquid Extraction and Gas Chromatography with Flame Photometric Detection (Greaves and Unger, 1988, *Biomed & Env Mass Spec* 15: 565–9).

#### Some alternative methods

No alternative methods have been recommended for tributyltin because no methods meet the required criteria.

### Health considerations

Available data suggest that organotins are poorly absorbed and they tend to be distributed primarily in the liver and kidney following oral administration in rodents. Following oral administration it appears that the principal route of excretion of organotins is in the faeces.

*In vitro*, tributyltin was metabolised to dibutyltin, hydroxybutyltins, butanol and butane. Carbon dioxide and butene were detected as metabolites of both dibutyltin diethanoate and tributyltin ethanoate in mice *in vivo.*

Tributyltin oxide is not genotoxic. Although one carcinogenicity study was reported in which neoplastic changes were observed in endocrine organs, the significance of these changes is considered questionable. The most sensitive end-point appears to be immunotoxicity.

Triphenyltin hydroxide is classified by the USEPA in Group 2B: a probable human carcinogen.

Organotins appear on many endocrine disruption compound lists.

EXTOXNET (1996) quotes a RfD of 0.00003 mg/kg/d for TBTO.

The lowest NOAEL detected in the toxicity tests was 0.1 mg triphenyltin hydroxide/kg body weight per day for maternal toxicity in a rabbit gavage study, based on decreased food consumption and body weight gain at 0.3 mg/kg body weight per day. The same value was obtained in an early two-year rat study in which a slight decrease in white blood cells was seen at higher doses. In a 52-week dog study, the NOAEL was estimated to be 0.21 mg triphenyltin hydroxide/kg body weight per day based on a decrease in relative liver weight in females at higher doses (WHO 1999a).

ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.005 mg/kg/day for intermediate-duration oral exposure (15–364 days) to dibutyl tin (see dialkyltin datasheet), and for tributyl tin oxide, ATSDR quotes a minimal risk level (MRL) of:

* 0.0003 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.0003 mg/kg/day for chronic-duration oral exposure (>364 days).

The following substances containing tin are on the EC List of 66 Category 1 substances (EC 2015) showing evidence of endocrine disrupting activity in at least one species using intact animals:

tributyltin, tributyltin oxide = bis(tributyltin) oxide; 2-propenoic acid, 2-methyl-, methyl ester = tributylmethacrylate stannane; methoxyethylacrylate tinbutyltin copolymer; 2-[[(tributylstannyl)oxy]carbonyl phenol; (benzoyloxy)tributyl-stannane; [1,2-phenylenebis(carbonyloxy) stannane; tributyl stannane = tributyltin naphthalate; mono(naphthenoyloxy tributyl-stannane; tributyl[(1-oxo-9,12-octadecad stannane; tributyl[(1-oxo-9-octadecenyl)-stannane; tributyl[[[1,2,3,4,4a,4b,5,6,1-stannane; tributylfluoro-stannane; tributyl[(2-methyl-1-oxo-2-propenyl)oxy]stannane; tributyltincarboxylate; tributyltinnaphthalate; tributyltinpolyethoxylate; tri-n-propyltin (TPrT); triphenyltin, fentin acetate; tetrabutyltin (TTBT).

### Derivation of Maximum Acceptable Value

No MAV.

WHO (2004 and 2011) did not establish a guideline value for tributyl tin because it is considered unlikely to occur in drinking-water.

However, in DWSNZ 2005, the provisional MAV had been derived as follows: a tolerable daily intake approach has been used for the derivation of the MAV of tributyltin oxide (TBTO) in drinking-water. The most sensitive end-point appears to be immunotoxicity in a 17-month feeding study in rats related to suppression of resistance to the nematode *Trichinella spiralis*. The significance of this finding to humans is not completely clear, but the no-observable-adverse-effects level is consistent within an order of magnitude with other levels from long-term toxicity studies.

0.025 mg/kg body weight per day x 70 kg x 0.2 = 0.002 mg/L

2 L x 100

where:

* no-observable-adverse-effect level = 0.025 mg/kg body weight per day in a 17‑month study in rats related to suppression of resistance to the nematode *Trichinella spiralis*
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 100 (for intra- and interspecies variation).

### References

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WHO. 2004. *Guidelines for Drinking-water Quality 2004* (3rd edition). Geneva: World Health Organization. Available at: [www.who.int/water\_sanitation\_health/dwq/gdwq3/en/print.html](http://www.who.int/water_sanitation_health/dwq/gdwq3/en/print.html) see also the addenda.

WHO. 2006. Mono- and disubstituted methyltin, butyltin, and octyltin compounds. *Concise International Chemical Assessment Document (CICAD)* 73. International Programme on Chemical Safety (IPCS). 73 pp. See: <http://www.who.int/entity/ipcs/publications/cicad/cicad73.pdf>.

# Trichloroacetaldehyde

CAS No. 302-17-0. Frequently called chloral hydrate or 2,2,2-trichloro-1,1-ethanediol (CAS), or 1,1,1-trichloro-2,2-dihydroxyethane or 2,2,2-trichloroethane-1,1-diol (IUPAC), or trichloroacetaldehyde monohydrate, chloral monohydrate and various tradenames.

Note that chloral (also known as anhydrous trichloroacetaldehyde, anhydrous chloral, or 2,2,2-trichloroacetaldehyde; trichloroethanal; 2,2,2-trichloroethanal) is CAS No. 75‑87-6. Chloral is (or was) used as an intermediate in the synthesis of the insecticides DDT, methoxychlor, naled, trichlorfon, and dichlorvos, and the herbicide trichloracetic acid. IARC (2013) evaluated chloral and chloral hydrate together since the two substances exist in equilibrium in aqueous solution.

### Maximum Acceptable Value

WHO (2005 and 2011) state that because trichloroacetaldehyde usually occurs in drinking-water at concentrations well below the health-based value of 0.1 mg/L (i.e., generally below 0.01 mg/L), it is not considered necessary to derive a guideline value.

In DWSNZ 2005, the provisional MAV had been 0.01 mg/L. The WHO (2004) guideline value had been designated as provisional because of limitations of the available database.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of chloral hydrate in drinking water should not exceed 0.02 mg/L. This has been changed to 0.1 mg/L (NHMRC, NRMMC 2016).

### Sources to drinking-water

#### 1. To source waters

Trichloroacetaldehyde is not known to occur as a natural product. Trichloroacetaldehyde may enter source waters from industrial discharges, where it is used in the manufacture of several pesticides; it was used in the production of DDT. Hydrated trichloroacetaldehyde (chloral hydrate) is used as a sedative and hypnotic in human and veterinary medicine (chloral was the first hypnotic drug – the conversion from chloral to chloral hydrate occurs spontaneously when chloral is placed in aqueous media). In its use as a sedative for people, the usual clinical dose of chloral hydrate is 250 mg, three times a day (equivalent to 10.7 mg/kg body weight per day). The metabolite trichloroethanol is responsible for the pharmacological effect. Chloral hydrate is also a metabolite of trichloroethene and tetrachloroethene. Technical grade chloral may contain traces of chloroform, dichloroacetaldehyde and phosgene.

#### 2. From treatment processes

Trichloroacetaldehyde may be formed as a by-product during disinfection (using chlorine or chloramine) of water containing natural organic matter and amino acids; adding ozone before the chlorine or chloramine favours its formation. Stockham and Morran detected trichloroacetaldehyde in water treated with polyDADMAC after chlorination. Drinking-water is the major route of exposure of the general public. Table 1.2 in IARC (2013) summarises recent measurements of chloral hydrate in drinking-water and swimming pool water in several countries.

In the WRF (2016) study, chloral hydrate concentrations varied with modelled water age, and no obvious degradation or accumulation was observed in samples from all utilities. But it was evident that chloral hydrate concentration increases with decreasing chlorine residual. The highest concentration found was 4.6 µg/L at water age of 68 hours.

#### 3. From the distribution system

No known sources. However, concentrations leaving the treatment plant appear to increase during distribution (WHO 2011).

### Form and fate in the environment

Chloral converts to chloral hydrate in water. Both are very soluble in water. It can break down to trichloroethanol, trichloroacetic acid and dichloroacetic acid in water.

If released to soil, chloral hydrate is expected to have high mobility based upon an estimated Koc of 82. Volatilisation from moist soil surfaces is expected to be an important fate process based upon an estimated Henry’s Law constant of 5.7 x 10-9 atm‑cu m/mole. Biodegradation data for chloral hydrate were not available. In water, chloral hydrate is formed from the exothermic reaction of chloral with water, in which chloral hydrate is in equilibrium. Chloral hydrate hydrolyses to form chloroform at high pH. Chloral hydrate is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilisation from water surfaces is expected to be an important fate process based on its estimated Henry’s Law constant. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 511 zones, found trichloroacetaldehyde concentrations to range from “not detectable” (nd) to 0.030 mg/L, with the median concentration being “nd” (limit of detection = 0.002 mg/L). The Priority 2 Identification Programme found 17 distribution zones supplying drinking-water to a total of 46,975 people with trichloroacetaldehyde at greater than the MAV, and 75 distribution zones supplied 233,249 people with >50 percent of the (then) MAV of 0.01 mg/L (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0003 mg/L trichloroacetaldehyde in the raw water the treated water, and up to 0.0008 mg/L in the distribution system.

The median chloral hydrate concentration reported under the US Information Collection Rule (ICR) in water leaving the treatment plant was 0.0017 mg/L, and the maximum was 0.046 mg/L.

The reported chloral hydrate concentration in distribution system samples ranged from the method reporting limit of 0.0005 mg/L up to 0.092 mg/L. Surface water showed higher concentrations (median value of 0.004 mg/L) than groundwater (median value of 0.0005 mg/L). Chloral hydrate concentrations in the distribution systems of surface water treatment plants (median value of 0.004 mg/L) were generally higher than those in the finished water at the treatment plants (median value of 0.0024 mg/L), suggesting that chloral hydrate concentrations increase somewhat through the distribution system (WHO 2005).

Canadian surveys conducted in 1995 and 1997 show levels of chloral hydrate in Canadian drinking water supplies ranging from 0.001 to 0.004 mg/L in winter and from 0.004 to 0.008 mg/L in summer, with a maximum level of 0.023 mg/L observed in winter. Although slightly higher levels may be associated with smaller-scale treatment plants with a limited ability to remove organic matter prior to the addition of the chlorine disinfectant, these levels are still expected to be much lower than any level of concern (Health Canada 2008).

NHMRC, NRMMC (2016) reports concentrations up to 0.088 mg/L chloral hydrate in South Australia and up to 0.04 mg/L in Victoria.

### Removal methods

Complete removal of 0.0045 mg/L of trichloroacetaldehyde by GAC over one year’s operation was reported from a pilot plant study.

However, as this compound arises in waters principally as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina orion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

### Analytical methods

#### Referee method

Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551.1). See also IARC (2013).

#### Some alternative methods

Method 5710 D of *Standard Methods for the Examination of Water and Wastewater* (APHA 2005) stipulates that chloral hydrate may be analysed with trihalomethanes (THMs) using a sulfite reducing solution to quench the reaction.

### Health considerations

Trichloroacetaldehyde is absorbed rapidly in humans with most of the dose being excreted in the urine as trichloroethanol glucuronide.

Trichloroacetaldehyde has been used widely as a sedative or hypnotic drug in humans at recommended oral doses of 0.25–1.0 g. Generally, single doses or daily dosages for adults should not exceed 2 g. Concentrated solutions are quite irritating to the gastrointestinal tract, and ingestion of undiluted preparations causes nausea and vomiting.

Adverse effects in patients given either 0.5 or 1.0 g trichloroacetaldehyde included central nervous system depression, minor sensitivity reactions, gastrointestinal disturbances and central nervous system excitement. Trichloroacetaldehyde induced arrhythmias have been described.

The chronic use of trichloroacetaldehyde may result in development of intolerance, physical dependence, and addiction.

No epidemiological or carcinogenic studies were found in humans associating exposure to trichloroacetaldehyde with cancer, despite the fact that trichloroacetaldehyde has been used for many decades (and still is used) as a sedative and hypnotic drug in adults and children (specifically for dental procedures).

IARC classified trichloroacetaldehyde as not classifiable as to its carcinogenicity to humans (Group 3) in 1995, based on inadequate evidence in humans and limited evidence in experimental animals for the carcinogenicity of trichloroacetaldehyde. There is equivocal evidence of genotoxicity for trichloroacetaldehyde. In 2004, IARC confirmed that chloral hydrate is not classifiable as to its carcinogenicity to humans (Group 3). IARC (2013) evaluated chloral and chloral hydrate as *probably carcinogenic to humans (Group 2A).*

USEPA (2000) states that although chloral hydrate and its metabolites, trichloroacetic acid and dichloroacetic acid, can induce a variety of mutational events, they do so with very low potency. Owing to the high concentration of chloral hydrate and its metabolites required to induce an observable effect in these assays, it is not likely that a genotoxic mode of action can be held responsible for the pituitary adenomas found in female mice or the hepatocellular tumours found in male mice.

Although the reference value of 0.1 mg/kg-day derived from the pharmacologically active dose in humans is an acute RfD, keeping exposure below this level will also be protective for any non-cancer health effect from chronic exposure. For example, chronic exposure to chloral hydrate does not cause adverse effects in the liver of rats or mice until the exposure approaches 135 or 160 mg/kg-day, respectively. Similarly, there are no reproductive, developmental, neurobehavioral, or immunological effects following long-term treatment of laboratory animals until the exposure approaches 160 mg/kg-day. Therefore, it is appropriate to use the acute RfD also as the chronic RfD (USEPA 2000).

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. One of their findings was that for cell cytotoxicity, the rank order from most cytotoxic to least cytotoxic for the DBP classes was haloacetaldehydes > haloacetamides > halonitromethanes > haloacetonitriles > >2C-haloacids > haloacetic acids > halomethanes.

### Derivation of Maximum Acceptable Value

No MAV.

WHO (2017) states a health-based value of 0.1 mg/l (rounded figure) can be calculated on the basis of a TDI of 0.0045 mg/kg body weight derived based on an increased incidence of liver histopathology observed in mice in a 2-year drinking-water study, allocating 80 percent of the TDI to drinking-water (because most exposure to chloral hydrate is from drinking-water) and assuming a 60 kg adult consuming two litres of water per day. However, because chloral hydrate usually occurs in drinking-water at concentrations well below those of health concern, it is not considered necessary to derive a guideline value.

WHO (2005) stated that a health-based value of 0.1 mg/L can be calculated. The health based value is expected to be protective of non-cancer end-points, including neurodevelopmental toxicity. However, because trichloroacetaldehyde usually occurs in drinking-water at concentrations well below this health-based value (ie, generally below 0.01 mg/L), it is not considered necessary to derive a guideline value.

The non-cancer end-point of histopathology in the liver was chosen for the risk assessment, based on a study where mice were treated in a lifetime study with trichloroacetaldehyde in drinking-water at a range of concentrations. At 120 mg/L (13.5 mg/kg of body weight per day and above), significant increases in the incidence of proliferative lesions were observed. Since these lesions were observed at all dose levels, no NOAEL could be derived; therefore, a LOAEL of 120 mg/L (13.5 mg/kg of body weight per day) was set.

A health-based value for trichloroacetaldehyde in drinking-water can be derived as follows (WHO 2005):

13.5 mg/kg body weight per day x 70 kg x 0.8 = 0.126 mg/L (rounded to 0.1 mg/L)

2 L x 3,000

where:

* lowest-observable-adverse-effect level = 13.5 mg/kg body weight per day chloral hydrate in drinking-water for 90 days
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.8
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 3,000 (10 each for intra- and interspecies variation, 10 for the use of a LOAEL instead of a NOAEL and 3 to account for limited evidence of carcinogenicity).

The PMAV in the DWSNZ (2005) had been based on the following:

A tolerable daily intake approach had been used for the derivation of the 0.01 mg/L PMAV for trichloroacetaldehyde in drinking-water. The lowest-observable-adverse-effect level used in the derivation was based on a study in which liver effects were seen in mice which received trichloroacetaldehyde in drinking-water for 90 days. The LOAEL was 16 mg/kg body weight per day, based on a study in which liver effects were seen in mice which received trichloroacetaldehyde in drinking-water for 90 days. An uncertainty factor of 10,000 had been used, and the proportion of TDI allocated to drinking-water was 0.2.

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# Trichloroacetic acid

CAS No. 76-03-9. Also called trichloroethanoic acid, trichloracetic acid, 2,2,2‑trichloracetic acid, trichloromethane carboxylic acid or TCA. Trichloroacetic acid is one of the USEPA’s 5 haloacids (HAA5); the others are monochloroacetic acid, dichloroacetic acid, monobromoacetic acid and dibromoacetic acid. Refer also to the haloacetic acids datasheet. CAS No. 650-51-1 is allocated to sodium trichloroacetate.

### Maximum Acceptable Value

Based on health considerations, the concentration of trichloroacetic acid in drinking-water should not exceed 0.2 mg/L.

The sum of the ratio of the concentrations of dichloroacetic acid, monochloroacetic acid and trichloroacetic acid to each of their respective MAVs must not exceed one. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, Section 10.2.5.3).

The total maximum contaminant level (MCL) for the five haloacetic acids (USEPA 2006/2009/2011) is 0.06 mg/L. The lifetime health advisory for trichloroacetic acid is 0.02 mg/L (USEPA 2006/2009/2011) where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The MAC in Canada is 0.3 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of trichloroacetic acid in drinking water should not exceed 0.1 mg/L.

### Sources to drinking-water

#### 1. To source waters

Trichloroacetic acid is not known to occur as a natural product. Trichloroacetic acid may enter raw water through its industrial (eg, as an auxiliary in textile dyes, wastewater from electroplating facilities, textile washing and pulp mills), agricultural and domestic use. It may be used as an intermediate in the synthesis of organic chemicals, laboratory reagent, herbicide, soil steriliser, antiseptic and treatment for warts.

Trichloroacetic acid is listed in ERMA’s Summary of Approvals of Substances transferred under the Hazardous Substances (Pesticides) Transfer Notice 2004 (as Amended) as at 22 May 2008, as a component in a product containing 2,2‑dichloropropionic acid (dalapon).

Table 1.2 in IARC (2013) lists, with concentrations, where trichloroacetic acid has been found in surface waters, groundwater and drinking-water worldwide.

#### 2. From treatment processes

Trichloroacetic acid is formed at microgram/L level from organic material during water disinfection using chlorine or chloramine. Levels of trichloroacetic acid tend to decline with length of residence in the distribution system, and tend to be higher in warmer seasons. Stockham and Morran detected trichloroacetic acid in water treated with polyDADMAC after chlorination. Being a disinfection by-product, the USEPA (2007) regulates trichloroacetic acid.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Trichloroacetic acid is stable in neutral solution and is classified as “non-biodegradable” with a “low bioaccumulation potential” for fish and a “high bioaccumulation potential” for terrestrial plants (OECD 2000). It is miscible in water. Trichloroacetic acid has been identified as a major chlorinated by-product of the photocatalytic degradation of tetrachloroethylene. It is also a metabolite of 1,1,1‑trichloroethane and chloral hydrate (trichloroacetaldehyde, qv) (IARC 2013).

NPIC (1994) quotes for TCA a soil half-life of 21 days, water solubility of 120 percent and a sorption coefficient (soil Koc) of 3. This resulted in a pesticide movement to groundwater rating of very high. Health Canada (2008) quotes a log octanol/water partition coefficient of 1.33.

In 2013/14 Hamilton’s six-monthly analyses have found <0.001 mg/L trichloroacetic acid in the raw water and the treated water.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 488 zones, found trichloroacetic acid concentrations to range from “not detectable” (nd) to 0.115 mg/L, with the median concentration being “nd” (limit of detection = 0.005 mg/L). The Priority 2 Identification Programme found one distribution zone supplying drinking-water to a total of 240 people with trichloroacetic acid at greater than the MAV, and 23 distribution zones supplied 41,100 people with >50 percent of the MAV (ESR 2001).

Trichloroacetic acid has been detected in US groundwater and surface water distribution systems at mean concentrations of 0.0053 mg/L (range <0.001–0.08 mg/L) and 0.016 mg/L (range <0.001–0.174 mg/L), respectively; the maximum concentration was 0.20 mg/L, measured in chlorinated water in Australia (WHO 2004).

IARC (2004) has tabulated trichloroacetic acid concentrations found in drinking-water. Several maximum levels have been found to exceed 0.1 mg/L, with some >0.2 mg/L.

10,691 water utilities in the US reported detecting trichloroacetic acid in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.176 mg/L.

### Removal methods

No information is available on how trichloroacetic acid might be removed from contaminated source waters.

However, as this compound arises in waters principally as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

### Analytical methods

#### Referee method

Ion Exchange Liquid-Solid Extraction and Gas Chromatography with Electron Capture Detection (EPA 552.1). DWI (2011) includes a thorough discussion on the analysis of the nine haloacetic acids. See also IARC (2013).

#### Some alternative methods

1. Micro Liquid–Liquid Extraction Gas Chromatographic Method (APHA 6251B; EPA 552.3).

### Health considerations

Trichloroacetic acid is absorbed rapidly from the intestinal tract and metabolism occurs primarily in the liver. Following oral and intravenous administration in rats, trichloroacetic acid appears to bind significantly to plasma proteins and is also distributed to the liver; plasma protein binding has been found to vary across species and is highest in humans. Trichloroacetic acid can be converted to carbon dioxide and chloride ion, or reduced to the aldehyde. Relatively small proportions of trichloroacetic acid are metabolised, and much of this compound is excreted unchanged in the urine.

Trichloroacetic acid is one of the major metabolites of trichloroethene (TCE) and tetrachloroethene (PCE) in humans.

The NOEL for repeated dose toxicity in a four-month feeding study with rats was 365 mg/kg bw/day, the NOEL in a two-year feeding study in rats was 80 mg/kg bw/day (OECD 2000).

Trichloroacetic acid administered in drinking water has consistently been shown to produce liver tumours in mice but not in rats; as a result Health Canada has classified trichloroacetic acid as possibly carcinogenic to humans. It has given mixed results in *in vitro* assays for mutations and chromosomal aberrations, and has been reported to cause chromosomal aberrations in *in vivo* studies.

IARC (2004) classified trichloroacetic acid in Group 3, not classifiable as to its carcinogenicity to humans. However, IARC (2013) states that trichloroacetic acid is *possibly carcinogenic to humans (Group 2B).* The weight of evidence indicates that trichloroacetic acid is not a genotoxic carcinogen. Health Canada (2008) considers TCA is to be a possible carcinogen in humans, based on limited evidence in experimental animals and inadequate evidence in humans. Animal studies have shown a link between exposure to TCA and liver tumours in mice only, but it is still uncertain whether the mechanism causing these tumours is relevant to humans.

In the Stage 1 D/DBPR, USEPA established an MCLG of 0.3 mg/L for TCAA based on developmental toxicity and limited evidence of carcinogenicity in animals. In the Stage 2 D/DBPR, USEPA proposed and finalised an MCLG of 0.02 mg/L for TCAA derived from an RfD of 0.03 mg/kg/day, using a NOAEL for liver histopathological changes in rats, an uncertainty factor of 1000, an additional risk management factor of 10 to adjust for “*suggestive evidence of carcinogenicity”*. This MCLG was based on this RfD, using adult tap water consumption of two litres/day for a 70 kg adult and a relative source contribution of 20 percent for drinking water exposure. The IRIS RfD of 0.02 mg/kg/day is based on a 95 percent lower confidence level on the modelled benchmark dose for a 10 percent decrease (BMDL10) in liver necrosis in the treated B6C3F1 mice of 18 mg/kg/day. The study used a drinking water route of exposure over a 60-week period. Copied from USEPA (2016).

Using a NOAEL of 32.5 mg/kg bw per day based on a chronic rat study, Health Canada (2008) calculated a TDI of 0.0325 mg/kg bw per day, and a health-based target of 0.3 mg/L.

The reference dose or RfD (USEPA 2006/2009/2011) for trichloroacetic acid is 0.03 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 1 mg/L.

### Derivation of Maximum Acceptable Value

Because the weight of evidence on the carcinogenicity of trichloroacetic acid is restricted to one species, a tolerable daily intake approach has been used for the derivation of the MAV for trichloroacetic acid in drinking-water. The no-observed-adverse-effect level used in the derivation is based on a study in which decreased body weight, increased liver serum enzyme activity and liver histopathology were seen in rats exposed to trichloroacetate in drinking-water for two years.

The MAV for trichloroacetic acid in drinking-water was derived as follows:

32.5 mg/kg body weight per day x 70 kg x 0.2 = 0.223 mg/L (rounded to 0.2 mg/L)

2 L x 1000

where:

* no-observable-adverse-effect level = 32.5 mg/kg body weight per day from a two-year study
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1000 (100 for intra- and interspecies variation and 10 for database deficiencies, including the absence of a multigeneration reproductive study, the lack of a developmental study in a second species, and the absence of full histopathological data in a second species).

WHO (2004) emphasised that difficulties in meeting the guideline value must never be a reason for compromising adequate disinfection.

The DWSNZ 1995 and 2000 had a provisional MAV for trichloroacetic acid of 0.1 mg/L, derived as follows:

178 mg/kg body weight per day x 70 kg x 0.2 = 0.1 mg/L

2 L x 10,000

where:

* lowest-observable-adverse-effect level = 178 mg/kg body weight per day from a study in which increased liver weight was seen in mice exposed to trichloroacetic acid in drinking-water for 52 weeks
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 10,000 (100 for intra- and interspecies variation and 100 for use of a slightly less than lifetime study, use of a LOAEL instead of a NOAEL, and possible carcinogenicity.

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# Trichloroacetonitrile

CAS No. 545-06-2. Also called cyanotrichloromethane, trichloromethylcyanide, trichloroethanenitrile and trichloromethylnitrile. IUPAC name is 2,2,2‑trichloroacetonitrile. Datasheets also exist for 2-chloroacetonitrile, 2,2‑dichloroacetonitrile, 2,2-dibromoacetonitrile, 2-bromo-2-chloroacetonitrile, and bromoacetonitriles.

### Maximum Acceptable Value

WHO (2004 and 2011) states that available data are insufficient to serve as a basis for derivation of a guideline value for trichloroacetonitrile.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set guideline values for haloacetonitriles in drinking water.

### Sources to drinking-water

#### 1. To source waters

Trichloroacetonitrile has been used in the past as an insecticide and therefore may have entered raw water as a contaminant.

#### 2. From treatment processes

Haloacetonitriles such as trichloroacetonitrile are formed from organic precursors during chlorination or chloramination of drinking-water. In general, increasing temperature and/or decreasing pH have been associated with increasing concentrations of halogenated acetonitriles. Ambient bromide levels appear to influence, to some degree, the speciation of halogenated acetonitrile compounds. Trichloroacetonitrile is probably the least predominant halogenated acetonitrile species detected in drinking-water.

#### 3. From the distribution system

No known sources.

### Form and fate in the environment

Haloacetonitriles are reported to undergo hydrolysis in water, yielding non-volatile products.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 209 zones, did not find trichloroacetonitrile at detectable concentrations (limit of detection = 0.0002 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with trichloroacetonitrile at >50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0003 mg/L trichloroacetonitrile in the raw water, the treated water and the distribution system.

Trichloroacetonitrile concentrations are likely to be much less than 0.001 mg/L (WHO 2004). Trichloroacetonitrile was detected in groundwater and surface water distribution systems at mean concentrations of 0.00014 and 0.00003 mg/L.

DWI (2012) reported a UK study. The lowland water sources that were included in the survey formed more N-DBPs than the upland and groundwater sources. The six treatment works that applied ozone were associated with higher concentrations of HANs and HAcAms than non-ozone treatment works, although this was potentially confounded because all the ozone works were treating lowland source waters which may have had higher N-DBP formation potential. None of the N-DBPs exhibited consistent links with total trihalomethanes (THMs). There were no trends linking HNMs to either THMs or HAA9. The mean HAN concentration was 0.0032 mg/L (3.2 µg/L). The main HANs were dichloroacetonitrile, bromochloroacetonitrile and dibromoacetonitrile. Trichloroacetonitrile was not detected.

### Removal methods

No information is available for methods to remove trichloroacetonitrile from contaminated source waters.

However, as this compound arises in water principally as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

### Analytical methods

#### Referee method

A referee method cannot be selected for trichloroacetonitrile because a MAV has not been established and therefore the sensitivity required for the Referee method is not known.

#### Some alternative methods

Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551). See also DWI (2010).

### Health considerations

The only known route of human exposure is through chlorinated drinking-water. Haloacetonitriles are rapidly absorbed from the gastrointestinal tract and metabolised to single carbon compounds. Insufficient data are available to determine whether haloacetonitriles can accumulate in specific organs.

No data are available on the health effects of haloacetonitriles (chloroacetonitrile, dichloroacetonitrile, trichloroacetonitrile, bromochloroacetonitrile and dibromoacetonitrile) in humans.

In a study in rats, trichloroacetonitrile decreased the percentage of females delivering litters and increased the percentage of foetal resorptions. Mean birth weights were reduced and postnatal survival was significantly reduced. Another study reported numerous cardiovascular and urogenital malformations in surviving foetuses.

IARC has concluded that dichloro-, dibromo-, bromochloro- and trichloroacetonitrile are not classifiable as to their carcinogenicity in humans, ie, Group 3. Dichloroacetonitrile and bromochloroacetonitrile have been shown to be mutagenic in bacterial assays, whereas results for dibromoacetonitrile and trichloroacetonitrile were negative. All four of these halogenated acetonitriles induced sister chromatid exchange and DNA strand breaks and adducts in mammalian cells *in vitro* but were negative in the mouse micronucleus test.

The UK Water Research Foundation (2009) analysed 66 USEPA priority drinking water disinfection by-products (DBPs) for their chronic cytotoxicity and acute genotoxicity in mammalian cells, and ranked the cytotoxicity and genotoxicity of the DBPs. They noted that the majority of DBPs have yet to be chemically characterised, and only a small fraction of DBPs have been evaluated for their biological and toxicological effects. One of their findings was that for cell induced genomic DNA damage, the rank order from the most genotoxic to the least genotoxic of the DBP classes was haloacetonitriles > haloacetamides > halonitromethanes > haloacetaldehydes > haloacetic acids > >2C‑haloacids > halomethanes.

2,2,2-Trichloroacetonitrile is cytotoxic and causes genotoxicity *in vitro*. There is little or no evidence for carcinogenicity. LOAELs for 2,2,2-trichloroacetonitrile were identified at 7.5 mg/kg of body weight per day for embryotoxicity and the NOAEL for teratogenic effects was set at 1 mg/kg of body weight per day. However, later studies suggest that these responses were dependent upon the vehicle used (DWI 2010).

### Derivation of Maximum Acceptable Value

No MAV.

The 1995 DWSNZ and datasheet stated *“Based on health considerations, the concentration of trichloroacetonitrile in drinking-water should not exceed 0.001 mg/L (1 g/L).”* This was a provisional MAV, and it still applied in the DWSNZ 2000.

A tolerable daily intake approach had been used for the derivation of the PMAV of trichloroacetonitrile in drinking-water. The no-observable-adverse-effects level used in the derivation was for decreases in foetal weight, and viability and cardiovascular and urogenital malformations in a teratology study in rats. The MAV had been designated as provisional owing to the lack of long-term studies in the available data base.

The MAV for trichloroacetonitrile in drinking-water had been derived as follows:

1 mg/kg body weight per day x 70 kg x 0.2 = 0.0014 mg/L (rounded to 0.001 mg/L)

2 L x 5000

where:

* no-observable-adverse-effect level = 1 mg/kg body weight per day for foetal resorption, decreased foetal weight and viability and numerous cardiovascular and urogenital malformations from a study in which rats were dosed on gestation days 6 to 8
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.2
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 5,000 (100 for intra- and interspecies variation and 10 for the severity of the effects at doses above the NOAEL and 5 for limitations of the data base ie, lack of a 90-day study).

WHO (2004 and 2011) states that available data are insufficient to serve as a basis for derivation of a guideline value for trichloroacetonitrile. The previous provisional guideline value of 0.001 mg/L was based on a developmental toxicity study in which trichloroacetonitrile was administered by gavage in tricaprylin vehicle, and a recent re-evaluation judged this study to be unreliable in light of the finding in a more recent study that tricaprylin potentiates the developmental and teratogenic effects of halogenated acetonitriles and alters the spectrum of malformations in the fetuses of treated dams.

This unreliability had resulted in trichloroacetonitrile being listed in Table A2.2 of the DWSNZ 2005, which was a list of determinands for which health concerns have been raised but for which no MAV has been set.

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# 2,3,6-Trichloroanisole and 2,4,6-Trichloroanisole

2,3,6-Trichloroanisole:

* CAS No. 50375-10-5. Synonym: 1,2,4-trichloro-3-methoxybenzene.

2,4,6-Trichloroanisole:

* CAS No. 87-40-1. Synonyms are 1,3,5-trichloro-2-methoxybenzene and 2,4,6‑trichloro-1-methoxybenzene.

Note that the dichloroanisoles have been reported to give rise to tastes or odours in drinking-water too – see datasheet.

Pentachloroanisole (CAS No. 1825-21-4) is a metabolite of pentachlorophenol (see datasheet in pesticides section); also called methyl pentachlorophenate, pentachloromethoxybenzene or PCA.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for any chlorinated anisoles; nor do the WHO Guidelines have a guideline value.

### Sources to drinking-water

#### 1. To source waters

2,3,6-Trichloroanisole and 2,4,6-trichloroanisole, described variously as causing musty, earthy mouldy odours in water, are metabolites from biological activity of micro-organisms such as fungi, and actinomycetes. Bromoanisoles can be produced as well in bromide-rich waters (Diaz et al 2004).

2,4,6-Trichloroanisole has been reported to have an odour threshold in water at around 1 ng/L (0.000001 mg/L). The odour threshold of 2,4,6-trichloroanisole has even been reported at about 0.08 pg/L (0.00000008 mg/L), Young et al (1996). The odour thresholds in wine are typically in the range of 2 ng/L for detecting difference and 6 ng/L for recognition (ETS 2011).

2,4,6-Trichloroanisole is the chief cause of cork taint in wine. Corked wine containing 2,4,6-trichloroanisole has a characteristic odour, variously described as resembling a mouldy newspaper, wet dog, damp cloth, or damp basement.

Monochloro, trichloro, tetrachloro and pentachloroanisoles have also been implicated with tastes and odours. Tetrachloroanisole appears approximately three times less potent in wine than 2,4,6-trichloroanisole, while pentachloroanisole is unlikely to reach its odour threshold of 4,000 ng/L (0.004 mg/L) in wine, but is a useful indicator of origins of contamination (ETS 2011).

#### 2. From treatment processes

The base compound, anisole (CAS No. 100-66-3) is also called methoxybenzene or phenyl methyl ether. It is manufactured to prepare fragrances. Because it is not thought to occur in water, disinfection processes are not likely to be the source of any chloroanisoles. Even if anisole appears in water, it only reacts with chlorine at low pH, much less than pH 3 to 4. In fact, according to Piriou et al (2001), due to the biological production of the haloanisoles, the presence of chlorine is more likely to reduce the risk of them forming.

#### 3. From the distribution system

A report of methylation of halogenated phenols by micro-organisms in the distribution system was presented by Karlsson et al (1995). 2,3,6-Trichloroanisole was considered to be the cause of odours in drinking-water in Paris, caused by biological methylation of 2,4,6-trichlorophenol.

### Forms and fate in the environment

Pentachloroanisole is a persistent degradate of PCP and is still found in sediments in US water systems; USGS (2006) gave the following values: log Kow = 5.66; log Koc (where Koc is in mL/g) = 4.62; water solubility = 0.2 mg/L; Henry’s law constant (KH in Pa.m3/mol) expressed as log KH = -2.91.

Pentachloroanisole has been known to taint fish flesh (Allard et al 1987).

### Typical concentrations in drinking-water

### Removal methods

A dose of around 10 mg/L of chlorine dioxide may reduce the 2,3,6-trichloroanisole concentration by about 50–60 percent, but at that dose, chlorite is likely to be excessive. Chlorine and ozone may reduce the concentration but only by about  
30–40 percent, even when dosed at more than 20 mg/L, and potassium permanganate was ineffective (Faust and Aly 1998).

2,4,6-Trichloroanisole has been removed from water using ‘tight’ hydrophobic ultrafiltration (Park et al 2007).

### Analytical methods

#### Referee method

Not needed.

#### Some alternative methods

Various GC/MS methods are used, measuring down to about 1 ng/L, eg, Palmentier and Taguchi (2001). Also see ETS Laboratories.

### Health considerations

Chlorinated anisoles do not present a health risk at the concentrations found in drinking-water.

### Derivation of Maximum Acceptable Value

No MAV.

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# Trichloroethanes

CAS No. 71-55-6: 1,1,1-Trichloroethane. Also called methylchloroform, methyl chloroform, alpha-trichloroethane, methyltrichloromethane, trichloromethylmethane,chlorothene, chloroethene or 1,1,1-TCE.

CAS No. 79-00-5: 1,1,2-Trichloroethane. Also called ethane trichloride, vinyl trichloride or 1,1,2-TCE.

### Maximum Acceptable Value

WHO (2004 and 2011) stated that because 1,1,1-trichloroethane occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a guideline value.

1,1,2-Trichloroethane is not mentioned in WHO.

In DWSNZ 2005, the provisional MAV for 1,1,1-trichloroethane in drinking-water had been 2 mg/L.

The maximum contaminant level or MCL in the US for 1,1,1-trichloroethane (USEPA 2006/2009/2011) is 0.2 mg/L, and 0.005 mg/L for 1,1,2-trichloroethane.

The USEPA (2006) established a lifetime health advisory of 0.2 mg/L for 1,1,1‑trichloroethane; this was removed from the 2011 edition.

The USEPA (2006/2011) established a lifetime health advisory of 0.003 mg/L for 1,1,2‑trichloroethane where the lifetime health advisory isthe concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set a health-based guideline value for 1,1,1-trichloroethane in drinking water.

1,1,1-Trichloroethane and 1,1,2-trichloroethane are “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

1,1,1-Trichloroethane can be released to the aquatic environment as an industrial contaminant. It was once used widely overseas as a cleaning solvent (substituting for carbon tetrachloride), as a solvent for adhesives, coatings and textile dyes, as a coolant and lubricant in metal cutting oils, and as a component in inks and drain cleaners. In order to prevent reaction with aluminium and alloys, commercial grades of 1,1,1‑trichloroethane contain an inhibitor such as nitromethane, N-methylpyrrole, 1,4‑dioxane, butylene oxide, 1,3-dioxolane, or a secondary butyl alcohol. 1,1,1‑Trichloroethane is implicated in health problems related to glue sniffing.

Trichloroethane is listed as a controlled substance in the New Zealand Ozone Layer Protection Act, 1990 and is now phased out so be imported only under permit. There has been little demand for 1,1,1-trichloroethane for several years so its occurrence in the New Zealand environment should have been decreasing for some time. Today it is almost entirely used as a precursor for hydrofluorocarbons, which also have restricted uses.

It may be found in groundwater as a consequence of surface spills or poor handling practice. Tributaries of the Rhine contained 1,1,1-trichloroethane at levels of 0.00005 to 0.002 mg/L. Surface waters in Switzerland contained an average of 0.00006 mg/L. In Europe, groundwater levels were in the range 0.00004 to 0.13 mg/L.

1,1,2-Trichloroethane has been used as a solvent in special industrial situations; its main use overseas is as an intermediate in the production of other chemicals such as vinylidene chloride.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

1,1,1-Trichloroethane is found mainly in the atmosphere, where it may act as a source of ClOx, which plays a vital role in ozone photochemistry. It is moderately soluble in water (about 1,300 to 1,500 mg/L) and can be anaerobically dechlorinated to 1,1‑dichloroethane. A low soil adsorption coefficient suggests that it is mobile in soils and readily migrates to groundwaters where its half life has been reported to be about 200–300 days. It does not bioaccumulate in animals.

If released to soil, 1,1,1-trichloroethane is expected to have high mobility based upon Koc values of 120-151 measured in soil. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 0.072 atm‑cu m/mole. 1,1,1-Trichloroethane may volatilise from dry soil surfaces based upon its vapour pressure. 1,1,1-Trichloroethane is expected to biodegrade slowly in soil with half-lifes of greater than 97 days and greater than 485 days, measured in two soils from Louisiana and Oklahoma, respectively. If released into water, 1,1,1-trichloroethane is not expected to adsorb to suspended solids and sediment based upon the Koc range. The biodegradation half-life of 1,1,1-trichloroethane in an unpolluted anaerobic aquifer was estimated to range from 9 days (1 percent organic carbon content) to 16 years (0.001 percent organic carbon content). Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 1 hour and 4.5 days, respectively. The hydrolysis half-life at pH 7 is 1.1 years for 1,1,1-trichloroethane. BCF values of 0.7 to 4.9 measured in fish, suggests bioconcentration in aquatic organisms is low (EAWAG accessed February 2015).

1,1,2-Trichloroethane solubility in water is about 3500 to 4500 mg/L. It is very rarely found in surface water, where it is classified as not readily biodegradable (half-life about 3 months). It does not adsorb readily to particulate matter. If it reaches groundwater it may persist for more than 16 weeks. The highest concentration found in US groundwater is 0.03 mg/L.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 332 zones, found 1,1,1-trichloroethane in one zone at a concentration of 0.003 mg/L, with the median concentration being “nd” (limit of detection = 0.001 mg/L) (ESR 2001).

257 water utilities in the US reported detecting 1,1,1-trichloroethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.06 mg/L.

31 water utilities reported detecting 1,1,2-trichloroethane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.0037 mg/L.

The maximum concentration of 1,1,1-trichloroethane found in 13,168 samples from 2,769 groundwaters in the UK was 0.126 mg/L, mean 0.0004 mg/L (DWI 2008).

The maximum concentration of 1,1,2-trichloroethane found in 8,366 samples from 2,597 groundwaters in the UK was 0.017 mg/L, mean 0.0008 mg/L (DWI 2008).

### Removal methods

Some removal can be achieved by adsorption on to granular activated carbon, although the adsorption is relatively weak. Some removal is also achievable by air-stripping.

1,1,1-Trichloroethane has been found in only a small proportion of surface waters and groundwaters, usually at concentrations of less than 0.02 mg/L; higher concentrations (up to 0.15 mg/L) have been observed in a few instances.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551).

### Health considerations

1,1,1-Trichloroethane is absorbed rapidly from the lungs and gastrointestinal tract, but only small amounts (about 6 percent in humans) are metabolised.

Large oral doses of 1,1,1-trichloroethane have produced nausea, vomiting and diarrhoea in humans. Acute inhalation exposures result in neurological effects. In fatalities resulting from inhalation, acute congestion of the lungs, fluid build-up and fatty deposits in the liver were reported. High concentrations of 1,1,1-trichloroethane in air can cause respiratory failure and problem with heart rhythm. Chronic exposure to low levels of 1,1,1-trichloroethane had no effect on serum and urine chemistry parameters, which are indicative of liver and kidney damage in humans.

In animals, long-term studies have reported diminished body-weight gains at high doses (above 350 mg/kg body weight). Liver tumours were observed in mice, but not in rats fed 1,1,1-trichloroethane for two years. However, the study reported a high number of accidental deaths in both the control group and the study groups, and the results may be insignificant.

IARC has placed 1,1,1-trichloroethane and 1,1,2-trichloroethane in Group 3 (not classifiable as to its carcinogenicity to humans). 1,1,1-Trichloroethane does not appear to be mutagenic.

In 1987 the USEPA classified 1,1,1-trichloroethane in Group D: not classifiable as to human carcinogenicity, and 1,1,2-trichloroethane in Group C: a possible human carcinogen.

USEPA (2007) found that “1,1,1-trichloroethane provides *inadequate information to assess carcinogenic potential*. Epidemiologic studies of humans chronically exposed to 1,1,1-trichloroethane are inconclusive. A two-year inhalation bioassay showed no treatment-related increase in tumours in rats and mice at an exposure concentration below the maximum tolerated dose. The two available oral cancer bioassays in rats and mice are considered inadequate for evaluation of carcinogenic potential. 1,1,1‑Trichloroethane has been tested extensively for genotoxic potential. The chemical has shown little capacity to produce genotoxic effects in bacteria or fungi. Results in mammalian test systems *in vitro* and *in vivo* were more mixed, but still predominantly negative for assays other than cell transformation. The chemical has been shown to interact weakly with DNA.”

USEPA (2009) states that a concentration of 0.06 mg/L 1,1,2-trichloroethane represents a 10-4 cancer risk.

In a 90 days drinking water study of mice, reduction of P-450 contents in the liver were observed and the NOEL was considered as 3.9 mg/kg/day. In a developmental toxicity study, the chemical was administered by gavage to mice on days 8 through 12 of gestation at dose of only 350 mg/kg/day. No changes including teratogenicity and embryo/fetal viability, and/or postnatal growth and viability were observed. Therefore, the NOEL for developmental toxicity was considered to be 350 mg/kg/day (OECD 2002).

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 20 mg/kg/day for intermediate-duration oral exposure  
(15–364 days) to 1,1,1-trichloroethane.

As at July 2013 ATSDR quotes a minimal risk level (MRL) for 1,1,2-trichloroethane of:

* 0.3 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.04 mg/kg/day for intermediate-duration oral exposure (15–364 days).

The oral reference dose or RfD (USEPA 2006) for 1,1,1-trichloroethane was 0.035 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006) was 1 mg/L. Then USEPA (2007) derived a chronic oral RfD of 2 mg/kg/d, based on a BMDL10 of 2155 mg/kg/d and an uncertainty factor of 1000. They also derived a subchronic RfD of 7 mg/kg/d, based on a BMDL10 of 2155 mg/kg/d and an uncertainty factor of 300. USEPA (2009/2011) also quotes the DWEL at 70 mg/L.

The reference dose or RfD (USEPA 1995/2006/2009/2011) for 1,1,2-trichloroethane is 0.004 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.1 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

WHO (2003 and 2011) states that because 1,1,1-trichloroethane occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value. WHO (2017) stated that a health-based value of 2 mg/L can be calculated for 1,1,1-trichloroethane on the basis of a TDI of 0.6 mg/kg body weight, based on changes in the kidney that were consistent with hyaline droplet nephropathy observed in a 13-week oral study in male rats, and taking into account the short duration of the study.

In DWSNZ 2000 and 2005, the provisional MAV had been derived as follows: a health-based MAV can be calculated for 1,1,1-trichloroethane based on changes in the kidney that were consistent with hyaline droplet nephropathy observed in a 13-week oral study in male rats, and taking into account the short duration of the study.

0.6 mg/kg body weight per day x 70 kg x 0.1 = 2.1 mg/L (rounded to 2 mg/L)

2 L

where:

* the calculation used a TDI of 0.6 mg/kg of body weight
* average weight of adult = 70 kg
* average quantity of water consumed by an adult = 2 L per day
* proportion of tolerable daily intake allocated to drinking-water = 0.1.

In the 1995 DWSNZ and datasheet, the provisional MAV for 1,1,1-trichloroethane had been derived as follows:

1365 mg/m3 x 1.043 m3/d x 0.3 x 70 kg x 0.1 = 2 mg/L

0.03 kg x 2 L/d x 1000

where:

* no-observable-adverse-effect-level = 1365 mg/m3 based on a 14-week inhalation study in male mice
* average mouse body weight = 0.03 kg
* breathing rate = 0.043 m3/d
* adsorption of air concentration by mouse = 0.3
* average weight of adult = 70 kg
* average quantity of water consumed by an adult = 2 L per day
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* uncertainty factor = 1000 (100 for intra- and interspecies variation, and 10 for possible carcinogenicity and short term duration of study.

This is a provisional MAV because of the use of an inhalation study instead of an oral study.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for 1,1,1-trichloroethane is 9 mg/L, and 0.003 mg/L for 1,1,2-trichloroethane. 1,1,1-Trichloroethane also has a subchronic limit of 20 mg/L.

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# Trichloroethene

CAS No. 79-01-6. Also called trichloroethylene, or occasionally TCE, 1,1-dichloro-2-chloroethylene, ethinyl trichloride, acetylene trichloride, ethylene trichloride, 1-chloro-2,2-dichloroethylene, or 1,1,2-trichloroethene and various trade names.

### Maximum Acceptable Value (provisional)

Based on health considerations, the concentration of trichloroethene in drinking-water should not exceed 0.02 mg/L. WHO (2005, 2011 and 2017) stated that the guideline value was designated as provisional because of deficiencies in the toxicological database.

In previous DWSNZ, the provisional MAV had been 0.08 mg/L.

The maximum contaminant level or MCL (USEPA 2006/2009/2011) is 0.005 mg/L. The maximum acceptable concentration in Canada is 0.005 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that data are inadequate to set a guideline value for trichloroethylene in drinking water.

The Prescribed Concentration or Value (PCV) for trichloroethene in England and Wales is 0.01 mg/L. See Notes.

WHO (2005) considers the odour threshold to about 0.3 mg/L in water; however, trichloroethene is not an aesthetic determinand in the DWSNZ.

Trichloroethene is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Trichloroethene can be released to the aquatic environment as an industrial contaminant. It was used in dry-cleaning, for degreasing of metal parts (major use), as a solvent for fats, waxes, resins, oils, rubber, paints, varnishes, the decaffeination of coffee, and as an inhalation analgesic and anaesthetic. Its use in industrialised countries has declined sharply since 1970. Commercial grades of trichloroethene, formulated to meet use requirements, differ in the amount and type of added inhibitor and stabiliser – see IARC (1995) and EU (2004) for a long list of impurities and chemicals added to trichloroethene.

Trichloroethene is expected to be <0.001 mg/L in natural freshwaters but has been measured at around 0.05 mg/L in rivers in industrial areas of Europe in the past.

EU (2004) includes discussion on the possibility that seawater algae may be a natural source of trichloroethylene.

#### 2. From treatment processes

Trichloroethene may be formed from naturally organic precursors during chlorination of drinking-water.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

It is expected that exposure to trichloroethene from air will be greater than that from food or drinking-water.

Trichloroethene is readily released to the atmosphere, where it is highly reactive. Its solubility in water is about 1,000–1,300 mg/L. Trichloroethene is removed from water mainly by volatilisation and possibly with some partitioning to sediment and suspended organic matter, photodegradation and hydrolysis play minor roles. Trichloroethene in anaerobic groundwater may degrade to more toxic compounds, including vinyl chloride. Trichloroethene is highly mobile in soil and may leach into groundwater supplies. Bioconcentration of trichloroethene in aquatic species is fairly low. It has a low n‑octanol/water partition coefficient (log Kow 2.29–2.42), a high vapour pressure  
(8.0–9.9 kPa at 20–25°C), and a Henry’s law constant of 1.1 kPa·m³/mol at 25°C.

If released to soil, trichloroethylene is expected to have high mobility based upon an average Koc of 101, measured in 32 soils, hence adsorption is not likely to be a significant removal process. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 9.85 x 10-3 atm‑cu m/mole. Trichloroethylene may volatilise from dry soil surfaces based upon its vapour pressure. Utilising the Japanese MITI test, 2.4 percent of the theoretical BOD was reached in two weeks indicating that biodegradation is not a fast environmental fate process. Cometabolic biodegradation of trichloroethylene has been reported under aerobic conditions where additional nutrients have been added. Under anaerobic conditions, as might be seen in flooded soils, sediments or aquifer environments, trichloroethylene is slowly biodegraded via reductive dechlorination; the extent and rate of degradation is dependent upon the strength of the reducing environment. If released into water, trichloroethylene is expected to adsorb to suspended solids and sediment based upon the average Koc (EU 2004 quotes logKoc = 2.2). Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lives for a model river and model lake are three hours and 4.6 days, respectively. BCFs of 4 to 39 suggest bioconcentration in aquatic organisms is low to moderate. Trichloroethylene is not hydrolysed by water under normal conditions. However, slow photooxidation in water has been noted (half-life of 10.7 months) (EAWAG accessed February 2015).

The biodegradation of tetrachloroethene (or perchloroethylene, PCE – qv) in groundwater may also lead to the formation of trichloroethene.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 505 zones, found trichloroethene twice in one zone at concentrations of 0.0016 and 0.0017 mg/L, with the median concentration being “nd” (limit of detection = 0.001 mg/L) (ESR 2001).

Trichloroethene has been detected frequently in natural water and drinking-water in Canada and other countries. Due to its high volatility, trichloroethene concentrations are normally low in surface water (0.001 mg/L). It is found mostly in groundwater from which it is not lost to air.

Recent data from Canada for raw (surface water and groundwater), treated and distributed water indicated that more than 99 percent of samples contained trichloroethene at concentrations less than or equal to 0.001 mg/L. The maximum concentration was 0.081 mg/L. Of those samples with detectable trichloroethene concentrations, most were from groundwater (WHO 2005).

In 1995, a national review of TCE occurrence data in Canada was carried out to determine the extent of groundwater contamination by TCE and the number of people potentially exposed to contaminated drinking water. The majority of sites were from Ontario and New Brunswick. The review was based on urban groundwater supplies. Of the 481 municipal/communal and 215 private/domestic groundwater supplies (raw water), 8.3 percent and 3.3 percent, respectively, contained TCE, at average maximum concentrations of 25 µg/L and 1,680 µg/L, respectively. A majority of all sites (93 percent) had non-detectable levels (<0.01–10 µg/L), 3.6 percent had a maximum concentration of <1 µg/L, 1.4 percent had a maximum of 1–10 µg/L, 0.43 percent had a maximum of 10–100 µg/L and 1.3 percent had a maximum of >100 µg/L. From Health Canada (2005).

In the USA, trichloroethene has been the volatile organic contaminant that is most frequently found in groundwater and the one present in the highest concentrations. Trichloroethene was detected (detection limit 0.0002 mg/L) in 91 of 945 (9.6 percent) samples of finished water using groundwater sources nationwide. The median level in positive samples was 0.001 mg/L, and the maximum was 0.13 mg/L (WHO 2005).

653 water utilities in the US reported detecting trichloroethylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.015 mg/L.

### Removal methods

Trichloroethene present in contaminated source waters may be removed by adsorption on to granular activated carbon or air stripping, or more effectively in combination. Treatment with ozone has been shown to be effective too. WHO (2005) summarises many case studies.

Although considered overseas to arise predominantly from waters containing industrial contamination, there is evidence that trichloroethene is also formed as a disinfection by-product. However, WHO (2005) makes no reference to this.

When its appearance in a water results from chlorination, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products.

Trichloroethene, once present as a disinfection by-product, can be removed by adsorption on to granular activated carbon, or by air stripping.

WRF (2014) reports that trichloroethylene is characterised with a relatively high Henry’s law constant (0.289 dimensionless air/water at 20°C). Low profile air stripping is very effective for trichloroethylene removal at high air to water ratios at all examined temperatures. The removal efficiency in the case of trichloroethylene seems to be more sensitive to the temperature change. The drop in removal efficiencies was noticeable even at the 150–167 range air to water ratios. 99.5 percent removal was achieved at 20°C (150 air to water ratio), 98.1 percent removal efficiency at 12°C (167 air to water ratio), and 96.6 percent at 4°C (162 air to water ratio). The removal efficiency at 20°C dropped to 94.7 percent at the lowest air to water ratio of 53. Both removal efficiencies at 12°C and 4°C at the two air to water ratios of about 53 and 70 were considered unreliable due to the inconsistent individual trays removal results obtained at these conditions.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

2. Liquid–Liquid Extraction and Gas Chromatography with Electron-Capture Detection (EPA 551).

3. Purge and Trap Capillary-Column Gas Chromatographic Method with photoionisation conductivity detection (EPA 503.1).

### Health considerations

At least 80 percent of ingested trichloroethene is absorbed and is distributed widely, with the highest concentration being in body fat. Inhalation studies with humans show that 40–75 percent of the retained dose is metabolised. Trichloroethene is eliminated with a biological half-life of 1.5 hours while the metabolites are excreted more slowly. Transplacental diffusion has been demonstrated in humans following inhalation.

Humans exposed to high concentrations of trichloroethene have experienced central nervous system depression. Oral exposure of humans to 15–25 mL (21–35 g) of trichloroethene resulted in vomiting and abdominal pain, followed by transient unconsciousness.

Humans exposed occupationally to trichloroethene had an increase in serum transaminases, which indicates damage to the liver parenchyma. Neurological abnormalities were associated with occupational exposure to 14–85 ppm trichloroethene for one month to 15 years, including decreased appetite, sleep disturbances, ataxia, vertigo, headache, and short-term memory loss.

In view of the sufficient weight of evidence of carcinogenicity in two species of experimental animals with supporting human data, IARC (1995) classified trichloroethene as Group 2A, probably carcinogenic to humans. In summary, although several studies have indicated a positive association between exposure to solvents, including trichloroethene, and human cancer, further study is still necessary to better specify the specific agents that confer this risk and to estimate the magnitude of that risk.

Trichloroethylene is weakly mutagenic *in vitro*. In the presence of metabolic activation, trichloroethylene tested positive in several bacterial and fungal gene mutation assays. Trichloroethylene also tested positive in a mouse lymphoma gene mutation assay, and unscheduled DNA synthesis (UDS) was reported in several studies (NICNAS 2000).

The USEPA (2009/2011) quotes a health advisory of 0.3 mg/L for trichloroethene, representing a 10-4 cancer risk.

Trichloroethene appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

As at October 2015 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.0005 mg/kg/day for intermediate-duration oral exposure (15–365 days) and chronic-duration oral exposure (>364 days) to trichloroethene.

The oral reference dose or RfD for trichloroethene (USEPA 2006/2009/2011) for trichloroethene is 0.007 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.2 mg/L. Note however that USEPA (2011a) quotes a chronic oral RfD of 0.0005 mg/kg/d.

### Derivation of Maximum Acceptable Value

WHO (2005) considered both cancer and non-cancer end-points in the derivation of the guideline value for TCE in drinking-water. The health-based value of 0.02 mg/L derived for reproductive effects was selected as the guideline value, as it is protective for both cancer and non-cancer end-points. It should be noted that the allocation factor of 50 percent of the TDI for drinking-water was used rather than the 20 percent that was used previously, since the discontinuation of TCE in many medical applications and some consumer products has decreased exposure to this contaminant in these situations. The guideline remains provisional on the basis of uncertainties in the toxicological database.

WHO (2005/2011/2017) used the benchmark dose or BMD10 approach, (the lower 95 percent confidence limit corresponding to a 10 percent increase in extra risk of fetal heart malformations over the background), using studies of the effects of trichloroethene on cardiac anomalies as well as the observation of similar malformations in studies of TCE metabolites.

The PMAV was calculated as follows:

0.00146 mg/kg body weight per day x 70 kg x 0.5 = 0.0219 mg/L (rounded to 0.02 mg/L)

2 litres per day

where:

* 0.00146 mg/kg of body weight per day is the TDI, as derived above
* 70 kg is the average body weight of an adult
* 0.5 is the proportion of total daily intake that is allocated to drinking-water
* 2 litres/day is the daily volume of water consumed by an adult.

The basis used for the PMAV in earlier editions of the DWSNZ had been:

A tolerable daily intake approach has been used for the derivation of the provisional MAV for trichloroethene in drinking-water. The lowest-observable-adverse-effect level used in the derivation is for minor effects on relative liver weight in a six-week study in mice.

The PMAV for trichloroethene in drinking-water was derived as follows:

100 x (5/7) mg/kg body weight per day x 70 kg x 0.1 = 0.08 mg/L

2 L x 3000

where:

* lowest-observable-adverse-effect level = 100 mg/kg body weight per day for minor effects on relative liver weight in a six-week study in mice (normalised for five days/week dosing in derivation)
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 3000 (100 for intra- and interspecies variation and 10 for limited evidence of carcinogenicity, and 3 in view of the short duration of the study and the use of a LOAEL rather than a NOAEL).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for trichloroethene is 0.005 mg/L, as are the short term and subchronic limits. A limit of 0.002 mg/L was established for cancer.

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# Trichloromelamine

CAS No. 7673-09-8 and 12379-38-3. Also called 2-N,4-N,6-N-trichloro-1,3,5-triazine-2,4,6-triamine, N,N’,N’’-trichloro-2,4,6-triamino-1,3,5-triazine, 2,4,6‑tris(chloroamine)triazine, chloromelamine, TCM and various trade names.

### Maximum Acceptable Value

The DWSNZ do not have a MAV for trichloromelamine; nor do the WHO Guidelines have a guideline value.

### Sources to drinking-water

#### 1. To source waters

Trichloromelamine is used as a disinfectant and sanitiser (producing chlorine), primarily on food-contact surfaces. It can also be used as an algicide. It can enter water systems from hotel glassware washing systems.

USEPA (2005) stated that none of the uses associated with trichloromelamine are expected to impact either surface water or groundwater resources, so no drinking water assessment was performed.

#### 2. From the distribution system

No known sources.

### Forms and fate in the environment

The half-life of trichloromelamine in soils has been estimated to be about 38 days and 150 days in sediments. With an estimated Koc of 150, it is likely to be immobile and persistent in soils and sediments and may not pose a concern for groundwater contamination. However, as trichloromelamine is immobile, it may pose a concern for surface water contamination due to soil erosion. Because use of trichloromelamine is limited to use as a food and food-contact surface sanitiser in restaurants and similar establishments, and in military mess halls, trichloromelamine is not expected to enter the environment and exposure to soil and water should be minimal. The major residue is melamine (qv).

Water solubility of trichloromelamine is 640 mg/L.

### Health considerations

For the chronic dietary endpoint, the uncertainty factor of 300 includes a 10x interspecies extrapolation, 10x intraspecies variation, and 3x extrapolation from a subchronic study to a chronic endpoint. The USEPA chose the NOAEL of 30 mg/kg/day from the subchronic study in rats as a conservative endpoint for all exposure scenarios. Residues of trichloromelamine are likely to disappear quickly based on the chemistry of trichloromelamine, and subchronic and chronic exposures are likely to be very low. The acute RfD (and PAD) is 0.3 mg/kg/d. The chronic RfD (and PAD) is 0.1 mg/kg/d.

Trichloromelamine is expected to break down rapidly into hypochlorous acid and melamine – refer to the melamine datasheet for further information.

### Derivation of Maximum Acceptable Value

No MAV.

### References

USEPA. 2005. *Re-registration Eligibility Decision for Trichloromelamine*. EPA739‑R‑05‑008. Washington, DC: US Environmental Protection Agency. 65 pp. Available at: http://archive.epa.gov/pesticides/reregistration/web/pdf/trichloromelamine\_red.pdf.

# 2,4,6-Trichlorophenol

CAS No. 88-06-2. Also called 2,4,6-TCP and various trade names.

### Maximum Acceptable Value

Based on health considerations, the concentration of 2,4,6-trichlorophenol in drinking-water should not exceed 0.2 mg/L.

The maximum acceptable concentration (MAC) for 2,4,6-trichlorophenol in Canadian drinking water is 0.005 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on aesthetic considerations (taste and odour), the concentration of 2,4,6-trichlorophenol in drinking water should not exceed 0.002 mg/L, and it would not be a health concern unless the concentration exceeded 0.02 mg/L.

2,4,6-Trichlorophenol is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

2,4,6-Trichlorophenol may occur in raw water as an industrial contaminant or from agricultural use. It may be used in the production of tetra-, and pentachlorophenol, as a germicide, glue and wood preservative, and an antimildew agent. However, it does not appear on NZFSA’s Full List of ACVM approved veterinary medicines and pesticides, as at 2009. 2,4,6-Trichlorophenol may also enter the environment as emissions from combustion of fossil fuels and incineration of municipal wastes.

#### 2. From treatment processes

Chlorophenols are most likely to occur in drinking-water as disinfection by-products through the reaction of naturally-occurring organic matter with chlorine.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Because 2,4,6-trichlorophenol is moderately water-soluble (400–500 mg/L), weakly acidic, and has a low vapour pressure, it is anticipated that volatilisation does not play a significant role in removing it from water. The importance of photolysis of trichlorophenols in the natural environment is unknown. Sorption of trichlorophenol to organic-rich sediments is likely to be a significant removal mechanism for trichlorophenols from water. Biodegradation has been reported. Aquatic biota may bioconcentrate chlorinated phenols with bioconcentration factors increasing with increasing chlorine substitution.

A pKa value of 6.23 indicates that the dissociated form of 2,4,6-trichlorophenol will be present in moist soils and aquatic systems. 2,4,6-Trichlorophenol is expected to have low mobility in soils based upon Koc values in the range of 150–2,200 depending on pH. This compound is not expected to volatilise from dry soil surfaces based on its vapour pressure. 2,4,6-Trichlorophenol may volatilise slowly from moist soil surfaces given its estimated Henry’s Law constant of 2.6 x 10-6 atm‑cu m/mole. This compound is expected to biodegrade in the environment with aerobic and anaerobic biodegradation half-lifes of about five and 20 days respectively in soils. In water, 2,4,6‑trichlorophenol is expected to dissociate somewhat to 2,4,6-trichlorophenolate based on its pKa. 2,4,6-Trichlorophenol is expected to adsorb to sediment and particulate matter. It is expected to volatilise slowly from water surfaces given its Henry’s Law constant. Estimated volatilisation half-lifes of 2,4,6-trichlorophenol for a model river and model lake are 20 and 150 days, respectively. 2,4,6-Trichlorophenol is expected to undergo photolysis in surface waters based on an aqueous photolysis half-life of 2.1 hours when irradiated with light at environmentally relevant wavelengths. 2,4,6-Trichlorophenol is expected to biodegrade in aquatic systems based on river die away tests with estimated half-lifes in the range of 3 to 70 days. The potential for bioconcentration is expected to be high based on BCF values of 250–310 measured in fish (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 494 zones, did not find any 2,4,6-trichlorophenol at detectable concentrations (limit of detection = 0.005 mg/L) (ESR 2001).

2,4,6-Trichlorophenol concentrations are generally less than 0.001 mg/L (WHO 2004/2011).

The maximum concentration found in 4863 samples from 1635 groundwaters in the UK was 0.0019 mg/L, mean 0.00004 mg/L (DWI 2008).

### Removal methods

Chlorophenols can be removed from contaminated source water by adsorption on to activated carbon. The effectiveness of the processes is pH dependent. Greater adsorption occurs as the pH is lowered.

However, as this compound is more likely to arise in New Zealand waters principally as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products. The formation of chlorophenols can be reduced largely by the use of chlorine dioxide in place of chlorine.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Chlorophenols can be removed by adsorption on to activated carbon. The effectiveness of the processes is pH dependent. Greater adsorption occurs as the pH is lowered.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

### Analytical methods

#### Referee method

Micro Liquid/Liquid Extraction Gas Chromatographic Method (APHA 6251B).

#### Some alternative methods

1. Liquid/Liquid Extraction Gas Chromatographic Method (APHA 6420).

2. Liquid/Solid Extraction Gas Chromatographic/Mass Spectrometric Method (EPA 526).

3. Liquid/Liquid Extraction Gas Chromatographic/Mass Spectrometric Method (APHA 6410B).

4. Acetylation Liquid/Liquid Extraction Gas Chromatographic/Mass Spectrometric Method (EPA 1653).

### Health considerations

Chlorophenols are well-absorbed after oral administration and they readily penetrate the skin. Chlorophenols do not appear to accumulate in body tissues in rats but are metabolised rapidly and eliminated from the body, principally in urine.

2,4,6-Trichlorophenol induced lymphomas and leukaemias in male rats and hepatic tumours in male and female mice. The compound has not been shown to be mutagenic in the Ames test but has shown weak mutagenic activity in other in vitro and in vivo studies. The International Agency for Research on Cancer has concluded that there is limited evidence in experimental animals for the carcinogenicity of 2,4,6-trichlorophenol, and that combined exposures to polychlorophenols or to their sodium salts are possibly carcinogenic to humans (Group 2B).

The USEPA has determined that 2,4,6-trichlorophenol is a probable carcinogen (B2) based on leukemias in male rats and hepatocellular adenomas or carcinomas in male mice. The USEPA (2009/2011) quotes a health advisory of 0.3 mg/L for 2,4,6‑trichlorophenol, representing a 10-4 cancer risk.

2,4,6-Trichlorophenol appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

The reference dose or RfD (USEPA 2006/2009/2011) for 2,4,6-trichlorophenol is 0.0003 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.01 mg/L.

### Derivation of Maximum Acceptable Value

A MAV for 2,4,6-trichlorophenol has been derived by applying the linearised multistage model to leukaemias in male rats observed in a 2-year feeding study. The hepatic tumours found in this study were not used in risk estimation, owing to the possible role of contaminants in their induction. The concentration in drinking-water associated with an excess lifetime cancer risk of one per 100,000 (10-5) is 0.2 mg/L.

The drinking water concentration of 2,4,6-trichlorophenol associated with an excess lifetime cancer risk of 10-4, 10-5, and 10-6 is 0.30 mg/L, 0.03 mg/L, and 0.003 mg/L, respectively (USEPA 1987).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The cancer health risk limit for 2,4,6-trichlorophenol is 0.03 mg/L.

The DWSNZ includes a Guideline Value of 0.002 mg/L for 2,4,6-trichlorophenol for taste. The Health Canada aesthetic objective is 0.002 mg/L.

The USEPA established an organoleptic effect criterion of 0.002 mg/L for 2,4,6‑trichlorophenol. Source: [*Quality Criteria for Water*, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

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# Trichlorophenols (other)

By far the most important trichlorophenol after 2,4,6-trichlorophenol (see previous datasheet) is 2,4,5-trichlorophenol, CAS No. 95-95-4. Also called 2,4,5-TCP.

The other trichlorophenols are:

* 2,3,4-trichlorophenol CAS No. 15950-66-0
* 2,3,5-trichlorophenol CAS No. 933-78-8
* 2,3,6-trichlorophenol CAS No. 933-75-5
* 3,4,5-trichlorophenol CAS No. 609-19-8.

### Maximum Acceptable Value

There are no MAVs for the above trichlorophenols in the DWSNZ, and the WHO Guidelines do not mention them.

### Sources to drinking-water

#### 1. To source waters

2,4,5-Trichlorophenol may occur in raw water as an industrial contaminant or from agricultural use where it has been used as an herbicide. The largest use for 2,4,5-TCP has been as an intermediate, especially in the production of the herbicides 2,4‑dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). It may be used as a fungicide/bactericide (not on the ERMA approved list as at 2009), an intermediate in the manufacture of herbicides (in which it may also appear as an impurity), and in hide and leather processing. It is also the predominant metabolite of chlorophenoxy pesticides, particularly fenoprop and 2,4,5-T.

#### 2. From treatment processes

Chlorophenols are most likely to occur in drinking-water as disinfection by-products through the reaction of naturally-occurring organic matter with chlorine.

#### 3. From the distribution system

No known sources.

### Forms and fate in the environment

Because 2,4,5-trichlorophenol is fairly water-soluble (about 900–1,000 mg/L), weakly acidic, and has a low vapour pressure, it is anticipated that volatilisation does not play a significant role in removing it from water. Photolysis of trichlorophenols in the natural environment is probably important. Sorption of trichlorophenol to organic-rich sediments is likely to be a significant removal mechanism for trichlorophenols from water. Biodegradation has been reported. Aquatic biota may bioconcentrate chlorinated phenols with bioconcentration factors increasing with increasing chlorine substitution.

IPCS (1989) reported that 3,4,5-trichlorophenol concentrations were little changed in clay loam soils after 160 days.

A pKa value of 7.4 indicates that the dissociated form of 2,4,5-trichlorophenol will be present in moist soils and aquatic systems. 2,4,5-Trichlorophenol is expected to have low mobility in soils based upon a Koc value of 2,300 measured in soil, but the mobility of the anion is expected to be greater that that of the neutral species. This compound is not expected to volatilise from dry soil surfaces given its vapour pressure. 2,4,5‑Trichlorophenol may volatilise slowly from moist soil surfaces given its estimated Henry’s Law constant of 1.6 x 10-6 atm‑cu m/mole. It is expected to biodegrade in the environment with aerobic half-lives of about 15 days in soil and 690 days in water. In water, 2,4,5-trichlorophenol is expected to dissociate somewhat to 2,4,5‑trichlorophenolate based on its pKa value. 2,4,5-Trichlorophenol is expected to undergo photolysis in surface waters with an estimated photolysis half-life of about one hour. This compound is expected to adsorb to sediment and particulate matter based on its Koc value. It is expected to volatilise from water surfaces given its Henry’s Law constant. Estimated volatilisation half-lifes of 2,4,5-trichlorophenol for a model river and model lake are 32 and 236 days, respectively. The potential for bioconcentration in aquatic organisms is expected to be high based on BCF values of 121–825 measured in carp (EAWAG accessed February 2015).

### Removal methods

Chlorophenols can be removed from contaminated source water by adsorption on to activated carbon. The effectiveness of the processes is pH dependent. Greater adsorption occurs as the pH is lowered.

However, as these compound may possibly arise in New Zealand waters principally as a disinfection by-product, the preferred method for minimising its formation is to reduce the concentration of natural organic matter (NOM) coming into contact with the chlorine. Removal of NOM can be achieved by coagulation/flocculation with aluminium or iron salts. In some cases, adequate removal of NOM may be attained using organic polyelectrolytes as coagulants. NOM can also be removed by adsorption on to activated carbon, activated alumina or ion exchange resins, however these methods are generally more expensive than coagulation.

Some reduction in disinfection by-product formation can be achieved by introducing the disinfectant into the water after the water has passed through all treatment steps, ie, avoiding prechlorination wherever possible.

Chlorinated disinfection by-product formation can be reduced by the use of an alternative disinfectant such as ozone or chlorine dioxide, although these too have their associated disinfection by-products. The formation of chlorophenols can be reduced largely by the use of chlorine dioxide in place of chlorine.

Where minimising disinfection by-product formation cannot reduce the concentration of disinfection by-products to a satisfactory level, methods to remove the disinfection by-products themselves may be considered. Chlorophenols can be removed by adsorption on to activated carbon. The effectiveness of the processes is pH dependent. Greater adsorption occurs as the pH is lowered.

Note that the application of chlorine-containing disinfectants to activated carbon adsorbers should be avoided because of the unknown health effects of compounds formed through surface reactions between adsorbed contaminants and the disinfectants.

### Analytical methods

#### Referee method

Micro Liquid/Liquid Extraction Gas Chromatographic Method (APHA 6251B).

#### Some alternative methods

1. Liquid/Liquid Extraction Gas Chromatographic Method (APHA 6420).

2. Liquid/Solid Extraction Gas Chromatographic/Mass Spectrometric Method (EPA 526).

3. Liquid/Liquid Extraction Gas Chromatographic/Mass Spectrometric Method (APHA 6410B).

4. Acetylation Liquid/Liquid Extraction Gas Chromatographic/Mass Spectrometric Method (EPA 1653).

### Health considerations

Chlorophenols are well-absorbed after oral administration and they readily penetrate the skin. Chlorophenols do not appear to accumulate in body tissues in rats but are metabolised rapidly and eliminated from the body, principally in urine.

The oral RfD for 2,4,5-trichlorophenol was calculated at 0.1 mg/kg/d (USEPA 1988).

The combined exposure of chlorophenols as a group has been classified as an IARC Group 2B carcinogen. This classification is based on limited evidence of carcinogenicity in humans exposed during the production of chlorophenoxy herbicides and sufficient animal evidence of carcinogenicity for 2,4,6-TCP. The evidence of carcinogenicity in animals for 2,4,5-TCP was considered inadequate.

Tri- and tetrachloro-dibenzo-p-dioxins are frequently found in trichlorophenol formulations (IPCS 1989).

### Derivation of Maximum Acceptable Value

No MAV.

The USEPA established an organoleptic effect criterion of 0.001 mg/L for 2,4,5‑trichlorophenol. Source: [*Quality Criteria for Water*, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

IPCS (1989) reports the organoleptic threshold in water for 2,3,6-trichlorophenol is 0.0005 mg/L, 0.001 mg/L for 2,4,5-trichlorophenol and 0.002 mg/L for 2,4,6‑trichlorophenol.

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# 1,2,3-Trichloropropane

CAS No. 96-18-4. Also called trichloropropane, allyl trichloride, glycerol trichlorohydrin, TCP, glyceryl trichlorohydrin and trichlorohydrin.

1,1,2-Trichloropropane may also be encountered, CAS No. 598-77-6. This chemical has also been called 2-chloropropylidene chloride.

All references in this datasheet are to 1,2,3-trichloropropane unless otherwise specified.

### Maximum Acceptable Value

1,2,3-Trichloropropane does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

The USEPA concluded on 22 September 2009 that 1,2,3-trichloropropane is known or anticipated to occur in public water supplies and may require regulation. Therefore they added 1,2,3-trichloropropane to their CCL 3 (Drinking Water Contaminant Candidate List 3, USEPA 2009b). The USEPA (2006) established a lifetime health advisory of 0.04 mg/L, where the lifetime health advisory is the concentration of a chemical in drinking water that is not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure, and is based on exposure of a 70‑kg adult consuming two litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity.

### Sources to drinking-water

There are no known natural sources of 1,2,3-trichloropropane. 1,2,3-Trichloropropane has been used as an industrial solvent for oils and fats, waxes and resins, in paint removers, thinners and cleaners. It is not known exactly how much of it is made or used now, but it may still be a large amount (ATSDR 1992). Most of the 1,2,3‑trichloropropane is used to make other substances such as polysulfide rubbers and some pesticides. Epichlorohydrin and certain soil fumigants (eg, dichloropropene – see datasheet in the pesticides section) and nematocides contain 1,2,3‑trichloropropane as an impurity.

### Form and fate in the environment

Using the Henry’s law constant, a half-life of 6.9 hours was calculated for evaporation from a model river 1 m deep, flowing at 1 m/sec, with a wind velocity of 3 m/sec, and neglecting adsorption to sediment. 1,2,3-Trichloropropane in surface water is not readily biodegraded but is expected to volatilise to the atmosphere before producing degradation products. 1,2,3-Trichloropropane might be leached from soil into groundwater, due to the low soil sorption coefficients (Koc). DWI (2014) quotes a Henry’s Law constant of 0.000343 atm.m3/mole at 25°C, a log Kow of 2.27, a Koc of 77 to 95. TCP is very recalcitrant in groundwater systems due to its long hydrolysis half-life and low biodegradability.

Water solubility – 1,750 mg/L.

### Typical concentrations in drinking-water

Data from the EPA STORET Data Base indicate that 1,2,3-trichloropropane was found in 39 percent of 941 samples of groundwater at a median concentration of 0.0007 mg/L, at an average concentration of 0.0001 mg/L. It was usually found where the pesticide dichloropropene had been used. 132 water utilities in the US reported detecting 1,2,3‑trichloropropane in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.04 mg/L.

Concentrations of 1,2,3-trichloropropane in drinking water range from <0.24 to 2.654 μg/L, in groundwater from 0.02 to >100 μg/L, in fresh surface water from 0.027 to 100 μg/L, in marine surface water from <0.1 to <0.5 μg/L and in effluents from 0 to <200 μg/L (DWI 2014).

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 36,848 drinking water samples for 1,2,3-trichloropropane between 2013 and 2015, and found 256 samples exceeded the minimum reporting level (MRL) of 0.03 µg/L, and 55 samples contained >0.0004 mg/L.

### Removal methods

1,2,3-Trichloropropane has low to moderate absorption capacity for granular activated carbon (GAC). 1,2,3-Trichloropropane is expected to undergo limited removal by air stripping. Advanced oxidation processes require high doses and long contact times (DWI 2014).

WRF (2014) reports that 1,2,3-trichloropropane is characterised with a low Henry’s law constant (0.0096 dimensionless air/water), which is the lowest among the 13 focus VOCs trialled. Low profile air stripping is effective for 1,2,3-trichloropropane removal at high air to water ratios, and at high temperatures at moderate air to water ratios. 1,2,3‑Trichloropropane was almost completely removed at the three temperatures and air to water ratio of above 500. Lower removal efficiencies (94.2 percent, 80.1 percent and 57.8 percent) were observed at lower air to water ratios (150, 167 and 162), sub ug/L effluent was not achieved at 12°C and 4°C, and the temperature change effect was significant at this range of air to water ratios. At the low air to water ratio of about 53, the removal efficiency dropped to between 50 percent and 20 percent, at temperatures between 20°C and 4°C, and no sub ug/L effluent was achieved. GAC filtration is the most cost effective treatment.

### Health considerations

Toxicokinetic data demonstrate the ability of 1,2,3-trichloropropane or its metabolites to bind to intracellular macromolecules such as proteins and nucleic acids (USEPA 2009a).

Clear evidence of carcinogenicity was found for 1,2,3-trichloropropane in male and female rats at doses of 3 mg/kg/day or more and in male and female mice at doses of 6 mg/kg/day or more in a 2-year gavage study. USEPA (2009a) states that 1,2,3‑trichloropropane is “likely to be carcinogenic to humans,” based on a statistically significant and dose-related increase in the formation of multiple tumours in both sexes of two species from an NTP (1993) chronic oral bioassay. Statistically significant increases in incidences of tumours of the oral cavity, forestomach, pancreas, kidney, preputial gland, clitoral gland, mammary gland, and Zymbal’s gland in rats, and the oral cavity, forestomach, liver, Harderian gland, and uterus in mice were reported. No human oral exposure studies are available.

1,2,3-Trichloropropane is reasonably anticipated to be a human carcinogen based on sufficient evidence of malignant tumour formation at multiple sites in multiple species of experimental animals. IARC considered 1,2,3-trichloropropane is probably carcinogenic to humans (Group 2A). In making the overall evaluation, the Working Group took into account the following evidence:

* 1,2,3-trichloropropane causes tumours at multiple sites and at high incidence in mice and rats
* the metabolism of 1,2,3-trichloropropane is qualitatively similar in human and rodent microsomes
* 1,2,3-trichloropropane is mutagenic to bacteria and to cultured mammalian cells and binds to DNA of animals treated *in vivo*.

DWI (2014) reports that overall, the data indicate that 1,2,3-trichloropropane requires metabolic activation to produce genotoxic effects *in vitro*. However, while *in vivo* studies indicate that 1,2,3-trichloropropane may bind to DNA following intraperitoneal administration, oral administration did not alter DNA synthesis or induce lethal mutations.

The USEPA (1990) derived a chronic oral RfD for 1,2,3-trichloropropane of 0.006 mg/kg/d based on a NOAEL of 8 mg/kg/day, five days/week converted to 5.71 mg/kg/day, and a UF of 1,000 (10 for intraspecies, 10 for interspecies extrapolation, and 10 for extrapolating subchronic to chronic exposures). USEPA (2009, 2009a and 2011) revised this to 0.004 mg/kg/d, based on a BMDLADJ of 1.1 mg/kg/d and an uncertainty factor of 300. The Drinking Water Equivalent Level or DWEL (USEPA 2006) was 0.2 mg/L, and in 2009/2011 this became 0.1 mg/L. The USEPA established a one-day health advisory of 0.6 milligrams per litre (mg/L) and a 10-day health advisory of 0.6 mg/L for TCP in drinking water for a 10-kilogram (kg) child (USEPA 2014).

DWI (2014) states that for Repeat Oral Dose Toxicity and Carcinogenicity a LOAEL may be derived of 3 mg/kg bw/day (approximately 2.1 mg/kg bw/day, adjusting to a seven-day/week dosing regimen) based on the occurrence of neoplasms at all doses. Using this LOAEL an oral Tolerable Daily Intake (TDI) of 0.002 mg/kg bw/day (2 μg/kg bw/day; rounded) is derived.

The USEPA (1988) derived a chronic oral RfD for 1,1,2-trichloropropane of 0.005 mg/kg/d based on a NOAEL of 15 mg/kg/day, finding mild lesions in the liver, kidney and thyroid in a rat oral subchronic study.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.08 mg/kg/day for intermediate-duration oral exposure (15–364 days) to 1,2,3-trichloropropane.

The USEPA previously established a lifetime exposure limit for 1,2,3-trichloropropane from drinking-water of 0.2 mg/L, changed this to 0.04 mg/L in 2006, but lists no lifetime exposure limit in 2009 or 2011.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The acute, short-term, chronic and subchronic health risk limits are 0.007 mg/L, and a limit of 0.000003 mg/L was set for cancer.

1,2,3-Trichloropropane is one of the Substances from the Carcinogenic Potency Database which are of particular concern even if ingested at doses at or below 0.0025 μg/kg body weight per day (EFSA 2016).

### Derivation of Maximum Acceptable Value

No MAV.

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# Triclosan

CAS No. 3380-34-5. Also called 5-chloro-2-(2,4-dichlorophenoxy)-phenol, 2,4,4’‑trichloro-2’-hydroxydiphenyl ether, TCS and various trade names.

### Maximum Acceptable Value

Triclosan does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

### Sources to drinking-water

Triclosan, a [polychlorophenoxy phenol](https://en.wikipedia.org/wiki/Polychloro_phenoxy_phenol), is an antibacterial active ingredient used in liquid soap formulations for hand disinfection, typically at 0.7 percent. Triclosan may also have virucidal and fungicidal activity. The triclosan molecule kills the bacterial cell by disturbing the function of the cell membrane.

About 90 percent of the triclosan used in soaps is washed off the body and enters the sewerage system. It is not fully removed in wastewater treatment plants and can be released into the environment.

Triclosan can also be found in products such as clothing, kitchenware, furniture, and toys where it is used as a preservative. It also may be added to body washes, toothpastes, mouthwash and some cosmetics (US FDA). In 2016 the US FDA banned its use in over-the-counter products, claiming it was likely to be doing more harm than good.

### Form and fate in the environment

If released to soil, triclosan is expected to have low to no mobility based upon Koc values of 2,400 to 15,892. The pKa of triclosan is 7.9, indicating that this compound will exist partially in anion form in the environment and anions generally do not adsorb more strongly to soils containing organic [carbon](https://pubchem.ncbi.nlm.nih.gov/compound/carbon) and clay than their neutral counterparts. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry’s Law constant of 2.1 x 10-8 atm‑cu m/mole and its pKa. Using the Japanese MITI test, 0 percent of the theoretical BOD was reached in four weeks indicating that biodegradation is not an important environmental fate process in soil. Triclosan had a photodegradation half-life of 17 days on dry loamy soil. If released into [water](https://pubchem.ncbi.nlm.nih.gov/compound/water), triclosan is expected to adsorb to suspended solids and sediment based on its Koc values. Triclosan was shown to biodegrade in grey [water](https://pubchem.ncbi.nlm.nih.gov/compound/water) under aerobic, anaerobic and anaerobic/aerobic conditions. Volatilisation from [water](https://pubchem.ncbi.nlm.nih.gov/compound/water) surfaces is not expected to be an important fate process based on its estimated Henry’s Law constant and pKa. Measured BCFs in orange-red killifish of 2.7 to 90 suggest bioconcentration in aquatic organisms is low to moderate. Hydrolysis is not expected to be an important environmental fate process since triclosan lacks functional groups that hydrolyse under environmental conditions. Photolytic degradation of triclosan was measured in fresh and sea [water](https://pubchem.ncbi.nlm.nih.gov/compound/water) with half-lifes of eight and four days, respectively, with a degradation product of [2,8-dichlorodibenzo-p-dioxin](https://pubchem.ncbi.nlm.nih.gov/compound/2%2C8-dichlorodibenzo-p-dioxin). NIH.

Due to the use pattern of triclosan, the main route to the aquatic environment will be via sewage effluents. Sewage treatment can remove >90 percent of the triclosan. For surface water, a realistic worst-case assumption (predicted no effect concentration or PNEC) for triclosan is 0.05 μg./L. Triclosan is not persistent; the metabolite methyl triclosan may be persistent or even very persistent (ECHA 2015).

Water solubility – 10 mg/L at 20°C; readily soluble in alkaline solutions.

### Typical concentrations in drinking-water

Triclosan was detected in finished [water](https://pubchem.ncbi.nlm.nih.gov/compound/water) at one of 18 drinking [water](https://pubchem.ncbi.nlm.nih.gov/compound/water) treatment plants across the US in samples taken in 2006 and 2007, at a concentration of 1.2 ng/L. Triclosan was detected in one of 15 finished drinking [water](https://pubchem.ncbi.nlm.nih.gov/compound/water) samples from four [water](https://pubchem.ncbi.nlm.nih.gov/compound/water) filtration plants in San Diego County, CA at a concentration of 0.73 ug/L (730 ng/L). NIH.

### Removal methods

Ozone has been reported to break down the triclosan molecule. Its pKa suggests that activated carbon will not be very effective.

### Health considerations

In humans, triclosan is rapidly and completely absorbed from the gastrointestinal tract; excretion is relatively rapid with the major route of excretion being the urine, while the faeces is of secondary importance. A number of *in vitro* and *in vivo* genotoxicity studies are available, and, although some positive results were obtained, overall, there is no evidence of an *in vivo* genotoxic potential. Oral carcinogenicity studies in the rat and hamster provide no evidence of a carcinogenic potential. No effects on fertility were seen in a two-generation study in the rat, and there was no evidence of teratogenicity in developmental toxicity studies conducted in rats and rabbits. The US Pharmacopoeia have set limits for dioxins, dibenzofurans and some chlorinated phenols as impurities in triclosan used in therapeutics. See NICNAS (2009).

Triclosan was detected in breast milk samples from Australian mothers at extremely low levels (less than one part per billion in most samples). There is no evidence of any potential harm to breastfed babies from the amount of triclosan detected in breast milk, and breastfeeding is recommended in accordance with Australian Dietary Guidelines (NICNAS 2013).

The USEPA maintains a table of Human Health Benchmarks for Pesticides that includes RfDs and ARfDs for (currently) 363 pesticides. These were originally developed in 2012. The table includes a column for “acute or one-day HHBPs”, another for “chronic or lifetime (non-cancer) HHBPs”, and one for “carcinogenic HHBPs”. Details can be accessed at <http://water.epa.gov/drink/standards/hascience.cfm> or <http://iaspub.epa.gov/apex/pesticides/f?p=HHBP:home:10911636297819:::::>. The USEPA acute one day HHBP (Human Health Benchmark for Pesticides) in drinking water for triclosan is 3.0 mg/L. This was based on the ARfD of 0.30 mg/kg/d. The chronic RfD is also 0.30 mg/kg/d.

The critical effects of triclosan in rats were determined in a two-year carcinogenicity study. The NOAEL was determined to be 40 mg/kg bw/day based on reduced white blood cell counts in female rats and increased clotting time/decreased monocyte count in male rats. Tumour induction was reported in the mouse, but no effects of these types were seen in rats and hamsters, and it was concluded that the mouse is uniquely sensitive to triclosan in the liver due to peroxisome proliferation as inducer of liver tumours in mice. No genotoxic or foetal effects were observed. There is a growing number of studies from the open literature showing potential problems with triclosan concerning endocrine disruption (ECHA 2015).

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Trihalomethanes

Trihalomethanes (THMs) are halogen-substituted single-carbon compounds with the general formula CHX3, where X represents a halogen, which may be fluorine, chlorine, bromine, or iodine, or combinations thereof. The four THMs most commonly present (and regulated) in drinking-water are:

* chloroform (CHCl3)
* bromodichloromethane or dichlorobromomethane or BDCM (CHBrCl2)
* dibromochloromethane or chloro-dibromomethane or DBCM (CHClBr2)
* bromoform (CHBr3).

Sometimes total trihalomethanes are referred to as TTHM. Refer to the individual datasheets for specific details of these determinands. This datasheet discusses these determinands as a group.

The six iodo-THMs have been implicated as DBPs (unregulated) in chlorinated and chloraminated waters, and are:

* bromochloroiodomethane
* chlorodiiodomethane
* dichloroiodomethane
* bromodiiodomethane
* dibromoiodomethane
* iodoform.

The iodo-THMs are not included in the MAV. Refer to the datasheet for iodinated DBPs for more information.

### Maximum Acceptable Value

To account for additive toxicity, the sum of the ratio of the concentration of each trihalomethane to its respective MAV should not exceed 1. When a determinand is reported to be less than its limit of detection (LoD), a value equal to half the LoD should be used in the calculation (see Guidelines, section 10.2.5.3).

Action to reduce THMs is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than THMs.

The maximum acceptable concentration for total THMs in Canada is 0.10 mg/L (considered as a locational running annual average of quarterly samples taken at the point in the distribution system with the highest potential THM levels). This maximum acceptable concentration for THMs is protective of the health effects of all THMs. Since chloroform is the trihalomethane most often found in drinking water, and generally at the highest concentrations, the trihalomethane guideline is based on health risks linked to chloroform.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of trihalomethanes, either individually or in total, in drinking water should not exceed 0.25 mg/L. Trihalomethane concentrations fluctuating occasionally (for a day or two annually) up to 1 mg/L are unlikely to pose a significant health risk.

The Prescribed Concentration or Value (PCV) for total trihalomethanes in England and Wales is 0.1 mg/L. See Notes.

### Sources to drinking-water

THMs are formed in drinking-water primarily as a result of chlorination of organic matter present in raw water supplies. The rate and degree of THM formation increase as a function of the chlorine and humic acid concentrations, temperature, pH, and bromide ion concentration. As well as being the most common THM, chloroform is also the principal disinfection by-product in chlorinated drinking-water. In the presence of bromides, brominated THMs are formed preferentially and chloroform concentrations decrease proportionally. Being disinfection by-products, the USEPA (2007) regulates trihalomethanes.

Also refer to the individual datasheets.

### Form and fate in the environment

Refer to individual datasheets.

### Typical concentrations in drinking-water

The Priority 2 Identification Programme found 2 distribution zones supplying drinking-water to a total of 4900 people with trihalomethanes where the sum of the ratios exceeded 1, and 34 distribution zones supplied 76,123 people where the sum of the ratios exceeded 0.5 (ESR 2001).

THMs are rarely found in raw water but are often present in finished water, more commonly when sourced from surface water than groundwater; concentrations are generally below 0.10 mg/L. In most circumstances, chloroform is the dominant compound. THMs tend to be higher in the summer, maybe double the winter concentrations. Concentrations tend to increase as the water passes through the distribution system.

Based on data received from eight Canadian provinces, the mean THM level was about 0.066 mg/L in drinking-water samples from all systems. Some systems had average values in the 0.40 mg/L range, and some systems had maximum or peak values in the 0.80 mg/L range; 3.4 percent of the sampled population served reported having mean THM levels greater than 0.10 mg/L (WHO 2005).

In major Australian reticulated supplies concentrations of total trihalomethanes range up to 0.6 mg/L.

The concentration of each of the THMs has been reported to reduce to a third or quarter by boiling (*IARC Monograph* 52).

25,794 water utilities in the US reported detecting total trihalomethanes (TTHMs) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.24 mg/L.

Also refer to the individual datasheets.

### Removal methods

Refer to the individual datasheets.

### Analytical methods

Refer to the individual datasheets.

### Health considerations

Refer to the individual datasheets.

### Derivation of Maximum Acceptable Value

The sum of the ratio of the concentration of each trihalomethane to its respective guideline value should not exceed 1.

The MAVs for the four main trihalomethanes are:

* bromoform 0.1 mg/L
* dibromochloromethane 0.15 mg/L (DBCM)
* bromodichloromethane 0.06 mg/L (BDCM)
* chloroform 0.4 mg/L

The sum of the individual MAVs does not take account of the additive toxicity of trihalomethanes. The following fractionation approach can be taken to determine the total trihalomethane “concentration”:

Concentration (bromoform) + Concentration (DBCM) + Concentration (BDCM) + Concentration (chloroform)

MAV (bromoform) MAV (DBMC) MAV (BDCM) MAV (chloroform) < 1

For example, say a water sample contained:

* 0.032 mg/L bromoform
* 0.015 mg/L dibromochloromethane
* 0.018 mg/L bromodichloromethane
* 0.14 mg/L chloroform

then the calculation becomes:

0.032 + 0.015 + 0.018 + 0.14

0.1 0.15 0.06 0.4 = 0.320 + 0.100 + 0.300 + 0.350 = 1.07

ie, the sum of the ratios of the THMs in this sample are than greater than 1.0, even though none of the individual THMs exceeded its MAV.

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# Trimethylbenzenes

There are three isomers, as follows. The CAS No. for the mixture is 25551-13-7. They are sometimes referred to as dimethyltoluenes or TMBs.

* 1,2,3-Trimethylbenzene (CAS No. 526-73-8) is sometimes called hemellitol, hemimellitene or 1,2,3-trimethylbenzol.
* 1,2,4-Trimethylbenzene (CAS No. 95-63-6) is sometimes called pseudocumene, pseudocumol, or asymmetrical trimethylbenzene.
* 1,3,5-Trimethylbenzene (CAS No. 108-67-8) is sometimes known as mesitylene or symmetrical trimethylbenzene.

A structurally similar chemical is n-propylbenzene (CAS No. 103-65-1) and is also called propylbenzene, isocumene, or 1-phenylpropane – see cumene datasheet.

Another structurally similar chemical is isopropylbenzene, (1-methylethyl)-benzene, 2‑phenylpropane, or cumene (qv, CAS No. 98-82-8).

The trimethylbenzenes comprise a benzene ring with a methyl group attached in three places. The propylbenzenes are made up of a benzene ring with a propyl group attached. The xylenes and trimethylbenzenes are part of a bigger family of related chemicals called the alkyl substituted benzene derivatives.

### Maximum Acceptable Value

The trimethylbenzenes do not have MAVs in the DWSNZ, and are not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1. To source waters

Production of 1,2,4-trimethylbenzene occurs during petroleum refining as a major component of the C9 aromatic hydrocarbon fraction (or simply the C9 fraction). It typically constitutes around forty percent of the C9 fraction, with the other trimethylbenzenes and ethyltoluenes making up the remainder of this fraction.

1,2,3-Trimethylbenzene is the least abundant of the three isomers. Trimethylbenzenes occur in vehicle exhaust.

Trimethylbenzenes sometimes appear in cleaners, dyes, inks, paints and pesticides. They have been used as UV stabilisers in plastics.

Trimethylbenzenes can enter groundwater from leaking underground storage tanks.

#### 2. From treatment process

No known sources.

### Forms and fate in the environment

Volatilisation is the major route of removal of 1,2,4-trimethylbenzene from soils; although, biodegradation may also occur (USEPA 1994).

When xylenes and trimethylbenzenes are released to surface water, they volatilise to air very rapidly. The volatilisation half-life of 1,2,4-trimethylbenzene from a model river is calculated to be 3.4 hours , however moderate adsorption to soils and sediments may occur (USEPA 1994).

Water solubility of 1,2,4-trimethylbenzene is about 57 mg/L. Water solubility of butylbenzene is about 12 mg/L.

### Typical concentrations in drinking-water

138 water utilities in the US reported detecting 1,2,4-trimethylbenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.065 mg/L.

Fifty-nine water utilities in the US reported detecting 1,3,5-trimethylbenzene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.12 mg/L.

### Removal methods

Xylenes can be removed from water by adsorption on to granular activated carbon or by air stripping; presumably trimethylbenzenes would behave in a similar manner.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Painters that worked for several years with a solvent containing 50 percent 1,2,4- and 30 percent 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; hematological effects may have been due to trace amounts of benzene.

USEPA (2011) quotes a health advisory of 10 mg/L for 1,3,5-trimethylbenzene for a one-day intake for a 10 kg child.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit for 1,2,4-trimethylbenzene and 1,3,5‑trimethylbenzene is 0.1 mg/L (each).

In 2001, the California Department of Health Services established a drinking water notification level for 1,2,4- and 1,3,5-trimethylbenzene of 0.33 mg/L.

USEPA (2016) set an RfD of 1 × 10−2 mg/kg-day based on liver effects, and a subchronic RfD of 4 × 10−2 mg/kg-day based on neurological effects following exposure to 1,2,4-TMB.

### Derivation of Maximum Acceptable Value

No MAV.

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# Urethane

CAS No. 51-79-6. The IUPAC name is ethyl carbamate. Also called carbamic acid ethyl ester.

### Maximum Acceptable Value

Urethane does not have a MAV in the DWSNZ, and is not mentioned in the WHO Guidelines.

Urethane is one of the fourteen VOCs on the USEPA’s 3rd Chemical Contamination List (CCL3).

### Sources to drinking-water

#### 1. To source waters

Despite its common name, urethane is not a component of [polyurethanes](https://en.wikipedia.org/wiki/Polyurethane).

Urethane is a naturally occurring compound that is formed during the fermentation of foods such as fruit, beer, wine, yoghurt, bread and soy sauce. The UK Food Standards Agency’s Food Advisory Committee discussed the presence of naturally occurring urethane and stated that levels of urethane in these foods should be reduced to the lowest technologically achievable concentrations. Historically industrial, medical and veterinary uses of urethane have been reported but there is little data on the uses of urethane at present. From DWI (2014).

In the US it is reported that urethane is widely used as an ingredient in paint. It is also used as a solvent for organic materials, an intermediate in organic synthesis, in the preparation and modification of amino resins, and as a solubiliser and co-solvent for pesticides, fumigants and cosmetics. From DWI (2014).

Concentrations of urethane in drinking water and groundwater are detected but not quantifiable and in effluents range from 18 to 37 μg/l. No data are available for fresh and marine surface water.

#### 2. From treatment process

No known sources.

### Forms and fate in the environment

DWI (2014) states:

* Log Kow = -0.15
* Koc = 20
* Henry’s Law constant = 6.43 x10-8 atm.m3/mole at 25°C
* Water solubility is about 48 percent.

### Removal methods

No data were located on the removal of urethane during drinking water treatment, however, some predictions on its fate during treatment can be made based on its physico-chemical properties.

A Koc of 20 has been reported for urethane, which would suggest that it has high mobility in the water column and therefore is unlikely to be amenable to removal by GAC.

Urethane has a vapour pressure of 0.262 mm Hg at 25°C and a Henry’s Law constant of 6.43 x 10-8 atm.m3/mole at 25°C. Therefore, it is unlikely to undergo significant removal by air stripping.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Ex DWI (2014):

B6C3F1 mice were administered urethane in drinking water at doses of approximately 0, 0.07, 0.36, 0.72, 7.2 and 72 mg/kg bw/day, respectively. A NOAEL approximately 0.07 mg/kg bw/day was identified based on dose-related increases in liver angiosarcomas. Using this NOAEL an oral Tolerable Daily Intake (TDI) of 0.00007 mg/kg bw/day (0.07 μg/kg bw/day) is derived.

Mixed results were observed in *in vitro* genotoxicity tests, indicating that urethane is not genotoxic in bacteria or yeast, however evidence of genotoxicity has been observed in mammalian assays. Clear evidence of genotoxicity can be observed in *in vivo* tests.

In 2010, the International Agency for Research on Cancer (IARC) evaluated the available data and classified urethane in Group 2A, (ie, probably carcinogenic to humans) based on “sufficient” and “inadequate” evidence for carcinogenicity in experimental animals and humans, respectively (IARC 2010). Therefore, although a TDI has been proposed, it may be appropriate to ensure the concentration of urethane in water is as low as reasonably practicable.

### Derivation of Maximum Acceptable Value

No MAV.

### References

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# Vinyl chloride

CAS No. 75-01-4. Also called vinyl chloride monomer, chloroethene, monochloroethene, chloroethylene, ethylene monochloride or VC or VCM.

Vinyl acetate (CAS No. 108-05-4) and vinyl bromide (CAS No. 593-60-2) are also encountered. This datasheet discusses vinyl chloride unless otherwise specified. Vinyl acetate is also called acetic acid, ethenyl ester; acetoxyethene and ethenyl ethanoate. Sometimes referred to as VAM: vinyl acetate monomer.

### Maximum Acceptable Value

Based on health considerations, the concentration of vinyl chloride in drinking-water should not exceed 0.0003 mg/L (0.3 g/L).

The maximum contaminant level or MCL in the US (USEPA 2006/2009/2011) is 0.002 mg/L. The maximum acceptable concentration in Canada is 0.002 mg/L, based on treatment and analytical achievability.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that no safe concentration for vinyl chloride in drinking water can be confidently set. However, for practical purposes, the concentration should be less than 0.0003 mg/L, which is the limit of determination.

The Prescribed Concentration or Value (PCV) for vinyl chloride in England and Wales is 0.0005 mg/L. See Notes.

Vinyl chloride is one of the “priority pollutants” under the US Clean Water Act.

### Sources to drinking-water

#### 1. To source waters

Vinyl chloride can be released to the aquatic environment as an industrial contaminant. It is used primarily (about 98 percent) for the production of polyvinyl chloride (PVC) where n = 700 to 1500, which is used extensively in the plastics, rubber, paper and glass industries. Since PVC is imported and not polymerised in New Zealand, the occurrence of vinyl chloride in the New Zealand environment is not expected to be extensive. Vinyl chloride may also be used as a co-monomer with vinyl acetate or vinylidene chloride to produce copolymers, and as a raw material in the manufacture of 1,1,1-trichloroethane and monochloracetaldehye.

Vinyl acetate, a similar chemical, has many uses too, eg, in the production of PVA glues, resins and polymers. The US Food and Drug Administration (FDA) has determined that vinyl acetate may be safely used as a coating or a part of a coating that is used in plastic films for food packaging, and as a modifier of food starch. Commercial vinyl acetate may contain up to 30 mg/L hydroquinone as an inhibitor, or 0.00015–0.002 percent w/w hydrochinone mono methyl ether as a stabilisator.

#### 2. From treatment processes

Chlorinated ethylenes such as vinyl chloride may be formed during the chlorination of water.

#### 3. From the distribution system

Vinyl chloride may be found in drinking-water from distribution systems constructed with some grades of PVC pipe or in water stored for long periods in PVC containers; see Chapter 16, section 16.2.6: Permeation and leaching. Vinyl chloride concentrations are best controlled by specification of material quality.

### Forms and fate in the environment

Volatilisation, followed by atmospheric oxidation, is considered to be the primary removal mechanism for vinyl chloride from the aquatic environment. Its water solubility has been reported to range from about 1,100–2,800 mg/L. When released to the ground, it does not absorb on to soil but migrates readily to groundwater, where it may be degraded to carbon dioxide and chloride ion, or it may be persistent for several years. Vinyl chloride has been reported to be a degradation product of trichloroethylene and tetrachloroethylene in groundwater.

If released to soil, vinyl chloride is expected to have high mobility based upon a Koc of 57. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 2.78 x 10-2 atm‑cu m/mole. The volatilisation half-life of vinyl chloride was estimated as 0.2 days when incorporated in a soil at a depth of 1 cm and 0.5 days at a depth of 10 cm. Vinyl chloride may volatilise from dry soil surfaces based upon its vapour pressure. Utilising the Japanese MITI test, 16 and 3 percent of the theoretical BOD was reached in 28 days indicating that biodegradation may be an important environmental fate process. If released into water, vinyl chloride is not expected to adsorb to suspended solids and sediment based upon the Koc. The biodegradation half-life of vinyl chloride in aerobic and anaerobic waters was reported as 28 and 110 days, respectively. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are two hours and three days, respectively. A BCF of <10 in golden ide fish suggests bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process based on hydrolysis half-lifes of 9.91 years (pH = 7, 25°C) and 10.7 years (pH = 7, 10°C) (EAWAG accessed February 2015).

Vinyl acetate (water solubility about 2 percent) undergoes hydrolysis in surface water and groundwater, producing acetic acid and acetic aldehyde. The hydrolytic half-life of the compound at 25°C and pH 7.0 has been estimated to be 7.3 days. Decreasing pH decreases the hydrolysis rate; for example, the rate is minimal at pH 4.4. Acetic acid and acetaldehyde are the main products of vinyl acetate hydrolysis (ATSDR 1992; IARC 1995). EU (2008) quotes: vapour pressure = 120 hPa at 20°C; partition coefficient = logPow = 0.7; estimated hydrolysis half-life = 17 days at standard conditions; vinyl acetate is classified as readily biodegradable.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 332 zones, did not find any vinyl chloride at detectable concentrations (limit of detection = 0.001 mg/L) (ESR 2001).

Rarely detected in surface waters, the concentrations measured generally not exceeding 0.010 mg/L; much higher concentrations found in groundwater and well water in contaminated areas; concentrations up to 0.010 mg/L detected in drinking-water (WHO 2004).

In a 1982 survey, vinyl chloride was found in fewer than 1 percent of the 945 groundwater supplies tested in the United States; the concentrations in groundwater were up to 0.008 mg/L. 121 water utilities in the US reported detecting vinyl chloride in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.006 mg/L.

It has occasionally been detected in drinking water supplies that use PVC pipes in the United States and Germany, with a maximum reported concentration of 0.01 mg/L.

### Removal methods

WRF (2014) reports that vinyl chloride is characterised with high Henry’s law constant (0.869 dimensionless air/water at 20°C). Low profile air stripping is very effective for vinyl chloride removal even at low temperatures and low air to water ratios (below 100). Vinyl chloride was completely removed in most of the tested scenarios, and showed a very high removal efficiency (99.6 percent) at the lowest temperature (4°C) and lowest air to water ratio (53) which is the worst case examined.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

Vinyl chloride is absorbed readily following ingestion or inhalation, is rapidly converted to water-soluble metabolites, bioactivated by the liver, and is rapidly excreted. The highest concentrations of metabolites are found in the liver, kidneys and spleen. Vinyl chloride is metabolised to chloroethylene oxide and can rearrange spontaneously to chloroacetaldehyde; both substances are highly reactive and mutagenic. Low doses administered by gavage are metabolised and eliminated primarily in the urine, whereas a substantial proportion of higher doses are excreted unchanged via the lung. No significant accumulation of vinyl chloride occurs in the body. In rats it is estimated to have a biological half life of 20 minutes.

Vinyl chloride has exhibited mutagenic activity in a variety of tests on bacteria and mammalian cells.

Vinyl chloride is a narcotic agent, and loss of consciousness can occur at 25,000 mg/m3 in air. Effects of chronic inhalation exposure include Raynaud’s phenomenon, a painful disorder of the hands. No data are available on oral exposure in humans.

There is sufficient evidence of carcinogenicity of vinyl chloride in humans from industrial populations exposed to high concentrations, and the International Agency for Research on Cancer has classified vinyl chloride in Group 1 (carcinogenic to humans).

Vinyl fluoride (CAS No. 75-02-5) and vinyl bromide (CAS No. 593-60-2) are gases used predominantly for the manufacture of their respective polymers. The IARC Working Group took into consideration that all available studies showed a consistently parallel response between these chemicals and vinyl chloride, and classified vinyl fluoride and vinyl bromide separately as “probably carcinogenic to humans” (Group 2A). They stressed that for practical purposes, these chemicals should be considered to act similarly to the human carcinogen, vinyl chloride. The International Agency for Research on Cancer (IARC) has determined that vinyl acetate is not classifiable as to its ability to cause cancer in humans (ATSDR 1992).

Causal association between vinyl chloride exposure and angiosarcoma is sufficiently proved, and some studies suggest that vinyl chloride can also be associated with heptacellular carcinoma, brain tumours, lung tumours and malignancies of the lymphatic and haemotopoietic tissues. This chemical appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. USEPA (2000) states that vinyl chloride is a *known human carcinogen* by the inhalation and oral routes of exposure and highly likely to be carcinogenic by the dermal route of exposure. The USEPA (2009/2011) quotes a health advisory of 0.002 mg/L for vinyl chloride, representing a 10-4 cancer risk.

For oral repeated dose, the critical target organ is the liver (liver cell polymorphism) with a lifetime NOAEL in the rat of 0.13 mg/kg/day. Vinyl chloride (and/or its metabolites) produces DNA adducts and has been positive in gene mutation and chromosomal aberration assays (OECD 2004).

Evidence indicates that vinyl chloride metabolites are genotoxic, interacting directly with DNA. DNA adducts formed by the reaction of DNA with a vinyl chloride metabolite have also been identified. Occupational exposure has resulted in chromosomal aberrations, micronuclei and sister chromatid exchanges; response levels were correlated with exposure levels.

Animal studies show vinyl chloride to be a multi-site carcinogen. Vinyl chloride administered orally or by inhalation to mice, rats and hamsters produced tumours in the mammary glands, lungs, Zymbal gland, and skin, as well as angiosarcomas of the liver and other sites.

Vinyl chloride has been detected in tobacco smoke.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.003 mg/kg/day for chronic-duration oral exposure (>364 days) for vinyl chloride. There are no oral MRLs for vinyl acetate.

The reference dose or RfD (USEPA 2000 and 2000/2006/2009/2011) is 0.003 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) is 0.1 mg/L.

Vinyl acetate was not lethal to rats or mice administered drinking-water that contained up to 5,000 ppm (equivalent to 684 to 950 mg/kg/day) for up to three months (ATSDR 1992). IARC (1995) states that the available data were too limited to form the basis for an evaluation of the carcinogenicity of vinyl acetate to humans.

### Derivation of Maximum Acceptable Value

The MAV of 0.0003 mg/L was based on an application of a linear extrapolation by drawing a straight line derivation between the dose, determined using a pharmocokinetic model, resulting in tumours in 10 percent of animals in rat bioassays involving oral exposure and the origin (zero dose), determining the value associated with the upper-bound risk of 10-5 and assuming a doubling of the risk for exposure from birth (WHO 2017).

The results of the linear extrapolation are nearly identical to those derived using the linearised multistage model. As vinyl chloride is a known human carcinogen, exposure to this compound should be avoided as far as practicable, and levels should be kept as low as technically feasible.

Vinyl chloride is primarily of concern as a potential contaminant from some grades of PVC pipe and is best controlled by specification of material quality.

The 0.005 mg/L MAV in the 1995 and 2000 DWSNZ had been derived as follows:

Because human data on carcinogenic risk following oral exposure to vinyl chloride are not available, estimation of risk of cancer in humans has been based on animal carcinogenicity bioassays involving oral exposure. Using results from a rat bioassay that yields the most protective value and applying the linear multi-stage model, the human lifetime exposure for a risk of one per hundred thousand of hepatic angiosarcoma was calculated to be 0.02 mg/person/day. It was assumed that, in humans, the numbers of cancers at other sites may equal that of angiosarcoma of the liver, and a correction factor of 2 for cancers other than angiosarcomas is justified. Thus using the lifetime exposure of 0.02 mg/person/day for an excess lifetime risk of hepatic angiosarcoma of one person per 100,000, the concentration of vinyl chloride in drinking-water should not exceed 0.005 mg/L.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for vinyl chloride is 0.01 mg/L. Their cancer health limit is 0.0002 mg/L.

Most people begin to taste vinyl chloride in water at 3.4 mg/L.

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# Xylenes

Collectively, the CAS No. is 1330-20-7 which usually means commercial xylene which often contains ethylbenzene as well. The IUPAC name for xylene is dimethylbenzene. The xylenes are for the most part manufactured and marketed as a mixture of the isomers, which will here be called xylene. Sometimes called xylol.

There are three possible xylene isomers: 1,2-, 1,3-, and 1,4-dimethylbenzene; these can also be referred to as o- (ortho), m- (meta), and p-(para) xylene, (or xylol or methyltoluene) respectively. Also 2-, 3- and 4-xylene (or xylol or methyltoluene).

* CAS No. o-xylene or 1,2-dimethylbenzene: 95-47-6.
* CAS No. m-xylene or 1,3-dimethylbenzene: 108-38-3.
* CAS No. p-xylene or 1,4-dimethylbenzene: 106-42-3.

### Maximum Acceptable Value

Based on health considerations, the concentration of xylenes (total) in drinking-water should not exceed 0.6 mg/L.

The Environmental Protection Authority of New Zealand ([www.epa.govt.nz](file:///C:\Users\sgilbert\AppData\Local\Microsoft\Windows\AppData\Local\Microsoft\Windows\Temporary%20Internet%20Files\Content.Word\www.epa.govt.nz)) has set (by an approval under Part 5 of the HSNO Act) a tolerable exposure limit (TEL) of 0.6 mg/L in drinking water.

The maximum contaminant level or MCL for total xylenes (USEPA 2006/2009/2011) is 10 mg/L.

The *Australian Drinking Water Guidelines* (NHMRC, NRMMC 2011) state that based on aesthetic considerations (taste and odour), the concentration of xylenes in drinking water should not exceed 0.02 mg/L. Xylenes would not be a health concern unless the concentration exceeded 0.6 mg/L.

EPA established an environmental exposure limit of 0.64 mg/L (640 µg/L) for o-xylene in fresh water (and <http://www.epa.govt.nz/search-databases/Pages/substance-exposure-limit-register.aspx)>, and 0.34 mg/l (340 µg/L for m/p-xylene.

### Sources to drinking-water

#### 1. To source waters

Xylenes may occur in raw water as contaminants from industrial activity. Xylenes occur naturally as a component of crude oil, and are present in petrol. Trucks burning diesel have been reported to produce 0.5 to 30 mg of xylenes per km (Environment Australia 2003). They are used in the manufacture of insecticides, pharmaceuticals, phthalates, detergents, paints, inks and adhesives, and involved in the rubber and leather industries. It is also used as a solvent, particularly for pesticides. Commercial (mixed) xylene usually contains ethylbenzene as well. Currently, nearly all mixed xylene is produced as a catalytic reformate of petroleum and consists of approximately 44 percent m-xylene, 20 percent o-xylene, 20 percent p-xylene, and 6 to 15 percent ethylbenzene, although these can vary with different manufacturers. Other minor contaminants of xylenes include toluene, trimethyl benzene and C9 aromatic fractions. p-Xylene has by far the largest market, with the most significant use of this isomer involving its oxidation to produce purified terephthalic acid (PTA). PTA is used in turn to make polymers such as polyethylene terephthalate (PET) and polybutylene terephthalate.

#### 2. From treatment process

No known sources.

#### 3. From the distribution system

Xylene can enter water from solvents used in adhesives for bonding plastic drinking-water fittings. They may also leach from some compounds used to seal water reservoirs. Xylenes can also penetrate plastic pipe from contaminated soil.

### Forms and fate in the environment

Releases of xylenes to the environment are largely to air because of their volatile nature. The distribution to water has been calculated to be less than 1 percent. Their solubility in water varies from about 130–210 mg/L. When xylenes are released to surface water, they volatilise to air very rapidly. Xylenes degrade in air and are also biodegraded readily in soils and surface waters. Xylene below the soil surface may travel down through the soil and enter groundwater. Xylene may remain in groundwater for several months. They can be degraded in aerobic groundwater, but no biotransformation is expected under anaerobic conditions. Their log octanol-water partition coefficients (Kow) are 2.77, 3.20 and 3.15 for the o-, m- and p-isomers, respectively.

If released to soil, 2-xylene is expected to have very high to moderate mobility in soil based upon Koc values ranging from 24–251. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 5.18 x 10-3 atm‑cu m/mole. 2-Xylene may volatilise from dry soil surfaces based upon its vapour pressure. 2-Xylene is expected to biodegrade in soil under both aerobic and anaerobic conditions. Under aerobic conditions, 70 percent degradation has occurred after 10 days. Under anaerobic conditions, a long lag period may be required before degradation commences. If released into water, 2-xylene is not expected to adsorb to suspended solids and sediment based upon the range of Koc values. 2-Xylene is expected to biodegrade in water under aerobic conditions based on reported half-lives ranging from days to weeks. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 3.2 hours and 4.1 days, respectively. BCF values ranging from 6.2–21 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

If released to soil, 3-xylene is expected to have moderate mobility based upon Koc values ranging from 166–182. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 7.18 x 10-3 atm‑cu m/mole. 3-Xylene may volatilise from dry soil surfaces based upon its vapour pressure. Biodegradation of 3-xylene in both soil and water is expected to be a major fate process. Under aerobic conditions, 3-xylene was biodegraded within several weeks; this compound was anaerobically biodegraded in aquifer studies within weeks to months. Metabolites include 3-methylbenzyl fumaric acid, 3-methylbenzyl succinic acid, 3-methylsalicylate, 3-methylbenzoate and 3-methylbenzaldehyde. If released into water, 3-xylene is not expected to adsorb to suspended solids and sediment based upon the range of Koc values. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 3.1 hours and 4.1 days, respectively. BCF values ranging from 6 to 23.4 suggest the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

If released to soil, 4-xylene is expected to have moderate mobility based upon Koc values of 246 and 540. Volatilisation from moist soil surfaces is expected to be an important fate process based upon a Henry’s Law constant of 6.90 x 10-3 atm‑cu m/mole. 4-Xylene may volatilise from dry soil surfaces based upon its vapour pressure. Biodegradation is an important environmental fate process for 4-xylene. In general, it has been found that 4-xylene is biodegraded in soil and groundwater samples under aerobic conditions and may be degraded under anaerobic denitrifying conditions. If released into water, 4-xylene is not expected to adsorb to suspended solids and sediment based upon the Koc values. Volatilisation from water surfaces is expected to be an important fate process based on its Henry’s Law constant. Estimated volatilisation half-lifes for a model river and model lake are 3.1 hours and 4.1 days, respectively. A BCF value of 15 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions (EAWAG accessed February 2015).

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 301 zones, found meta- and para-xylene in four zones at concentrations ranging from 0.0012 to 0.022 mg/L, with the median concentration being “nd”, and ortho-xylene in three zones at concentrations ranging from 0.0006 to 0.01 mg/L, with the median concentration being “nd” (Limits of detection for all isomers = 0.0005mg/L) (ESR 2001).

Concentrations of up to 0.008 mg/L xylenes have been reported in surface water, groundwater and drinking-water; levels of a few milligrams per litre were found in groundwater polluted by point emissions (WHO 2004).

The mean concentrations of xylenes in three groundwater monitoring wells at a Shell Oil service station site in San Diego, California were 0.39, 1.23, and 19.35 mg/L.

712 water utilities in the US reported detecting o-xylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.025 mg/L.

91 water utilities in the US reported detecting m-xylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.024 mg/L.

140 water utilities in the US reported detecting p-xylene in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the highest concentration being 0.024 mg/L.

The maximum concentration of 1,2-xylene found in 8615 samples from 2,620 groundwaters in the UK was 0.00095 mg/L, mean 0.095 mg/L (DWI 2008).

The maximum concentration of 1,3-xylene or 1,4-xylene found in 8,672 samples from 2,621 groundwaters in the UK was 0.125 mg/L, mean 0.0015 mg/L (DWI 2008).

### Removal methods

Xylenes can be removed from water by adsorption on to granular activated carbon or more cost-effectively by air stripping.

IEH (2014) reports that the concentration of xylenes may be reduced by up to 50 percent by conventional water treatment and chlorination; PAC will enhance this.

### Analytical methods

#### Referee method

Purge and Trap Capillary Column Gas Chromatographic/Mass Spectrometric Method (APHA 6200B, EPA 524.2).

#### Some alternative methods

1. Purge and Trap Capillary-Column Gas Chromatographic Method (APHA 6200C, EPA 502.2).

### Health considerations

Exposure to xylenes is mainly from air, and exposure is increased by smoking.

Data on absorption of xylene after ingestion are not available. However, xylene isomers are readily absorbed after inhalation, with retention of 60–65 percent in humans. Isomers of xylene have similar toxicokinetic properties and elicit similar toxicological effects, with no single isomer consistently exhibiting the greatest potency. The few data available indicate rapid intake of the compound after intake and metabolism principally to methylbenzoic acid. Xylene can cross the placenta.

No data are available for the effects of ingestion of xylenes. In acute inhalation studies, irritation of eyes and throat were observed. After short-term exposure, abnormalities in psychometric functions were observed. Controlled studies of longer duration are lacking.

A two-year study using rats and mice reported decreased growth at high doses (500 mg/kg body weight per day) but no xylene-related lesions.

There was no evidence of carcinogenicity in oral and skin administration studies using rats and mice, and xylene was not mutagenic in tests using bacteria and mammalian cells.

The International Agency for Research on Cancer has concluded that xylene is not classifiable as to its carcinogenicity in humans (Group 3).

USEPA (2003) states that *data are inadequate for an assessment of the carcinogenic potential* of xylenes. Adequate human data on the carcinogenicity of xylenes are not available, and the available animal data are inconclusive as to the ability of xylenes to cause a carcinogenic response. Evaluations of the genotoxic effects of xylenes have consistently provided negative results.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) for mixed xylenes of:

* 1 mg/kg/day for acute-duration oral exposure (1–14 days)
* 0.4 mg/kg/day for intermediate-duration oral exposure (15–364 days)
* 0.2 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD for the sum of the three xylenes (USEPA 2003 and 2006/2009/2011) is 0.2 mg/kg/d, based on a NOAEL of 179 mg/kg/d and an uncertainty factor of 1000. The Drinking Water Equivalent Level or DWEL (USEPA 2006/2009/2011) for the three xylenes is 7 mg/L.

### Derivation of Maximum Acceptable Value

A tolerable daily intake approach has been used for the derivation of the MAV for xylene in drinking-water. The no-observable-adverse-effect level used in the derivation is based on decreased body weight in a 103-week gavage study in rats.

The MAV for xylenes in drinking-water was derived as follows:

250 x (5/7) mg/kg body weight per day x 70 kg x 0.1 = 0.625 mg/L (rounded to 0.6 mg/L)

2 L x 1000

where:

* no-observable-adverse-effect level = 250 mg/kg body weight per day based on decreased body weight in a 103-week gavage study in rats (normalised for five days/week dosing in the derivation)
* average weight of an adult = 70 kg
* proportion of tolerable daily intake allocated to drinking-water = 0.1
* average quantity of water consumed by an adult per day = 2 L
* uncertainty factor = 1,000 (100 for intra- and interspecies variation and 10 for limited toxicological end-point).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for xylenes is 10 mg/L.

The odour threshold for xylene isomers has been reported to range between 0.02 and 1.8 mg/L, and at 0.3 mg/L they produce a detectable taste. The aesthetic objective in Canada is not greater than 0.3 mg/L for total xylenes, for both taste and odour. IEH (2014) reports p-xylene having a taste and odour threshold of 0.53 to 1.0 mg/L. For o‑xylene and mixed xylene, the taste and odour thresholds are 0.45 to 1.8 mg/L and 0.20 to 1.8 mg/L, respectively.

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# Xylenols

Xylenol is an abbreviation and combination of ‘xylene’ and ‘phenol’, and covers the six isomers of dimethylphenol.

The CAS No. for the xylenols collectively is 1300-71-6.

* CAS No. for 2,3-dimethylphenol or 2,3-xylenol: 526-75-0.
* CAS No. for 2,4-dimethylphenol or 2,4-xylenol: 105-67-9.
* CAS No. for 2,5-dimethylphenol or 2,5-xylenol: 95-87-4.
* CAS No. for 2,6-dimethylphenol or 2,6-xylenol: 576-26-1.
* CAS No. for 3,4-dimethylphenol or 3,4-xylenol: 95-65-8.
* CAS No. for 3,5-dimethylphenol or 3,5-xylenol: 108-68-9.

2,4-Dimethylphenol is one of the “priority pollutants” under the US Clean Water Act.

Several chlorinated xylenols exist too, such as 4-chloro-3,5-xylenol or p-chloro-m-xylenol, CAS No. 88-04-0; sometimes loosely called chloroxylenol; this datasheet includes some information.

### Maximum Acceptable Value

There are no MAVs for the xylenols in the DWSNZ; the WHO Guidelines do not mention xylenols.

### Sources to drinking-water

#### 1. To source waters

Xylenols are an important class of phenolics with wide industrial usage. Xylenols are also used as [pesticides](http://en.wikipedia.org/wiki/Pesticide) and used in the manufacture of [antioxidants](http://en.wikipedia.org/wiki/Antioxidant). They are found in household cleansers, oven cleaners and disinfectants, and are a component of creosote. 2,6-Xylenol is the most important of the isomers.

M-cresol and 2,4 xylenol are formulated together as one product that has bacteriostatic activity against the causal agents for crown gall and olive knot and control of the genetic/physiological disorder, burr knot on fruit, ornamental and shade trees and ornamental woody shrubs and vines. The USEPA (1994) reviewed the available data for m-cresol and xylenol and has determined that the uses described and modified in the reregistration eligibility decision document will not cause unreasonable risk to humans or the environment and that these uses are eligible for re‑registration. This product does not seem to be registered for use in New Zealand.

4-Chloro-3,5-xylenol is used in hospitals/medical institutions as an antimicrobial and antifungal agent that operates by denaturing proteins and inactivating enzymes. It is a major component in products such as Dettol. It also has a range of industrial uses such as an antimicrobial in adhesives, emulsions, and paints. Chloroxylenol appears as an antimicrobial, ectoparasiticide and antifungal agent on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2009 (see [https://eatsafe.nzfsa.govt.nz/web/public/acvm-register and select entire register](http://www.nzfsa.govt.nz/acvm/registers-lists/acvm-register/index.htm)).

#### 2. From treatment process

No known sources. In theory, 4-chloro-3,5-xylenol could result from the chlorination of water containing 3,5-xylenol.

### Forms and fate in the environment

Xylenols have low vapour pressures (under 1 kPa) and high water solubilities (in the 3,450 mg/L to 7,870 mg/L range). Oxidation and microbial degradation are the main fates of phenols in the environment. Sorption is low as are log Kow values; generally under 2.0, reaching only 2.5 as a maximum. Adsorption of xylenols to sediment and particulate matter in the water column will only be moderate but they will be readily biodegradable; the half life should be less than several days in humic waters due to photo-oxidation by alkylperoxy radicals. If spilled on soil, 2,4-dimethylphenol will probably adsorb moderately to the soil and biodegrade in several days (Spectrum Fact Sheet).

The water solubility of 4-chloro-3,5-xylenol is about 300 mg/L.

### Removal methods

The concentration of xylenol is reduced by treatment with ozone, however, for effective removal, free radicals are required, by using ozone in conjunction with UV light or hydrogen peroxide (advanced oxidation process), with CO2 the main breakdown product.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Xylenols are suspected gastrointestinal or liver toxicants.

2,4-Dimethylphenol: male and female albino mice were dosed by gavage for 90 days with 5.0, 50.0, or 250 mg 2,4-dimethylphenol/kg/day. Toxicologically relevant clinical signs observed only after week 6 in the high-dose groups of both genders included: squinting, lethargy, prostration, and ataxia, with onset shortly after dosing. Statistically significant hematological changes (p<0.05) included lower mean corpuscular volume and mean corpuscular hemoglobin concentration in females at terminal, but not interim, sacrifice. Significant differences were not found in gross necropsy or histopathological evaluations, or in organ weights, except for an increase in adrenal weights of low-dose females. The LOAEL and NOAEL for this study were 250 and 50 mg/kg/day, respectively. An uncertainty factor of 3000 was established: 10 each for inter- and intraspecies variability and 30 for lack of chronic toxicity data, data in a second species and reproductive/developmental studies, giving an oral RfD of 0.02 mg/kg/day (USEPA 1990).

2,6-Dimethylphenol: rats were administered orally 0, 0.6 and 6.0 mg/kg/day 2,6‑dimethylphenol over an eight-month period. No effects were reported at 0.6 mg/kg/day. Animals receiving 6 mg/kg/day exhibited body weight changes, blood pressure changes, changes in protein sulfhydryl groups in blood serum and internal organs, and histopathological changes in liver, kidney and spleen. Dividing the NOEL of 0.6 mg/kg/day by an uncertainty factor of 1000 results in an oral RfD of 0.0006 mg/kg/day (USEPA 1988).

3,4-Dimethylphenol: was administered orally to white rats for an eight-month period. No toxic effects were seen at 1.4 mg/kg/day (NOEL) but a dose of 14 mg/kg/day resulted in body weight, blood pressure and histopathological changes in internal organs (LOAEL). Applying an uncertainty factor of 1000 to the NOEL of 1.4 mg/kg/day results in the RfD of 0.001 mg/kg/day (USEPA 1989).

4-Chloro-3,5-xylenol: a developmental toxicity study on rats with dose levels of 0, 100, 500, or 1000 mg/kg given by gavage on gestation days 6–15. The maternal NOEL was 100 mg/kg/day (USEPA 1994a).

### Derivation of Maximum Acceptable Value

No MAVs.

The North Carolina groundwater quality standard for the protection of the groundwaters for 2,4-dimethylphenol (m-xylenol) is 0.14 mg/L.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in drinking water. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for 2,4-dimethylphenol is 0.1 mg/L.

The USEPA established an organoleptic effect criterion of 0.4 mg/L for 2,4‑dimethylphenol. Source: [*Quality Criteria for Water*, 1986 (“Gold Book”)](http://nepis.epa.gov/Exe/ZyPDF.cgi?Dockey=00001MGA.txt), <http://www.epa.gov/wqc/national-recommended-water-quality-criteria-organoleptic-effects>.

The odour threshold for xylenol isomers has been reported to be about 0.4 mg/L.

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