Volume 3 Datasheets – Chemical and physical determinands

Part 2.1: Inorganic chemicals

2019

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Inorganic Determinands (including aesthetic determinands that have a MAV)

**Notes**

1. 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.

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.

2. The WHO states that their guideline value normally represents the concentration of a constituent that does not result in any significant risk to health over a lifetime of consumption.

3. Some datasheets include the USEPA’s MCL. Title 40, Protection of Environment, Chapter I: Environmental Protection Agency, Part 141, National Primary Drinking Water Regulations, § 141.1 40 CFR Ch. I (7–1–02 edition) defines MCL (maximum contaminant level) as the maximum permissible level of a contaminant in water which is delivered to any user of a public water system. The US has secondary standards (non-enforceable), based on cosmetic or aesthetic effects.

Some datasheets also include the reference dose or RfD or chronic 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”.

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

4. 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>

# Antimony

CAS No. 7440-36-0.

### Maximum Acceptable Value

Based on health considerations, the concentration of antimony in drinking-water should not exceed 0.02 mg/L (20 g/L).

The maximum contaminant level or MCL in the USA (USEPA 2009/2011) is 0.006 mg/L. The USEPA also established a lifetime health advisory of 0.006 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 2 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 0.006 mg/L, adding that plumbing should be thoroughly flushed before water is used for consumption.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that because of the increasing use of antimony-tin solder, based on health considerations, the concentration of antimony in drinking water should not exceed the limit of determination of 0.003 mg/L.

The Prescribed Concentration or Value (PCV) for antimony in England and Wales is 0.005 mg/L. See Notes.

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

### Sources to drinking-water

#### 1. To source waters

Antimony can reach the aquatic environment from the weathering of minerals and rocks, run-off from soils and atmospheric deposition. Over 100 antimony containing minerals occur in nature. In New Zealand stibnite (Sb2S3), antimony sulphide, is the chief ore of antimony and is found in many quartz lodes, especially in the Reefton Goldfield and in Otago. Other examples of known major occurrence in New Zealand include near Russell, Reefton, Westland, Great Barrier Island and on the Coromandel Peninsula.

Geothermal waters in the Kawerau area can contain up to 0.2 µg/L Sb (GNS 2015).

The concentration of antimony in seawater is about 0.0004 mg/L. The concentrations of antimony detected in Canadian surface waters range from 0.001 to 9.1 mg/L. Concentrations are typically less than 0.01 mg/L and are often closer to 0.001 mg/L.

Antimony can also enter water via the discharge of wastes from industries in which it is used. These include the production of semi-conductors, batteries, solder, pewter, safety matches, electronic equipment, paint pigments, ceramic enamels, glass and pottery, plastics, ammunition primers, antifriction materials, cable sheathing, flame-proofing compounds (major use), and fireworks. Antimony is released into the atmosphere from coal-fired power plants and inorganic chemical plants. It is also found in petrol, diesel and their exhausts. It is alloyed with lead and other metals to increase their hardness, mechanical strength, corrosion resistance, and electrochemical stability or decrease their coefficient of friction. Some antimony alloys expand slightly upon cooling, a valuable property for use in type metal and other castings. Potassium antimony tartrate is used in analytical laboratories and the pharmaceutical industry.

Pewter hip flasks have been observed to release antimony to fluids; WQRA (2009) reports 0.024 mg/L being found in water.

#### 2. From treatment processes

No known sources.

#### 3. From the distribution system

The dissolution of antimony-tin solder, used in household plumbing, by corrosive water is a potential source of antimony from the distribution system. Antimony–tin solder is beginning to replace lead solder and hence exposure to antimony in drinking-water may be increasing.

### Forms and fate in the environment

The most common oxidation state of antimony in water is expected to be +5 (antimonate). In the normal redox range found in surface waters, most of the antimony present in the aquatic environment probably remains in solution. However, antimony has an affinity for clay and mineral surfaces and may co-precipitate with hydrous iron, manganese and aluminium. In fact, more than half of naturally-occurring antimony in sediments is bound to extractable iron and aluminium. Under reducing or anaerobic conditions, trivalent species such as Sb(OH)3, Sb(OH)4-, and Sb2S44- may be significant.

In the atmosphere antimony may be present in gaseous, vapour and particulate forms. Smoking can result in antimony contamination of indoor air.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 897 zones, found antimony concentrations to range from “not detectable” (nd) to 0.012 mg/L, with the median concentration being “nd” (Limit of detection = 0.0005 mg/L). The Priority 2 Identification Programme found six distribution zones supplying drinking-water to a total of 2,300 people with antimony at greater than the MAV (ESR 2001).

WHO (2017) reports concentrations in groundwater are normally less than 0.000001 mg/L, less than 0.0002 mg/L in surface water, and less than 0.005 mg/L in drinking-water.

The NZ Institute of Environmental Health reported in its March 2006 newsletter that water from a German well contained 4 parts per trillion or ppt (0.000004 mg/L) of antimony. A bottle (made of PET) of water bought from a supermarket contained 360 ppt (0.00036 mg/L) and three months later the same water contained 0.00063 mg/L. Antimony trioxide is a catalyst in the manufacture of PET.

972 water utilities in the US reported detecting antimony (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.018 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found 0.0006 to 0.0008 mg/L antimony in the raw water and treated water.

### Removal methods

Coagulation/filtration and membrane filtration will only remove antimony associated with particulate matter. However, antimony is not normally found in source water. Otherwise reverse osmosis should reduce the concentration of dissolved antimony in the raw water.

Where antimony enters the water post-treatment, ie, from dissolution of antimony-tin solder in the distribution system, control of the corrosiveness of the water and of product specification will minimise the presence of antimony in the drinking-water.

### Analytical methods

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113) (pre‑concentration may be necessary).

#### Some alternative methods

1. EPA Method 200.8, Determination of Trace Elements in Waters and Wastes by Inductively Coupled Plasma – Mass Spectrometry.

### Health considerations

Total exposure from environmental sources, food and drinking-water is very low compared with occupational exposure (WHO 2017).

The form of antimony in drinking-water is a key determinant of the toxicity, and it would appear that antimony leached from antimony-containing materials would be in the form of the antimony(V) oxo-anion, which is the less toxic form. The subchronic toxicity of antimony trioxide is lower than that of potassium antimony tartrate, which is the most soluble form. Antimony trioxide, owing to its low bioavailability, is genotoxic only in some in vitro tests, but not in vivo, whereas soluble antimony(III) salts exert genotoxic effects in vitro and in vivo. Animal experiments from which the carcinogenic potential of soluble or insoluble antimony compounds may be quantified are not available (WHO 2017).

Antimony is not absorbed readily from the gastro-intestinal tract, with absorption ranges of <5 percent in cows to 15 percent in rats being reported. Its distribution is highest in spleen, liver and bone, and it is excreted in faeces and urine. Antimony can cross the placenta. Daily oral uptake of antimony appears to be significantly higher than exposure by inhalation, although total exposure from environmental sources, food and drinking-water is very low compared with occupational exposure.

Acute antimony poisoning in humans may result in vomiting, diarrhoea and death. Swallowing a single, high dose of antimony (ie, 30 milligrams) can cause nausea and vomiting; vomiting reduces the amount of antimony taken up by the body. The effects of long-term human exposure to antimony have been investigated in a number of studies. In one study, six adult males who had worked in an antimony smelter for 2–13 years reported no adverse effects. However, other studies have reported increased blood pressure and heart irregularities, ulcers and increased incidence of spontaneous late abortions among female workers. One study, where workers were exposed to dust containing a mixture of antimony trioxide and antimony pentoxide for 9–31 years in an antimony smelting plant, resulted in reports of symptoms such as chronic coughing, bronchitis and emphysema, conjunctivitis, staining of front tooth surface, inactive tuberculosis and pleural adhesions. A dermatitis condition was observed in more than half the exposed workers. Antimony can have beneficial effects when used for medical reasons; it has been used as a medicine to treat people infected with parasites.

Animal studies have shown that antimony may cause sterility, fewer offspring and foetal damage.

Mutagenic activity in tests with bacteria have been demonstrated using trivalent and pentavalent antimony salts. In addition they have been found to induce chromosomal aberrations in cultured human leucocytes and rat bone marrow cells.

A study found that antimony (III) oxide and antimony ore concentrate caused lung tumours in female rats exposed by inhalation. In ingestion studies on rats and mice, antimony did not appear to cause tumours. Antimony leached into water from antimony-containing materials would be in the form of antimony(V) oxo-anion which is the less toxic form.

The reference dose or RfD (USEPA 2009/2011) is 0.0004 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.01 mg/L.

In April 2017 ATSDR established minimal risk levels (MRL) for antimony:

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

0.0006 mg/kg/day for intermediate-duration oral exposure (15–365 days)

The International Agency for Research on Cancer (IARC) has concluded that, by inhalation exposure, antimony trioxide is possibly carcinogenic to humans (Group 2B) and antimony trisulphide is not classifiable as to its carcinogenicity to humans (Group 3). Although there is some evidence for the carcinogenicity of certain antimony compounds by inhalation, there are no data to indicate carcinogenicity by the oral route (WHO 2017).

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

Antimony has been reported (summarised in WQRA 2012) to leach from plastic bottles (polyethylene terephthalate or PET) when reused; the concentration was reported to be 0.25 µg/L (0.00025 mg/L), ie, about 1 percent of the MAV.

### Derivation of Maximum Acceptable Value

As there is limited evidence of the carcinogenicity of antimony to humans, a tolerable daily intake approach has been used for the derivation of the MAV. In a limited life-time study in which rats were exposed to antimony in drinking-water at a single dose level of 0.43 mg antimony/kg body weight per day, reduced longevity and altered blood levels of glucose and cholesterol were observed. The incidence of benign or malignant tumours was not affected. This study has been used as the basis for the lowest-observable-adverse-effects level of antimony used in the derivation of the MAV.

The MAV for antimony 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 per day x 1000

where:

* No Observed Adverse Effect Level = 6 mg/kg body weight per day for decreased body weight gain and reduced food and water intake in a 90-day study in which rats were administered potassium antimony tartrate in drinking-water
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* uncertainty factor = 1,000 (100 for inter- and intraspecies variation and 10 for the short duration of the study)
* average amount of water consumed by an adult = 2 L per day.

The possibility of co-exposure of consumers to arsenic and antimony in drinking-water would necessitate an assessment of the local geological conditions on a case-by-case basis. If both elements were found to be present, case-specific risk evaluations for possible additivity and synergistic effects would need to be performed.

The MAV of 0.003 mg/L for antimony in the 1995 and 2000 DWSNZ was derived as follows:

0.43 mg/kg body weight per day x 70 kg x 0.1 = 0.003 mg/L

2 L per day x 500

where:

* Lowest Observed Adverse Effect Level = 0.43 mg/kg body weight per day observed in a limited lifetime study in rats resulting in decreased longevity and altered blood levels of glucose and cholesterol
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* uncertainty factor = 500 (100 for inter- and intraspecies variation and 5 for the use of a LOAEL instead of a NOAEL
* average amount of water consumed by an adult = 2 L per day.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for antimony is 0.006 mg/L.

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# Arsenic

CAS No. 7440-38-2 (the metal).

### Maximum Acceptable Value (provisional)

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

The WHO guideline value is designated as provisional in view of treatment performance and analytical achievability.

The maximum contaminant level or MCL (USEPA 2009/2011) is 0.01 mg/L total arsenic.

The maximum acceptable concentration in Canada is 0.010 mg/L, based on municipal- and residential-scale treatment achievability. Certified residential treatment devices are commercially available to remove arsenic to well below this concentration. Every effort should be made to maintain arsenic levels in drinking water as low as reasonably achievable.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of arsenic in drinking water should not exceed 0.01 mg/L.

The Prescribed Concentration or Value (PCV) for arsenic in England and Wales is 0.01 mg/L. See Notes.

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

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

The 1984 DWSNZ allowed up to 0.05 mg/L of arsenic.

### Sources to drinking-water

#### 1 To source waters

Arsenic is found widely in Earth’s crust in oxidation states of –3, 0, +3 and +5, often as sulfides or metal arsenides or arsenates. In water, it is mostly present as arsenate (+5), but in anaerobic conditions, it is likely to be present as arsenite (+3). It is usually present in natural waters at concentrations of less than 0.001 to 0.002 mg/L. However, in waters, particularly groundwaters, where there are sulfide mineral deposits and sedimentary deposits deriving from volcanic rocks, the concentrations can be significantly elevated.

The concentration of arsenic in seawater is about 0.01 mg/L.

Arsenic can enter the aquatic environment by the weathering of minerals and rocks, run-off from soils, from geothermal fluids or atmospheric deposition. The mineralised zones of sulphitic ores probably contain the highest concentrations of arsenic although high levels of arsenic may also occur in some coals (recorded at up to 1,500 mg/kg) and peats. In New Zealand, arsenic occurs in greywacke and schists and in tertiary volcanics. In greywacke and schist it occurs as arsenopyrite and loellingite in gold-bearing lodes of the Reefton and Otago goldfields. It also occurs in auriferous quartz lodes associated with volcanics in the Hauraki goldfield, especially in the Tokatea-Coromandel area. Geothermal fluids contain elevated concentrations of arsenic and water bodies such as the Waikato River subjected to their discharge have typically high arsenic concentrations. Levels of around 8–9 mg/L of arsenic have been recorded in geothermal springs in the Tokaanu area. It was estimated that about 300 tonnes per annum entered the Waikato River (Reay 1973).

Arsenic can also be released to the aquatic environment via the discharge of wastes from industries in which it is used. Arsenic and its compounds are used in the production of transistors, lasers, semiconductors, pigments, for medical purposes, in glassmaking, in alloys with lead and copper, rodenticides, insecticides, herbicides, and as timber preservatives (the major use). Sodium cacodylate 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)). Sodium cacodylate is the sodium salt of cacodylic acid (dimethylarsenic acid, CAS No. 75-60-5), a methylated arsenical compound that the USEPA has classified in Group B2: a probable human carcinogen.

USEPA (2008) describes chromated arsenicals as including chromated copper arsenate (CCA), ammoniacal copper zinc arsenate (ACZA), ammoniacal copper arsenate (ACA), and acid copper chromate (ACC). Ammoniacal copper arsenate (ACA) was withdrawn in the US due to lack of use. The registered active arsenical ingredients are orthoarsenic acid and arsenic pentoxide (arsenic acid anhydride). CCA generally contains about  
20–25 percent arsenic pentoxide.

Sheep dip chemicals in New Zealand were arsenic-based until the 1950s. In soil sampling studies at former sheep dip sites in Canterbury, arsenic concentrations of up to 4,390 mg/kg have been recorded, decreasing with distance from the dip to normal soil background concentrations, generally within 15–25 metres from the dip site. Groundwater sampled for arsenic beneath three dipping sites in Canterbury had total arsenic concentrations ranging from 0.15 to 2.42 mg/L (ECan 2003).

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; arsenic levels range from 17 to 70 mg/kg depending on land use.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources, despite the use of arsenical brasses.

### Forms and fate in the environment

The most common oxidation states of arsenic are +3 and +5 although it can also exist in the 0 and -3 states. Arsenic (V) is the stable form of arsenic in aerobic water while arsenic (III) is the predominant form of arsenic under anaerobic conditions such as in groundwaters. In surface waters, the majority of arsenic occurs in a soluble form which can be removed from the water by co-precipitation with hydrated iron and aluminium oxides, or adsorbed/chelated by suspended organic matter in water or humic substances in bottom sediments. Arsenic in aquatic systems partitions preferentially to the sediment. An increase in pH may increase the concentration of dissolved arsenic in water.

Arsenic is more strongly bound to soils that have a high clay or high organic matter content and, in these circumstances, is less available to plants. Arsenic is phytotoxic. Plants take up arsenic in proportion to the soil concentration, except at very high soil concentrations. Plants growing on mine or smelter wastes have developed resistance to arsenic toxicity; such plants sometimes have concentrations of arsenic (6,000 mg/kg has been found) that may be toxic to animals eating the plants. Arsenic taken up by plants is distributed to all tissues (IPCS 1992).

Some micro-organisms can generate arsine from arsenite and arsenate in anaerobic conditions. Arsine decomposes on exposure to light or when it comes into contact with moisture in the air, depositing shiny black arsenic; in water, it rapidly hydrolyses to other arsenic compounds (WHO 2002).

Studies have demonstrated that under certain circumstances copper, arsenic, and/or chromium can leach from treated wood into the surrounding soil or water. In general, most leaching takes place in the first few days and the extent and rate of leaching being highest for copper and lowest for chromium. Available field and laboratory studies suggest that leaching of metals is highly variable and is dependent on environmental conditions. Studies on sorption into soils from utility poles, have shown that the release of metals into soils/sediments from the base of treated wood, decks or utility poles or from the pressure treatment facilities, do not show a high degree of migration, either to groundwater or to the surface. In most cases, after migration of the metals a few meters down into soil, these metals attain the background level concentration of soil (USEPA 2008).

### Typical concentrations in drinking-water

Arsenic was routinely measured in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. Of 1895 samples analysed between 1983 and 1989, 13 samples (1.3 percent of supplies) had concentrations equal to or exceeding the 1984 guideline value of 0.05 mg/L. The majority of drinking-water supplies in New Zealand have arsenic concentrations of less than 0.001 mg/L. However, supplies using source waters significantly contaminated with arsenic such as the Waikato River, that do not fully treat their water have been reported to contain up to 0.15 mg/L in reticulated water.

It is recommended that people using roof water should not burn treated wood, and if water is stored in a timber storage tank, the liner integrity is checked.

The P2 Chemical Determinand Identification Programme, sampled from 342 zones, found arsenic concentrations to range from “not detectable” (nd) to 0.10 mg/L, with the median concentration being “nd” (Limit of detection = 0.001 mg/L). The Priority 2 Identification Programme found 28 distribution zones supplying drinking-water to a total of 21,284 people with arsenic at greater than the MAV (ESR 2001).

Levels in natural waters generally range between 0.001 and 0.002 mg/L, although concentrations may be elevated (up to 12 mg/L) in areas containing natural sources (WHO 2004).

11,173 water utilities in the US reported detecting arsenic (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the average concentration being about 0.03 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found 0.020 to 0.027 mg/L arsenic in the raw water, and <0.001 to 0.002 mg/L in the treated water.

Concentrations of arsenic in groundwater samples in the Waikato region showed a highly (p<0.001) significant positive correlation to concentrations of lithium, and to ammonia, dissolved Fe and Mn.

The less mobile and less toxic arsenate form dominated in 70 percent of cases (14/20 groundwater samples), but:

* substantial conversion to the more mobile and toxic arsenite form had occurred in 30 percent of cases (6/20 samples)
* in the three cases showing very high arsenic (239, 571 and 1210 μg/L total arsenic) there was no consistent pattern. One of these three was entirely arsenate, another entirely arsenite, and the third 86 percent arsenite and 14 percent arsenate. Total arsenic is therefore unreliable as a means of guessing likely speciation. [Perhaps dissolved oxygen and/or redox should have been measured too.]

Due to the possibility of variation in arsenic concentrations in a given bore, it is recommended that groundwater from new drinking water bores developed in the Waikato region should be tested four times for the first year, once during each quarter, to ensure the results are representative (Waikato Regional Council 2006).

Inorganic AsIII, AsV, organic monomethylarsonic (MA) and dimethylarsinic acid (DMA) were determined in geothermal waters as part of the REGEMP III study. Inorganic AsIII was the dominant species present in all except four of the features, which were mainly overflows in which oxidation in air had occurred. Progressive oxidation to AsV was also shown for drainage downstream of Champagne Pool at Waiotapu. Very little DMA was detectable, but MA was measured in a number of features, though never more than 28 percent of the total arsenic present (Waikato Regional Council 2012).

Results of a survey of 638 waters (both source water and drinking water zone) were reported by ESR (2016). In most waters (83 percent of sources and 85 percent of zones) the arsenic concentration was below the limits of detection of the method used. In almost all cases this means the arsenic concentration was less than 0.001 mg/L. Arsenic concentrations were found to exceed 50 percent of the MAV in only 5 percent of source waters and 2 percent of zone waters. The geographical areas in which arsenic concentrations most frequently exceeded 50 percent of the MAV, or the MAV itself, are those known for geothermal activity: parts of the Waikato and Bay of Plenty. In the lower half of the North Island and all of the South Island, no arsenic concentration was found to exceed 50 percent of the MAV. The highest recorded concentration of arsenic in a source water was 0.027 mg/L. This was from the Waikato River near Tuakau. The highest recorded concentration in a zone was reported in the Rangitaiki Plans Rural zone in the Bay of Plenty (0.014 mg/L). Twenty-one source and five zone waters were found to contain arsenic at a concentration exceeding 50 percent of the MAV. Water suppliers were already aware of the elevated arsenic concentrations in the five zones because all have arsenic assigned as a Priority 2 determinand.

### Removal methods

WHO (2017) states that it is technically feasible to achieve arsenic concentrations of 0.005 mg/L or lower using any of several possible treatment methods. However, this requires careful process optimisation and control, and a more reasonable expectation is that 0.01 mg/L should be achievable by conventional treatment (eg, coagulation).

Conventional coagulation treatment with iron or aluminium can achieve good removal of arsenic, depending on: the oxidation state of the arsenic (trivalent arsenic should be converted to pentavalent arsenic by oxidation with chlorine or potassium permanganate); the pH at which the process is carried out; and whether iron or aluminium is used as the coagulant.

Lime-softening, ion exchange resins, and activated alumina can also be used to remove arsenic. The removal of arsenic from water by ion exchange and alumina depends upon the arsenic being present as the negatively-charged arsenate ion, AsO43-. This ion contains arsenic in the highest oxidation state (5+), and oxidation of any arsenic in the 3+ oxidation state is required if it is to be removed by these two processes.

Arsenic removal at Hamilton City’s conventional water treatment plant has been measured.

Arsenic in the Waikato River and Hamilton drinking water, and apparent treatment efficiency, for 1993–1994 and 2002

|  |  |  |
| --- | --- | --- |
| **Monitoring year** | **1993–1994** | **2002** |
| River concentration (μg/L) | 32 | 23 |
| Treated water concentration (μg/L) | 6.2 | 2.2 |
| Percent removal (%) | 80.6 | 90.4 |

Waikato Regional Council (2006).

USEPA (2000 and 2011a) describe arsenic removal technologies. Also see USEPA (2007) for a link to dozens of papers. WRF (2015) describes removing arsenic from a deep groundwater by recharging it to a shallow groundwater system.

### Analytical methods

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Hydride Generation / Atomic Absorption Spectrometric Method (APHA 3114B).

2. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

3. See IARC (2004) for a review. Also FAO/WHO (2011b).

### Health considerations

Arsenic has not been demonstrated to be essential in humans. Arsenic is found in the diet, particularly in fish and shellfish, in which it is found mainly in the less toxic organic form. There are only limited data on the proportion of inorganic arsenic in food, but these indicate that approximately 25 percent is present in the inorganic form, depending on the type of food. Apart from occupational exposure, the most important routes of exposure are through food and drinking-water, including beverages that are made from drinking-water. Where the concentration of arsenic in drinking-water is 0.01 mg/L or greater, this will be the dominant source of intake. In circumstances where soups or similar dishes are a staple part of the diet, the drinking-water contribution through preparation of food will be even greater (WHO 2011a, 2017).

The highest total arsenic concentrations have been found in seaweed, fish and shellfish, mushrooms and fungi, rice and rice products and some meat products. The levels in the remaining food products usually do not exceed 1 mg/kg. Major organic arsenicals present in fish when ingested undergo very little biotransformation and are excreted almost entirely unchanged. FAO/WHO concluded that there had been no reports of ill-effects among populations consuming large quantities of fish that result in organoarsenic intakes of about 0.05 mg/kg bw per day, but further investigation would be desirable to assess the implications for human health of exposure to naturally occurring organoarsenic compounds in marine products. Levels of inorganic arsenic in foods and beverages usually do not exceed 0.1 mg/kg, with mean values generally less than 0.03 mg/kg. However, seaweed, rice and some fish and seafood commodities have higher inorganic arsenic levels, as do food crops grown in arsenic-contaminated soils (FAO/WHO 2011a).

Drinking water is considered to be the major source of exposure to arsenic only in populations living near a source of arsenic (either a natural geological source or a site of contamination). For most Canadians, the primary source of exposure to arsenic is food, followed by drinking water, soil, and air (Health Canada 2006).

EFSA (2014) reports where a study of over 100,000 food samples (including drinking water) from 21 European countries were used to calculate dietary exposure to inorganic arsenic. For all the age classes except infants and toddlers, the main contributor to dietary exposure to inorganic arsenic was the food group ‘Grain-based processed products (non rice-based)’, in particular, wheat bread and rolls. Other food groups that were important contributors to inorganic arsenic exposure were rice, milk and dairy products. Milk and dairy products were the main contributor in infants and toddlers, followed by drinking water.

Both pentavalent and trivalent soluble arsenic compounds are rapidly and extensively absorbed from the gastrointestinal tract. Metabolism is characterised by 1) reduction of pentavalent to trivalent arsenic and 2) oxidative methylation of trivalent arsenic to form monomethylated, dimethylated and trimethylated products. Methylation of inorganic arsenic facilitates the excretion of inorganic arsenic from the body, as the end-products monomethylarsonic acid and dimethylarsinic acid are readily excreted in urine. There are major qualitative and quantitative interspecies differences in methylation, but in humans and most common laboratory animals, inorganic arsenic is extensively methylated, and the metabolites are excreted primarily in the urine. There is large interindividual variation in arsenic methylation in humans, probably due to a wide difference in the activity of methyltransferases and possible polymorphism. Ingested organoarsenicals are much less extensively metabolised and more rapidly eliminated in urine than inorganic arsenic (WHO 2017).

Although the results of available studies indicate that arsenic may be an essential element for several animal species (eg, goats, minipigs, rats, chicks), arsenic has not yet been demonstrated to be essential in humans. The acute toxicity of arsenic compounds in humans is predominantly a function of their rate of removal from the body. Arsine is considered to be the most toxic form, followed by the arsenites, the arsenates and organic arsenic compounds. Acute arsenic intoxication associated with the ingestion of well water containing very high concentrations (21.0 mg/L) of arsenic has been reported.

Drinking water rich in arsenic over a long period leads to arsenic poisoning or arsenicosis. Many waters contain some arsenic and excessive concentrations are known to occur naturally in some areas. The health effects are generally delayed and the most effective preventive measure is supply of drinking water low in arsenic concentration.

The health considerations apply mainly to the inorganic arsenic compounds. These are more likely to be present in drinking-water supplies than the organic compounds. Except for individuals who are occupationally exposed to arsenic, the most important route of exposure is through the oral intake of food and beverages.

Ingested elemental arsenic (which is not very likely to be encountered) is poorly absorbed and is largely eliminated unchanged. Soluble arsenic compounds are readily absorbed from the gastro-intestinal tract. Inorganic arsenic may accumulate in skin, bone and muscle. In humans, inorganic arsenic does not appear to cross the blood-brain barrier but transplacental transfer of arsenic has been reported. The organic forms of arsenic do not readily convert to inorganic forms.

Early symptoms of acute arsenic intoxication include abdominal pain, vomiting, diarrhoea, pain in the muscles, weakness and flushing of the skin. Signs of chronic arsenicalism include dermal lesions, peripheral neuropathy, skin cancer and peripheral vascular disease. Effects on the cardiovascular system were observed in children consuming arsenic-contaminated water (mean concentration 0.6 mg/L) for an average of seven years.

Arsenic does not appear to be mutagenic in bacterial and mammalian assays although it can induce chromosomal aberrations in a variety of cultured cell types, including human cells.

Arsenic is an important drinking-water contaminant, as it is one of the few substances shown to cause cancer in humans through consumption of drinking-water. There is overwhelming evidence from epidemiological studies that consumption of elevated levels of arsenic through drinking-water is causally related to the development of cancer at several sites, particularly skin, bladder and lung. In several parts of the world, arsenic-induced disease, including cancer, is a significant public health problem. WHO (2001) states that approximately 1 in 100 people who drink water containing 0.05 mg/L arsenic or more for a long period may eventually die from arsenic related cancers.

Because trivalent inorganic arsenic has greater reactivity and toxicity than pentavalent inorganic arsenic, it is generally believed that the trivalent form is the carcinogen. However, there remains considerable uncertainty and controversy over both the mechanism of carcinogenicity and the shape of the dose-response curve at low intakes.

IARC (2009) states that non-occupational exposure to arsenic is mainly through food, except in areas with high levels of arsenic in the drinking-water, eg, Taiwan, Bangladesh, West Bengal, northern Chile, and Cordoba Province (Argentina). Epidemiological studies have shown that exposure to arsenic through inhalation or drinking-water causes cancer of the lung, skin, and urinary bladder. Evidence suggests an association between exposure to arsenic in drinking-water and the development of tumours at several other sites; however, various factors prevent a conclusion. Analytical studies have provided only limited information to support an association with kidney cancer, causes of liver cancer can be difficult to elucidate in groups that are high-risk for hepatitis B, and data on prostate cancer and arsenic exposure are not consistent between countries. Overall, the Working Group classified arsenic and inorganic arsenic compounds as “carcinogenic to humans” (Group 1) on the basis of sufficient evidence for carcinogenicity in humans and limited evidence for carcinogenicity in animals.

Over the past 30 years, up to one quarter of the population of Bangladesh has been exposed to levels of arsenic above 0.05 mg/L due to drinking water from contaminated tubewells. One of the motivations for adoption of tubewells in Bangladesh was to reduce of diarrhoeal diseases caused by drinking from undisinfected surface water sources with high levels of microbial contamination. The high arsenic content of the tubewells was not discovered until the adverse effects of arsenic exposure began to be visible in the population after a decade or more of exposure. The health effects include cardiovascular disease, skin lesions, various forms of cancer and reduced intellectual function in exposed children. Summary of study in WQRA (2012).

Inorganic arsenic compounds appear on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. The USEPA (2011) quotes a health advisory of 0.002 mg/L for arsenic, representing a 10-4 cancer risk.

MfE (2011) states:

Arsenic is considered to be a non-threshold contaminant with internal cancers, such as bladder and liver cancers, the most sensitive endpoints. Estimates of carcinogenic potency are primarily derived from human epidemiological data from exposure via drinking water. A daily risk-specific dose of 0.0086 micrograms per kilogram bodyweight (µg/kg bw), derived from the arsenic concentration in drinking water determined to represent “negligible risk” by Canadian agencies (0.3 micrograms per litre, µg/L), is recommended. This value is based on the most current risk modelling data, and includes an external comparison population. Dermal absorption is considered to be negligible, although the skin absorption factor of 0.5 percent could be used as a refinement in the development of soil contaminant standards.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes minimal risk levels (MRLs) for arsenic:

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

0.0003 mg/kg/day for chronic-duration oral exposure (>364 days).

As at July 2013 ATSDR quotes a minimal risk level (MRL) for dimethylarsenic acid (DMA, CAS No. 75-60-5) of 0.02 mg/kg/day for chronic-duration oral exposure (>364 days).

As at July 2013 ATSDR quotes a minimal risk level (MRL) for monomethylarsenic acid (MMA, CAS No. 124-58-3) of:

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 for arsenic (USEPA 2009/2011) is 0.0003 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.01 mg/L.

The livestock guideline value is 0.5 mg/L total arsenic in drinking water; if arsenic is not provided as a food additive and natural levels of arsenic in the diet are low, a level of 5 mg/L in drinking water may be tolerated (ANZECC/ARMCANZ 2000). These guidelines were to be updated in 2012.

WHO (2011b) includes a 164 page paper on the health effects of arsenic in food.

### Derivation of Maximum Acceptable Value

WHO (2017) states that there remains considerable uncertainty over the actual risks at low concentrations, and available data on mode of action did not provide a biological basis for using either linear or non-linear extrapolation. In view of the significant uncertainties surrounding the risk assessment for arsenic carcinogenicity, the practical quantification limit in the region of 0.001–0.01 mg/L and the practical difficulties in removing arsenic from drinking-water, the guideline value of 0.01 mg/L was retained. In view of the scientific uncertainties, the guideline value was designated as provisional.

The practical quantification limit for arsenic is in the region of 0.001 to 0.010 mg/L, and removal of arsenic to concentrations below 0.01 mg/L is difficult in many circumstances. In view of the practical difficulties in removing arsenic from drinking-water, particularly from small supplies, and the practical quantification limit for arsenic, the guideline value of 0.01 mg/L is retained as a goal and designated as provisional (WHO 2011a/2017).

The provisional guideline value of 0.01 mg/L was previously supported by a JECFA provisional tolerable weekly intake (PTWI) of 0.015 mg/kg of body weight (15 μg/kg bw per week) for inorganic arsenic, assuming an allocation of 20 percent to drinking-water. However, JECFA recently re-evaluated arsenic and concluded that the existing PTWI was very close to the lower confidence limit on the benchmark dose for a 0.5 percent response (BMDL0.5) – computed to be 3.0 μg/kg bw per day or 2.0 to 7.0 μg/kg bw per day based on the range of estimated total dietary exposure, calculated from epidemiological studies (specifically for an increased risk of lung cancer) and was therefore no longer appropriate. The PTWI was therefore withdrawn (FAO/WHO, 2011a, b). JECFA concluded that for certain regions of the world where concentrations of inorganic arsenic in drinking-water exceed 0.05 to 0.10 mg/L, some epidemiological studies provide evidence of adverse effects. There are other areas where arsenic concentrations in water are elevated (eg, above the WHO guideline value of 0.01 mg/L), but are less than 0.05 mg/L. In these circumstances, there is a possibility that adverse effects could occur as a result of exposure to inorganic arsenic from water and food, but these would be at a low incidence that would be difficult to detect in epidemiological studies (WHO 2011a/2017).

Therefore, given that, in many countries, even the provisional guideline value may not be attainable, it is retained on the basis of treatment performance and analytical achievability with the proviso that every effort should be made to keep concentrations as low as reasonably possible (WHO 2011a/2017).

The possibility of co-exposure of consumers to arsenic and antimony in drinking-water would necessitate an assessment of the local geological conditions on a case-by-case basis. If both elements were found to be present, case-specific risk evaluations for possible additivity and synergistic effects would need to be performed (WHO 2003a).

The 1995 *Guidelines for Drinking-water Quality Management for New Zealand* had stated:

Data on the association between internal cancers and ingestion of arsenic in drinking-water are insufficient for quantitative assessment of risk. Instead, owing to the documented carcinogenicity of arsenic in the drinking-water of human populations, the lifetime risk of skin cancer has been estimated using a multistage model that is both linear and quadratic in dose. On the basis of observations in a Taiwanese population ingesting arsenic contaminated water, the levels associated with lifetime skin cancer risks of 10-4, 10-5 and 10-6 are 1.7, 0.17 and 0.017 g/L. These values may, however, overestimate the actual risk of skin cancer owing to the possible contribution of other factors to disease incidence in the Taiwanese population and to possible dose-dependent variations in the metabolism that could not be taken into consideration. Moreover, 1–14 percent of arsenic-induced skin cancers are fatal.

WHO has established a provisional guideline value of 0.01 mg/L for arsenic in drinking-water. The estimated lifetime skin cancer risk associated with exposure to this concentration is six per 10,000 (6 x 10-4, or 6 x 10-6 to 8.4 x 10-5 lifetime risk of fatal skin cancers).

The WHO provisional guideline value agrees with the value derived on the basis of the provisional maximum tolerable daily intake for inorganic arsenic of  
2 g/kg body weight, established by the Joint FAO/WHO Expert Committee on Food Additives in 1983, and assuming a 20 percent allocation to drinking-water.

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# Asbestos

CAS No. 1332-21-4 (for asbestos as a group). The six types of asbestos also have individual CAS numbers – see ATSDR (2001).

Chrysotile is also known as white asbestos (NICNAS 1999). Crocidolite is also known as blue asbestos.

### Maximum Acceptable Value

There are insufficient data to derive a health based MAV for asbestos in drinking-water. WHO (2017) states that there is no consistent evidence that ingested asbestos is hazardous to health.

The maximum contaminant level or MCL for asbestos fibres >10 microns in length (USEPA 2009/2011) is 7 million fibres per litre.

Asbestos (friable) is one of the “priority pollutants” under the US Clean Water Act.

Crocidolite, actinolite, anthophyllite and tremolite 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 Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that there are insufficient data to establish a health-based guideline value.

### Sources to drinking-water

#### 1 To source waters

Asbestos is a common term describing a variety of naturally formed hydrated silicates of which the two fundamental varieties are serpentine (chrysotile) and amphibole (actinolite, amosite, anthophyllite, crocidolite, tremolite). The most commonly mined forms of fibrous asbestos are chrysotile, an iron–magnesium silicate white in colour; crocidolite, an iron–sodium silicate blue in colour; and amosite, an iron and magnesium silicate grey-brown in colour. Actinolite, tremolite and anthophyllite occur in both fibrous and non-fibrous forms and have rarely been mined as commercial asbestos.

Asbestos minerals consist of thin, separable fibres that have a parallel arrangement. Non‑fibrous forms of tremolite, actinolite, and anthophyllite also are found naturally. However, because they are not fibrous, they are not classified as asbestos minerals. Amphibole asbestos fibres are generally brittle and often have a rod- or needle-like shape, whereas chrysotile asbestos fibres are flexible and curved. Chrysotile is the predominant commercial form of asbestos; amphiboles are of minor commercial importance.

Chrysotile asbestos is formed in serpentine veins during the alteration and metamorphism of basic igneous rocks rich in ferromagnesium silicates. The amphibole asbestoses occur as a result of metamorphism, resulting, in particular, in the formation of schists and gneisses in association with limestones, argillites and igneous rocks. Examples in New Zealand of such mineral associations include the Dun Mountain ultramafics near Nelson, small lenses of mafic and ultramafic rocks at the base of the Routeburn Formation and in the Red Mountain Ultramafics (in the Livingstone Mountains and West Dome) in West Otago. Natural erosion of these rocks would result in the introduction of asbestos fibres to the aquatic environment.

Asbestos fibres could have been introduced to raw waters from industrial effluents resulting from its use in the production of fire-proofing materials, heat-resistant textiles for fire-proof curtains, garments and gloves, building materials including insulation against heat and noise, floor and ceiling tiles, asphalt felts, coating and patching compounds, sheets and pipes. Asbestos is also used in brake linings and clutch facings (gaskets) because of its friction resistant qualities. Other uses include electrical insulation and certain paper products. Asbestos fibres may also be introduced to raw waters from sewage effluents dissolving any asbestos cement pipes through which they pass. In general, use of asbestos is falling worldwide.

In 1964 a New Zealand occupational standard was set for asbestos fibres in air and, in 1984, the import of raw friable crocidolite and amosite was banned by a Custom Prohibition Order. In 1989 this ban was extended to chrysotile and was confirmed again in 2005 by Customs Import Prohibition Order. The importation of raw asbestos is now prohibited by the Hazardous Substances and New Organisms Act 1996.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

Asbestos fibres can be introduced to the treated water in the distribution system directly from the leaching of asbestos cement (fibrolite) pipes by corrosive waters, ie, water that dissolves the lime/concrete substrate, such as low pH water and water containing free CO2. Studies in the United States and Canada have reported typical asbestos fibre numbers in drinking-water of less than 1 MFL (million fibres per litre). Severe deterioration of asbestos cement pipes has been known to produce fibre numbers of up to 2000 MFL.

The contribution of A/C pipe to the asbestos content of water is dependent upon its aggressivity, which varies as a function of pH, alkalinity and water hardness. In a national survey of 71 locations across Canada, erosion of A/C pipe appeared to contribute measurably to the asbestos content of water supplies at only two locations, even though it is used in about 19 percent of water supplies.

A study on asbestos-cement products in Western Australia found that deteriorating asbestos-cement roofs were common and that asbestos was present in the gutters and run-off water. The highest concentration of asbestos was from roofs 10–17 years old; younger and older roofs produced lower concentrations. (Taken from MoH 2013.)

### Forms and fate in the aquatic environment

Asbestos fibres are insoluble and inert, except chrysotile is soluble in acidic conditions at high temperatures (NICNAS 1999). Not much is known about the properties of asbestos in the aquatic environment although the fate of asbestos fibres is thought to be affected by sedimentation, resuspension and migration processes.

### Typical concentrations in drinking-water

Asbestos has not been measured routinely in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. However, in 1992 a survey was carried out of 18 water supplies (161 samples) to determine the extent to which asbestos was being released into the supplies through dissolution of asbestos cement pipes in the distribution system. In the majority of samples no asbestos fibres were detected. Chrysolite asbestos fibres were detected in 10 samples, and amphibole asbestos fibres were detected in one sample.

In 2017 in Temuka some AC pipes were found to be disintegrating, with fibres blocking filters. The concentration of fibres would be highly variable; one sample was reported to contain 57 MFL. The pipes would be at least 25 years old; there have been other reports of AC pipe disintegration in New Zealand.

103 water utilities in the US reported detecting asbestos in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 19 MFL.

### Removal techniques

For situations where asbestos has entered the source water (weathering of ferromagnesium silicates), coagulation/flocculation with filtration will remove fibres. Asbestos can also arise from corrosion of asbestos cement lined pipes. Corrosion should be minimised by pH and lime correction, allowing a calcium carbonate deposit to line the pipes, or AC pipes should be replaced with plastic pipes where a problem has been shown to exist.

### Analytical methods

#### Referee method

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

#### Some other alternative methods

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

For initial screening purposes, asbestos analysis may be performed using Scanning Electron Microscopy-Energy Dispersive X-ray Analysis (SEM-EDXA). It should be noted that the results of analysis by SEM cannot be compared directly with results achieved using Transition Electron Microscopy (TEM). TEM gives greater than an order of magnitude improvement in resolution, enabling the identification and counting of finer fibres. Therefore, in samples where fibres are detected, the fibre count determined by SEM should be multiplied by 100.

The method is capable of identifying fibres greater than 5 microns in length and greater than 0.15 microns in diameter. The detection limit for the method was estimated at around 300,000 fibres per litre, and the cost of using this technique is considerably less than the TEM method.

Samples found to contain asbestos above 3.5 MFL should be confirmed using TEM. Asbestos can be analysed by transmission electron microscopy with identification by selected-area electron diffraction. This procedure is both costly and time consuming and is not suitable for routine analysis. The limit of detection is about 0.3 MFL.

Also, see NICNAS (1999).

### Health considerations

Asbestos fibres have always existed naturally in air due to weathering of asbestos bearing rock, ie, there is a natural background level of asbestos fibres in air. Normal healthy human lungs can contain significant loading of fibres without harm.

The health hazards associated with inhalation of asbestos have been recognised for a long time. They include asbestosis, cancer of the bronchial tubes, malignant mesothelioma and possible cancers of the gastrointestinal tract and larynx.

Asbestos did not exhibit mutagenic activity in tests with bacteria, but has induced chromosomal aberrations, malignant transformation of mammalian cells *in vitro*, and various biochemical alterations associated with tumour promoters.

The International Agency for Research on Cancer has concluded that asbestos (actinolite, amosite, anthophyllite, chrysotile, crocidolite and tremolite) is carcinogenic to humans by the inhalation route and has classified it in Group 1. IARC (2009) states that “Epidemiological evidence has increasingly shown an association of all forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and anthophyllite) with an increased risk of lung cancer and mesothelioma. Although the potency differences with respect to lung cancer or mesothelioma for fibres of various types and dimensions are debated, the fundamental conclusion is that all forms of asbestos are “carcinogenic to humans” (Group 1). Mineral substances (eg, talc or vermiculite) that contain asbestos should also be regarded as carcinogenic to humans”.

Asbestos appears on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. The USEPA (2011) quotes a health advisory of 700 million fibres/L for asbestos, representing a 10-4 cancer risk.

There is potential for exposure to asbestos fibres in drinking water by inhalation of aerosol droplets or from fibres that are trapped on clothing during washing and which are subsequently released into the atmosphere. This has been studied and except in an extreme case there was no measurable increase in the number of fibres in the indoor atmosphere of houses. In addition, the fibres in drinking water consist almost entirely of short fibres, which are considered to contribute little or no risk to public health (DWI 2002).

Asbestos is a known human carcinogen by the inhalation route. Although it has been well studied, there is little convincing evidence of the carcinogenicity of ingested asbestos in epidemiological studies of populations with drinking-water supplies containing high concentrations of asbestos. Moreover, in extensive studies in experimental animal species, asbestos has not consistently increased the incidence of tumours of the gastrointestinal tract (DWI 2002). There is therefore no consistent evidence that ingested asbestos is hazardous to health, and thus it is concluded that there is no need to establish a health-based guideline value for asbestos in drinking-water. The primary issue surrounding asbestos-cement pipes is for people working on the outside of the pipes (eg, cutting pipe), because of the risk of inhalation of asbestos dust. Limited data indicate that exposure to airborne asbestos released from tap water during showers or humidification is negligible.

Moreover, in extensive studies in animal species, asbestos has not consistently induced increases in the incidence of tumours of the gastrointestinal tract. The weight of the evidence shows that ingested asbestos is not hazardous to health.

All forms of asbestos can cause cancer, but blue asbestos (crocidolite) is more potent than white asbestos (chrysotile) (MoH 2007). James Hardie Ltd used mainly chrysotile in their asbestos cement pipes; manufacturing ceased about 1982–84. Their autoclave curing process produced a stronger product than the typical air drying processes used overseas.

Three common asbestos types have been used industrially. Amosite and crocidolite are of the amphibole variety – they have straight fibre structures and are highly insoluble in lung fluid, and thus can persist in lung tissues for decades after inhalation. The third, and by far the most commonly used type in New Zealand, is chrysotile, which has a curly fibre structure and is relatively more soluble and more readily cleared from the lungs than the amphiboles. Estimates from different studies vary, but it is generally acknowledged that the cancer risk is higher from amphibole exposure than from chrysotile exposure. One estimate of the ratio of the potency for inducing mesothelioma suggested that chrysotile is up to 500x less potent than crocidolite, and 100x less potent than amosite. Nonetheless, all forms of asbestos are considered to be carcinogenic, and therefore hazardous (RSNZ/PMCSA 2015).

The next two paragraphs are taken from MoH (2007 and repeated in MoH 2013):

Most asbestos fibres in water are chrysotile and are <5 microns in length (ATSDR 2001). Available data on effects of exposure to chrysotile asbestos specifically in the general environment, including data from ecological studies of populations in Connecticut, Florida, California, Utah and Quebec and from case control study in Puget Sound, Washington State, are restricted to those populations exposed to relatively high concentrations of chrysotile asbestos in drinking water, particularly from serpentine deposits or asbestos cement pipes. WHO (1998) concluded that convincing evidence of an association between asbestos in public water supplies and cancer induction was scant.

The adverse effects following ingestion of asbestos have not been documented clearly. However, there is some evidence that acute oral exposure may induce precursor lesions of colon cancer, and chronic oral exposure may increase the risk of GI tumours (ATSDR 2001). The National Academy of Sciences (NAS 1983) and the United States Environmental Protection Agency (USEPA 1980) estimate that lifetime ingestion of water containing 1.0 MF/L (million fibres per litre of water) would result in an excess incidence of GI cancer of about 1–10 cases per 100,000 people exposed. No studies were located regarding death in humans or animals after acute or intermediate oral exposure to asbestos (ATSDR 2001).

### Derivation of Maximum Acceptable Value

No MAV.

WHO (2003) states:

Although asbestos is a known human carcinogen by the inhalation route, available epidemiological studies do not support the hypothesis that an increased cancer risk is associated with the ingestion of asbestos in drinking-water. Moreover, in extensive feeding studies in animals, asbestos has not consistently increased the incidence of tumours of the gastrointestinal tract. There is therefore no consistent, convincing evidence that ingested asbestos is hazardous to health, and it is concluded that there is no need to establish a guideline for asbestos in drinking-water.

The 1995 DWSNZ and datasheet included a maximum desirable target value of 7 million fibres per litre. Maximum desirable target values were not defined.

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# Barium

CAS No. 7440-39-3.

### Maximum Acceptable Value

Based on health considerations, the concentration of barium in drinking-water should not exceed 0.7 mg/L.

The maximum contaminant level or MCL (USEPA 2009/2011) is 2 mg/L. The maximum acceptable concentration in Canada is 1 mg/L.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of barium in drinking water should not exceed 2 mg/L (the previous version stated 0.7 mg/L).

### Sources to drinking-water

#### 1 To source waters

Barium, as the metal, does not occur freely in nature, but predominantly as the minerals barite (barium sulphate) and witherite (barium carbonate) in igneous rocks, sandstone and shale, making up 0.05 percent of the earth’s crust. It also occurs in ores of lead, zinc and silver and is often associated with fluorspar. Barium is also found in sedimentary rocks replacing the potassium in feldspar. The weathering of barite and witherite, as well as other barium-containing minerals releases barium to surface waters.

The concentration of barium in seawater is about 0.03 mg/L.

There are more than 20 known isotopes, but most of them are highly radioactive and have half-lives ranging from several milliseconds to several minutes.

In New Zealand the only major occurrence of barite is near Nelson. All other occurrences are very small with numerous veinlets in rocks of various ages, as vesicles in volcanic rocks and as concretions in Cretaceous and Tertiary sediments. Naturally occurring barium is a mix of seven stable isotopes.

Barium can also be found in raw water as the result of industrial discharges. Barium is used as a filler in paints, plastics and rubber products, in the production of glass, ceramics, photographic paper, soap, cement, metal alloys, green flares, explosives, lubricating oils and drilling muds, cosmetics, diesel fuels, optical glasses, in the case hardening of steel, and as a rat poison. Barium metaborate is used in some of these applications (USEPA 1994). It may also enter water close to drilling operations where it is used as an oil drilling mud.

#### 2 From treatment processes

Depending on the quality of the hydrated lime used, barium may become significant in lime softening plants.

#### 3 From the distribution system

Barium is used in the manufacture of plastics from which guttering and down-piping are made. However, leaching tests have indicated that significant concentrations of barium are not released from the plastic.

### Forms and fate in the environment

Barium is present in water primarily from natural sources. It occurs in both the 0 and +2 oxidation states and its concentration in water is limited by the presence of sulphate and carbonate ions which cause it to precipitate. Some barium salts (eg, acetate, nitrate, and chloride) are quite soluble in water, whereas others (eg, arsenate, carbonate, oxalate, chromate, fluoride, sulfate, and phosphate) are very poorly soluble. The water solubility of barium salts, except for barium sulfate, increases with decreasing pH. Generally barium is found in only trace amounts in surface waters because of this tendency to precipitate or to partake in adsorption or sedimentation processes. While some barium in water is removed by precipitation, exchange with soil, or other processes, most barium in surface waters ultimately reaches the ocean. Once freshwater sources discharge into seawater, barium and the sulfate ions present in salt water form barium sulfate.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 841 zones, found barium concentrations to range from “not detectable” (nd) to 0.082 mg/L, with the median concentration being 0.008 mg/L (limit of detection = 0.01 mg/L). The Priority 2 Identification Programme found one distribution zone supplying drinking-water to a total of 30 people with barium at greater than the MAV (ESR 2001).

Barium levels were determined in 122 municipalities in 10 provinces in samples of raw, treated and distributed drinking water serving approximately 36 percent of the Canadian population. The provincial median concentrations of barium in distributed water ranged from not detectable (0.005 mg/L) to 0.084 mg/L. The Canada-wide median concentration was 0.018 mg/L. The maximum level of barium in a sample of distributed water was 0.60 mg/L. Barium levels in raw water were not significantly different from those in treated water (Health Canada 1990).

WHO (2004/2017) stated that concentrations in drinking-water are generally below 0.1 mg/L, although concentrations above 1 mg/L have been measured in drinking-water derived from some groundwater.

Barium concentrations in drinking-water in the United States typically average 0.03 mg/L, but can average as high as 0.3 mg/L. However, individuals residing in certain regions of Kentucky, northern Illinois, New Mexico, and Pennsylvania who rely on groundwater for their source of drinking-water may be exposed to barium concentrations as high as 10 times the maximum contaminant level (MCL) in drinking-water of 2.0 mg/L.

21,744 water utilities in the US reported detecting barium (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 3.9 mg/L.

The maximum concentration found in 21,954 samples from 3,043 groundwaters in the UK was 25.4 mg/L, mean 0.095 mg/L (DWI 2008).

In 2013/14 Hamilton’s six-monthly analyses have found 0.015 to 0.019 mg/L barium in the raw water, and 0.013 to 0.014 mg/L in the treated water.

### Removal methods

Cation exchange softening for the removal of barium is excellent. Barium is removed in preference to other major cations such as calcium and magnesium. Regeneration of the resin requires special attention because of the resin’s capacity and affinity for barium ions.

Lime-softening at pH 9.5 to 11.5 effectively removes barium, and is pH dependent with optimum removal at pH 10.5. Reverse osmosis and electrodialysis also remove barium.

Conventional alum and iron sulphate coagulation is not effective for the removal of barium, even though insoluble barium sulphate is expected to form. Two-stage coagulation is up to 80 percent effective, but is slow and very costly.

### Analytical methods

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Flame Atomic Absorption Spectrometric Method (APHA 3111).

2. Inductively Coupled Plasma (ICP) Method (APHA 3120).

### Health considerations

Barium is not considered to be an essential element for human nutrition. The degree of absorption of barium from the gastro-intestinal tract depends on the solubility of the barium compound, the animal species, diet and age. [Barium sulphate](http://en.wikipedia.org/wiki/Barium_sulfate) (insoluble) is used as a [radiocontrast](http://en.wikipedia.org/wiki/Radiocontrast) agent for [X-ray imaging](http://en.wikipedia.org/wiki/Medical_imaging) of the digestive system ([barium meals](http://en.wikipedia.org/wiki/Barium_meal) and [barium enemas](http://en.wikipedia.org/wiki/Barium_enema)) and no adverse systemic effects have been reported. Following absorption, barium is deposited in bone and teeth and the principal route of excretion is faecal.

Food is the primary source of intake for the non-occupationally exposed population. In most foods, the barium content is relatively low (<3 mg/100 g) except in Brazil nuts, which have a very high barium content (150–400 mg/100 g). Bread is considered the largest source of dietary barium, contributing an estimated 20 percent of total intake. However, where barium levels in water are high, drinking-water may contribute significantly to total intake.

At high concentrations, barium causes vasoconstriction (constriction of blood vessels), peristalsis (contractions of the alimentary canal), convulsions and paralysis.

Although an association between mortality from cardiovascular disease and the barium content of the drinking-water has been reported in an epidemiological study, these results were not confirmed in an analytical epidemiological study of individuals in the same population. Moreover, in a short-term study in a small number of volunteers, there was no consistent indication of adverse cardiovascular effects following exposure to up to 10 mg/L in water.

Long-term studies with rats have shown that relatively low doses of barium in drinking-water can result in significant and persistent increases in systolic blood pressure. This has significance to humans as an increase in systolic blood pressure can increase the risk of heart attack.

There is no evidence that barium can cause an increase in the incidence of cancer. Barium chloride is not mutagenic in tests with bacteria and does not damage DNA.

There is no conclusive evidence that barium compounds induce reproductive, teratogenic, or carcinogenic effects in human beings. Barium has been shown to cause nephropathy in laboratory animals, but the toxicological end-point of greatest concern to humans appears to be its potential to cause hypertension (increased blood pressure).

The reference dose or RfD (USEPA 1998 revised/confirmed 2005, 2006, 2009, 2011) is 0.2 mg/kg/d, based on a BMDL05 of 63 mg/kg/d and an uncertainty factor of 300. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 7 mg/L.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes minimal risk levels (MRLs) for soluble salts of barium of:

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

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

### Derivation of Maximum Acceptable Value

WHO (2017) developed a guideline value of 1.3 mg/L, based on a TDI of 0.21 mg/kg bw per day, derived by applying an uncertainty factor of 300 to account for intraspecies variation (10), interspecies variation (10) and database deficiencies (3 for the lack of a developmental toxicity study) to a BMDL05 of 63 mg/kg bw per day for nephropathy in mice in a two-year study. This guideline value based on the long-term mouse study is not inconsistent with health-based values that could be derived from limited human studies.

The MAV in the 2008 DWSNZ for barium in drinking-water was a concentration of 0.7 mg/L. This value has been derived using the no-observed-adverse-effects level (NOAEL) of 7.3 mg/L from the most sensitive epidemiological study conducted to date. This study reported no significant differences in blood pressure or the prevalence of cardiovascular disease between a population drinking-water containing a mean barium concentration of 7.3 mg/L and one ingesting water containing barium at 0.1 mg/L, and incorporating an uncertainty factor of 10 to account for intraspecies variation.

The MAV for barium is based on an epidemiological study in which no adverse effects were observed, although the study population was relatively small and the power of the study was limited. As a consequence, an uncertainty factor of 10 was applied to the level of barium in the drinking-water of the study population. However, the level at which effects would be seen may be significantly greater than this concentration, so the guideline value for barium may be highly conservative and the margin of safety is likely to be high (WHO 2004 – first addendum).

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

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# Beryllium

CAS No. 7440-41-7 (the metal).

### Maximum Acceptable Value

Because beryllium occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value.

DWSNZ 2005 had retained the DWSNZ 2000 provisional MAV of 0.004 mg/L. WHO 1996 and 2004 excluded beryllium from guideline derivation because it was considered unlikely to occur in drinking-water.

However, beryllium is included on the plan of work of the rolling revision of the WHO Guidelines because: “WHO has received correspondence noting that although the *Guidelines for Drinking-water Quality* state that beryllium is unlikely to be present in drinking-water, over the past five years in Europe, geological studies show concentrations ranging from <0.000005 to 0.0027 mg/L (median 0.00001 mg/L) across Europe.”

The maximum contaminant level or MCL (USEPA 2009/2011) is 0.004 mg/L.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of beryllium in drinking water should not exceed 0.06 mg/L (the previous version stated that data are insufficient to set a health-based guideline value for beryllium).

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

### Sources to drinking-water

#### 1 To source waters

Beryllium enters natural waters through the weathering process and through atmospheric deposition. The combustion of fossil fuels (particularly coal) is the major source of beryllium to the environment. Other less significant sources are slag and ash dumps. Beryllium is found in the Earth’s crust at an average concentration of approximately 2.8–5.0 mg/kg.

The principal beryllium containing mineral in New Zealand is the precious gemstone, beryl, which is very resistant to weathering. Major occurrence of beryl includes in granitic pegmatites in two places in New Zealand: Charleston and Stewart Island. In addition, beryllium replaces the silicon in feldspar minerals and it is estimated that  
85–98 percent of the total crustal beryllium may be bound in these minerals.

The concentration of beryllium in seawater is about 0.0000008 mg/L.

Beryllium may also be present in raw water from the discharge of industrial and municipal wastes. Beryllium is used in the production of light alloys, copper and brass, in the production of X-ray tubes and neon sign electrodes, electric contacts and switches, and as a catalyst in the manufacture of organic chemicals. It is also a component of diesel exhaust. Beryllium metal is used primarily in the form of beryllium-copper and other alloys in the aerospace, weapons and nuclear industries. The primary source of beryllium compounds in water appears to be release from coal burning and other industries using beryllium.

Beryllium concentrations ranged from <4 to 120 ng/L (ie, up to 0.00012 mg/L) in the Great Lakes in the USA and from <10 to 120 ng/L (10 to 30 ng/L average) in Australian river waters. The maximum concentration found in a large survey in the Czech Republic was 0.035 mg/L.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

At neutral pH, most beryllium salts dissolved in water will be hydrolysed to insoluble beryllium hydroxide, and only trace quantities of dissolved beryllium will remain. However, at high pH, water-soluble complexes with hydroxide ions may form, increasing the solubility and mobility of beryllium. Solubility may also increase at low pH; detectable concentrations of dissolved beryllium have been found in acidified waters.

Except for its metallic form, beryllium exists primarily in the +2 oxidation state. In aqueous solution beryllium does not exist as free Be(II) but as hydrated complexes, in particular the insoluble beryllium hydroxide. In most aqueous environments, beryllium is present in particulate rather than dissolved form, primarily because of the insolubility of beryllium oxides; therefore beryllium will settle out into the sediments, and is not likely to find its way to groundwater.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 831 zones, found beryllium concentrations to range from “not detectable” (nd) to 0.012 mg/L, with the median concentration being “nd” (Limit of detection = 0.001 mg/L). Beryllium concentrations in drinking water overseas are generally very low, usually less than 0.001 mg/L.

The Priority 2 Identification Programme found no distribution zones supplying drinking-water with beryllium at greater than the detection limit (ESR 2001).

682 water utilities in the US reported detecting beryllium (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.012 mg/L.

The maximum concentration found in 16,163 samples from 2,707 groundwaters in the UK was 0.067 mg/L, mean 0.0006 mg/L (DWI 2008).

In 2013/14 Hamilton’s six-monthly analyses have found <0.0001 mg/L beryllium in the raw water and treated water.

### Removal methods

Coagulation/filtration, lime softening, activated alumina, ion exchange and reverse osmosis are methods that have been used for the removal of beryllium from drinking-water.

### Analytical methods

#### Referee method

A referee method cannot be selected for beryllium 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 beryllium for the above reason. However, the following methods are used to analyse for beryllium:

1. Electrothermal Atomic Absorption Spectrometric Method (APHA 3113B).

2. Flame Atomic Absorption Spectrometry (APHA 3111).

3. Inductively Coupled Plasma Method (APHA 3120B).

4. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

### Health considerations

Beryllium is poorly absorbed from the gastrointestinal tract, probably because as soluble beryllium compounds pass into the intestine, which has a higher pH, the beryllium is precipitated as the insoluble phosphate and thus is no longer available for absorption.

No studies are available on the health effects on humans of beryllium following ingestion. As gastrointestinal absorption is poor, toxicity is expected to be low via this route. Inhalation is known to cause serious health effects, with long-term exposure resulting in pulmonary granulomatosis (a type of lung tumour). Between 1.6 percent and 10 percent of the beryllium content, or 0.008 to 0.074 μg/cigarette, was reported to pass into the smoke during smoking. Assuming the smoke is entirely inhaled, an average smoker (20 cigarettes per day) might take in approximately 1.5 μg of beryllium per day (three times the combined total of the other routes). Because of the long storage of beryllium in the skeleton and in the lungs, its biological half-life is extremely long. In the human skeleton, it has been calculated to be 450 days.

Short-, medium-, and long-term studies in animals showed that the gastrointestinal and skeletal systems are target organs for beryllium following oral exposure. Dogs chronically exposed to soluble beryllium sulfate in the diet developed gastrointestinal lesions and bone marrow hypoplasia. Rickets were observed in rats exposed to sparingly soluble beryllium carbonate in the diet for 3–4 weeks, possibly due to decreased gastrointestinal absorption of phosphorus subsequent to formation of insoluble beryllium phosphate in the intestine (WHO 2001).

Beryllium interacts with DNA and causes gene mutations, chromosomal aberrations, and sister chromatid exchange in cultured mammalian somatic cells, although it is not mutagenic in bacterial test systems.

IARC (2009) states that “The Working Group reaffirmed the classification of beryllium and its compounds, cadmium and its compounds, chromium (VI) compounds, and nickel compounds as “carcinogenic to humans” (Group 1). Studies involved complex occupational exposures to a metal and its compounds, making it impossible to separately assess their carcinogenicity.”

WHO (2009) states: the International Agency for Research on Cancer evaluated the carcinogenicity of beryllium and assigned beryllium and beryllium compounds to Group 1, concluding that they are carcinogenic to humans. The assessment was based on sufficient evidence for carcinogenicity in humans and sufficient evidence for carcinogenicity in animals. However, this relates to its carcinogenicity through the inhalation route and not the oral route. The oral carcinogenicity database is considered inadequate for assessing the carcinogenic potential of ingested beryllium. No human data are available, and the animal studies produced only negative results and were limited by failure to achieve the MTD. Derivation of a quantitative cancer risk estimate for oral exposure is therefore precluded.

USEPA (1998) states that based on the weight of evidence (limited human and sufficient animal), beryllium can be classified as a probable human carcinogen (B1) according to the 1986 guidelines. According to the 1996 proposed guidelines, inhaled beryllium would be characterised as a “likely” carcinogen; the human carcinogenic potential of ingested beryllium cannot be determined because of inadequate data. The lack of adequate oral carcinogenicity data is an area of scientific uncertainty for this assessment.

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

The chronic reference dose or RfD (USEPA 1998, 2006 and 2009/2011) is 0.002 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.07 mg/L.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.002 mg/kg/day for chronic-duration oral exposure (>364 days) to beryllium.

### Derivation of Maximum Acceptable Value

No MAV.

WHO (2009/2017) stated that beryllium is rarely, if ever, found in drinking-water at concentrations of concern, therefore, it is not considered necessary to set a formal guideline value. A health-based value for beryllium in drinking-water would be 0.012 mg/L based on an allocation of 20 percent of the TDI of 0.0002 mg/kg body weight to drinking water and assuming a 60 kg adult drinking 2 litres of water per day. This allocation is probably conservative since the limited data on food indicate that exposure from this source is likely to be well below the TDI. A rounded health-based value for 70 kg body weight would be 0.01 mg/L.

Although beryllium appears to be found in drinking-water sources and drinking-water at low concentrations, the database on occurrence is limited and there may be specific circumstances in which concentrations can be elevated due to natural sources where the pH is either below 5 or above 8 or there is high turbidity.

The 2000 and 2005 DWSNZ included a provisional MAV of 0.004 mg/L for beryllium, derived in-house, possibly based on the USEPA MCL.

There was no MAV in the 1995 DWSNZ. The datasheet in 1995 stated: there are no suitable oral data on which to base a toxicologically supportable MAV. However, the low concentrations of beryllium normally found in drinking-water seem unlikely to pose a hazard to consumers.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The cancer health risk limit for beryllium is 0.00008 mg/L.

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# Bismuth

### Maximum Acceptable Value

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

### Sources to drinking-water

#### 1 To source waters

A fairly new range of biocides, eg, bismuth-2,3-dimercaptopropanol, is in production, primary for use in water supplies in buildings to control biofilms and slime build up in hot water systems.

Bismuth is an environmentally friendly substitute for lead in plumbing and many other applications, including fishing weights, hunting ammunition, lubricating greases, and soldering alloys.

The concentration of bismuth in seawater is about 0.0001 mg/L.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

Bismuth has been used in several applications designed to provide nontoxic substitutes for lead. The US Safe Drinking Water Act Amendment of 1996 required that all new and repaired fixtures and pipes for potable water be lead-free after August 1998. Bismuth use in water meters was one particular application that increased. Other major areas of development include bismuth-containing solders, and lubricating greases, especially extreme pressure lubricants. The use of new zinc-bismuth alloys in galvanising to achieve better processing is growing.

A new generation of reasonably priced brass alloys is now available for plumbing fittings and in-line devices. These “very low lead” brasses contain <0.25 percent lead, and bismuth (up to 7 percent by weight), or a combination of bismuth and selenium, replaces the lead in the alloy (AWWARF 2005). Lead, bismuth and selenium discharges from these materials is said to be extremely low (Mass et al 2002).

The growth of bismuth-based brass fittings may be limited by the availability of sufficient quantities of bismuth, and manufacturing difficulties, eg, it is said that bismuth brasses can only be cast, not forged.

The EU allows bismuth containing fittings to contribute up to 90 percent of the bismuth found at consumers’ taps, and recommends that the concentration of bismuth in the water does not exceed 0.01 mg/L.

### Typical concentrations in drinking-water

Five samples from British Columbia water supplies were tested during 2004/2005 and all contained <0.00002 mg/L Bi.

### Analytical methods

#### Referee method

A referee method cannot be selected for bismuth 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 bismuth for the above reason.

### Health considerations

Bismuth subsalicylate has been used for the treatment of gastrointestinal problems, dosage two 262-mg tablets four times a day for up to three weeks. Such long-term use can, however, darken the tongue and stool, produce tinnitus and cause reactions in salicylate-sensitive patients.

### Derivation of Maximum Acceptable Value

There is no MAV.

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# Boron

CAS No. 7440-42-8.

### Maximum Acceptable Value

The MAV for boron in the 2000, 2005 and 2008 DWSNZ, based on health considerations, is (or was) 1.4 mg/L.

If the next DWSNZ adopt the WHO (2011) Guideline Value the MAV will probably become 2.4 mg/L.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that considering that boron may be an essential trace element for humans and based on an acceptable range of oral intake, a concentration of up to 4 mg/L in water would not pose a human health risk.

The USEPA (2006) 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 2 litres of water per day. USEPA (2008b) derived a Drinking Water Equivalent Level (DWEL) of 6.7 mg/L rounded to 7 mg/L. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity. USEPA (2009/2011) revised the lifetime health advisory to 6 mg/L.

The maximum acceptable concentration in Canada is 5 mg/L.

The WHO (2004) had a provisional Guideline Value of 0.5 mg/L. This was changed to 2.4 mg/L in their 2011 and 2017 editions.

The Prescribed Concentration or Value (PCV) for boron in England and Wales is 1 mg/L. See Notes.

Boron 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

Boron is a naturally-occurring element that is widespread in nature at relatively low concentrations. Boron concentrations in rocks and soils are typically less than 10 ppm (mg/kg), although concentrations as high as 100 ppm have been reported in shales and some soils. The overall average concentration in the earth’s crust has been estimated to be 10 ppm. The most common boron containing mineral is tourmaline which is present in igneous and some sedimentary rocks. The weathering of both of these rock types releases boron, which is then transported in solution. Soil leaching and volcanic activity may also add boron to water.

Boron has been found in hot springs and brines at high concentrations, indicating that hydrothermal and geothermal fluids are also a source of boron. Ngawha Springs in Northland contains about 1000 mg/L boron, about 25 times that found in the Taupo Volcanic Zone geothermal fluids, and discharges 7600 tonnes a year (GNS 2015).

Boron is found in the environment primarily combined with oxygen in compounds called borates. Common borate compounds include boric acid and sodium tetraborate (borax).

Boron is actually a mixture of two stable isotopes, 10B (19.8 percent) and 11B (80.2 percent).

The concentration of boron in seawater is about 4.0–4.5 mg/L.

Boron enters the environment mainly through the weathering of rocks, boric acid volatilisation from seawater, and volcanic and geothermal activity. Boron may also be released to water from the discharge of industrial and domestic wastewaters, coal-fired power stations, or in agricultural run-off. In industry, boron is used in fire retardants, borosilicate glass (Pyrex), fibreglass insulation, porcelain enamels, ceramic glazes and antioxidants for soldering, detergents and in the photographic, cosmetic, leather, textile, paint and wood-processing industries. Boron compounds are also found in some fertilisers, herbicides and insecticides. Boric acid use is approved in New Zealand as a parenteral nutrient/electrolyte. Boron compounds are also used in eyedrops, ant poison and in the preparation of mild disinfectants and drugs and in some synthetic rocket fuels.

Elemental boron is used to harden metals, in nuclear reactors for neutron absorption and in agriculture to improve crop yields. For some of these uses, barium metaborate is used (USEPA 1994).

Boric acid is used as an enzyme stabiliser in liquid fabric softeners, laundry and dishwasher detergents, cosmetics and oral care products. DWI (2014) considers boric acid to be one of the commonest substances to enter water from the use of personal care products and domestic cleaning products. It is not extensively removed during wastewater treatment.

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; boron levels can exceed 10,000 mg/kg for all the land uses.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

The chemical behaviour of boron in the aquatic environment is poorly understood, but it is thought that the predominant species is boric acid, a very weak acid which is moderately soluble in water and does not dissociate readily, ie, it is represented better as B(OH)3 rather than H3BO3. Borates and metaborates (which are ionic and therefore soluble) form when the pH exceeds 9.

The adsorption of borates and boric acids to soils is controlled by the presence of aluminium and iron oxides and, to a lesser extent, organic matter. Soils rich in these oxides may experience significant adsorption of available borates (USEPA 2008).

### Typical concentrations in drinking-water

Boron was routinely measured in New Zealand drinking-water supplies as part of the Department of Health three-yearly surveillance programme. Of 1,904 samples analysed between 1983 and 1989, 35 samples (1.3 percent of supplies) contained concentrations equal to or exceeding the 1984 guideline value of 0.5 mg/L.

The P2 Chemical Determinand Identification Programme, sampled from 297 zones, found boron concentrations to range from “not detectable” (nd) to 11 mg/L, with the median concentration being “nd” (limit of detection = 0.06 mg/L). The Priority 2 Identification Programme found three distribution zones supplying drinking-water to a total of 1,225 people with boron at greater than the MAV of 1.4 mg/L (ESR 2001).

The USEPA evaluated boron occurrence in drinking water using data collected from 989 groundwater public water systems by the National Inorganics and Radionuclides Survey. The data indicate that approximately 4.3 percent of the groundwaters had detections of boron at levels greater than 0.70 mg/L, ie, half the health reference level (HRL), affecting approximately 2.9 percent of the population served by these groundwater systems. Approximately 1.7 percent of the groundwaters had detections of boron at levels greater than 1.40 mg/L (the HRL), affecting approximately 0.4 percent of the population served by these groundwater systems (USEPA 2008).

Harari et al (2012) report that in San Antonio de los Cobres (Northwestern Argentina) the public drinking water, supplied from a natural spring outside the village, has about 1 mg/L of lithium and 6 mg/L of boron. The authors note that at present there are no specific data to suggest adverse effects in children born in the San Antonio de los Cobres area compared to other areas in the study.

### Removal methods

There are, at present, no economically feasible methods of removing boron from source waters, other than changing or diluting the source water. Boron concentrations can be reduced by granular activated carbon (although not very practical or efficient), high pH processes such as anion exchange or lime-softening. See WHO (2009) for further discussion. Blending with low-boron supplies may be the only economical method to reduce boron concentrations in waters where these concentrations are high.

### Analytical methods

#### Referee method

Colorimetric Method, Azomethine-H Parts C, D (Boron in Waters, Effluents, Sewage and Some Solids, 1980, HMSO, UK). Also described in ISO (1990).

#### Some alternative methods

1. Colorimetric Method (APHA 4500-B B). WHO (2009) discusses ICP-MS.

### Health considerations

Boron is an essential element for plant growth and is sometimes applied to the soil as a plant fertiliser. Some sensitive plants cannot tolerate high levels of boron, eg, over 5 mg/L – this situation normally arises when boron accumulates in the soil in glasshouses. Boron is present naturally in many food products. The richest sources of boron are fruits, vegetables, pulses, legumes, and nuts. Dairy products, fish, meats, and most grains are poor sources of boron. It has been estimated that intake of boron from food is about 10 times that from water. Boric acid and sodium tetraborate are used to preserve sturgeon eggs (caviar).

WHO (2009) reports that boron appears to be an essential nutrient for humans, in that dietary deprivation of boron consistently results in changed biological functions that are detrimental and that can be corrected by increasing boron intake. Similar effects have been shown in animal models. However, as yet, no specific biochemical function for boron has been discovered. The signs of boron deficiency in animals are variable in nature and severity, being dependent on dietary intake of aluminium, calcium, cholecalciferol, magnesium, methionine and potassium. Variables affected by dietary boron include plasma and organ calcium and magnesium concentrations, plasma alkaline phosphatase and bone calcification. Consistent signs of deficiency include depressed growth and a reduction in some blood indices, particularly steroid hormone concentrations.

Numerous studies have shown that boric acid and borax are absorbed from the gastrointestinal tract and from the respiratory tract, as indicated by increased levels of boron in the blood, tissues or urine or by systemic toxic effects in exposed individuals or laboratory animals. Boric acid can form complexes with carbohydrates and proteins in the body. Boron, when administered as borates or boric acid, is rapidly and almost completely adsorbed from the gastrointestinal tract. Boron excretion occurs mainly through the kidney.

The Institute of Medicine of the National Academies categorises boron as a possible trace mineral nutrient for humans. It may interact with vitamin D and calcium homeostasis, influence estrogen metabolism, and play a role in cognitive function. The estimated average dietary intake of boron in US male adults is 1.5 mg/day. Large doses (of the order of 20 mg/kg or more) can cause nausea and vomiting (USEPA 2008).

Studies in both humans and animals show that boron is readily absorbed from the gastrointestinal tract, distributing evenly throughout the soft tissues, and also showing some accumulation in bone (USEPA 2008a).

WHO (2009) reports a two-year study where rats (35 per sex per dose) were administered weight-normalised boron doses of 0, 5.9, 18 or 59 mg/kg body weight per day in the diet. High-dose animals had coarse hair coats, scaly tails, hunched posture, swollen and desquamated pads of the paws, abnormally long toenails, shrunken scrotum, inflamed eyelids and bloody eye discharge. The haematocrit and haemoglobin levels were significantly lower, the absolute and relative weights of the testes were significantly lower, and relative weights of the brain and thyroid gland were higher than in controls. In animals in the middle- and low-dose groups, no significant effects on general appearance, behaviour, growth, food consumption, haematology, serum chemistry or histopathology were observed.

Long term exposure of humans to boron compounds leads to mild gastrointestinal irritation. In short-term and long-term animal studies and in reproductive studies with rats, testicular atrophy was observed. Boric acid and borates were not mutagenic in various *in vitro* test systems. No increased tumour incidence was observed in long-term carcinogenicity studies in mice and rats.

Acute boron poisoning has been reported after application of dressings, powders or ointments containing borax and boric acid to large areas of abraded skin and following ingestion. Symptoms of boron poisoning include gastrointestinal disturbances, skin eruptions, and central nervous system stimulation followed by depression.

Tests for mutagenicity using bacteria and mammalian cells have been mostly negative. Neither boric acid nor borate induce chromosomal aberrations in mammalian cells.

A survey of Turkish sub-populations compared fertility rates of 1,068 families living in two Turkish villages having drinking-water boron levels of 2–29 mg/L (from nearby geological deposits of calcium borate) with 610 families living in three other villages having drinking-water boron levels of 0.03–0.4 mg/L. Assuming 70 kg body weights and 2 L/day drinking-water consumption, these boron levels would results in an estimated range of daily doses of 0.06–0.8 mg/kg/day. Three generations of families were represented. No significant differences in frequencies of infertility were observed between high and low-exposure village groups. A separate analysis of the same sub-population found no association of higher drinking-water borate concentrations with increased rates of spontaneous abortions, stillbirths, or infant death. A follow-up study of this population reported no significant differences in infertility frequencies between the two populations (ATSDR 2007).

MfE (2011) states:

Boron is considered to be a threshold contaminant, with foetal weight decrease in rats the most sensitive endpoint. A tolerable daily intake of 0.2 mg/kg bw, based on benchmark dose modelling in two studies by the USEPA, is recommended. Inhalation exposure and dermal absorption of boron are expected to be negligible and are not considered relevant here. Dietary intake is expected to be the primary source of background exposure to boron and, in the absence of information specific to New Zealand, it is recommended that TDIs of 0.08 mg/kg bw for children and 0.017 mg/kg bw for adults, based on international data, are used.

The reference dose or RfD (USEPA 2004, 2006, 2009 and 2011) is 0.2 mg/kg/d, based on developmental defects in rats. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 7 mg/L.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes minimal risk levels (MRLs) of:

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

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

EFSA (2013) states that based on the NOAEL of 9.6 mg boron/kg bw/day, derived from a developmental toxicity study in rats, and application of an uncertainty factor of 60, a group ADI of 0.16 mg boron/kg bw/day can be established.

USEPA (2004 and 2008b) state that the data are considered to be inadequate for an assessment of the human carcinogenic potential of boron. No data were located regarding the existence of an association between cancer and boron exposure in humans. Studies available in animals were inadequate to ascertain whether boron causes cancer.

The livestock guideline value is 5 mg/L (ANZECC/ARMCANZ 2000). These guidelines were to be updated in 2012.

### Derivation of Maximum Acceptable Value

Adopting the WHO (2011/2017) guideline value, based on WHO (2009), a MAV for boron could be derived as follows:

10.3 mg/kg body weight per day x 70 kg x 0.4 = 2.4 mg/L

2 L per day x 60

where:

* Benchmark Dose (5 percent) or BMDL05 = 10.3 mg/kg body weight per day, based on developmental toxicity (decreased foetal body weight in rats)
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.4
* uncertainty factor = 60 (10 for interspecies variation and 6 for intraspecies variation)
* average amount of water consumed by an adult = 2 L per day.

WHO (2017) added that it should be noted that because it will be difficult to achieve the guideline value of 2.4 mg/L in some desalinated supplies and in areas with high natural boron levels, local regulatory and health authorities should consider a value in excess of 2.4 mg/L, by assessing exposure from other sources.

The MAV for boron in drinking-water was derived (in-house for the 2000 DWSNZ, and still in the 2008 DWSNZ) as follows:

10.3 mg/kg body weight per day x 70 kg x 0.2 = 1.4 mg/L

2 L per day x 50

where:

* Benchmark Dose (5 percent) = 10.3 mg/kg body weight per day
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.2
* uncertainty factor = 50
* average amount of water consumed by an adult = 2 L per day.

WHO (2003) had derived a guideline value of 0.5 mg/L as follows (but not adopted in any DWSNZ):

9.6 mg/kg body weight per day x 60 kg x 0.1 = 0.5 mg/L (0.6 mg/L using 70 kg bw)

2 L per day x 60

where:

* NOAEL = 9.6 mg/kg body weight per day, for the critical effect, which is developmental toxicity (decreased foetal body weight in rats, affecting 5 percent of the animals)
* average weight of an adult = 60 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* uncertainty factor = 60, 10 for interspecies variation (animals to humans), and 6 for intraspecies variation
* average amount of water consumed by an adult = 2 L per day.

In the 1995 DWSNZ and datasheet the MAV for boron had been 0.3 mg/L, derived as follows:

8.8 mg/kg body weight per day x 70 kg x 0.1 = 0.3 mg/L

2 L per day x 100

where:

* NOAEL = 8.8 mg/kg body weight per day, from a two-year study using dogs
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* uncertainty factor = 100, 10 for interspecies variation (animals to humans), and 10 for intraspecies variation
* average amount of water consumed by an adult = 2 L per day.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for boron is 1 mg/L.

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# Bromate

CAS No. 7758-01-2 (potassium salt); or 15541-45-4 (bromate ion).

### Maximum Acceptable Value (provisional)

Based on health considerations, the concentration of bromate in drinking-water should not exceed 0.01 mg/L. The WHO (2004/2011/2017) guideline value is provisional because of limitations in available analytical and treatment methods and uncertainties in the toxicological data.

Note that “*Bromate in bottled water*” 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*.

The maximum contaminant level or MCL (USEPA 2009/2011) is 0.01 mg/L, and the maximum contaminant level goal (MCLG) is 0 mg/L. The maximum acceptable concentration in Canada is 0.01 mg/L. The levels also apply to bottled water.

The Prescribed Concentration or Value (PCV) for bromate in England and Wales is 0.01 mg/L. See Notes.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of bromate in drinking water should not exceed 0.02 mg/L.

### Sources to drinking-water

#### 1 To source waters

Bromate is not a normal component of water. Potassium bromate, at 30–50 mg/kg, is used primarily as a maturing agent for flour and as a dough conditioner (it breaks down to bromide during baking). It is also used as a laboratory reagent and oxidising agent, in permanent-wave compounds, as a food additive and in explosives. Sodium bromate can be used in conjunction with sodium bromide to extract gold from gold ores. Bromate is also used in cleaning boilers and in the oxidation of sulfur and vat dyes.

#### 2 From treatment processes

Disinfection/oxidation systems producing highly oxidising species, such as ozonation (the main source), are known to produce bromate in bromide-containing waters. Laboratory studies indicate that the rate and extent of bromate formation increases depending on ozone concentration, bromide, pH, temperature, alkalinity, and contact time, and decreases as ammonia increases. Being a disinfection by-product, bromate is regulated in the US, see USEPA (2007).

Bromate has been found after ozonation at concentrations ranging from less than 2 to 293 μg/L, depending on bromide ion concentration, ozone dosage, pH, alkalinity and dissolved organic carbon; it can also be formed in the electrolytic generation of chlorine and hypochlorite from brine with a high level of bromide contamination (WHO 2017).

It is not practicable to remove bromide from raw water, and it is difficult to remove bromate once formed, although GAC filtration has been reported to be effective under certain circumstances. Bromate formation can be minimised by using lower ozone dose, shorter contact time and a lower residual ozone concentration. Operating at lower pH (eg, pH 6.5) followed by raising the pH after ozonation also reduces bromate formation, and addition of ammonia can also be effective. Addition of hydrogen peroxide can increase or decrease bromate formation (WHO 2005).

Some bromate formation may also arise from the chlorination of bromide-containing waters through reaction between hypobromite and hypochlorite. It can also form in the electrolytic generation of chlorine if bromide is present in the brine. Chlorine dioxide does not oxidise bromide.

Although bromate is unlikely to be formed during water chlorination, evidence has been found in US and British studies that water treatment grade sodium hypochlorite solutions may contain bromate as a contaminant. Data collected suggest that bromate concentrations range from <2 to 51 mg/L in the United States. In the United Kingdom, ranges from 50 to 1150 mg/L\* were noted. Other researchers have found bromate concentrations much greater than 10 µg/L in sodium hypochlorite solutions. Since chlorination activity of the solution decreases with time, it may be necessary to use larger quantities of the sodium hypochlorite solution in order to obtain the required level of disinfection. As a result, bromate levels could be high as a result of bromate’s stability during long-term storage (as occurs in smaller municipalities). Taken from Health Canada (1998).

\* This has been copied correctly, but it is more likely they meant 50 to 1150 µg/L.

#### 3 From the distribution system

No known sources.

### Forms and fate in the aquatic environment

Because bromate is a strong oxidant, its chief fate is probably reaction with organic matter resulting in the formation of the bromide ion. Bromate probably does not volatilise and only slightly absorbs to soils and sediment. Bromate is very soluble in water.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme did not find bromate at detectable concentrations in the six zones sampled from supplies treated with ozone (Limit of detection = 0.008 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with bromate at >50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.005 mg/L bromate in the raw water and the treated water.

Bromate has been reported in drinking-water with a variety of source water characteristics after ozonation at concentrations ranging from <0.002 to 0.29 mg/L, depending on bromide ion concentration, ozone dosage, pH, alkalinity and dissolved organic carbon (WHO 2005/2011). In the USA the annual mean bromate concentration in finished surface waters was 0.003 mg/L, with a range of <0.0002 to 0.025 mg/L. The average bromate concentration in ozonated bottled water in Canada was 0.018 mg/L, ranging from 0.004 to 0.037 mg/L.

Eighty-three water utilities in the US reported detecting bromate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.029 mg/L.

### Removal methods

Bromate is difficult to remove once formed. By appropriate control of disinfection conditions, it is possible to achieve bromate concentrations below 0.01 mg/L. Methods for the removal of bromate are still under investigation at present. Those that have shown some promise are membrane filtration (probably needs nanofiltration and RO), and ion-exchange.

Some reduction in bromate production during ozonation can be achieved by keeping the pH as low as possible during treatment.

### Analytical methods

#### Referee method

Ion chromatography. See USEPA method 300.1.

#### Some alternative methods

1. Ion chromatography. See Hautman and Bolyard (1992).

2. ICP/MS (USEPA Method 328.1).

### Health considerations

In a survey of retail bread samples in the United Kingdom in 1989, potassium bromate was found in all six unwrapped breads analysed, with a median concentration of 35 μg/kg (range 17 to 317 μg/kg), and in seven of 22 wrapped breads, with a median concentration of <12 μg/kg (range <12 to 238 μg/kg). In a second survey of the same brands in 1992, all samples contained less than the detection limit of 12 μg/kg flour. Most bromate is converted to bromide in the baking process.

Rats absorb bromate rapidly from their gastrointestinal tract. Although bromate was not subsequently detected in tissue, bromide concentrations were increased significantly in plasma, red blood cells, pancreas, kidney, stomach and small intestine.

Exposure to potassium bromate may occur during its production and use as a dough conditioner and food additive. Most cases of human poisoning from bromate are due to accidental or intentional ingestion of home permanent-wave solutions, which can contain 2–10 percent bromate. Toxic effects include nausea, abdominal pain and diarrhoea, central nervous system depression, and pulmonary oedema, most of which are reversible. Irreversible effects include kidney failure and deafness.

In rats exposed to bromate in drinking-water for 15 months, adverse effects included inhibited body-weight gain and changes to the kidney. Kidney tumours have been reported in studies using male and female rats, but not with female mice. There is evidence that tumours occur only after a minimum total cumulative dose has been exceeded.

Bromate exhibited mutagenic activity in tests using bacteria, and caused chromosomal aberrations in cultured mammalian cells. Some evidence of DNA damage has been reported in rats given potassium bromate.

The International Agency for Research on Cancer (IARC) considers that there is sufficient evidence in experimental animals for the carcinogenicity of potassium bromate, and has concluded that potassium bromate is possibly carcinogenic to humans (Group 2B).

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

USEPA (2001) states that bromate should be evaluated as a likely human carcinogen by the oral route of exposure. Although no epidemiological studies or studies of long-term human exposure to bromate are available, bromate is carcinogenic to male and female rats following exposure in drinking water. Given the limited data on possible mechanisms of carcinogenic action for bromate, it is a reasonable assumption that the production of tumours in rats occurs by a mode of action that is relevant to humans. With the lack of human data and the uncertainty surrounding the mode of action, the human relevance of the rat data relies on the assumption that the rat data are relevant to humans. The USEPA (2011) quotes a health advisory of 0.005 mg/L for bromide, representing a 10-4 cancer risk.

In the Stage 1 D/DBPR, USEPA established an MCLG of zero for bromate based on a weight of evidence evaluation of both the cancer and non-cancer effects indicating that bromate is a “probable or likely human carcinogen”. The MCLG was based on an increase in kidney and thyroid tumours in several rat studies. Insufficient evidence exists regarding the mode of carcinogenic action of bromate; thus, the low-dose extrapolation approach was used because it is more protective of public health. A USEPA IRIS assessment established an RfD of 0.004 mg/kg/day for bromate in 2001 based on a NOAEL of 1.5 mg/kg/day for potassium bromate (equivalent to 1.1 mg/kg/day bromate) for renal effects and the application of an uncertainty factor of 300. The RfD value did not change in the Stage 2 D/DBPR due to the lack of significant new health effects data for systemic effects and USEPA did not revise the MCLG at that time (USEPA 2003c, 2006a).

The reference dose or RfD (USEPA 2001, 2006, 2009 and 2011) is 0.004 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.14 mg/L.

### Derivation of Maximum Acceptable Value

WHO (2005/2011/2017) states:

The upper-bound estimate of cancer potency for bromate is 0.19 per mg/kg of body weight per day, based on low-dose linear extrapolation (a one-stage Weibull time-to-tumour model was applied to the incidence of mesotheliomas, renal tubule tumours and thyroid follicular tumours in male rats given potassium bromate in drinking-water, using the 12-, 26-, 52- and 77-week interim kill data). A health-based value of 0.002 mg/L is associated with the upper-bound excess cancer risk of 10-5. A similar conclusion may be reached through several other methods of extrapolation, leading to values in the range 0.002–0.006 mg/L.

The practical quantitation limit (PQL) of 0.001 mg/L may be difficult to achieve in many laboratories; a more attainable PQL is around 0.005 mg/L. In addition, it is now considered that 0.01 mg/L is a technically achievable value for the removal of bromate from drinking-water. The health-based value of 0.002 mg/L should therefore be raised to 0.01 mg/L, on the basis of analytical and technological feasibility. A provisional guideline value of 0.01 mg/L is therefore recommended. This value is associated with an upper-bound excess lifetime cancer risk of 10-4.

The MAV of 0.025 mg/L in the 1995 and 2000 DWSNZ had been derived as follows:

To estimate cancer risks, the linearised multi-stage model was applied to a study on the incidence of renal tumours in male rats given potassium bromate in drinking-water, although it was noted that if the mechanism of tumour induction is determined to be oxidative damage in the kidney, the application of the low-dose cancer risk model may not be appropriate. The concentration in drinking-water associated with an excess lifetime cancer risk of 10-5 is 0.003 mg/L. Because of limitations in available analytical and treatment methods, a provisional MAV of 0.025 mg/L is recommended. This value is associated with a lifetime excess cancer risk of 7 per one hundred thousand (7 x 10-5).

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# Bromide

### Maximum Acceptable Value

WHO (2011/2017) states that bromide occurs in drinking-water at concentrations well below those of health concern.

### Sources to drinking-water

#### 1 To source waters

Bromide commonly exists, along with chloride, as salts with sodium, potassium and other cations. Bromide concentrations in seawater range from 65 mg/L to well over 80 mg/L, in fresh water from trace amounts to about 0.5 mg/L and in desalinated waters up to 1 mg/L.

Geothermal waters in the Kawerau area can contain 4.5 mg/L Br (GNS 2015).

Analogous to chloride, bromide forms the strong hydrobromic acid (HBr), and the weaker hypobromous (HOBr), bromous (HBrO2) and bromic (HBrO3) oxyacids. Bromide commonly exists as salts with sodium, potassium and other cations, which are usually very soluble in water. Naturally occurring bromine and bromine compounds consists of 50.57 percent 79Br and 49.43 percent 81Br isotopes.

Freshwater concentrations range typically from a trace to about 0.5 mg/L.

#### 2 From treatment processes

Traces can be added to the water when electrolytically generating chlorine from salt, the quantity depending on the grade of salt being used, and to a lesser extent, the dose rate.

#### 3 From the distribution system

No known sources.

### Forms and fate in the aquatic environment

Bromide is stable in drinking-water.

### Typical concentrations in drinking-water

Generally less than 0.5 mg/L; desalinated water may contain up to 1 mg/L.

152 water utilities in the US reported detecting bromide in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 4 mg/L.

Eight water utilities in the US reported detecting bromide-82 in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 1.85 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found <0.05 to 0.05 mg/L bromide in the raw water, and <0.05 mg/L in the treated water.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Bromide was once used as an anticonvulsant and sedative at doses as high as 6 g/day. Clinical symptoms of bromide intoxication have been reported from its medicinal uses. Large doses of bromide cause nausea and vomiting, abdominal pain, coma and paralysis. Doses of bromide giving plasma levels of 12 mmol/L (96 mg/L of plasma) produce bromism, and plasma levels greater than 40 mmoL/l (320 mg/L of plasma) are sometimes fatal. The signs and symptoms relate to the nervous system, skin, glandular secretions and gastrointestinal tract.

The typical daily dietary intake of bromide in the US is 2 to 8 mg from grains, nuts and fish; the average bromide intake in the UK is reported as 8.4 mg/day. Limited findings suggest that bromide may be nutritionally beneficial; for example, insomnia exhibited by some haemodialysis patients has been associated with bromide deficiency.

Inorganic bromide was evaluated by the Joint FAO/WHO Meeting on Pesticide Residues (JMPR) in 1966, which recommended an acceptable daily intake (ADI) for humans of 0 to 1.0 mg/kg bw, based on a minimum pharmacologically effective dosage in humans of about 900 mg of KBr, equivalent to 600 mg of bromide ion. Although some effects were observed, no adverse effects were observed in the 12‑week humans studies at up to 9 mg/kg bw per day. The ADI of 0 to 1 mg/kg bw set in 1966 was reaffirmed with new data in 1988.

A no-observed-adverse-effect level (NOAEL) for sodium bromide of 300 mg/kg diet (equivalent to 240 mg/kg diet as bromide; 12 mg/kg body weight per day) for effects on the thyroid was determined; also a conservative no-observed-effect level (NOEL) (for marginal effect within normal limits of electroencephalograms in females at 9 mg/kg body weight per day) of 4 mg/kg body weight per day (Sangster et al 1986) suggests an ADI of 0.4 mg/kg body weight (EMEA 1997), including a safety factor of 10 for population diversity (quoted in WHO 2009).

The results of human studies suggest a conservative no-observed-effect level (NOEL) (for marginal effect within normal limits of electroencephalograms in females) of 4 mg/kg body weight per day, giving an ADI of 0–0.4 mg/kg body weight, including a safety factor of 10 for population diversity (WHO 2017).

The upper limit of the ADI of 0–0.4 mg/kg body weight yields an acceptable total daily intake of 24 mg/person for a 60 kg person. Assuming a relative source contribution of 50 percent, the drinking-water value for a 60 kg adult consuming 2 litres/day would be up to 6 mg/L; for a 10 kg child consuming 1 litre/day, the value would be up to 2 mg/L. However, the dietary bromide contribution for a 10 kg child would probably be less than that for an adult. These are reasonably conservative values, and they are unlikely to be encountered in drinking-water supplies (WHO 2017).

The Acceptable Daily Intake (ADI) adopted in Australia for bromide is 1 mg/kg body weight, with a NOEL of 0.22 mg/kg bw.

The main health issues arising from bromide in drinking-water relate to the production of disinfection by-products (DBPs), particularly if the drinking-water has been prepared by desalinating seawater.

Ozone oxidises bromide to produce hypohalous acids, which react with precursors to form brominated THMs. A range of other DBPs, including aldehydes and carboxylic acids, may also be formed. Of particular concern is bromate, formed by oxidation of bromide.

Trihalomethanes (THMs) are formed in drinking-water primarily as a result of chlorination of organic matter present naturally in raw water supplies. The rate and degree of THM formation increase as a function of the chlorine and humic acid concentration, temperature, pH and bromide ion concentration.

As chlorine dioxide does not oxidise bromide (in the absence of sunlight), water treatment with chlorine dioxide will not form bromoform or bromate.

See individual datasheets for further information.

### Derivation of Maximum Acceptable Value

No MAV.

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# Bromine

### Maximum Acceptable Value

There is no MAV for bromine in the DWSNZ. The WHO 2004/2011 Guidelines did not include a GV for bromine.

### Sources to drinking-water

#### 1 To source waters

Bromine is used as a general disinfectant and sanitiser for cooling towers and other indoor applications such as in commercial establishments, hospitals and households.

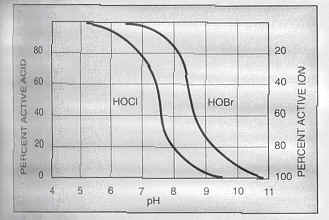
#### 2 From treatment processes

Bromine was first registered for use in the US in 1976 for use in treating potable water through a polybrominated ion exchange resin on naval vessels, with a food tolerance of 1 mg/L residual bromine. Weight-for weight, bromine has about half the disinfecting power of chlorine. Chlorine dissolves in water to form hypochlorous acid; bromine dissolves in water to form hypobromous acid, the disinfectant. And like chlorine, bromine is ineffective at inactivating most protozoa.

Bromine is sometimes used as a disinfectant in swimming pools. NZS 5826 states that the lowest bromine content in swimming pools and spa pools should be 4 mg/L, and the highest 10 mg/L. Bromine is used to disinfect swimming pool water in France where the standard is 0.7 mg/L. Concentrations of 0.5 mg/L may lead to irritations on mucous membranes, eyes and odour nuisance.

Today bromination is mainly achieved using bromochlorodimethylhydantoin (BCDMH) – see datasheet. Another process involves adding bromide, some of which can be oxidised by chlorine or ozone to form hypobromous acid.

Hypobromous acid is slightly less effective than hypochlorous acid in killing microorganisms. Both molecules dissociate into the ineffective ion as the pH increases:



#### 3 From the distribution system

No known sources.

### Forms and fate in the aquatic environment

Bromide is the predominant end-product in the environment. Bromine water solubility is about 3.5 percent; like chlorine, hydrogen bromide and hypobromous acid are formed:

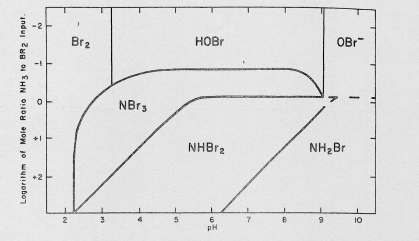
Br2 + H2O ↔ HOBr + HBr (i)

HOBr ↔ H+ + OBr- (ii)

### Typical concentrations in drinking-water

If drinking-water is disinfected using bromine, the residual bromine concentration is generally less than 0.2–0.5 mg/L. Free bromine (Br2) is not really used in drinking water treatment because it too quickly with organic substances, and no residue will remain. Bromine also gives drinking water a medicine-like taste. Bromine should only be used in emergency cases.

Just as chlorine can form chloramines, bromine can form bromamines; however, bromamines are better disinfectants than chloramines, in fact, monobromamine is as effective as hypobromous acid. Dibromamine (also called dibromoamine and bromimide), CAS No. 14519-03-0, is the most common form when the pH value is between 7 and 8.5. Dibromoamine is almost as effective as free chlorine in killing microorganisms. Dibromoamine is very active and usually dissociates quickly into bromide ions. Because of this, no bromine remains in the water. The following chart shows the different forms of bromine at various pH values and various concentrations of ammonia. Both charts were copied (in 2013) from <http://www.lenntech.com/processes/disinfection/chemical/disinfectants-bromine.htm>.



Both chlorination and bromination can give rise to a range of disinfection by-products.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Bromine reacts with other substances in the body to form bromide (qv). Due to the high reactivity of bromine, the main health concerns are related to the end-product, bromide. Bromate (qv) and other brominated disinfection by-products can be formed as well.

Toxicity studies in humans or animals for bromine per se via ingestion are very limited; this is mostly due to the corrosiveness and high reactivity of bromine; it quickly forms bromide in living tissues. Human studies with sodium bromide have allowed derivation of an ADI for bromide of 0.4 mg/kg bw based on the most sensitive toxicological endpoint relating to changes within electroencephalograms (WHO 2018).

### Derivation of Maximum Acceptable Value

No MAV.

A drinking-water guideline value has not been proposed for bromide in the WHO GDWQ as it occurs in drinking-water at concentrations well below those of health concern. However, the GDWQ includes a health-based value of 6 mg/L for adults and 2 mg/L for children (WHO 2017).

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# Cadmium

CAS No. 7440-43-9 (metal).

### Maximum Acceptable Value

Based on health considerations, the concentration of cadmium in drinking-water should not exceed 0.004 mg/L (4 g/L).

The maximum contaminant level or MCL (USEPA 2009/2011) is 0.005 mg/L. The USEPA also established a lifetime health advisory of 0.005 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 2 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 Prescribed Concentration or Value (PCV) for cadmium in England and Wales is 0.005 mg/L. See Notes.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of cadmium in drinking water should not exceed 0.002 mg/L.

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

Cadmium 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

Cadmium can enter water from the weathering of rocks and minerals and run-off from soils. The only naturally-occurring cadmium compound of significance, the sulphide greenockite, CdS, which is fairly rare, is almost always associated with the polymetallic sulphide ores of zinc, lead and copper.

Longhurst (2006) reports that the cadmium concentration found in Hawkes Bay soils to 75 mm depth were considerably lower with a mean (and median) of 0.30 mg/kg (range 0.04–0.63) compared with the mean of 0.44 mg/kg (range 0.4–1.53) found for pastoral soils throughout New Zealand.

The concentration of cadmium in seawater is about 0.0005 mg/L.

NZFSA reported cadmium levels of 0.02 to 0.06 mg/kg in wheat in their 2010 season 1 food residues surveillance programme.

Cadmium has a wide range of sources and may enter water in industrial and domestic discharges or from street and agricultural run-off. Domestic discharges generally contain high levels of cadmium. Its principal industrial uses include electroplating other metals or alloys for corrosion protection, in solders and in amalgam used in dentistry. It is also used in the manufacture of pigments, nickel-cadmium storage batteries (major use), electronic equipment, lubricants, photography supplies, glass, ceramics, biocides and as a stabiliser in plastics.

It is likely to be present in waste discharged from fertiliser factories using phosphate ores containing cadmium. In agriculture, farm run-off containing these fertilisers is an important source of diffuse pollution by cadmium. The average cadmium content of superphosphate fertiliser in New Zealand from 2001 to 2005 was 180 mg/kg of P (ESR 2014). Cadmium applied to the soil by these means may leach into groundwater or enter surface water with run off. In the latter case the cadmium will be associated with particulate matter, and will probably remain so.

Exhaust emission and tyre wear contribute a significant amount of cadmium to street run-off.

MAF (2011) developed soil guidelines; MAF (2011a) proposed a strategy for long-term risk management; MPI (2012) worked towards risk-based soil guideline values. MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; cadmium levels range from 0.3 to 1300 mg/kg depending on land use. NZWWA (2003) includes the statement that setting a guideline level of 1 mg Cd/kg soil should also protect the microbial population of the soil, and should not result in significant leaching of Cd from the soil into groundwater.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

Cadmium may enter drinking-water from the dissolution of galvanised pipes in which it is an impurity when associated with the zinc. It may also be present as a result of cadmium-containing solders in fittings, water heaters, water coolers and taps.

### Forms and fate in the environment

In fresh waters, cadmium exists principally as the free Cd(II) ion, cadmium chloride and cadmium carbonate. Adsorption is probably the most important process for removal of cadmium from the water column. Exchange of cadmium for calcium ions in the lattice structure of carbonate minerals can remove cadmium from solution. In natural waters, co-precipitation with hydrous iron, aluminium and manganese oxides occurs. Alternatively, in waters of high organic content, adsorption of cadmium to humic substances and other organic complexing agents can be significant.

### Typical concentrations in drinking-water

Cadmium was routinely measured in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. Typical concentrations of cadmium in New Zealand drinking-water supplies are <0.005 mg/L (<5 g/L).

The P2 Chemical Determinand Identification Programme, sampled from 898 zones, found cadmium concentrations to range from “not detectable” (nd) to 0.26 mg/L, with the median concentration being “nd” (limit of detection = 0.0005 mg/L). The Priority 2 Identification Programme found 30 distribution zones supplying drinking-water to a total of 64,750 people with cadmium at greater than the MAV (ESR 2001).

A NAQUADAT survey of 3,067 samples of raw water taken across Canada found only four samples containing cadmium at concentrations higher than the detection limit (0.01 mg/L), the highest concentration found being 0.061 mg/L (Health Canada 1986).

1,247 water utilities in the US reported detecting cadmium (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.012 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found <0.000053 mg/L cadmium in the raw water and treated water.

Due to the concern about cadmium levels in superphosphate, ESR (2014) reported to MoH on the cadmium levels in New Zealand groundwaters. A lot of the data came from MAF (2008) and Daughney and Randall (2009). Key findings were:

1. The majority of median concentrations reported in the national groundwater quality indicators report are below the limit of detection of the test methods used.

2. The cadmium concentration in 87 percent of samples analysed by regional councils is below the limit of detection.

3. Of 1,283 results in the combined regional council dataset, only three exceeded 50 percent of the cadmium MAV of 0.004 mg/L. No cadmium concentrations exceeded the MAV.

4. Taranaki and Waikato were the only regions in which data had been collected that allowed testing for trends in the cadmium concentration. No trends were found at the 95 percent confidence level.

5. A qualitative assessment of geographical patterns in cadmium concentrations showed that the median concentrations were the same in all regions and below the limit of detection. The 90th percentile values of groundwater cadmium concentrations in Southland and Canterbury were also below detection, but the 90th percentile concentrations were above the limit of detection in Taranaki, Waikato and the Bay of Plenty, regions where the 90th percentile of soil cadmium concentrations (all land uses) exceeds 1 mg/kg.

The data available to this study show that, generally, groundwaters do not presently contain cadmium concentrations that would constitute a risk to public health if these waters were used for water supply purposes.

### Removal methods

Lime-softening achieves good removal of cadmium, provided it is applied to hard waters; 0.003 mg/L should be achievable.

Ion exchange resins can remove cadmium, provided the resins are not overwhelmed by other cations such as calcium and magnesium. This form of treatment may be useful for the removal of heavy metals that have entered the water post-treatment.

Some adsorption of cadmium on to PAC, GAC and oxides of Mn(IV), Fe(III) and Al(III) has been reported.

Chemical coagulation with aluminium and iron salts is limited as a viable option for the removal of soluble cadmium. The effectiveness of removal is dependent on the pH at which the process is carried out. In both cases, the effectiveness increases with increasing pH.

In situations where the dissolution of poor-quality zinc from galvanised pipes is a source of cadmium, adjustment of the water chemistry to reduce its corrosiveness will minimise cadmium concentrations.

### Analytical methods

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Inductively Coupled Plasma (ICP) Method (APHA 3120).

2. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

### Health considerations

Food is the main source of cadmium intake for individuals who are not occupationally exposed. A joint FAO/WHO expert committee has estimated a provisional tolerable weekly intake of cadmium for an adult to be from 0.4 to 0.5 mg. Because it would be difficult to reduce cadmium intake in food, intake from water should be as low as possible. Daily consumption of water containing the maximum acceptable concentration of cadmium would result in the ingestion of about 12 percent of the above estimated tolerable intake (Health Canada 1986).

Absorption of cadmium compounds is dependent on the solubility of the compounds. Cadmium accumulates primarily in the kidneys and has a long biological half-life in humans of about 10 to 35 years.

The kidney is the main target organ for cadmium toxicity. In humans long-term exposure can cause kidney dysfunction leading to the excretion of protein in the urine. This may occur in about 10 percent of the population if the amount of cadmium exceeds 200 mg/kg. Other effects may include the formation of kidney stones and softening of the bones (osteomalacia).

Itai-Itai disease has been reported in Japan among people exposed to cadmium via food and drinking-water. Symptoms were similar to osteomalacia accompanied by kidney dysfunction.

The reference dose or RfD (USEPA 2009/2011) is 0.0005 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.02 mg/L.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes minimal risk levels (MRLs) of:

0.0005 mg/kg/day for intermediate-duration oral exposure (15–364 days)

0.0001 mg/kg/day for chronic-duration oral exposure (>364 days).

Evidence concerning the mutagenicity of cadmium is unclear with many tests reporting negative results although some report gene mutation and chromosome abnormalities in mammalian cells. However the positive results are reported as being weak and seen only at high concentrations.

There is evidence for the carcinogenicity of cadmium by the inhalation route, and the International Agency for Research on Cancer has classified cadmium and cadmium compounds in Group 2A (probably carcinogenic to humans). However, there is no evidence of carcinogenicity by the oral route. Food is the main source of daily exposure to cadmium. The daily oral intake is 0.01–0.035 mg. Smoking is a significant additional source of cadmium exposure. IARC (2009) states that “The Working Group reaffirmed the classification of beryllium and its compounds, cadmium and its compounds, chromium (VI) compounds, and nickel compounds as “carcinogenic to humans” (Group 1). Studies involved complex occupational exposures to a metal and its compounds, making it impossible to separately assess their carcinogenicity.”

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

MfE (2011) states:

Cadmium is considered to be a threshold contaminant, with kidney damage as a result of long-term exposure considered the most sensitive endpoint. Unlike for most other substances, toxicokinetic modelling has typically been used to estimate tolerable intakes. Given the long-term effects of cadmium, it is more appropriate to express intakes as monthly intakes. The Joint Expert Committee on Food Additives (JECFA) of the Food and Agriculture Organization (FAO) and the World Health Organization (WHO) recommend a provisional tolerable monthly intake (PTWI) of 25 µg/kg bw and it is recommended that this value is used for the derivation of soil contaminant standards. Dermal absorption is expected to be negligible, although a dermal absorption factor of 0.0012 could be used. Dietary intake is the primary source of background exposure to cadmium and was estimated to be 12.5 µg /kg bw/month for a child (aged  
1–3 years, 13 kg) and 7.9 µg/kg bw/month for an adult.

Cadmium chloride and cadmium sulfate are two 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 livestock guideline value is 0.01 mg/L (ANZECC/ARMCANZ 2000). These guidelines were to have been updated in 2012.

### Derivation of Maximum Acceptable Value

As there is no evidence of carcinogenicity by the oral route and no evidence for the genotoxicity of cadmium, a provisional tolerable monthly intake (PTMI) approach has been used for the derivation of the MAV in WHO (2017). Assuming an absorption rate for dietary cadmium of 5 percent and a daily excretion rate of 0.005 percent of body burden, the Joint FAO/WHO expert Committee on Food Additives concluded that, if levels of cadmium in the renal cortex are not to exceed 50 mg/kg, a total intake of cadmium should not exceed 0.001 mg/kg body weight per day. This total daily intake has been used to derive the MAV.

Although new information indicates that a proportion of the general population may be at increased risk for tubular dysfunction when exposed at the current PTMI, the risk estimates that can be made at present are imprecise. It is recognised that the margin between the PTMI and the actual monthly intake of cadmium by the general population is small and that this margin may be even smaller in smokers.

The MAV for cadmium in drinking-water was derived as follows:

0.001 mg/kg body weight per day x 70 kg x 0.1 = 0.0035 mg/L (rounded to 0.004 mg/L)

2 L per day

where:

* PTMI = 0.025 mg/kg, so the tolerable daily intake = 0.001 mg/kg body weight per day, based on the relationship between β2-microglobulin excretion in urine and cadmium excretion in urine for individuals who are 50 years of age and older
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* average amount of water consumed by an adult = 2 L per day.

Previous WHO GVs had been based on a PTWI of 0.007 mg/kg bw.

The MAV for cadmium in the 1995 and 2000 DWSNZ was 0.003 mg/L because the result of the calculation had been rounded down.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for cadmium is 0.004 mg/L.

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# Caesium

### Maximum Acceptable Value

There is no MAV in the DWSNZ for caesium, and it is not mentioned in the WHO Guidelines. The caesium level in drinking-water is controlled via the radiochemical MAVs.

### Sources to drinking-water

#### 1 To source waters

Cesium (US spelling) is a naturally-occurring element found in rocks, soil, and dust at low concentrations. Granites contain an average cesium concentration of about one part of cesium in a million parts of granite (ppm) and sedimentary rocks contain about 4 ppm. The most important source of commercial cesium is a mineral known as pollucite (the largest deposits of pollucite are located in Manitoba, Canada and account for about two-thirds of the world’s known supply), which usually contains about 5 to 32 percent cesium oxide (Cs2O).

The concentration of caesium in seawater is about 0.001 mg/L.

There are relatively few commercial uses for cesium metal and its compounds. Sometimes cesium is used as a getter for residual gas impurities in vacuum tubes and as a coating in tungsten filaments or cathodes of the tubes. Crystalline cesium iodide and cesium fluoride are used in scintillation counters, which convert energy from ionising radiation into pulses of visible light for radiation detection and spectroscopy. Cesium is also used in highly accurate atomic clocks.

Natural caesium is present in the environment in only one stable form, as the isotope 133Cs. Caesium-134 is a radioactive chemical found in the environment due to fallout from nuclear power station accidents and nuclear weapons testing.

The two most important radioactive isotopes of cesium are 134Cs and 137Cs. Radioactive isotopes are constantly decaying or changing into different isotopes by giving off radiation. Each atom of 134Cs changes into either xenon (134Xe) or barium (134Ba), neither of which is radioactive, while each atom of 137Cs decays to 137Ba, which is also not radioactive. As 134Cs and 137Cs decay, beta particles and gamma radiation are given off. The half-life is the time it takes for half of that cesium isotope to give off its radiation and change into a different element. The half-life of 134Cs is about two years and the half-life of 137Cs is about 30 years.

Naturally-occurring cesium occurs in the environment mostly from the erosion and weathering of rocks and minerals. The mining and milling of certain ores can also release cesium to the air, water, and soil. Radioactive cesium is released to the environment during the normal operation of nuclear power plants, explosion of nuclear weapons, and accidents involving nuclear power plants or nuclear powered satellites or submarines. Cesium has also been detected in the fly ash of coal burning power plants.

Geothermal waters in the Taupo Volcanic Zone can contain 0.5 to 3 mg/L Cs (GNS 2015).

Studies from five distinct geochemical areas of the semi-arid endorheic region of the Famatina Range (La Rioja, Argentina) have shown some areas contain high levels of 133Cs in natural waters and sediment. The cesium concentration in fresh water systems of this region ranged from 0.58 to 3.69 μg/L.

#### 2 From treatment processes

No known sources.

### Forms and fate in the environment

Cesium binds strongly to most soils and does not travel far below the surface of the soil. Consequently, cesium is not readily available for uptake by vegetation through roots. However, radiocesium can enter plants upon falling on to the surface of leaves. Cesium salts and most cesium compounds are generally very water soluble.

Cesium has very low mobility in soil surfaces. Clay minerals and soils rich in exchangeable potassium adsorb cesium by binding the cations to interlayer positions of the clay particles. The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays, which can result in limited uptake of cesium by grass and plant material. Continental dust and soil erosion are the main emission sources of naturally-occurring cesium that is present in the environment.

### Typical concentrations in drinking-water

The amount of cesium in drinking water is ordinarily about 0.001 mg/L.

One water utility in the US reported detecting cesium-134 in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, the single sample containing 10 mg/L.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

On average, a person swallows about 10 µg of stable caesium per day in food and water, and breathes about 0.025 µg per day. Chemically, caesium behaves in a similar manner to potassium.

Radioactive cesium has been detected in surface water and in many types of food. This includes breast milk and pasteurised milk. The amount of radioactive cesium in food and milk is highly dependent upon several factors. The most important factor is whether or not there has been recent fallout from a nuclear explosion such as a weapons test or an accident that has occurred at a nuclear power plant. However, atmospheric testing of nuclear weapons was halted many years ago, and there have only been two major reactor accidents at nuclear plants where radiocesium was released in significant amounts. The two accidents occurred in Windscale, England in 1957 and Chernobyl, Russia in 1986. Cesium only contributed a small fraction of the total radioactivity released following these events. The radiological impacts in Europe from 137Cs and 134Cs released from the Chernobyl accident, however, were great. These included environmental dispersion of radiocesium and uptake in reindeer, caribou, and livestock. Furthermore, the consequences of external exposure to gamma radiation and beta particles are not unique to 137Cs and 134Cs, but are very similar for all gamma and beta emitting radionuclides.

Due to its low relative abundance, limited use in industry, and relatively low level of toxicity observed in animal studies, exposure to stable cesium is not considered to be a significant public health concern.

No acute-, intermediate-, or chronic-duration oral MRLs were derived for stable cesium due to the lack of suitable human or animal data regarding health effects following oral exposure to stable or radioactive cesium. Reports of health effects following oral exposure to stable cesium are limited. Daily ingestion of approximately 68 mg Cs/kg (as cesium chloride) for up to 36 days resulted in decreased appetite, nausea, and diarrhoea, as well as neurological signs within 15 minutes following ingestion.

The USEPA has not derived reference concentrations (RfCs) or reference doses (RfDs) for stable or radioactive cesium.

### Derivation of Maximum Acceptable Value

No MAV.

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# Carbon disulfide

CAS No. 75-15-0. Also spelt carbon disulphide, and in early days called carbon bisulphide.

For further information, also refer to the Sodium Tetrathiocarbonate datasheet in the Pesticides section.

### Maximum Acceptable Value

There is no MAV in the DWSNZ for carbon disulfide, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1 To source waters

Carbon disulfide is an industrial chemical used in the manufacture of rayon fibres (major use), cellophane, pesticides, in the production of cellulose and rubber chemicals, as a solvent for cleaning and extraction; as an extractant for olive oil, and in the production of adhesives. Carbon disulfide is a natural product of anaerobic biodegradation. In nature, minute amounts occur in coal tar and in crude petroleum. Other sources include animal waste, in particular pig urine and faeces, fish processing, plastic and refuse combustion, synthetic fibre and starch manufacture, natural gas and volcanoes.

Carbon disulphide is produced naturally by soil and sediment micro-organisms, vegetation, forest and grass fires and volcanic activity. The microbial reduction of sulphate in soil produces fluxes of carbon disulphide. The annual global emission from this source has been estimated to be 9 x 105 tonnes pa (5.29–5.63 times the total volume used in the EU in 2001). Other natural sources include volcanic emissions, estimated to be 2 x 104 tonnes pa, and marshlands, estimated emissions 1 x 105 tonnes pa. Worldwide, at least 40 percent, and possibly, as much as 80 percent of the releases are a result of natural or biogenic activity (EC 2002).

Sodium tetrathiocarbonate (see datasheet in pesticides section) 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)). Sodium tetrathiocarbonate breaks down to carbon disulphide which is effectively the pesticide; also see datasheets for dazomet, mancozeb, metam sodium, metiram and propineb.

#### 2 From treatment processes

No known sources.

#### 3 Forms and fate in the environment

Carbon disulfide is not expected to be removed significantly from the aquatic phase through adsorption. The low Koc value (54), calculated from water solubility data, indicates high soil mobility, but it probably will be less mobile in soils of high organic content. Carbon disulfide released to soils in spills should rapidly volatilise to the atmosphere due to its Henry’s Law Constant of 1.42 x 103 Pa-m3 mol-1 (1.44 x 10-2 atm‑m3 mol-1) at 24°C, but a portion of the compound remaining on soil surfaces could be available for transport into groundwater since it does not have much affinity for soil particles. Carbon disulfide is rapidly metabolised by organisms and does not bioconcentrate or biomagnify.

Carbon disulfide is stable to hydrolysis in the pH region of environmental concern (pH 4 to 10). The volatilisation half-life from a saturated water solution has been estimated to be 11 minutes. The compound apparently does not undergo biodegradation at rates that are competitive with its volatilisation from surface waters (ATSDR 1996).

In the aquatic environment photolysis is not considered to be a significant loss mechanism but carbon disulphide can be hydrolysed to carbon dioxide and hydrogen sulphide in alkaline (pH 9) solutions with a half-life of 1.1 years. Carbon disulphide has been shown to be readily biodegradable and is not expected to sorb to organic carbon based on a low organic carbon water partition coefficient (log Koc) of 1.79. The octanol-water partition coefficient (log Kow) is 1.94–2.14. EC (2002).

NPIC (1994) quotes for carbon disulfide a soil half-life of 1.5 days, water solubility of 2,300 mg/L and a sorption coefficient (soil Koc) of 60. This resulted in a pesticide movement to groundwater rating of very low.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Inhalation of carbon disulfide is the commonest route of absorption of carbon disulphide in man, and the main health risk (see IPCS 1993). The oral reference dose (RfD) is 0.1 mg/kg/d based on foetal toxicity/malformations (USEPA 1990). No reports are available to indicate any carcinogenic or mutagenic effects of carbon disulfide.

A minimal oral risk level (MRL: an estimate of daily human exposure to a dose of a chemical that is likely to be without an appreciable risk of adverse non-cancerous effects over a specified duration of exposure) of 0.01 mg/kg/day was derived for acute exposure to carbon disulfide. This MRL was derived based on the inhibition of enzyme activities, specifically decreases in the activities of several hepatic microsomal cytochrome P-450-dependent drug-metabolising enzymes and cytochrome P‑450 content. A LOAEL of 3 mg/kg/day was established for this effect. Also, the effect was minimal since the inhibition of enzyme activities was selective and reversible. This dose was divided by an uncertainty factor of 300 (three for use of a minimal LOAEL, 10 for extrapolation from animals to humans, and 10 for interhuman variability) to yield the calculated MRL of 0.01 mg/kg/day.

Carbon disulphide has been shown to be capable of penetrating the placenta and has been detected in the urine of infants of exposed mothers and in the breast milk (EC 2002).

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/index.html>) quotes a minimal risk level (MRL) of 0.01 mg/kg/day for acute-duration oral exposure (1–14 days) for carbon disulfide.

Carbon disulfide 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 2002).

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for carbon disulfide is 0.7 mg/L.

### Derivation of Maximum Acceptable Value

No MAV.

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WHO. 2002. *Carbon Disulfide. Concise International Chemical Assessment Document (CICAD) 46*. International Programme on Chemical Safety (IPCS). 49 pp. See: <http://www.who.int/ipcs/publications/cicad/cicads_alphabetical/en/index.html> and select carbon disulfide, or go to http://www.inchem.org/documents/cicads/cicads/cicad46.htm.

# Chloramines

The commonest and simplest chloramines (the inorganic chloramines) are:

* Monochloramine
* Dichloramine
* Trichloramine

See individual entries.

The maximum acceptable concentration in Canada is 3 mg/L for total chloramines based on health effects associated with monochloramine and analytical achievability. See http://www.hc-sc.gc.ca/ewh-semt/water-eau/index-eng.php.

Total chloramines represent the difference between the total chlorine concentration and the FAC:

Total chlorine = FAC + chloramines

Total chloramines comprise the organic and inorganic chloramines.

# Chlorate

CAS No. 7775-09-9 (sodium salt), or 3811-04-9 (potassium salt).

### Maximum Acceptable Value (Provisional)

Based on health considerations, the concentration of chlorate in drinking-water should not exceed 0.8 mg/L.

WHO (2017) stated that the guideline value for chlorate is designated as provisional because use of aged hypochlorite or of chlorine dioxide as disinfectants may result in the chlorite and chlorate guideline values being exceeded, and difficulties in meeting the guideline values must never be a reason for compromising adequate disinfection.

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

The maximum acceptable concentration in Canada is 1 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 (Health Canada 2008).

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that data are insufficient to set a guideline value for chlorate in drinking water).

Refer to the chlorite and chlorine dioxide datasheets for further information.

### Sources to drinking-water

#### 1 To source waters

Chlorate does not occur naturally but may enter source waters in industrial discharge from industries in which it is used or from agricultural practices.

Sodium chlorate has been used as a herbicide, fungicide and defoliant, and in the manufacture of dyes, matches and explosives. When used as a herbicide it contains fire retardants, up to about 50 percent of the weight. Fire retardants used were sodium metaborate or ammonium phosphates.

In 2005, the use of sodium chlorate as a pesticide/fumigant was banned under the Montreal Protocol on Substances that Deplete the Ozone Layer, apart from quarantine and shipping uses. However in March 2010 these uses were also banned because of its adverse effects.

Chlorate is formed from disproportionation of chlorine dioxide which is used commercially as bleaching agent in paper production, paper pulp, and cleaning and tanning of leather.

#### 2 From treatment processes

##### From gaseous chlorine

Gas chlorinated systems do not show the presence of chlorate as the high hypochlorous acid/hypochlorite concentrations formed during injection are rapidly diluted (Health Canada 2008).

##### From chlorine dioxide

Chlorate usually appears in waters treated with chlorine dioxide, generally around 0.1 mg/L, (WHO 2017). Chlorate levels are generally about <20 percent of the chlorine dioxide dose (WHO 2016).

The concentration of chlorate depends heavily on process conditions (in both the chlorine dioxide generator and the water treatment plant) and the applied dose of chlorine dioxide. As there is no viable option for reducing chlorate concentrations, control of chlorate concentration must rely on preventing its formation (from chlorine dioxide).

There is always a small amount of chlorate produced during the generation of chlorine dioxide. In addition, photochemical decomposition of chlorine dioxide can lead to chlorate formation in sections of the treatment process exposed to sunlight. Chlorate may also be created in chlorine dioxide-treated waters if chlorine comes in contact with the chlorine dioxide, or through disproportionation of the chlorine dioxide.

##### From hypochlorites

Concentrations of chlorate above 1 mg/L have been reported when hypochlorite was used, but such high concentrations would be unusual unless hypochlorite is stored under adverse conditions, especially if warm. As the solution ages and the available chlorine concentration decreases, it is necessary to dose more product to achieve the desired residual chlorine concentration, with a consequent increase in the amount of chlorate added to the treated water. The decomposition of solid calcium hypochlorite is much slower, and consequently contamination with chlorate is less likely to be significant. However, if calcium hypochlorite solutions are prepared and stored before use, then decomposition to form chlorate would also slowly occur (WHO 2017).

Chlorate has been detected in waters treated with hypochlorite. The older the solution the higher the chlorate concentration. Hypochlorite solutions should (from Health Canada 2008):

* contain less than 1,500 mg chlorate/L
* have a pH greater than 12
* be used within a relatively short time frame after delivery (within three months)
* be stored in a cool (<30°C) dry location, away from sunlight
* contain less than 0.08 mg/L of transition metals.

Also, AWWA (2004) states that organic matter can accelerate the decomposition of hypochlorites. They (and Gordon et al 1995) describe the transition metals mentioned above as copper, nickel and cobalt.

Chlorate is also a contaminant produced during on-site generation of hypochlorite solutions and the subsequent decomposition of the hypochlorite.

Chlorate concentrations in the sodium hypochlorite solutions used for chlorinating in a New Zealand study ranged from 315 to 31,400 mg/L. The proposed SILs (Water NZ 2016) for chlorate in hypochlorite products are 2000 mg/L for chlorate in 13 percent sodium hypochlorite solution and 9000 mg/kg in 60 percent calcium hypochlorite solid (ESR 2019).

Perchlorate can also be formed (DWI 2013), see datasheet for further details.

#### 3 From the distribution system

No known sources. However, if chlorine dioxide and chlorite ion are not removed prior to secondary disinfection with chlorine, they will react with free chlorine to form chlorate ion.

### Forms and fate in the environment

Sodium chlorate may persist in soil for six months to five years, depending on rate applied, soil type, fertility, organic matter, moisture, and weather conditions. Toxicity in soil is decreased considerably by a high nitrate content, alkaline conditions, and high soil temperatures. Decomposition of the compound occurs more readily in moist soils (EXTOXNET 1996). Chlorate is susceptible to biodegradation to chloride by reductive processes in the environment. Thus, pesticidal and industrial releases of chlorate may not entirely survive to reach drinking-water sources (WHO 2016).

The major route of environmental exposure to sodium chlorate is through drinking-water.

### Typical concentrations in drinking-water

The P2 Chemical Determinand Identification Programme, sampled from 130 zones, has found chlorate concentrations to range from “not detectable” (nd) to 2.2 mg/L, with the median concentration being “nd” (Limit of detection = 0.05 mg/L). The Priority 2 Identification Programme found eight distribution zones supplying drinking-water to a total of 1,505 people with chlorate at greater than the MAV, and 32 distribution zones supplied 423,526 people with >50 percent of the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found <0.1 mg/L chlorate in the treated water.

Studies have shown that chlorate ion concentrations ranging from 0.1 to 0.5 mg/L can be found in finished water treated with sodium hypochlorite.

Forty-four water utilities in the US reported detecting chlorate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 1.06 mg/L.

A 1996 Information Collection Rule survey of chlorate in disinfected drinking-water in the USA reported that in WTPs using hypochlorite, the median chlorate concentration was 99 μg/L, the 90th percentile concentration was 239 μg/L and the maximum concentration was 502 μg/L. In water treatment plants using chlorine dioxide, the median chlorate concentration was 129 μg/L, the 90th percentile concentration was 264 μg/L and the maximum concentration was 691 μg/L (from WHO 2016).

Results of two studies were reported in EFSA (2015):

* chlorate was analysed in drinking water (n = 1,199) from different Italian cities by ion chromatography with conductivity detection. Samples had been collected between October 1999 and September 2000. Chlorate was detected in 34 percent of the samples (LOD = 20 μg/L) with a median concentration of 76 μg/L (range: 20–1,500 μg/L).
* chlorate was measured by ion chromatography with conductivity detection (LOD = 1.0 μg/L) in 509 drinking water samples taken in 2007 and 2008 in Castilla y Léon, Spain. Chlorate was detected in 65 percent of the samples with a mean concentration of 224 μg/L (range: 2–4,340 μg/L).

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 62,859 drinking water samples for chlorate between 2013 and 2015, and found 34,426 samples exceeded the minimum reporting level (MRL) of 0.020 mg/L, and 1,896 samples contained >0.21 mg/L.

Chlorate was detected within the reticulation system of all hypochlorite-treated supplies included in a New Zealand study (n = 25). Three supplies were identified with particularly high chlorate concentrations (6.5, 42.9 and 92.1 mg/L) on one sampling occasion. These three supplies did not have remarkably high chlorate concentrations on a second sampling occasion (0.10, 0.50 and 0.08 mg/L, respectively). Six of 47 (13 percent) analytical results for chlorate in reticulated water exceeded the provisional maximum acceptable value (PMAV) of 0.8 mg/L. However, only one supply contained chlorate concentrations that exceeded the PMAV on both sampling occasions (ESR 2019).

### Removal methods

There is no satisfactory method for the removal of chlorate from water; once chlorate ion is present in water, it is very persistent and very difficult to remove. pH values above about 9 and high carbonate conditions should be avoided after chlorine dioxide is added to the water. If high pH processes are used, the formation of chlorate can be minimised by minimising the concentration of chlorine dioxide during the high pH step. Treatment plants employing chlorine dioxide should be designed with a minimum of water surface area exposed to sunlight minimised to reduce chlorine dioxide loss through photo-decomposition. This precaution will also minimise chlorate formation through photo-decomposition.

As much as 35 percent of the chlorate concentration found in a distribution system can be attributed to the type and performance (tuning) of the chlorine dioxide generator. If chlorite ion is present in water and is not removed, it will react with any applied free chlorine to produce chlorate and chloride ions.

Current commercial chlorine dioxide generators may be classified broadly as chlorite based, chlorate based, or electrochemical systems.

1. Chlorite ion-based systems rely on the oxidation of chlorite ion to chlorine dioxide through the use of an acid, which may attain a maximum conversion efficiency of 80 percent by stoichiometry; or through the use of chlorine gas, which can result in chlorite carry through if the chlorine gas feed is too low and chlorate formation if the chlorine gas feed is too high.

2. Recently developed chlorate ion-based systems typically depend on the reduction of chlorate ion through the reaction of sodium chlorate with an acid and hydrogen peroxide. The product may be quite acidic, and the risk of high hydrogen peroxide and perchlorate levels in the water may detract from the viability of this method.

3. Electrochemical systems can either directly or indirectly generate chlorine dioxide. The direct method involves the electrolysis of chlorite ion to chlorine dioxide at the anode, and the indirect method is the production of an acid or chlorine gas as a precursor chemical, resulting in the formation of chlorine dioxide, again at the anode. When the chlorine dioxide is formed at the anode, it must be extracted as a gas from the solution by gas-stripping columns, eductors/venturis, low-pressure air flow over a packed bed, or perstraction, which involves the use of a gas-permeable hydrophobic membrane. Proper balance and control are required with these systems to prevent the formation and carry-through of impurities such as acid, chlorate ion, perchlorate ion, and chlorine.

Where chlorination is carried out by hypochlorite dosing, the use of old hypochlorite solutions should be avoided.

### Analytical methods

#### Referee method

Ion chromatography is the recommended method, having reported detection limits as low as 0.009 mg/L (9 g/L) as chlorate, see USEPA 300.1.

#### Some alternative methods

1. Other (older) ion chromatography methods include: Hautman and Bolyard (1992), USEPA Method 317.0 Rev. 2.and USEPA Method 326.0.

The determination of individual chlorine-containing species in a chlorine dioxide-treated water presents a complex analytical problem. As a general comment, expensive instrumentation is required to obtain accurate results. Methods requiring simple apparatus (eg, amperometric titration) have been developed, and rely on the manipulation of pH to differentiate between the chlorine-containing species. Consequently, the final result involves the subtraction of a number of different readings, leading to a large uncertainty in the result. Considerable analytical skill and experience is required to obtain good precision and accuracy with these methods.

Flow injection analysis provides a detection limit of approximately 0.1 mg/L as chlorate.

**Note:** concentrations for chlorine dioxide, chlorite and chlorate determined by amperometric and DPD titrations are often expressed as mg/L of chlorine. This can lead to confusion, and when reporting results the units in which the results are expressed must be clearly stated, ie, mg/L as Cl2 or mg/L as chlorate.

### Health considerations

Chlorate may occur in foods as a result of the uses of chlorine dioxide, sodium chlorate or sodium chlorite in flour processing, as a decolorising agent for carotenoids and other natural pigments (from chlorine dioxide), and from food cooked in water containing chlorate.

Following oral ingestion by monkeys, chlorine dioxide was rapidly converted into chloride ion and, to a lesser extent, chlorite and chlorate and are excreted primarily in the urine; however most is degraded to chloride.

Although chlorate has also been reported to have effects on red blood cells, the most sensitive effects observed in rats administered sodium chlorate in drinking-water for 21 or 90 days were changes in thyroid histology (eg, colloid depletion, hypertrophy, incidence and severity of hyperplasia) and in thyroid hormones. As with chlorite, a chlorate dose of 36 μg/kg bw per day for 12 weeks did not result in any adverse effects in human volunteers (WHO 2017).

Chlorate is absorbed rapidly by the gastro-intestinal tract into blood plasma and distributed to the major organs. It is metabolised rapidly, and is excreted mainly in the form of chloride in the urine.

Because of its (mostly historical) use as a weed killer, a large number of cases of chlorate poisoning have been reported. Symptoms include methaemoglobinaemia, anuria, abdominal pain and renal failure. For an adult human, the oral lethal dose is estimated to be as low as 20 g of sodium chlorate (230 mg of chlorate per kg of body weight).

Several studies have been carried out on human volunteers administered doses of sodium chlorate in drinking-water. The consumption of high doses of chlorate was associated with a change in serum urea nitrogen and corpuscular haemoglobin.

A tolerable daily intake (TDI) of 3 μg chlorate/kg body weight (b.w.) was set by read-across from a TDI of 0.0003 mg/kg b.w. derived for this effect for perchlorate, multiplied by a factor of 10 to account for the lower potency of chlorate. Formation of methaemoglobin was identified as the critical acute effect of chlorate. Like perchlorate, chlorate is a competitive inhibitor of iodine uptake in the thyroid. An acute reference dose (ARfD) of 0.036 mg chlorate/kg b.w. was derived from a no-observed-effect-level (NOEL) for chlorate in a controlled clinical study. Chronic exposure of adolescent and adult age classes did not exceed the TDI. However, at the 95th percentile the TDI was exceeded in all surveys in ‘Infants’ and ‘Toddlers’ and in some surveys in ‘Other children’. Chronic exposures are of concern in particular in younger age groups with mild or moderate iodine deficiency (EFSA 2015).

No data are available on the mutagenic activity of chlorate.

### Derivation of Maximum Acceptable Value

WHO (2016/17) considered recent data where JECFA established an ADI of  
0–0.01 mg/kg bw for chlorate on the basis of the BMDL10 of 1.1 mg/kg bw per day for non-neoplastic effects on the thyroid of male rats in a carcinogenicity study (NTP 2005), a safety factor of 10 to allow for intraspecies variability and an additional factor of 10 to allow for the deficiencies in the database. The rationale for selection of a tenfold uncertainty factor (as opposed to, for example, a threefold uncertainty factor) was not additionally specified by JECFA.

Using the upper bound of the unrounded ADI of 11 μg/kg bw, a typical human body weight of 60 kg, the assumption that drinking-water contributes 80 percent (default ceiling value based on drinking-water as the predominant source of exposure) of the total exposure and a typical consumption of 2 L of water per day, **a health-based value of 0.3 mg/L (rounded figure) could be calculated**. Chlorate concentrations arising from the use of sodium hypochlorite are generally below the health-based value, although higher concentrations have been noted. As well, the concentration of chlorate arising from the use of hypochlorite as a disinfectant depends heavily on process conditions, and control of chlorate concentrations must rely on preventing its formation. Control of storage conditions is considered to be most difficult in small, resource-limited water supplies, and so the potential for the health-based value to be exceeded is also greater under these circumstances.

**In view of the above considerations, the previous provisional guideline value of 0.7 mg/L is retained**. It is essential to ensure the availability of hypochlorite and chlorine dioxide for disinfection purposes. The guideline value is designated as provisional because use of aged hypochlorite or of chlorine dioxide as disinfectants may result in the chlorate guideline value being exceeded, and difficulties in meeting the guideline value must never be a reason for compromising adequate disinfection.

The provisional MAV for chlorate in drinking-water in the 2008 DWSNZ was derived as follows (WHO 2004/2011):

30 mg/kg body weight per day x 70 kg x 0.8 = 0.84 mg/L (rounded to 0.8 mg/L)

2 L per day x 1000

where:

* NOAEL = 30 mg/kg/day in a well-conducted 90-day study in rats, based on thyroid gland colloid depletion at the next higher dose
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.8
* average amount of water consumed by an adult = 2 L per day
* uncertainty factor = 1000 (10 each for inter- and intraspecies variation and 10 for the short duration of the study).

The basis for the provisional MAV of 0.3 mg/L for chlorate in the 2000 DWSNZ appeared in the 1995 datasheet:

Available data for the effects of chlorate in humans and experimental animals are considered by WHO to be insufficient to develop a MAV. Until further data are available, it is considered prudent to keep chlorate levels at or below the provisional for chlorite of 0.3 mg/L.

WHO (1996) stated available data on the effects of chlorate in humans and experimental animals are considered insufficient to permit the development of a guideline value. Data on accidental poisonings indicate that the lethal dose to humans is about 230 mg/kg of body weight per day. This is of the same order of magnitude as the NOAELs identified from studies in rats and dogs. Although no effects were observed in a 12-week clinical study in a small number of human volunteers ingesting 36 μg/kg of body weight per day, a guideline value was not derived from these results because no adverse effect level was determined.

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# Chlorine

CAS No. 7782-50-5.

### Maximum Acceptable Value

Based on health considerations, the concentration of chlorine in drinking-water should not exceed 5 mg/L.

Based on aesthetic considerations, the concentration in drinking water should not exceed 0.6 – 1.0 mg/L, but microbiological quality must not be compromised.

The maximum residual disinfectant level (USEPA 2009/2011) is 4 mg/L. The USEPA also established a lifetime health advisory of 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 2016) state that based on health considerations, the guideline value for total chlorine in drinking water is 5 mg/L, except for chloraminated systems.

Health Canada states that no MAC is required, and adds that “*Any measures taken to limit the concentration of chlorine or its by-products in drinking water supplies must not compromise the effectiveness of disinfection.”*

There is no Prescribed Concentration or Value (PCV) for chlorine in England and Wales.

Refer to cyanuric acid in the organic chemicals section for a datasheet for the disinfectant sodium dichloroisocyanurate.

### Sources to drinking-water

#### 1 To source waters

Chlorine may be present in source waters as a result of its discharge from industries in which it is used. These include the food industry where chlorine and hypochlorites are used for general sanitation and odour control, in the production of industrial and domestic disinfectants and bleaches, and in the synthesis of a large range of chemical compounds. Chlorine is widely used to disinfect drinking-water, cooling water, sewage, wastewater and swimming pool water. Its high reactivity would suggest it is unlikely to be found in raw water.

#### 2 From treatment processes

Chlorine and hypochlorites are widely used as disinfectants for drinking-water supplies. Chlorine is purchased as a gas in pressurised cylinders/drums or manufactured on-site by electrolysis of salt. Sufficient chlorine is usually added at the treatment plant to maintain a residual in the distribution system. NHMRC (2009) established an aesthetic guideline value based on the odour threshold which is generally 0.6 mg/L but 0.2 mg/L for some people; in some supplies it may be necessary to exceed the aesthetic guideline in order to maintain an effective disinfectant residual throughout the system.

DWI (2013) states that BS EN 937 limits nitrogen trichloride to 20 mg/kg of liquid chlorine.

#### 3 From the distribution system

No known sources, except in some large water supplies where supplementary chlorination is used to maintain a residual at remote parts of the system.

### Forms and fate in the environment

In environmental waters, chlorine exists as hypochlorous acid and hypochlorite ion. Both species exist in equilibrium, with their relative concentrations depending upon pH. Hypochlorous acid shows some volatility and some may be lost by aeration or water turbulence. Chlorine can react with ammonia or organic nitrogen compounds in the water to form chloramines. See Chapter 15.

Concentrations of chlorate and some perchlorates (qv) increase in hypochlorite solutions upon storage at high ambient temperatures or when new hypochlorite is added to old hypochlorite.

### Typical concentrations in drinking-water

Free available chlorine concentrations in chlorinated waters should range from about 0.2 to 1 mg/L. However, the sampling point in the reticulation may have an effect on the residual measured; high levels may be found at points close to the treatment plant, while very low levels, or the absence of chlorine, may be found at the extremes of the distribution system.

Free available chlorine levels in Australian waters have been found to range from 0.1 to 4 mg/L. Most Canadian drinking water supplies maintain free chlorine residuals in the 0.04–2.0 mg/L range in the distribution system. At these concentrations, taste and odour related to chlorine or its by-products are generally within the range of acceptability for most consumers (Health Canada 2009).

### Removal methods

If necessary, chlorine in water can be neutralised by the addition of a number of reducing agents, including sodium thiosulphate, sulphur dioxide, sodium bisulphite. Exposure of chlorinated water to sunlight will result in photodecomposition of the chlorine. The volatility of chlorine is pH dependent; it is more easily lost from water in pH regions where hypochlorous acid is the predominant form. Some chlorine can be lost by aeration of water, but stripping is not an efficient way of removing chlorine. Activated carbon can reduce the chlorine concentration. Boiling the water breaks down the chlorine to form chloride.

### Analytical methods

Chlorine cannot be preserved in a sample. Analysis must be carried out as soon after sampling as possible, and if a delay between sampling and analysis is unavoidable the sample must be kept dark and chilled.

#### Referee method

DPD Ferrous Titrimetric Method (APHA 4500-Cl F).

The limit of detection for this method is approximately 0.1 mg/L for field use, although lower levels can be determined under laboratory conditions and with care and experience. Analytical texts indicate that by manipulation of the conditions of the analysis, measurement of monochloramine, dichloramine and trichloramine can be made. These methods are of use when ammonia only is in the water being chlorinated. In most natural waters nitrogen-containing organic compounds are also present. Organic chloramines are formed from these compounds when chlorine reacts with them. Organic chloramines also produce colour during the DPD test and make attempts to differentiate between the different inorganic chloramines of little value. It is recommended, therefore, that only the total combined chlorine, ie, total chloramine concentration, is reported.

#### Some alternative methods

1. Amperometric Titration Method (APHA 4500-Cl D and E).

While more accurate than the DPD methods, expensive equipment and a high degree of skill and care are required for this method. The limit of detection for method APHA 4500-Cl D is better than 0.1 mg/L. APHA 4500-Cl D describes variations that will allow the determination of mono- and di-chloramine. APHA 4500-Cl E can measure down to 0.01 mg/L but it is not possible to differentiate between free and combined forms of chlorine. Interferences due to organic chloramines may also cause interferences with these methods.

2. DPD Colorimetric Method (APHA 4500-Cl G).

This method requires a spectrophotometer for the colorimetric measurements, although hand-held comparators do offer a cheaper, though less reliable variation for field use. The limit of detection with, instrumental assistance, is better than 0.05 mg/L.

The limit of detection for the comparator depends on the colour disc in use. FAC concentrations as low as 0.1 mg/L should be detectable, but the accuracy of the method depends upon use of the correct lighting (natural lighting should be used with the sun behind the viewer), the individual’s ability to match colours and judge their intensity, and ensuing that readings are taken as soon after colour development as possible. Using a Nessleriser will enable concentrations of 0.05 mg/L to be read.

3 Syringaldazine (FACTS) Method (APHA 4500-Cl H).

This colorimetric method is less prone to interference from combined chlorine than the DPD methods, but there can be problems with the solubility of the syringaldazine indicator. The limit of detection is 0.1 mg/L or better.

### Health considerations

Chlorine, or hypochlorites, are strong oxidising agents that readily react with organic molecules to produce a variety of chlorinated compounds. This reactivity makes it difficult to separate the effects of chlorine from those of its metabolites. In animal studies using a naturally-occurring non-radioactive chlorine isotope, chlorine was found to be absorbed rapidly by the gastrointestinal tract, and highest concentrations of the isotope were found in blood plasma.

It is assumed that the toxicity of aqueous solutions containing chlorine, hypochlorous acid or hypochlorite are similar since they are in dynamic equilibrium. Chlorine concentrations therefore refer to free available chlorine.

Very few toxic effects have been associated with drinking-water containing high chlorine concentrations. In one report, 150 people drank water with 50 mg/L during a period of mains disinfection, with no adverse effects. Several instances have been reported where military personnel drank water with chlorine concentrations up to 32 mg/L for several months with no ill-effects. Momentary constriction of the throat, and mouth irritation were observed when the chlorine concentration exceeded 90 mg/L. Most people would refuse to drink water with a chlorine concentration over 25 mg/L (NHMRC, NRMMC 2016).

Individuals that are more likely to dislike or show some form of reaction to high levels of chlorine in drinking-water include those with respiratory conditions such as asthma, hay fever, chronic bronchitis, and heavy smokers.

The reference dose or RfD (USEPA 2009/2011) is 0.1 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 5 mg/L.

In the Stage 1 D/DBPR, USEPA finalised an MRDLG of 4 mg/L for chlorine based on a weight of evidence evaluation of both the cancer and non-cancer effects and classified chlorine as “not classifiable as to human carcinogenicity”. The MRDLG was based on the RfD of 0.1 mg/kg/day, an adult tap water consumption of two litres/day for a 70 kg adult and an assumed drinking water contribution of 80 percent of total exposure. The RfD was based on a NOAEL of 14 mg/kg/day for no treatment-related effects from NTP (1992a), a two-year drinking water study in rats and mice, with the application of an uncertainty factor of 100. Due to a lack of significant new health effects data available for the Stage 2 D/DBPR, the RfD value did not change, and USEPA did not revise the MRDLG for chlorine at that time. Taken from USEPA (2016).

A number of studies have suggested an association between chlorination by-products and various cancers. This association has been consistent in relation to cancers of the bladder and rectum, but there are insufficient data to determine concentrations at which chlorination by-products might cause increased risk to human health.

Long-term animal toxicity studies have shown no specific effects from the ingestion of chlorine. Chlorine, hypochlorous acid and hypochlorite did not act as carcinogens or tumour initiators.

Assessment of the mutagenicity of chlorine is complicated by the reactivity of chlorine. Hypochlorite was found to be mutagenic in tests with one strain of bacteria but not with another. Chromosome aberrations were reported in tests with mammalian cells.

The International Agency for Research on Cancer (IARC) has concluded that hypochlorites are not classifiable as to their carcinogenicity to humans, Group 3.

Buildings containing chlorine gas must be very well ventilated. Chlorine in air is corrosive and is very hazardous. ICPS (1998) reports:

1 to 3 ppm Mild mucous membrane irritation after 1 hour

5 to 15 ppm Moderate irritation of upper respiratory tract

30 ppm Immediate chest pain, vomiting, and coughing

40 to 60 ppm Toxic pneumonitis and pulmonary oedema

430 ppm Lethal after 30 minutes

1,000 ppm Fatal within a few minutes.

### Derivation of Maximum Acceptable Value

The MAV for chlorine has been derived as follows:

15 mg/kg body weight per day x 70 kg x 1 = 5.25 mg/L, rounded to 5 mg/L

2 L per day x 100

where:

* no-observable-adverse-effects level = 15 mg/kg body weight per day. This is from a study which reported the absence of toxicity in rodents that received chlorine as hypochlorite in drinking-water for up to two years
* average adult weight = 70 kg
* the proportion of the total intake of chlorine to drinking-water = 1
* the average quantity of water consumed by an adult = 2 L per day
* uncertainty factor = 100 for intra- and interspecies variation.

It should be noted that this value is conservative as no adverse effect level was identified in this study. Most individuals are able to taste chlorine at the MAV. WHO (2004) indicated an increased risk of unacceptability at free chlorine residuals between 0.6 and 1.0 mg/L. NHMRC (2016) reported an odour threshold of 0.6 mg/L for chlorine in drinking water but noted some people are particularly sensitive and can detect amounts as low as 0.2 mg/L; whereas a study by AwwaRF suggested thresholds as low as 0.05–0.1 mg/L. There is a wide variability of taste and odour thresholds in the population, depending on individual sensitivities (Health Canada 2009).

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# Chlorine dioxide

CAS No. 10049-04-4.

### Maximum Acceptable Value

A MAV has not been established for chlorine dioxide because of the rapid breakdown of chlorine dioxide and because the chlorite MAV is adequately protective for potential toxicity from chlorine dioxide.

Any chlorine dioxide remaining at the consumer’s tap will be reduced to chlorite and chloride upon ingestion (WHO 2017).

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state: based on aesthetic considerations, the concentration in drinking water should not exceed 0.4 mg/L.

Health Canada states that no MAC is required because of its rapid reduction to chlorite in drinking water. Health Canada (2008) states that treatment plants using chlorine dioxide as primary disinfectant should not exceed a maximum feed dose of 1.2 mg/L, which will ensure that the chlorite and chlorate guidelines can be met, and that consumers are not exposed to concentrations of chlorine dioxide that could pose health risks.

The maximum residual disinfectant level (USEPA 2009/2011) is 0.8 mg/L. The USEPA also established a lifetime health advisory of 0.8 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 health advisory for Group C carcinogens includes an adjustment for possible carcinogenicity.

Refer to the chlorate and chlorite datasheets for further information.

### Sources to drinking-water

#### 1 To source waters

The major use for chlorine dioxide is as a bleaching agent in paper production, paper pulp and for the cleaning and tanning of leather. However, in wastewaters from these industries it is generally chlorite which is detected because of its rapid formation from the reactions of chlorine dioxide with compounds in the waste.

Chlorine dioxide appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2017 (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 sold as a sodium chlorite solution to which is added citric acid, thereby generating the ClO2. It is used as fungicide on fruit, vegetables and flowers.

#### 2 From treatment processes

Chlorine dioxide can be used as a disinfectant for drinking-water supplies. Chlorine dioxide is explosive under pressure so it usually has to be manufactured in situ. In smaller plants it is made by adding acid to sodium chlorite; this process does not produce chlorine. Another small plant process involves the addition of sodium chlorite to a persulphate. Bigger plants make chlorine dioxide by adding chlorine to sodium chlorite. Chlorine dioxide can be stored as a liquid below 4°C; although fairly stable, it breaks down to chlorine and oxygen. It can be marketed and transported as a stabilised aqueous solution, generally less than 1 percent w/v (more concentrated forms are explosive). See Section 15.5.3 in Chapter 15: Disinfection Processes in the Guidelines.

The total dosage of chlorine dioxide could be an indicator of the potential upper-bound concentrations of chlorate and chlorite; chlorite levels can generally range from 30 percent to 70 percent of the chlorine dioxide dose and chlorate levels are generally less than that, at about <20 percent of the dose (WHO 2016).

Chlorine dioxide is quite soluble in water (but unlike chlorine, it does not react with water), typically 1–3 g/L (1000–3000 mg/L), depending on the temperature and pressure.

The melting point of chlorine dioxide is 11°C. For safety reasons chlorine dioxide in air should be <10 percent by volume.

Chlorine dioxide reacts with natural organic matter in the water (but less reactive than chlorine), and other oxidisable materials to produce primarily chlorite and to a lesser extent chloride; approximately 70 percent of the applied chlorine dioxide will eventually form chlorite, while about 10 percent will form chlorate. Chlorate may also be present in these waters as a by-product of the reaction used to generate the chlorine dioxide.

#### 3 From the distribution system

No known source.

### Forms and fate in the environment

Chlorine dioxide is unlikely to be long-lived in the environment. This is partly because it reacts with organic matter in water, primarily to produce chlorite, and to some extent chloride, and partly because it will be photodecomposed readily by sunlight. The major photodecomposition products are: chloride, chlorate and molecular oxygen.

Chlorine dioxide is stable in pure water in the absence of reducing agents and ultraviolet light; however, in normal water, its disproportionation to chlorite and chlorate is a function of basicity and is catalysed by transition metal ions such as iron and copper, as well as by hypobromite and hypochlorite ions. Chlorine dioxide is also reduced to chlorite and chloride by reactions with total organic carbon components. Chlorine dioxide is stable in pure water in the dark, but it is photoreactive in sunlight, producing chlorate, chlorite and chloride, especially in alkaline solution (WHO 2016).

### Typical concentrations in drinking-water

Only one public water supply has used chlorine dioxide for water treatment on a long-term basis in New Zealand, and no data are available on typical chlorine dioxide concentrations.

Chlorine dioxide residuals in treated Australian waters typically have been between 0.2 and 0.4 mg/L. It is particularly effective in the control of manganese-reducing bacteria.

The need to minimise chlorite production is a major factor that influences the doses of chlorine dioxide that can be used.

Chlorine dioxide decomposes into chlorite, chlorate and chloride ions in treated water, chlorite being the predominant species. This reaction is favoured by alkaline conditions.

### Removal methods

Chlorine dioxide can be removed by the addition of chemical reducing agents, which include sodium thiosulphate, sulphur dioxide, sodium bisulphite, and ferrous chloride, to the water. The chemical reduction of chlorine dioxide can have unacceptable consequences on the water quality, because reduction often leads to the production of chlorite, and although the chlorite can be reduced to chloride under some conditions, the presence of oxygen in the water can produce chlorate.

Decomposition of the chlorine dioxide with light could be considered, but chlorate, as well as chloride and oxygen, is formed in this process.

### Analytical methods

#### Referee method

A referee method cannot be selected for chlorine dioxide 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 chlorine dioxide for the above reason. However, the following information may be useful:

Chlorine dioxide cannot be preserved in a sample. Analysis must be carried out as soon after sampling as possible, and if a delay between sampling and analysis is unavoidable the sample must be kept dark and chilled. Aeration of the sample must be avoided.

The analysis of chlorine dioxide in a water sample can be complex if chlorine and chloramines are present. The DPD and amperometric methods noted are extensions of the equivalent methods for chlorine.

1. Amperometric Method (APHA 4500-ClO2 C and E).

This method requires more sophisticated equipment and greater skill on the part of the analyst than either of the other two methods. By performing four titrations, free chlorine, chloramines, chlorite and chlorine dioxide can be determined separately. The uncertainty in the result can be larger than the result itself.

2. DPD Method (APHA 4500-ClO2 D).

A number of pH adjustments have to be made with this method during the measurement, which adds to the difficulty of its use. The limit of detection is approximately 0.1 mg/L as Cl2, or 0.19 mg/L as ClO2.

3. Lissamine Green B (USEPA 2005).

This method can measure chlorine dioxide and chlorite, overcoming interference from free available chlorine. It is primarily intended to be used at water treatment plants in conjunction with daily monitoring requirements. Method performance was demonstrated over a combined concentration range of 0.2 to 2.2 mg/L.

4. Chlorophenol Red Method (Harp et al).

This is a relatively simple colorimetric method that utilises the quantitative reduction in the colour of chlorophenol red resulting from its reaction with chlorine dioxide. The method is specific for chlorine dioxide; other chlorine compounds do not react with chlorophenol red in this way. The limit of detection for this method has been reported to be 0.004 mg/L as ClO2, but these were under laboratory conditions during development, and the sensitivity would probably be less for field analysis.

#### Note concerning units of measurement

Some methods measure chlorine dioxide as chlorine equivalent. The conversion factor for chlorine dioxide expressed in mg of Cl2/L to chlorine dioxide expressed in mg of ClO2/L is:

Chlorine dioxide (as mg of Cl2/L) = chlorine dioxide (as mg of ClO2/L) x 2.6

This assumes the result in mg of Cl2/L was calculated to yield the full oxidising capability (5 electron transfer) of ClO2. The complication of knowing how the ClO2 concentration expressed as Cl2 was determined is a very good reason for ensuring that all these determinands are expressed in units of their own weight per volume, rather than equivalent weight of Cl2/volume, wherever possible.

For bacterial and protozoal compliance testing, the DWSNZ require on-line measurement.

### Health considerations

Chlorine dioxide is absorbed rapidly from the gastro-intestinal tract and no particular organ appears to concentrate the dose selectively following exposure. Animal studies have shown that following ingestion, chlorine dioxide is converted rapidly to the chloride ion, and to a lesser extent chlorite and chlorate. There is a ready interconversion among these species in water (before administration to animals) and in the gut (after ingestion). Therefore, what exists in water or the stomach is a mixture of these chemical species (ie, chlorine dioxide, chlorite, chlorate) and possibly their reaction products with the gastrointestinal contents (USEPA 2000). Excretion is primarily via urine.

In a study with human volunteers, no adverse effects were observed after drinking 0.5 L per day of water containing chlorine dioxide concentrations up to 5 mg/L for periods of 12 weeks. In a series of extensive human volunteer studies on water disinfectants, groups of 10 males received aqueous chlorine dioxide in drinking-water by a range of different protocols (a sequence of rising concentrations of up to around 0.34 mg/kg body weight over a 16-day period, approximately 0.035 mg/kg body weight on every third day for 12 weeks, or approximately 3.6 × 10–5 mg aqueous chlorine dioxide/kg body weight per day daily for 12 weeks). Observations included physical examination (blood pressure, respiration rate, pulse, oral temperature, and electrocardiography), extensive blood biochemistry, haematology, and urinalysis, and the subjective recording of taste. There were no significant adverse effects recorded for any of the parameters measured (ex WHO 2002).

In rats exposed from birth, high concentrations of chlorine dioxide may impair neurobehavioural and neurological development. Significant depression of thyroid hormones have been observed in rats, pigeons and monkeys exposed to doses of approximately 10 mg/kg body weight per day for long periods of time.

The results of repeated oral exposure studies in rats and primates are generally of limited design and/or quality but show no evidence of systemic toxicity associated with chlorine dioxide administered in the drinking-water or by gavage. No tumours were observed in rats following two year exposures to chlorine dioxide in drinking-water. Chlorine dioxide was mutagenic in tests with one bacterium strain. It did not induce chromosomal aberrations in tests with mouse bone-marrow cells.

USEPA (2000) states that the human carcinogenicity of chlorine dioxide cannot be determined because no satisfactory human or animal studies assessing the chronic carcinogenic potential of chlorine dioxide were located.

In the Stage 1 D/DBPR, USEPA proposed a MRDLG of 0.3 mg/L for chlorine dioxide based on an RfD of 0.01 mg/kg/day from a developmental rat study. USEPA reviewed and completed a peer review of a two-generation reproductive study of chlorite in Sprague-Dawley rats. In this study, male and female rats were administered sodium chlorite in drinking water at doses ranging up to 300 ppm. Reproduction, fertility, clinical signs and histopathology were evaluated in two generations of offspring. These data are relevant to chlorine dioxide because chlorine dioxide is rapidly reduced to chlorite and chlorite is oxidised to chlorate. In the Stage 1 D/DBPR, USEPA finalised an MRDLG of 0.8 mg/L for chlorine dioxide based on the same data used to derive the MCLG for chlorite; the RfD of 0.03 mg/kg/day, an adult tap water consumption of two litres/day for a 70 kg adult and an assumed drinking water contribution of 80 percent of total exposure. The RfD was derived based on a NOAEL of 35 ppm (3 mg/kg/day for the chlorite ion) for decreases in absolute brain and liver weight and lowered auditory startle amplitude at 70 and 300 ppm and the application of an uncertainty factor of 100. In the Stage 1 D/DBPR, the final MRDLG was not changed from the proposed value because a lower uncertainty factor (100 vs 300) was applied with the use of the multigeneration CMA (1996) study. USEPA did not revise the MRDLG for chlorine dioxide in the Stage 2 D/DBPR. Taken from USEPA (2016).

The reference dose or RfD (USEPA 2000, 2006, 2009 and 2011) is 0.03 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 1 mg/L.

### Derivation of Maximum Acceptable Value

A MAV has not been established for chlorine dioxide because of the rapid breakdown of chlorine dioxide and because the chlorite MAV is adequately protective for potential toxicity from chlorine dioxide.

The taste and odour threshold for chlorine dioxide in water is 0.2–0.4 mg/L. Although chlorine dioxide can reduce odour from some water components, it has also been reported to have caused strong chlorinous odours in some residences during distribution. Chlorine dioxide’s water solubility – and hence its odour threshold – is decreased with reduced pressure and increased temperature. The sudden evaporation of chlorine dioxide from cold water (<8 to 10°C) released at the tap can lead to a chlorine dioxide odour, and this effect may be increased by heating the water to 40°C. Kerosene-like and cat urine–like odours were produced in some homes with new carpets when volatising chlorine dioxide reacted with airborne volatiles (WHO 2016).

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# Chlorite

CAS No. 7758-19-2.

### Maximum Acceptable Value (Provisional)

Based on health considerations, the concentration of chlorite in drinking-water should not exceed 0.8 mg/L. The provisional MAV for chlorite in the 1995 and 2000 DWSNZ had been 0.3 mg/L.

WHO (2004 and 2011) stated that the guideline value for chlorite was designated as provisional because use of chlorine dioxide as a disinfectant may result in the chlorite guideline value being exceeded, and difficulties in meeting the guideline value must never be a reason for compromising adequate disinfection.

Being a disinfection by-product, chlorite is regulated in the US, see USEPA (2007). The maximum contaminant level (USEPA 2009/2011) is 1.0 mg/L. The lifetime health advisory is 0.8 mg/L (USEPA 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 Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of chlorite in drinking water should not exceed 0.8 mg/L (the previous version stated 0.3 mg/L).

The maximum acceptable concentration in Canada is 1 mg/L. Health Canada (2008) states that 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.

Refer also to the chlorate and chlorine dioxide datasheets.

### Sources to drinking-water

#### 1 To source waters

Chlorite may enter raw waters in industrial discharges when it is used in the production of paper, textiles and straw products and in the manufacture of waxes, shellacs, and varnishes. Chlorine dioxide is used to bleach cellulose, paper pulp, flour and oils.

Chlorine dioxide appears on the NZFSA’s complete database of Agricultural Compounds and Veterinary Medicines (ACVM) as at 2017 (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 sold as a sodium chlorite solution to which is added citric acid, thereby generating the ClO2. It is used as fungicide on fruit, vegetables and flowers.

#### 2 From treatment processes

The appearance of chlorite in treated water is inevitable when using chlorine dioxide. There are three reasons for chlorite appearing through the use of this treatment process.

Chlorine dioxide is generated by the reaction of chlorine with chlorite. Poor control over the process, so that insufficient chlorine is added for complete reaction of the chlorite, may lead to excess chlorite being carried into the treated water.

Chlorine dioxide itself will undergo a base-catalysed decomposition to form chlorite. The rate of the reaction is also increased by the presence of carbonate. It is therefore important that processes raising the pH above 9, and/or using carbonate, such as lime-soda softening, not be used after chlorine dioxide dosing.

Chlorite is also produced from the chemical reduction of chlorine dioxide during the oxidation of organic matter.

Chlorite levels can generally range from 30 percent to 70 percent of the chlorine dioxide dose. When chlorine dioxide is used carefully as the final disinfectant at typical doses, the resulting chlorite concentration would normally be less than 0.2 mg/L.

Chlorite can also be produced during the slow decomposition of sodium hypochlorite solution. Chlorite is present at steady state in hypochlorite solutions and is an intermediate between hypochlorite and chlorate and, ultimately, perchlorate (WHO 2017).

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

Chlorine dioxide is stable in pure water in the dark, but it is photoreactive in sunlight and dissociates into chlorite, chlorate and chloride ion, especially in alkaline solution, with chlorite being the predominant species. Chlorite ions will be mobile in soils and may leach into groundwater; however, oxidation–reduction reactions may reduce the concentration of chlorite ions capable of leaching into groundwater (WHO 2016).

The major route of environmental exposure to sodium chlorite is through drinking-water.

### Typical concentrations in drinking-water

Chlorite was not routinely measured in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. However, a small programme was undertaken to monitor the chlorite and chlorate levels in a supply receiving water treated by chlorine dioxide. In ten samples the chlorite concentrations ranged from 0.6 to 2.1 mg/L as chlorite. Such a small data set does not provide a good basis for a comment on what might be typical in New Zealand; a number of factors, including raw water quality, will control the sorts of levels that will appear in a particular supply.

In 2013/14 Hamilton’s six-monthly analyses have found <0.005 mg/L chlorite in the treated water.

113 water utilities in the US reported detecting chlorite in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 1.03 mg/L.

### Removal methods

It is preferable to avoid forming chlorite rather than trying to remove it once formed. This is best achieved by: removing as much organic matter from the water as possible before the chlorine dioxide is introduced; ensuring that the pH of the water is kept below 9; and avoiding high carbonate concentrations in the water (which is easy in New Zealand), and by fine-tuning the ClO2 generator. If high pH and high carbonate conditions are unavoidable, the chlorine dioxide concentration at this part of the process should be minimised.

Some chemical reductants used in water treatment have been found to produce unacceptably high concentrations of chlorate when used to remove chlorite from water. Thiosulphate and ferrous iron appear to be reductants that could be used with minimal formation of chlorate.

Activated carbon will remove chlorite ion through adsorption and chemical reduction. Early break-through has been reported in granular activated carbon (GAC) filters when the adsorptive sites have been exhausted, perhaps by competing organic compounds, and only the reduction mechanism remains. The performance of GAC filters for chlorite removal is further complicated by the oxidation of chlorite to chlorate, which may occur if free chlorine is present in the feed water. Short bed life, high operating costs, and the potential for chlorate formation make GAC an impractical choice for chlorite removal at the municipal scale.

Sulfur agents such as sulfite, metabisulfite, and thiosulfate will reduce chlorine dioxide and chlorite ion, thereby lowering their concentrations in water. In the presence of dissolved oxygen, sulfite and metabisulfite will reduce chlorite to form chloride ion and the undesirable chlorate ion and, as such, is not recommended for the removal of chlorite in drinking water. Thiosulfate is effective at reducing chlorine dioxide and chlorite and does not form chlorate as a by-product, but it requires a long contact time and is pH dependent, which may limit its effectiveness.

Ferrous iron will chemically reduce chlorite ion, thereby lowering its concentrations in water. Chlorate ion will form only if the pH drops below 5, which can occur at localised application points where acidic reducing agents such as ferrous chloride are added to the water. Good application and rapid mix and/or pH adjustment to neutral pH 7 may prevent the occurrence of micro-regions of low pH and the subsequent formation of chlorate. When the pH exceeds 7, the subsequent reaction of chlorite and ferrous iron forms insoluble ferric hydroxide, which may be beneficial by aiding clarification. Any residual chlorite will react with chlorine to form chlorate and should be removed before post-chlorine disinfectant is applied. Ferrous iron or thiosulfate, when used as treatment options for chlorite removal, may be fed in excess of the demand and can complicate post disinfection.

### Analytical methods

#### Referee method

Ion chromatography is the recommended method, having reported detection limits as low as 0.009 mg/L (9 g/L) as chlorate, see USEPA 300.1.

#### Some alternative methods

1. Other (older) ion chromatography methods include: Hautman and Bolyard (1992), USEPA Method 317.0 Rev. 2.and USEPA Method 326.0.

The determination of individual chlorine-containing species in a chlorine dioxide-treated water can be complex. As a general comment, expensive instrumentation is required to obtain accurate results. Methods requiring simple apparatus have been developed, and rely on the manipulation of pH to differentiate between the chlorine-containing species. Consequently, the final result involves the subtraction of a number of different readings, leading to a large uncertainty. Considerable analytical skill and experience is required to obtain good precision and accuracy with these methods when concentrations less than 1 mg/L are of interest.

Chlorite can be determined by: amperometric titration (APHA 4500-ClO2 D); DPD titration (APHA 4500-ClO2 E); polarography; specific ion electrode (using a chlorine-specific electrode); flow injection analysis; and ion chromatography. There is a range of accuracy and reliability within this set of methods.

Flow injection analysis is also reliable but its reported detection limit of approximately 0.1 mg/L as chlorite is poorer than that of ion chromatography.

*NOTE:* concentrations for chlorine dioxide, chlorite and chlorate determined by amperometric and DPD titrations are often expressed as mg/L as Cl2. This can lead to confusion, and when reporting results, the units in which the results are expressed must be stated clearly, ie, mg/L as Cl2 or mg/L as chlorite.

### Health considerations

Chlorite may occur in foods as a result of the uses of chlorine dioxide, sodium chlorate or sodium chlorite in flour processing, as a decolorizing agent for carotenoids and other natural pigments (using chlorine dioxide), as a bleaching agent in the preparation of modified food starch (sodium chlorite), and as an indirect additive in paper and paperboard products used for food packaging (sodium chlorite) (WHO 2016).

Chlorite was readily absorbed when administered to rats, then randomly distributed throughout the tissues. It was transformed mainly into chloride; chlorate may be formed in oxidising situations. There is a ready interconversion among these species in water (before administration to animals) and in the gut (after ingestion). Therefore, what exists in water or the stomach is a mixture of these chemical species (ie, chlorine dioxide, chlorite, chlorate) and possibly their reaction products with the gastrointestinal contents (USEPA 2000). Excretion is mainly via urine, about 90 percent in the form of chloride.

In a study with human volunteers, no adverse effects were observed after drinking-water with either chlorine dioxide or chlorite concentrations up to 5 mg/L for periods of 12 weeks.

Chlorite affects red blood cells, resulting in methaemoglobin formation in cats and monkeys. The primary and most consistent finding arising from exposure to chlorite in a number of species was oxidative stress resulting in changes in the red blood cells. This observation was supported by a number of biochemical studies conducted in vitro. Studies with human volunteers for up to 12 weeks did not identify any effect on blood parameters at the highest dose tested, 36 μg/kg bw per day (WHO 2017).

In the Stage 1 D/DBPR, USEPA proposed an MCLG for chlorite of 0.08 mg/L based on neurodevelopmental effects in a rat study. Subsequent to the proposal, USEPA reviewed and completed a peer review of a two-generation reproductive study of chlorite in Sprague-Dawley rats. In this study, male and female rats were administered sodium chlorite in drinking water at doses ranging up to 300 mg/L. Reproduction, fertility, clinical signs and histopathology were evaluated in two-generations of offspring. In the Stage 1 D/DBPR, USEPA finalised an MCLG of 0.8 mg/L for chlorite based on the RfD (0.03 mg/kg/day), an adult tap water consumption of two litres/day for a 70 kg adult and an assumed drinking water contribution of 80 percent of total exposure. The RfD of 0.03 mg/kg/day was derived based on a NOAEL of 35 ppm (3 mg/kg/day for the chlorite ion) for decreases in absolute brain and liver weight and lowered auditory startle amplitude at 70 and 300 ppm and the application of an uncertainty factor of 100. Although the RfD differed from that derived from Mobley et al (1990) of 0.003 mg/kg/day the use of a lower uncertainty factor in the assessment based on the CMA study yielded the same MCLG. USEPA did not revise the MCLG for chlorite in the Stage 2 D/DBPR. Copied from USEPA (2016).

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 chlorite.

The reference dose or RfD (USEPA 2000, 2006, 2009 and 2011) is 0.03 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 1 mg/L.

USEPA (2000) states that the human carcinogenicity of chlorite cannot be determined because of a lack of human data and limitations in animal studies.

Sodium chlorite was not carcinogenic following a number of long-term studies, although these were not conducted to current standards. The International Agency for Research on Cancer concluded in 1991 that sodium chlorite was not classifiable with respect to carcinogenicity to humans (Group 3). Sodium chlorite has given positive results in some, but not all, in vitro genotoxicity assays and in one of the two available in vivo mouse micronucleus assays involving intraperitoneal administration (WHO 2016).

### Derivation of Maximum Acceptable Value

WHO (2016) used the JECFA ADI of 0–0.03 mg/kg bw on the basis of the NOAEL of 3 mg/kg bw per day for reduced liver weight of F0 females and F1 males and females in a two-generation reproductive toxicity study in rats and a safety factor of 100 to allow for interspecies and intraspecies variability. This ADI is supported by the results of studies in human volunteers showing no adverse effects at this intake. Using the upper bound of the ADI of 0.03 mg/kg bw, a typical human body weight of 60 kg, the assumption that drinking-water contributes 80 percent of the total exposure and a typical consumption of 2 L of water per day, the provisional guideline value is calculated to be 0.7 mg/L (rounded figure). This guideline value is designated as provisional because use of chlorine dioxide as a disinfectant may result in the chlorite guideline value being exceeded, and difficulties in meeting the guideline value must never be a reason for compromising adequate disinfection.

The provisional MAV in the 2008 DWSNZ for chlorite in drinking-water was derived as follows (from WHO (2004/2011):

2.9 mg/kg body weight per day x 70 kg x 0.8 = 0.812 mg/L (rounded to 0.8 mg/L)

2 L per day x 100

where:

* no-observable-adverse-effects level = 2.9 mg/kg body weight per day, identified in a two-generation study in rats, based on lower startle amplitude, decreased absolute brain weights in two generations and altered liver weights in two generations
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.8
* average amount of water consumed by an adult = 2 L per day
* uncertainty factor = 100 (10 each for inter- and intraspecies variation).

The MAV is designated as provisional because use of chlorine dioxide as a disinfectant may result in the chlorite MAV being exceeded, and difficulties in meeting the MAV must never be a reason for compromising adequate disinfection.

The provisional MAV for chlorite in the 1995 and 2000 DWSNZ had been 0.3 mg/L, and was derived as follows:

1 mg/kg body weight per day x 70 kg x 0.8 = 0.3 mg/L

2 L per day x 100

where:

* no-observable-adverse-effects level = 1 mg/kg body weight per day based on a 90-day study in rats
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.8
* average amount of water consumed by an adult = 2 L per day
* uncertainty factor = 100 (10 each for inter- and intraspecies variation).

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# Chromium

CAS No. 7440-47-3.

### Maximum Acceptable Value (provisional)

Based on health considerations, the concentration of total chromium in drinking-water should not exceed 0.05 mg/L. WHO (2004/2011/2017) states that their guideline value is designated as provisional because of uncertainties in the toxicological database.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of hexavalent chromium (Cr(VI)) in drinking water should not exceed 0.05 mg/L. If the concentration of total chromium exceeds this value then a separate analysis for hexavalent chromium should be undertaken.

The maximum contaminant level or MCL for total chromium (USEPA 2009/2011) is 0.1 mg/L. The maximum acceptable concentration in Canada is 0.05 mg/L; chromium (III) is an essential element, the MAC is protective of health effects from chromium (VI).

The Prescribed Concentration or Value (PCV) for chromium in England and Wales is 0.05 mg/L. See Notes.

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

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

In August 2013 the California Department of Public Health proposed a maximum MCL for hexavalent chromium of 0.01 mg/L Cr (VI).

### Sources to drinking-water

#### 1 To source waters

Chromium is present in most soils and rocks and it can enter water naturally from weathering and run-off from soils. Chromite, the main chromium containing mineral, is found in ultramafic rocks such as dunites, peridotites, pyroxenites and serpentinites, or as the detrital mineral from weathering of these rocks. Most of the known chromite in New Zealand occurs as disseminated grains in dunites, or segregated in small discontinuous lenses in dunites and serpentinites. Chromite is highly resistant to weathering. Examples of chromite occurring in ultramafic rocks include d’Urville Island; Croisilles Harbour, Nelson; Whangamoa, Nelson; Dun Mountain Area, Nelson; Red Hill, Wairau Valley; Red Mountain, Northwest Otago. Detrital chromite is found in the Wellsford–Silverdale area in Northland; from near Te Kuiti; Lee River near Nelson; and in beach sands between Greymouth and Hokitika.

In general, the concentration of chromium in ocean water is much lower than that in lakes and rivers. The mean total chromium concentration in ocean water is 0.3 μg/L, with a range of 0.2–50 μg/L. In the suspended materials and sediment of water bodies, total chromium levels ranged from 1 to 500 mg/kg.

Chromium is used in a range of industries, especially leather tanning and timber treatment, eg, copper-chrome-arsenic (CCA). USEPA (2008) states that CCA usually contains 28–35 percent chromic acid (which is CrVI). Hexavalent chromium compounds are used in the metallurgical industry for stainless steel, chrome alloy and chromium metal production, in the chemical industry as oxidising agents in chrome plating, and in the production of other chromium compounds used in paints, dyes, explosives, ceramics and paper. The primary sources of hexavalent chromium in the atmosphere are chromate chemicals used as rust inhibitors in cooling towers and emitted as mists, particulate matter emitted during manufacture and use of metal chromates, and chromic acid mist from the plating industry.

In the environment, almost all naturally occurring chromium is in the trivalent form. There is a wide variety of commercially available trivalent chromium compounds, the most important ones being chromium(III) oxide and basic chromium sulfate. Trivalent chromium salts are used in textile dyeing, in the ceramic and glass industry and in photography. Chromium compounds are also added to drilling muds to reduce corrosion fatigue of drilling pipes, in heating and cooling coils, in sprinkler systems and it is present in some fertilisers and pesticides.

In the hexavalent state, chromium exists as oxo species such as CrO3 and CrO42- that are strongly oxidising. In solution, hexavalent chromium exists as hydrochromate (HCrO4-), chromate (CrO42-), and dichromate (Cr2O72-) ionic species. The proportion of each ion in solution is pH dependent. In basic and neutral pH, the chromate form predominates. As the pH is lowered (6.0 to 6.2), the hydrochromate concentration increases. At very low pH, the dichromate species predominate (taken from USEPA 1998).

Chromium is emitted into the air, not only by anthropogenic sources, but also by every combustion process, including forest fires, and even with volcanic dust.

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; chromium VI levels range from 290 to 6,300 mg/kg depending on land use, and chromium III can exceed 10,000 mg/kg for all the land uses.

#### 2 From treatment processes

The main contributors of chromium are as impurities in alum and steel pickle liquor-derived ferric chloride. It is likely some will end up in the sludge (DWI 2013).

#### 3 From the distribution system

It is possible that corrosion of metals in reticulation and plumbing systems could lead to levels of chromium at the tap being higher than those leaving the plant.

### Forms and fate in the environment

Chromium is present in the environment in the trivalent and hexavalent states although trivalent chromium occurs more commonly. Hexavalent chromium occurs infrequently in nature; its presence in water is generally the result of industrial and domestic chromium waste discharges.

Chromium in soil is present mainly as insoluble oxide and is not very mobile. Chromium(VI) appears to be much less strongly adsorbed to soils than chromium(III). The mobility of soluble chromium in soil will depend on the sorption characteristics of the soil. Living plants and animals absorb the hexavalent form in preference to the trivalent form, but once absorbed, the hexavalent chromium is reduced to the more stable trivalent state (INCHEM 2013).

Industrial effluents containing chromium, some of which is in the hexavalent form, are emitted into surface waters. Whether the chromium remains hexavalent until it reaches the ocean depends on the amount of organic matter present in the water. If organic matter is present in large quantities, the chromium(VI) may be reduced by, and chromium(III) adsorbed on, the particulate matter. If it is not adsorbed, the chromium(III) will form large, polynucleate complexes that are no longer soluble. The reduction of chromium(VI) to chromium(III) occurs rapidly under anaerobic conditions, and reducing conditions generally exist in deeper groundwaters. Most of the chromium released into water will ultimately be deposited in the sediment. The major dissolved species of chromium(III) are Cr3+, CrOH2+, Cr(OH)30, and Cr(OH)4-.

Total chromium concentrations in lake water generally do not exceed 0.005 mg/L. Mean chromium(III) concentrations of up to 0.002 mg/L have been reported for surface waters. Higher levels of chromium can be related to sources of anthropogenic pollution, with the highest levels of up to 40 mg chromium(III)/L near tannery discharges.

Studies have demonstrated that under certain circumstances copper, arsenic, and/or chromium can leach from treated wood into the surrounding soil or water. In general, most leaching takes place in the first few days and the extent and rate of leaching being highest for copper and lowest for chromium. Available field and laboratory studies suggest that leaching of metals is highly variable and is dependent on environmental conditions. Studies on sorption into soils from utility poles, have shown that the release of metals into soils/sediments from the base of treated wood, decks or utility poles or from the pressure treatment facilities, do not show a high degree of migration, either to groundwater or to the surface. In most cases, after migration of the metals a few meters down into soil, these metals attain the background level concentration of soil (USEPA 2008).

### Typical concentrations in drinking-water

Chromium was measured routinely in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. Of 1904 samples analysed between 1983 and 1989, six samples (0.3 percent) were equal to or exceeded the MAV of 0.05 mg/L.

The P2 Chemical Determinand Identification Programme, sampled from 897 zones, found chromium concentrations to range from “not detectable” (nd) to 0.12 mg/L, with the median concentration being “nd” (Limit of detection = 0.002 mg/L). The Priority 2 Identification Programme found 1 distribution zone supplying drinking-water to a total of 150 people with chromium at greater than the MAV (ESR 2001).

It is recommended that people using roof water should not burn treated wood, and if water is stored in a timber storage tank, the liner integrity is checked.

Total chromium concentrations in drinking-water are usually less than 0.002 mg/L although concentrations between 0.06 mg/L to 0.12 mg/L have been reported overseas. In major Australian reticulated supplies concentrations of total chromium range up to 0.03 mg/L, with typical concentrations usually less than 0.005 mg/L. A survey of Canadian drinking water supplies suggested that the maximum concentrations of chromium in raw water and in treated and distributed waters were 0.014 and 0.009 mg/L, respectively; the median concentration of chromium in three types of water was 0.002 mg/L. The median concentration in the US has been reported to be 0.01 mg/L.

247 water utilities in the US reported detecting chromium (hexavalent) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.093 mg/L.

6,995 water utilities in the US reported detecting chromium (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 1.80 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found <0.00053 mg/L chromium in the raw water and treated water.

DWI (2015) found (using their method referred to below) that chromium (VI) is the dominant species in raw and drinking water in the UK. There was little evidence of seasonal effects on the concentrations of chromium (III) and chromium (VI) in raw and finished drinking water. Generally, chromium concentrations reported were <1 μg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 62,917 drinking water samples for chromium (total) between 2013 and 2015, and found 31,773 samples exceeded the minimum reporting level (MRL) of 0.2 µg/L, and one sample contained >0.10 mg/L.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 62,837 drinking water samples for chromium VI between 2013 and 2015, and found 47,503 samples exceeded the minimum reporting level (MRL) of 0.03 µg/L (USEPA 2017).

### Removal methods

Cr(III) is more easily removed from drinking-water than Cr(VI). Being soluble, very little removal of Cr(VI) occurs by coagulation with alum or ferric sulphate. However, nearly 100 percent removal can be achieved by coagulation with ferrous sulphate. In this process, the Cr(VI) is reduced to Cr(III) by the ferrous ions, then precipitated as insoluble chromium hydroxide. Some pH manipulation is required to obtain the optimum pH conditions for the different steps of reduction and precipitation.

Lime-softening is effective for removal of Cr(III) but not Cr(VI).

Some chromium removal can be achieved by the use of activated carbon, but the efficiency of removal depends strongly on the pH and the type of carbon used.

Initial studies have shown that Cr(VI), in the form of the chromate ion, is removed efficiently by a variety of anion exchange techniques.

To avoid chromium entering the water as the result of corrosion of metals in the reticulation, the corrosiveness of the water should be minimised.

At bench-scale, commercially available SBA-IX resins were able to achieve 15,000 to 30,000 bed volumes of treatment prior to an 8 μg/L Cr(VI) treatment threshold. These high treatment capacity results are likely attributable to raw water quality conditions that are favourable for SBA-IX treatment, namely low concentrations of co-occurring parameters including sulfate and nitrate (WRF 2014).

DWI (2015) reports that the USEPA estimated the efficiency for chromium removal at 82–97 percent for RO. Nanofiltration has also been used for chromium removal and shows similar efficiency for both chromium (III) and chromium (VI).

WRF (2015 and 2016) describe the reduction/coagulation/filtration (RCF) process that uses ferrous iron to reduce Cr(VI) to Cr(III) (within five minutes) and co-precipitate and/or adsorb Cr(III) with iron hydroxide particles. Pilot testing showed that Cr(VI) values at or below 0.001 mg/L could be consistently achieved.

WRF (2016) found that weak base anion exchange proved to be an effective Cr(VI) treatment technology. After one year of testing and over 200,000 bed volumes, all the resins evaluated continued to produce treated water below the 0.01 mg/L MCL. Controlling pH was critical for this technology, though variations in water quality are less so. The primary impact of water quality was alkalinity, which affects the cost of achieving the pH target. Disposal considerations were examined, and all resins accumulated uranium, and could potentially become low-level radioactive wastes.

### Analytical methods

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Flame Atomic Absorption Spectrometric Method (APHA 3111).

2. Inductively Coupled Plasma Method (APHA 3120B).

3. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

DWI (2015) trialled an ICP-MS method to directly measure total chromium concentrations in water, then in combination with an ion chromatographic separation, measured the individual chromium species (chromium (III) and chromium (VI). The method was shown to be reproducible and precise, to have a linear range up to 50 μg/L and a minimum detectable amount for chromium of 0.1 μg/L. The limit of quantitation was 0.5 μg/L.

### Health considerations

Trivalent chromium is an essential trace element for humans, playing an essential role in insulin metabolism, with food being the major source of intake. Hexavalent chromium is not considered to be an essential nutrient and harmful effects due to chromium have been attributed to this form. Chromium(VI) is unstable in the body and is reduced to chromium(V), chromium(IV) and ultimately to chromium(III) by many substances, including ascorbate and glutathione. It is believed that the toxicity of chromium(VI) compounds results from damage to cellular components during this process (eg, generation of free radicals) (INCHEM 2013).

The absorption of chromium after oral exposure is relatively low and depends on the speciation. Cr(VI) is more readily absorbed from the gastrointestinal tract than Cr(III) and is able to penetrate cellular membranes. In general, food appears to be the major source of intake. The general population is exposed to trivalent chromium mainly from the daily diet. Chromium(III) is thought to be an essential nutrient required for normal energy metabolism (being involved in lipid and glucose metabolism), although reports of chromium(III) deficiency are rare and there is no recognised disease that is attributed to chromium deficiency as there is with most other essential minerals. See DWI (2015) for an extensive discussion.

INCHEM (2013) reports an oral tolerable daily intake for non-cancer effects of 0.9 μg chromium(VI) per kilogram body weight (bw) per day, derived from findings of diffuse epithelial hyperplasia in the duodenum observed in female mice after exposure to sodium dichromate dihydrate in drinking-water. This was based on a lower limit on the benchmark dose for a 10 percent response (BMDL10) of 0.094 mg/kg bw per day and application of an uncertainty factor of 100.

EFSA (2014) reports a TDI of 0.3 mg/kg bw per day for Cr(III) from the lowest NOAEL identified in an NTP chronic oral toxicity study in rats. Under the assumption that all chromium in food is Cr(III), the mean and 95th percentile dietary exposure across all age groups were well below the TDI and therefore does not raise concerns for public health. On the basis of a BMDL10 value of 0.11 mg Cr(VI)/kg b.w. per day for diffuse epithelial hyperplasia of the duodenum in female mice they also report low concern regarding Cr(VI) intake via drinking water (assuming chromium in all water intended for human consumption and natural mineral waters is CrVI) for all age groups when considering the mean chronic exposure values with the exception of infants at the upper bound (UB) exposure estimates.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes minimal risk levels (MRLs) for hexavalent chromium of:

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

0.0009 mg/kg/day for chronic-duration oral exposure (>364 days).

The reference dose or RfD for total chromium (USEPA 1998, 2006, 2009 and 2011) is 0.003 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.1 mg/L. As at May 2014, <http://water.epa.gov/drink/standards/hascience.cfm> quotes a RfD of 0.009 mg/kg/d for the calcium, magnesium and potassium chlorates, and 0.03 for sodium chlorate. These are their Human Health Benchmarks for Pesticides.

There are no adequate toxicity studies available to provide a basis for a no-observable-adverse-effects level. In a long-term carcinogenicity study in rats by the oral route with Cr(III), no increase in tumour incidence was observed. In rats, Cr(VI) is a carcinogen via the inhalation route, although the limited data available do not show evidence for carcinogenicity via the oral route. In epidemiological studies, an association has been found between exposure to Cr(VI) and lung cancer. The International Agency for Research on Cancer has classified Cr(VI) in Group 1 (carcinogenic to humans) and Cr(III) in Group 3 (not classifiable as to its carcinogenicity to humans).

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

USEPA (1998) states that applying the criteria outlined in the 1986 *Guidelines for Carcinogen Risk Assessment* for evaluating the overall weight of evidence for carcinogenicity to humans, hexavalent chromium is most appropriately designated a Group A – Known Human Carcinogen. Using the proposed 1996 *Guidelines for Carcinogen Risk Assessment*, hexavalent chromium is most appropriately designated a known human carcinogen by the inhalation route of exposure on the following basis. The potential carcinogenicity of chromium by the oral route of exposure cannot be determined at this time.

Cr(VI) compounds are positive in a wide range of *in vitro* and *in vivo* genotoxicity tests, whereas Cr(III) compounds are not. The mutagenic activity of Cr(VI) can be decreased or abolished by reducing agents, such as human gastric juices.

IARC (2009) states that “The Working Group reaffirmed the classification of beryllium and its compounds, cadmium and its compounds, chromium (VI) compounds, and nickel compounds as “carcinogenic to humans” (Group 1). Studies involved complex occupational exposures to a metal and its compounds, making it impossible to separately assess their carcinogenicity.”

MfE (2011) states:

Chromium in its trivalent state is an essential element, but at high concentrations, and particularly in its hexavalent state, it is toxic. There is limited data on which to base tolerable daily intakes for chromium. The United States Environmental Protection Agency (USEPA) recommends a toxicological intakes of 1,500 µg Cr(III)/kg bw/day and 3 µg Cr(VI)/kg bw/day and these values are recommended for use in New Zealand. Dermal absorption of chromium (III) is expected to be a negligible route of exposure for soil contamination and is not considered relevant here. It is recommended that the adverse effects arising from dermal exposure to chromium (VI) are considered separately to those arising from oral exposure and that allergic contact dermatitis is the main effect of interest. A soil contaminant standard protective from allergic contact dermatitis could be established, but as these effects are likely to be elicited at higher concentrations than those arising from oral exposure, a soil contaminant standard protective against effects arising from oral exposure will also protect against allergic contact dermatitis. Estimates of dietary intake of chromium (III) are based on nutrient reference values for different age groups from the US Institute of Medicine (IOM) as recommended by the Australian National Health and Medical Research Council (NHMRC).

The livestock guideline value for chromium (total) is 1 mg/L (ANZECC/ARMCANZ 2000). These guidelines were to have been updated in 2012.

### Derivation of Maximum Acceptable Value

The provisional MAV for chromium in drinking-water comes from the following WHO (2003/2017) assessment:

There are no adequate toxicity studies available to provide a basis for derivation a NOAEL. The guideline value was first proposed in 1958 for hexavalent chromium, based on health concerns, but was later changed to a guideline for total chromium because of difficulties in analysing for the hexavalent form only. In principle, because the health effects are determined largely by the oxidation state, different guideline values for chromium(III) and chromium(VI) should be derived. However, current analytical methods and the variable speciation of chromium in water favour a guideline value for total chromium.

Because of the carcinogenicity of Cr(VI) by the inhalation route and its genotoxicty, the 1984 MAV of 0.05 mg/L has been questioned, but the available toxicological data do not support the derivation of a new value. However, as a practical measure, 0.05 mg/L, which is considered to be unlikely to give rise to significant risks to health, has been retained as the provisional MAV until additional information becomes available and chromium can be re-evaluated.

WHO states that separate guideline values for chromium (III) and chromium (VI) should be derived, however, there are technical difficulties in analytically measuring chromium at different valencies. DWI (2015) reported a study to understand the significance of chromium in drinking water. On the assumption of drinking water only accounting for 20 percent of total exposure to chromium (VI), a health-based value of 5.4 µg/L can be derived. If more realistic assumptions were applied, ie, drinking water accounts for 80 percent of total exposure to chromium (VI), a derived health-based value of 21.6 μg/L would apply.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for chromium VI is 0.1 mg/L, and 20 mg/L for chromium III.

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# Cobalt

CAS No. 7440-48-4 (the metal).

### Maximum Acceptable Value

There is no MAV in the DWSNZ; the WHO Guidelines do not mention cobalt.

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

### Sources to drinking-water

#### 1 To source waters

Small amounts of cobalt are naturally found in most rocks, soil, water, plants, and animals. It is the 33rd most abundant element. Small amounts of cobalt may be released into the atmosphere from coal-fired power plants, incinerators and vehicular exhaust.

The concentration of cobalt in seawater is about 0.0003 mg/L.

Cobalt metal is mixed with other metals to form alloys, which are harder or more resistant to wear and corrosion. These alloys are used in a number of military and industrial applications such as gas turbine aircraft engines, magnets, and grinding and cutting tools. They are also used in artificial hip and knee joints. Cobalt compounds are used to colour glass (blue), ceramics, and paints, as catalysts (mainly in the petroleum industry), and as paint driers. Cobalt compounds are also used as trace element additives in agriculture and medicine.

The US Geological Survey (USGS) assessed data for 6,805 ambient surface water stations and estimated the geometric mean and median dissolved cobalt concentration as 0.003 and 0.002 mg/L respectively.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

Traces may be released from metallic components – see below.

### Forms and fate in the environment

Cobalt is a naturally-occurring element that has properties similar to those of iron and nickel. Cobalt deposited on soil is often strongly attached to soil particles and therefore does not travel very far and is not likely to reach groundwater. Most cobalt salts are soluble, with the cobalt cation occurring in the +2 valence state. Factors affecting the speciation and fate of cobalt in water, sediments, and soil include organic ligands such as humic acids, anions, pH, and redox potential. The soil mobility of cobalt is inversely related to the strength of adsorption by soil constituents. Although plants may take up cobalt from the soil, the translocation of cobalt from the roots to other parts of the plant is not significant.

### Typical concentrations in drinking-water

The concentration of cobalt in groundwater in the United States is generally low, usually between 0.001 and 0.01 mg/L.

Of 380 drinking-waters in the US, only 0.5 percent contained cobalt levels exceeding 0.001 mg/L; the maximum concentration found was 0.03 mg/L. These values are higher than the respective median and maximum levels of <0.002 and 0.006 mg/L found in Canadian finished drinking water.

In the US 3,834 grab samples of household tap water from 35 geographical areas were analysed. Cobalt was found in 9.8 percent of the samples at concentrations ranging from 0.003 to 0.11 mg/L. Presumably up to 0.1 mg/L of cobalt may be picked up from the distribution system or plumbing.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 62,982 drinking water samples for cobalt between 2013 and 2015, and found 833 samples exceeded the minimum reporting level (MRL) of 1 µg/L, and three samples contained >0.07 mg/L Co.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

In general, intake of cobalt from food sources is much greater than from drinking-water and air. The cobalt intake in food has been estimated to be 0.005–0.04 mg/day, mainly as bakery goods, cereal and vegetables. A biochemically important cobalt compound is vitamin B12 or cyanocobalamin. Vitamin B12 is essential for good health in animals and humans. Cobalt (at 0.16–1.0 mg/kg of body weight) has been used as a treatment for anaemia.

Approximately 50 percent of the cobalt that enters the gastrointestinal tract will be absorbed. Cobalt absorption is increased among individuals who are iron deficient. Water-soluble forms are better absorbed than insoluble forms.

In the 1960s, some breweries added cobalt salts to beer to stabilise the foam (resulting in exposures of 0.04–0.14 mg cobalt/kg). Some people who drank excessive amounts of beer (8–25 pints/day) experienced serious effects on the heart. In some cases, these effects resulted in death. Nausea and vomiting were usually reported before the effects on the heart were noticed. The effects on the heart, however, may have also been due to the fact that the beer drinkers had protein-poor diets and may have already had heart damage from alcohol abuse. Effects on the heart were not seen in people with anaemia treated with up to 1 mg cobalt/kg, or in pregnant women with anaemia treated with 0.6 mg cobalt/kg. Effects on the thyroid were found in people exposed to 0.5 mg cobalt/kg for a few weeks.

Short-term exposure of rats to high levels of cobalt in the food or drinking-water results in effects on the blood, liver, kidneys, and heart. Longer-term exposure of rats, mice, and guinea pigs to lower levels of cobalt in the food or drinking-water results in effects on the same tissues (heart, liver, kidneys, and blood) as well as the testes, and also causes effects on behaviour; relatively high doses are needed to effect these changes, usually >20 mg/kg/day.

Based on the animal data, the International Agency for Research on Cancer (IARC) has determined that cobalt is possibly carcinogenic to humans, Group 2B. Cobalt oxide and sulphate 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 0.01 mg/kg/day for intermediate-duration oral exposure (15–364 days) to cobalt.

The livestock guideline value is 5 mg/L (ANZECC/ARMCANZ 2000). These guidelines were to have been updated in 2012.

Cobalt sulfate 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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# Copper

CAS No. 7440-50-8 (metal). Copper naphthenate is discussed in the Pesticides section.

### Maximum Acceptable Value

Based on health considerations, the concentration of copper in drinking-water should not exceed 2 mg/L.

Based on aesthetic considerations (eg, staining of laundry and sanitary ware), the concentration of copper in drinking water should not exceed 1 mg/L (Guideline Value).

The action level (USEPA 2006/2011) is 1.3 mg/L at the tap; they also have a secondary drinking water regulation of 1.0 mg/L for copper. The aesthetic objective in Canada is not greater than 1 mg/L after flushing.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of copper in drinking water should not exceed 2 mg/L. Based on aesthetic considerations, the concentration of copper in drinking water should not exceed 1 mg/L.

The Prescribed Concentration or Value (PCV) for copper in England and Wales is 2 mg/L. See Notes.

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

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

The Environmental Protection Authority of New Zealand ([www.epa.govt.nz](http://www.epa.govt.nz)) has established an environmental exposure limit (EEL) for copper in fresh water (set by an approval under Part 5 of the HSNO Act) of 0.0014 mg/L (1.4 µg/L).

The USEPA established an organoleptic effect criterion of 1.0 mg/L for copper. 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>.

### Sources to drinking-water

#### 1 To source waters

Copper occurs widely in nature in rocks and soils as sulphide and carbonate minerals. Weathering of these minerals releases copper to the aquatic environment. In New Zealand the typical geological settings for the occurrence of copper are associated with volcanic and metamorphic rocks. Areas likely to have high levels of copper include Northland, Great Barrier Island, Ruahine Ranges, Cape Colville, Coromandel Peninsula, Egmont National Park, Nelson, Stewart Island, Southland, Northwest Otago, South Westland and Fiordland. NZFSA reported copper levels of 3.0 to 4.5 mg/kg in wheat in their 2010 season 1 food residues surveillance programme.

Geothermal waters in the Taupo Volcanic Zone can contain up to about 3 mg/L Cu (GNS 2015).

The concentration of copper in seawater is about 0.05 mg/L.

Copper is used in a range of industries including timber treatment, eg, as copper-chrome-arsenic where the copper oxide content is about 10–14 percent (USEPA 2008), the manufacture of electrical wiring, electroplating, production of alloys (eg, bronze and brass) and items made from these alloys, photography, utensils, antifouling paint, pesticide formulations, and textiles. It is also used in construction, roofing materials and brass and copper plumbing. It is used in gardening and horticulture as a fungicide where constant use has caused concern about the build-up of copper in soils having a detrimental effect on earthworms and bacterial activity, resulting in the search for degradable organic alternatives (Everett et al 2008). Severe effects of copper are expected at greater than 500 ppm copper in soil.

Copper salts may be added seasonally in small amounts to water supply reservoirs and lakes to suppress the growth of algae.

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; copper levels can exceed 10,000 mg/kg for all the land uses.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

An important source of copper is from the dissolution of domestic water pipes and plumbing fixtures. A number of factors determine the extent to which copper is dissolved from pipes and fittings. The chemical factors include: pH, carbon dioxide concentration, dissolved oxygen concentration, chlorine residual, hardness and alkalinity. Studies have shown that high carbon dioxide content is a major factor in determining the corrosiveness of a water towards copper tubing. In addition, the copper concentration in the water can be expected to be higher if the water in contact with the copper remains stagnant. Aggressive carbon dioxide should be removed from bore waters, or consumers should be advised to use plastic pipes and fittings.

### Forms and fate in the environment

The most common oxidation states of copper are +1 (cuprous) and +2 (cupric) with the cupric ion being dominant in aerated waters. Copper exhibits complex behaviour in the aquatic environment and may be present as the free cupric ion or complexed with inorganic or organic ligands. At pH levels and inorganic carbon concentrations characteristic of natural fresh waters, most of the soluble copper is present as complexes of cupric carbonate. Less than 1 percent of the total copper exists in the free ionic form in natural water.

Copper has a high affinity for hydrous iron and manganese oxides, clays, carbonate minerals and organic matter. Even though copper binds strongly to suspended particles and sediments, there is evidence to suggest that some water soluble copper compounds do enter groundwater. Copper that enters surface water eventually collects in the sediments of rivers, lakes, and estuaries. In reducing acidic environments, remobilisation of adsorbed or co-precipitated copper can occur. In the presence of soluble organic matter, adsorption of copper may be relatively ineffective, resulting in an increase in soluble forms in the water column.

Studies have demonstrated that under certain circumstances copper, arsenic, and/or chromium can leach from treated wood into the surrounding soil or water. In general, most leaching takes place in the first few days and the extent and rate of leaching being highest for copper and lowest for chromium. Available field and laboratory studies suggest that leaching of metals is highly variable and is dependent on environmental conditions. Studies on sorption into soils from utility poles, have shown that the release of metals into soils/sediments from the base of treated wood, decks or utility poles or from the pressure treatment facilities, do not show a high degree of migration, either to groundwater or to the surface. In most cases, after migration of the metals a few meters down into soil, these metals attain the background level concentration of soil (USEPA 2008).

### Typical concentrations in drinking-water

Copper was routinely measured in New Zealand drinking-water supplies as an aesthetic parameter as part of the Department of Health three yearly surveillance programme. Copper is now considered to be an inorganic parameter of health significance. Of 1143 samples analysed from 913 supplies, between 1983 and 1989, 2284 samples (31 percent of supplies) exceeded the then highest desirable 1984 MAV of 0.05 mg/L.

The P2 Chemical Determinand Identification Programme, sampled from 897 zones, found copper concentrations to range from “not detectable” (nd) to 10 mg/L, with the median concentration being 0.032 mg/L (Limit of detection = 0.002 mg/L). The Priority 2 Identification Programme found 24 distribution zones supplying drinking-water to a total of 13,405 people with copper at greater than the MAV (ESR 2001).

20,241 water utilities in the US reported detecting copper in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 7.0 mg/L.

Health Canada (1992) reports that the median copper concentration in distributed water was 0.27 mg/L (range <0.01 to 0.90 mg/L) for 27 supplies with acid or neutral pH (90 percent with pH 5.3 to 7.5), adding that distributed water contains considerably more copper than the original water supply because of the dissolution of copper from copper piping, which is used extensively in domestic plumbing in Canada.

Accurately measuring copper levels in tap water fed from copper tubing is almost pointless if there is any sign of corrosion. Concentrations will depend on retention time, water velocity, volume collected, age of copper pipe, seriousness of corrosion taking place, and the length and design of the plumbing.

### Removal methods

The copper found in drinking-waters is usually not the result of the metal being present in the source water. It generally arises because of corrosion of fittings in the consumers’ plumbing.

Correct pH adjustment, removal of dissolved carbon dioxide, maintenance of a moderate alkalinity, avoidance of high chloride levels, and minimisation of aluminium carry-over from the coagulation process are ways in which a water’s tendency to dissolve copper can be reduced. In some instances, replacement of copper piping with plastic piping may be required to reduce severe copper problems.

Flushing pipes before drinking-water is drawn from the tap can help to reduce high copper concentrations that arise from corrosive water being in contact with the piping for extended periods of time.

Should copper be present in the raw water, alum coagulation at pH 7 is effective in its removal, provided the copper is in a particulate form or is complexed to organic matter.

### Analytical methods

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Flame Atomic Absorption Spectrometry (APHA 3111).

2. Inductively Coupled Plasma Method (APHA 3120B).

3. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

### Health considerations

Copper is an essential element, and the intake from food is normally 1–3 mg/day. The WHO has recommended a daily intake of 30 µg/kg body weight per day (or 2.1 mg/day) for an adult male and 80 µg/kg body weight per day for infants. Copper is particularly rich in shellfish (especially oysters), and nuts.

Copper is generally not an issue where CCA-treated timber has been used or disposed of if health issues related to arsenic and chromium have been addressed.

In adults, the absorption and retention rates of copper depend on the daily intake; as a consequence, copper overload is unlikely. The absorption of copper by the gastro-intestinal tract is dependent on a number of factors including pH and copper speciation, but is probably 25–60 percent effective. Copper is stored in the brain and muscle tissue. High concentrations can also be found in the kidneys, heart and hair.

IPCS (1998) concluded that the upper limit of the acceptable range of oral intake in adults is uncertain but is most likely in the range of several (meaning more than two or three) but not many milligrams per day in adults. This evaluation was based solely on studies of gastrointestinal effects of copper-contaminated drinking-water. The available data on toxicity in animals were not considered helpful in establishing the upper limit of the acceptable range of oral intake due to uncertainty about an appropriate model for humans, but they help to establish a mode of action for the response.

The data on the gastrointestinal effects of copper must be used with caution, since the effects observed are influenced by the concentration of ingested copper to a greater extent than the total mass or dose ingested in a 24-hour period. For example, a single glass of tap water with a concentration greater than 3 mg of copper per litre is more likely to elicit nausea than a litre of water containing the same amount (mass) of copper, but ingested episodically throughout a day.

Recent studies have delineated the threshold for the effects of copper in drinking-water on the gastrointestinal tract, but there is still some uncertainty regarding the long-term effects of copper on sensitive populations, such as carriers of the gene for Wilson disease and other metabolic disorders of copper homeostasis.

Acute gastric irritation may be observed in some individuals at concentrations in drinking-water above 3 mg/L. Brodlo et al (2005) reported a case in an Australian school where several pupils became ill. Copper concentrations in the range of  
6–43 mg/L were found in the initial response sampling. Copper levels fell to 1–3 mg/L after remedial action, with no further illness.

In adults with Wilson’s disease and other metabolic disorders of copper homeostasis, the copper regulatory mechanism is defective, and long-term ingestion can give rise to liver cirrhosis.

Copper metabolism in infants, unlike that in adults, is not well-developed, and the liver of the newborn contains about 90 percent of the body burden, with much higher levels than in adults. Since 1984, there has been some concern regarding the possible involvement of copper from drinking-water in early childhood liver cirrhosis in bottle fed infants, although this has yet to be confirmed.

The Acceptable Daily Intake (ADI) adopted in Australia for copper is 0.2 mg/kg body weight.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes minimal risk levels (MRLs) of:

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

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

Recent studies in rabbits have suggested a link between copper in drinking-water and Alzheimer’s disease. However, levels in the rabbits’ drinking-water were well below the current WHO guideline of 2 mg/L, which is based on acute gastrointestinal effects. It has not been established whether rabbits are an appropriate model. In addition, a communication received by WHO Headquarters suggested the need to review the guideline value and text on copper with regard to toxicity in the preparation of formula for bottle-fed infants.

Copper was not found to be carcinogenic in tests with mice and dogs. The results of mutagenicity tests with different strains of bacteria are generally negative. Tests for mutagenicity using mammalian cells, both *in vitro* and *in vivo*, give predominantly positive results.

MfE (2011) states:

Copper is an essential element, and adverse effects can arise both from copper deficiency and from excess copper intake. Liver damage is the critical endpoint for intake of high levels of copper in animal and human studies. The tolerable upper limit of 10 mg/day, based on liver function and converted using a 70-kg bodyweight, is used to derive a toxicological intake value of 0.15 mg/kg bw/day. Dermal absorption and inhalation are expected to be negligible routes of exposure and are not considered relevant for soil contamination. Dietary intake is the primary source of background exposure to copper. Estimated dietary intake for a child aged 5–6 years was 0.06 mg/kg bw/day and for an adult (25–44 years) was 0.02 mg/kg bw/day, which is within the recommended dietary intake for copper.

The livestock guideline value is 0.4 mg/L for sheep, 1 mg/L for cattle and 5 mg/L for poultry (ANZECC/ARMCANZ 2000). If animal diets are high in copper, the levels in drinking water should be revised downwards. Animal intake of sulfur and molybdenum should also be considered in conjunction with copper. These guidelines were to have been updated in 2012.

### Derivation of the Maximum Acceptable Value

Basis of guideline value (WHO 2017): to be protective against acute gastrointestinal effects of copper and provide an adequate margin of safety in populations with normal copper homeostasis.

For adults with normal copper homeostasis, the MAV should permit consumption of two or three litres of water per day, use of a nutritional supplement, and copper from foods, without exceeding the tolerable upper intake level of 10 mg/day or eliciting an adverse gastrointestinal response.

The data on the gastrointestinal effects of copper must be used with caution, as the effects observed are influenced by the concentration of ingested copper to a greater extent than the total mass or dose ingested in a 24-hour period (WHO 2017). A concentration of 2 mg/L should contain a sufficient margin of safety for bottle-fed infants, because copper intake from other sources is usually low.

The aesthetic guideline value for copper is 1 mg/L for the staining of laundry and sanitary ware, and at levels above 2.5 mg/L, copper imparts an undesirable bitter taste to the water. At concentrations of about 1 mg/L Cu, a pH of 7 is the approximate dividing line between soluble (pH <7) and particulate (pH >7). Alkalinity and carbonate also influence the solubility of copper at basic pH values; taken from WRF (2015). While copper is considered to have a taste, copper actually has only a slight taste and a significant retronasal odour that provides its flavour. The cause of the retronasal odour is lipid oxidation that is induced by copper ions reacting with lipids and salivary components in the oral cavity. Recent flavour tests reveal that the human threshold for soluble copper is in the range of 0.4 to 0.8 mg/L in distilled water or tap water. Particulate copper is poorly detected by taste or flavour and may not be perceived when ingested, even up to concentrations of 8 mg/L. Although unusual in most homes, high levels of electric current (AC greater than 5 amps and DC greater than 2 amps) can lead to release of copper from copper pipe when they are used as a ground for home electrical wiring.

The 1995 version of the *Guidelines* explained the derivation of the 2 mg/L MAV as follows:

As copper has not been found to be carcinogenic, a tolerable daily intake (TDI) approach has been used for the derivation of the MAV. In 1982, the Joint FAO/WHO Expert Committee on Food Additives proposed a provisional maximum tolerable daily intake of 0.5 mg/kg body weight, based on end-point liver toxicity in a rather old study in dogs and considering the essentiality of copper. This value has been used for the derivation of the MAV. However, in view of the remaining uncertainties regarding copper toxicity in humans, the MAV is considered provisional.

The 1995 MAV for copper in drinking-water was derived as follows:

0.5 mg/kg body weight per day x 70 kg x 0.1 = 2 mg/L

2L per day

where:

* tolerable daily intake = 0.5 mg/kg body weight per day (WHO 1982)
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* average amount of water consumed by an adult = 2L per day.

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# Cyanide

CAS No. 57-12-5 (metal).

### Maximum Acceptable Value

Cyanide occurs in drinking-water at concentrations well below those of health concern, except in emergency situations following a spill to a water source. Because cyanide is unlikely to occur in drinking-water at concentrations of health concern, it is considered unnecessary to derive a formal guideline value for short-term exposure to cyanide; a health-based value of 0.5 mg/L for short-term exposure can be calculated (WHO 2011/2017).

The DWSNZ in 2008 had stated: based on health considerations, the concentration of cyanide (as total CN) in drinking-water should not exceed 0.6 mg/L. This is a short term MAV, and was based on WHO (2004), adjusted for 70 kg bw.

In DWSNZ 2005, the MAV had been 0.08 mg/L.

The Prescribed Concentration or Value (PCV) for cyanide in England and Wales is 0.05 mg/L. See Notes.

The maximum contaminant level or MCL for free cyanide\* (USEPA 2009/2011) is 0.2 mg/L. The USEPA (2009) also established a lifetime health advisory of 0.2 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 2 litres of water per day. The Lifetime HA for Group C carcinogens includes an adjustment for possible carcinogenicity. USEPA (2011) no longer lists this lifetime health advisory.

\* Free cyanide is defined as the sum of the cyanide present as either HCN or CN-. Under typical conditions in natural waters (pH 6 to 8.5 and 4 to 10°C), over 90 percent of cyanide is in the form of molecular HCN. Conversion of cyanide to a less toxic cyanate occurs at pH levels of 8.5 and above.

The maximum acceptable concentration in Canada is 0.2 mg/L.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of cyanide in drinking water should not exceed 0.08 mg/L.

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

EPA established an environmental exposure limit of 0.00018 mg/L (0.18 µg/L) for cyanide in fresh water (<http://www.epa.govt.nz/search-databases/Pages/substance-exposure-limit-register.aspx>).

### Sources to drinking-water

#### 1 To source waters

Hydrogen cyanide is ubiquitous in nature. The natural decomposition of some plants that synthesise cyanoglycosides produce free cyanide as a result of their metabolic processes and may release cyanide to source waters. Apricot pips are one of the richest sources of plant-origin cyanide found in New Zealand, containing 89–2170 mg/kg of cyanide, and cherry juice has been reported to contain about 5 mg/L (WHO 2007; NZFSA). Almonds, millet sprouts, lima beans, soy, cabbage, spinach, sorghum, bamboo shoots, and cassava (also called tapioca and manioc) roots are also reported to contain cyanide or contain cyanogen glycosides that can release hydrogen cyanide upon biodegradation or ingestion. See also cyanogen chloride datasheet.

Some micro-organisms, such as the cyanobacterium *Anacystis nidulans* and the bacterium *Chromobacterium violaceum*, produce free cyanide.

Cyanide may also enter water via the discharge of wastes from industries in which it is used (eg, electroplating and metal cleaning), and from agricultural practices. These uses, and transport, could give rise to spills, which result in short-term emergency situations, hence the derivation of a short-term MAV.

Various cyanide-containing products 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)). A slow-release product, ferotox, is an encapsulated cyanide pellet containing up to 80 percent potassium cyanide used for possum and rat control. Secondary poisoning of predators is unlikely to occur when baiting with cyanide (DoC 2005).

Cyanide is used to manufacture a range of other chemicals, in some chemical industries, in insecticides and rodenticides, and sodium cyanide is used in the extraction of gold and silver from low-grade ores. In uncontaminated water sources, free cyanide concentrations are usually less than 0.01 mg/L. While many chemical forms of cyanide are used in industrial application or are present in the environment, the cyanide anion CN- is the primary toxic agent, regardless of origin.

Hydrogen cyanide is formed during the incomplete combustion of nitrogen-containing polymers such as certain plastics, polyurethanes, and wool. Hydrogen cyanide is present in cigarette smoke. One cigarette without a filter liberates 0.5 mg hydrogen cyanide, while filter cigarettes liberate only 0.1 mg in mainstream smoke. The average rate of emission of hydrogen cyanide by automobile exhaust was reported to be  
7–9 mg/km for cars not equipped with catalytic converters and on the order of 0.6 mg/km for cars with catalytic converters operating under optimum conditions in the mid- to late 1970s (ex WHO CICAD 61, 2004).

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

The Priority 2 Identification Programme found no distribution zones supplying drinking-water with cyanide at greater than the MAV (ESR 2001).

Cyanides are a diverse group of organic and inorganic compounds, characterised by the -CN group. The form of cyanide in water is dependent primarily on pH, and also on temperature, dissolved oxygen, salinity and the presence of other ions, complexing agents and sunlight. Most cyanide in surface water will form hydrogen cyanide and evaporate. Although there appears to be no specific data, the half-life of cyanides in water is likely to be weeks.

Cyanides are readily soluble in water and fairly mobile in soil. Some cyanide compounds in soil can form hydrogen cyanide and evaporate, whereas some cyanide compounds will be transformed into other chemical forms by micro-organisms in soil. Consequently, cyanides usually do not seep into groundwater.

### Typical concentrations in drinking-water

Cyanide was not measured routinely in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme.

During the P2 Chemical Determinand Identification Programme, cyanide was found in only one zone, at a concentration equal to the limit of detection (0.01 mg/L).

In major Australian reticulated supplies cyanide concentrations range up to 0.05 mg/L, with typical concentrations usually less than 0.02 mg/L.

Cyanides are usually found only at very low concentrations in drinking-water sources. According to the USEPA STORET database, the mean cyanide concentration in most surface waters in the USA is less than 3.5 μg/L, ie, less than 0.0035 mg/L (WHO 2007).

787 water utilities in the US reported detecting cyanide in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.57 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found <0.001 mg/L total cyanide in the raw water and the treated water.

### Removal methods

Chlorine oxidation, reverse osmosis, and ion exchange have been used for the removal of cyanide from drinking-water. Note that some cyanide-containing complexes are resistant to oxidation by chlorine. Cyanide in this form, however, is unlikely to present a health concern as it will be very strongly bound to the complexing metal ion. Chlorine converts cyanide to cyanogen chloride almost instantaneously, but the pH needs to be above 8 to oxidise this to cyanate. Ozone is also an effective oxidant, particularly if the water is alkaline.

Activated carbon can be used to adsorb cyanide. The amount adsorbed has been reported to be 2–3 mg/g, as determined by adsorption isotherm tests. The addition of copper increased the capacity to 25 mg/g (WHO 2007). The USEPA, see <http://water.epa.gov/drink/contaminants/basicinformation/cyanide.cfm> states that granular activated carbon in combination with packed tower aeration have proven to be effective for removing cyanide to below 0.2 mg/L.

Regular low levels of cyanide are rare. Cyanide is more likely to reach drinking-water sources, particularly surface waters, as a consequence of accidental releases that can be very large. Under these circumstances, rather than attempting to remove the cyanide, there is a need to consider whether abstraction should be stopped for a limited period while the highest concentrations in a plume of pollution pass the intake to the drinking-water treatment works.

### Analytical methods

#### Referee method

Weak Acid Dissociable Cyanide (4500-CN I)  
This method measures the concentration of HCN and forms of cyanide that readily convert to HCN or CN-, and therefore best represents acutely toxic cyanide.

#### Some alternative methods

The following methods may be useful for the analysis of different forms of cyanide:

1. Cyanides Amenable to Chlorination after Distillation (APHA 4500-CN G).

After part of the sample is chlorinated to decompose the cyanides, both the chlorinated and untreated samples are distilled as described above. The difference in cyanide concentration between the two samples is expressed as the cyanides amenable to chlorination.

Some organic compounds may oxidise or breakdown during chlorination giving higher results for cyanide after chlorination than before chlorination.

2. Cyanides Amenable to Chlorination without Distillation (APHA 4500-CN H).

This method covers the determination of HCN and of CN complexes that are amenable to chlorination and also thiocyanates. The cyanides are oxidised by chloramine T after the sample has been heated. Addition of pyridine-barbituric acid reagent produces a red-blue colour which is measured photometrically at 578 nm. Thiocyanate interferes with this method and it is not applicable if the ratio of SCN to CN is greater than 3.

3. Total Cyanide after Distillation (APHA 4500-CN C).

This method includes complex forms of cyanide that do not readily dissociate to HCN or CN- in drinking-water.

### Health considerations

Cyanide is highly acutely toxic. It is detoxified in the liver by first-pass metabolism following oral exposure. As a consequence, exposure to a dose spread over a longer period, through a day, for example, will result in lower toxicity, or higher tolerance, than the same dose given in a single bolus dose (WHO 2009/2017).

It is readily absorbed by the gastrointestinal tract and metabolised to the much less toxic thiocyanate. Although thiocyanates are less harmful than cyanide in humans, they are known to affect the thyroid glands, reducing the ability of the gland to produce hormones that are necessary for the normal function of the body. Cyanide can deplete vitamin B12 levels and has been linked to an increased incidence of goitre and cretinism. Nutritionally inadequate people are most at risk. The major source of cyanide exposure for the general population not exposed to high levels of cyanogenic glycosides in food, is cigarette smoke. Long term exposure to lower concentrations of cyanide in occupational settings can result in a variety of symptoms related to central nervous system effects.

It has been estimated that death occurred after absorption of an average of 1.4 mg of hydrogen cyanide per kilogram of body weight; the lowest fatal absorbed dose was 0.54 mg of hydrogen cyanide per kilogram of body weight. Some individuals ingesting 1–3 g of cyanide salts have survived. Daily oral doses of 2.9–4.7 mg cyanide are generally considered to be non-injurious to humans owing to the efficient detoxification of cyanide to thiocyanate (WHO 2007).

Effects on the thyroid and particularly the nervous system were observed in some populations in the tropics as a consequence of the chronic consumption of inadequately processed cassava containing high cyanide concentrations.

Animal studies have suggested that oral exposure to cassava (a cyanide-containing vegetable) may be associated with malformations in the fetus and low fetal body weights. However, the USEPA has classified cyanide as a Group D, not classifiable as to human carcinogenicity. Tests for mutagenicity with different strains of bacteria have been mostly negative.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.05 mg/kg/day for intermediate-duration oral exposure (15–364 days) to sodium cyanide.

The reference dose or RfD (USEPA 2009) was 0.02 mg/kg/d as hydrogen cyanide. The Drinking Water Equivalent Level or DWEL (USEPA 2009) was 0.8 mg/L. The RfD (USEPA 2010/2011) has been changed to 0.0006 mg/kg/d as HCN. The Drinking Water Equivalent Level or DWEL has been removed from USEPA (2011).

### Derivation of Maximum Acceptable Value

WHO (2011/2017) states:

The data on acute exposure to cyanide are unsuitable for use in deriving a health-based value for short-term exposure because of the high uncertainty surrounding the data. Using the NOAEL for effects on the reproductive organs of male rats in a 13-week study and an uncertainty factor of 100, a TDI of 0.045 mg/kg body weight can be derived. Because this health-based value is intended for short-term use and exposure would not exceed 5 days, it is considered to be acceptable to allocate 40 percent of the TDI to drinking-water to allow for exposure to cyanogenic glycosides in food. Therefore, assuming a 60 kg adult drinking 2 litres of water per day with an allocation of 40 percent of the TDI to drinking-water, a health-based value of 0.5 mg/L (rounded value) for short-term exposure can be calculated. (This equates to 0.6 mg/L for 70 kg bw).

This health-based value is well below the level that is normally considered to be of health concern for humans. Cyanide is rapidly detoxified, and exposure spread throughout the day will further reduce the potential for effects. This health-based value would be suitable for use for a limited period of up to five days, which is the longest period likely to be required under the circumstances of such an emergency. However, it is probable that, in most circumstances, this value will be highly conservative for short-term exposure.

It should be noted that the lowest reported odour threshold for cyanide in drinking-water is 0.17 mg/L, which is below the short-term health-based value. It is therefore possible that a small number of individuals will detect cyanide by odour at concentrations below the health-based value. The health-based value relates to total cyanide concentration at the tap, including cyanide from cyanogen chloride in drinking-water as a by-product of disinfection with chlorine.

The 2004 WHO guideline value for cyanide had been considered to be protective for acute and long-term exposure.

However, the WHO (2007) background document states that as the low levels of cyanide found in drinking-water are mostly as a consequence of the presence of cyanogen chloride, it is not considered necessary to develop a guideline for long-term exposure to cyanide. This publication states that while low concentrations of cyanide in water sources can occur, these are easily removed by treatment, such as with chlorine. The objective of this document is, therefore, to consider cyanide in the context of short-term exposure following a significant spill of cyanide to a drinking-water source water.

The short-term exposure level for cyanide in drinking-water, derived by WHO (2007) and adjusted for 70 kg bw, is:

4.5 mg/kg body weight per day x 70 kg x 0.4 = 0.63 mg/L (rounded to 0.6 mg/L)

2 L per day x 100

where:

* no-observed-adverse-effect level = 4.5 mg cyanide per kg body weight, based on a 13‑week study
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.4
* uncertainty factor = 100 (10 for intraspecies variation and 10 for interspecies extrapolation)
* average amount of water consumed by an adult = 2 L per day.

This value is well below the level that is normally considered to be injurious to health for humans. Cyanide is rapidly detoxified, and exposure spread throughout the day will further reduce the potential for effects. It would be suitable for use for a limited period of up to five days, which is the longest period likely to be required under the circumstances of such an emergency. However, it is probable that, in most circumstances, this value will be highly conservative for short-term exposure.

The DWSNZ 2005 MAV for cyanide in drinking-water had been derived as follows:

There is a very limited number of toxicological studies suitable for deriving a MAV. There is, some indication in the literature that pigs may be more sensitive than rats. There is only one study available in which a clear effect level was observed, at 1.2 mg/kg body weight per day, in pigs exposed for six months. The effects observed were in behavioural patterns and serum biochemistry. This study has been used as the basis of the lowest-observable-adverse-effects level used in the derivation of the MAV.

1.2 mg/kg body weight per day x 70 kg x 0.2 = 0.084 mg/L (rounded to 0.08 mg/L)

2 L per day x 100

where:

* lowest-observable-adverse-effect level = 1.2 mg/kg body weight per day in a six-month study in pigs
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.2
* uncertainty factor = 100 (for inter- and intraspecies variation; no additional factor for the use of a LOAEL was considered necessary because of doubts over the biological significance of the observed changes)
* average amount of water consumed by an adult = 2 L per day.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for free cyanide is 0.1 mg/L.

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# Cyanogen chloride

CAS No. 506-77-4. The IUPAC name is cyanic chloride; also called chloromethanenitrile and chlorine cyanide.

Cyanogen (CAS No. 460-19-5) is also called carbon nitrile, dicyanogen, ethane dinitrile, and oxalic acid dinitrile. See pesticide datasheet, listed as ethanedinitrile.

Cyanogen bromide (CAS No. 506-68-3) may also be encountered.

### Maximum Acceptable Value

WHO (2011/2017) states that cyanogen chloride occurs in drinking-water at concentrations well below those of health concern, hence a guideline value does not need to be established.

DWSNZ (2008) state: based on health considerations, the concentration of cyanogen chloride in drinking-water should not exceed 0.4 mg/L.

In DWSNZ 2005, the MAV had been 0.08 mg/L.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of total cyanogenic compounds (measured as cyanide) in drinking water should not exceed 0.08 mg/L.

### Sources to drinking-water

#### 1 To source waters

Cyanogen chloride may be found in raw water as an industrial contaminant. It is used in tear gas, in fumigant gases, and as a reagent in the synthesis of other compounds.

Cyanogen is used as a fumigant, as a fuel gas for welding and cutting heat-resistant metals, and as a rocket and missile propellant. Cyanogen is generated in the combustion of nitrogen–carbon compounds and appears in automobile exhaust gases and gases from blast furnaces.

#### 2 From treatment processes

Cyanogen chloride may be formed as a by-product of chloramination or chlorination of water, with higher formation at acidic and neutral pH; it is unstable in the presence of free chlorine. It is also formed by the chlorination of any cyanide ion present in the raw water.

Cyanogen halide formation was observed at the treatment plants where chloramines were used, although cyanogen halide precursors were present in most of the water sources under investigation. In the chloraminated waters, the formation of cyanogen halides ranged from 0.7 to 8.4 μg/L, among which cyanogen chloride (CNCl) was the predominant species. Low formation of cyanogen halide was found in treatment plants where ozone was applied prior to biofiltration, suggesting that biological treatment can effectively remove formadehyde and other cyanogen halide precursors resulting from ozonation. The nitrogen in cyanogen halides can be derived from both chloramines, through the formaldehyde chloramination pathway, and from nitrogenous precursors. In the latter case, cyanogen halide formation was found to be dependent on the type and structure of N-organic compounds; only certain amino acids, such as glycine, serine, and threonine were reported to yield significant quantities of cyanogen halides; glycine may be the most important cyanogen halide precursor tested. Tyrosine also produced significant cyanogen halide levels, indicating that phenolic groups in such compounds can also generate cyanogen halides (DWI 2010).

Bromine species such as monobromamine and dibromamine, formed during ozonation, were also found to outcompete chloramines in reacting with cyanogen ion, and thus forming CNBr.

Cyanogenic glycosides or cyanoglycosides account for approximately 90 percent of the wider group of plant toxins known as cyanogens. The key characteristic of these toxins is cyanogenesis, the formation of free hydrogen cyanide, and is associated with cyanohydrins that have been stabilised by glycosylation (attachment of sugars) to form the cyanogenic glycosides. Examples of cyanogenic glycosides include linamarin from cassava and amygdalin from the seeds of stone fruit. The amount of cyanogenic glycosides in plants is usually reported as the level of releasable hydrogen cyanide. The major edible plants in which cyanogenic glycosides occur are almonds, sorghum, cassava, lima beans, stone fruits and bamboo shoots. Potential toxicity of cyanoglycosides arises from enzymatic degradation to produce hydrogen cyanide, resulting in acute cyanide poisoning. The enzyme responsible (β-glucosidase) may arise from the plant material or from gut microflora. See Cyanide datasheet for further information. NZFSA.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

Cyanogen chloride is a volatile gas. Hydrolysis is an important removal mechanism for cyanogen chloride in water.

Water solubility of cyanogen chloride is 15.8 percent, cyanogen bromide 10.8 percent and cyanogen 45 percent.

### Typical concentrations in drinking-water

Studies from the USA indicate that concentrations found in finished waters are below 0.01 mg/L. Concentrations tend to be higher in chloraminated waters than in chlorinated waters.

Cyanogen chloride was not detected in any zones sampled during the P2 Chemical Determinand Identification Programme (Limit of detection = 0.01 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with cyanogen chloride at >50 percent of the MAV of 0.08 mg/L (ESR 2001).

WHO (2004) reported cyanogen chloride concentrations in drinking-water treated with chlorine and chloramine at 0.0004 and 0.0016 mg/L, respectively.

Four water utilities in the US reported detecting cyanogen 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 0.0037 mg/L.

DWI (2012) reported a UK study on nitrogen-containing disinfection by-products. The ozonated systems produced higher cyanogen chloride concentrations which agreed with the previous understanding of the formation of this compound. Cyanogen chloride was detected less frequently than the other N-DBPs and with larger variability in the detected concentrations between and within sampling rounds. The mean concentration was 0.0058 mg/L (5.8 µg/L).

### Removal methods

No information is available for methods to remove cyanogen chloride from contaminated source waters.

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 chlorine or chloramine.

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 the primary coagulant. 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 difficulties.

Cyanogen chloride is known to be unstable in chlorinated water, and can be removed by chemical reduction agents such as sodium sulphite, sodium bisulphite and sodium thiosulphate (WHO 2007).

### Analytical methods

#### Referee method

APHA 5710 D, GC/MS purge and trap.

#### Some alternative methods

1. USEPA Method 524.2, in which purge-and trap gas chromatography is combined with mass spectroscopy, can be used for the determination of cyanogen chloride. This method has a practical quantification limit of 0.0003 mg/L (USEPA 1991).

2. Cyanogen chloride colorimetric method (APHA 4500-CN J).

3. See DWI (2010).

### Health considerations

Cyanogen chloride is rapidly metabolised to cyanide in the body.

Effects of ingested cyanogen chloride in humans have not been reported. Cyanogen chloride was used as a nerve gas in World War I. A concentration of 48 ppm in air was lethal and inhalation of concentrations above 1 ppm causes irritation.

No data are available on the carcinogenicity or mutagenicity of cyanogen chloride.

Based on a daily drinking-water consumption of two litres for an adult, the daily intake of cyanogen chloride is estimated to be 0.9–1.6 μg (equivalent to 0.4–0.7 μg of cyanide) for cyanogen chloride concentrations in water of 0.45–0.80 μg/litre  
(0.19–0.34 μg cyanide/litre) (ex WHO CICAD 61, 2004).

The USEPA Office of Water derived a 10-day health advisory value for cyanogen chloride of 0.1 mg/L base on a LOAEL of 14 mg/kg/day in a study of cyanide by Palmer and Olsen (1979), with the application of a 1,000-fold uncertainty factor. Cyanogen chloride is transformed to cyanide in water with any residuals metabolised to cyanide when consumed. Free cyanide in water is currently regulated with a MCL/MCLG of 0.2 mg/L based on protection against nerve damage and thyroid problems (USEPA 1992b). The recent IRIS assessment (USEPA 2010b) updated the hydrogen cyanide and cyanide salts RfD to a value of 0.0006 mg/kg/day based on testicular effect in male rats (NTP 1993). The new RfD supports a lowering of the cyanide MCLG to 0.004 mg/L assuming the application of the same water intake, body weight and RSC variables as those used when deriving the current MCLG/MCL (from USEPA 2016).

The RfD for cyanide (USEPA 2011) was changed to 0.0006 mg/kg/d as HCN. The RfDs for simple cyanide salts like NaCN and KCN, which freely dissociate into cyanide, are calculated from the RfD for CN– by adjusting for molecular weight (ie, the RfD is multiplied by the ratio of the total molecular weight of the compound to the molecular weight of the CN–):

RfD for cyanogen (CN)2 = 6.3 × 10-4 × 52/26 = 1 × 10-3 mg/kg-day = 0.001 mg/kg/d.

USEPA (1988) established a low confidence RfD of 0.09 mg/kg/day for cyanogen bromide based on a NOAEL for weight loss, thyroid effects and myelin loss in rats with than uncertainty factor of 100 and a modifying factor of 5 to account for the use a study of cyanide for the assessment. CNBr dissociates into cyanide in water. USEPA examined cyanogen bromide (CNBr) under the program for peer reviewed provisional threshold values and chose not to establish an p-RfD for settings relevant to the Superfund Program due to the lack of pharmacokinetic, dissociation rates, issues linking CNBr to simple cyanides and lack of toxicological data specific to CNBr. The USEPA assessment calls attention to the current IRIS RfD for cyanide of 0.00063 mg/kg/day for decreased cauda epididymis weight in male #344/N rats (USEPA 2010b) as a value that could be applicable in scenarios where dissociation of cyanogen bromide dissociation is expected (from USEPA 2016).

### Derivation of Maximum Acceptable Value

The WHO (2017) health-based value of 0.6 mg/L for cyanogen chloride is based on a NOAEL for cyanide of 4.5 mg/kg body weight per day for minor changes in the testis in a subchronic study in which rats were exposed through their drinking-water and an uncertainty factor of 100, a TDI for cyanide of 0.045 mg/kg body weight (corresponding to a cyanogen chloride dose of 0.11 mg/kg body weight) can be derived. In view of the minor nature of the changes observed and the NOAEL in a previous chronic study, it is not considered necessary to include an additional uncertainty factor to allow for the length of the study. Further, it appears that a dose that may be toxic in acute poisoning would certainly be tolerated under chronic conditions, owing to efficient detoxification. Assuming a 60 kg adult drinking two litres of water per day and allowing 20 percent of the TDI to come from water because of the potential for exposure to cyanogenic glycosides in food, the health-based value for long-term exposure is 0.3 mg/L for cyanide or 0.6 mg/L for cyanogen chloride (rounded values).

WHO (2011) states that as cyanogen chloride is unlikely to be found in drinking-water at concentrations that are of health concern, it is considered unnecessary to develop a formal guideline value for cyanogen chloride. Instead, for guidance purposes, a health-based value is derived based on cyanide. Hence the “new WHO guideline value” for cyanogen chloride (as at 2007) becomes the WHO health-based value (in 2011).

The DWSNZ (2008) MAV was based on the WHO (2007) background document which derived a new guideline value for cyanogen chloride, measured as cyanide (CN-), and adjusted for 70 kg bw:

5.4 mg/kg body weight per day x 70 kg x 0.2 = 0.378 mg/L (rounded to 0.4 mg/L)

2 L per day x 100

where:

* no-observed-adverse-effect level = 5.4 mg cyanide per kg body weight, based on minor changes in the testes of rats
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.2
* uncertainty factor = 100 (10 for intraspecies variation and 10 for interspecies extrapolation)
* average amount of water consumed by an adult = 2 L per day.

The MAV for cyanogen chloride in 1995, 2000 and 2005 had been 0.08 mg/L, measured as CN. The 1995 datasheet stated:

There are few data on the oral toxicity of cyanogen chloride, and the proposed MAV is therefore based on cyanide. The MAV of 0.08 mg/L for cyanide as total cyanogenic compounds was developed (see cyanide).

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# Dichloramine

### Maximum Acceptable Value

There are insufficient data to establish a health-based MAV for dichloramine.

Dichloramine has not been extensively studied, and available data are inadequate to permit derivation of health-based guideline value (WHO 2017).

Dichloramine has been reported to impart a taste/odour to water at low concentrations (<0.1 mg/L); any health-based value is likely to be much higher than that.

### Sources to drinking-water

#### 1 To source waters

Chloramines may be present in source waters as a result of their discharge from industries in which they are used. Their principal use is as intermediates in the manufacture of hydrazine.

Comparatively little is known about the physical properties of pure dichloramine because of its instability and difficulty of preparation. Its odour, volatility from aqueous solution, and relative solubility in various solvents are intermediate between those of monochloramine and trichloramine. Under equilibrium conditions at pH 4 it is the only product of the reaction of equimolar concentrations of chlorine and ammonia. Under normal conditions, however, dichloramine solutions are unstable. USEPA (1994).

#### 2 From treatment processes

Dichloramine can be formed in chlorinated water that contains ammonia and some organic nitrogen compounds. The concentration depends upon the pH and chlorine to nitrogen ratio.

Ammonia may be intentionally added to the water to produce the chloramines as disinfectants.

#### 3 From the distribution system

It is possible that reactions of chlorine with nitrogenous material in the distribution system may produce chloramines.

### Typical concentrations in drinking-water

No typical value data are available for New Zealand. Chloramination is not used intentionally at present as a disinfectant in New Zealand, and the concentrations of inorganic chloramines present in waters depends upon the concentrations of inorganic and some organic nitrogen compounds present in the raw water, and control of the chlorination process.

### Removal methods

Chemical reducing agents, including sodium thiosulphate, sulphur dioxide, and sodium bisulphite can be used to remove dichloramine. Activated carbon adsorbs dichloramine.

### Analytical methods

#### Referee method

DPD Ferrous Titrimetric Method (APHA 4500-Cl F).

The limit of detection for this method is approximately 0.2 mg/L for field use, although lower levels can be determined under laboratory conditions and with care. Analytical texts indicate that by manipulation of the conditions of the analysis measurement of monochloramine, dichloramine and trichloramine can be made. These methods are of use when ammonia only is in the water being chlorinated. In most natural waters nitrogen-containing organic compounds are also present. Organic chloramines are formed from these compounds when chlorine reacts with them. Organic chloramines also produce colour during the DPD test and make attempts to differentiate between the different inorganic chloramines of little value. Unless investigating taste and odour problems, it is recommended that only the total combined chlorine, ie, total chloramine concentration, is reported.

These methods measure dichloramine in terms of mg Cl as Cl2/L.

#### Some alternative methods

1. Amperometric Titration Method (APHA 4500-Cl D and E).

While more accurate than the DPD methods, expensive equipment and a high degree of skill and care are required for this method. The limit of detection is better than 0.1 mg/L (0.01 mg/L for method APHA 4500-Cl E). The APHA method describes variations that will allow the determination of mono- and di‑chloramine. Interferences due to organic chloramines may also cause interferences with these methods.

2. DPD Colorimetric Method (APHA 4500-Cl G).

This method requires a spectrophotometer for the colorimetric measurements, although hand-held comparators do offer a cheaper, though less reliable variation for field use. The limit of detection (LOD), with instrumental assistance, is approximately 0.1 mg/L.

The LOD for the comparator depends on the colour disc in use. Chloramine concentrations as low as 0.2 mg/L approximately should be detectable, but the accuracy of the method depends upon use of the correct lighting (natural lighting should be used with the sun behind the viewer), the individual’s ability to match colours and judge their intensity, and ensuing that readings are taken as soon after colour development as possible. The LOD may be about 0.10 mg/L when using a Nessleriser.

The same comment on the usefulness of trying to determine the individual chloramine concentrations made for the referee method, also applies to this method.

### Health considerations

Studies have revealed equivocal evidence of carcinogenic activity of chloraminated drinking-water in female rats, as indicated by an increase in incidence of mononuclear cell leukaemia.

Epidemiological studies did not report an association between ingestion of chloraminated drinking-water and increased urinary bladder mortality rates in humans.

When tap-water containing chloramines was used for dialysis, acute haemolytic anaemia, characterised by denaturation of haemoglobin and lysis of red blood cells, was reported in haemodialysis patients.

Dichloramine has been reported to cause sore eyes in pool water at low concentrations (<0.1 mg/L). For dichloramine, the organoleptic effects between 0.1 and 0.5 mg/L were found to be “slight” and “acceptable”. Odour and taste thresholds of 0.15 and 0.13 mg/L were reported, respectively.

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# Fluoride

### Maximum Acceptable Value

Based on health considerations, the concentration of fluoride in drinking-water should not exceed 1.5 mg/L.

The maximum contaminant level or MCL in the US (USEPA 2009/2011) is 4 mg/L. USEPA (2011) lists the MCLG as 4 mg/L based on skeletal fluorosis. The USEPA established in 1986 a secondary MCL of 2.0 mg/L to prevent tooth enamel discoloration and pitting (also called fluorosis) (from WRF 2015).

A recent study (National Research Council 2006) recommends reducing the MCL to 2 mg/L. The National Research Council is the principal operating arm of the National Academy of Sciences and the National Academy of Engineering. It is a private, non-profit institution that provides science and technology advice under a congressional charter. The report was sponsored by the US Environmental Protection Agency. The USEPA (2009) has a secondary drinking water regulation (SDWR) level of 2.0 mg/L.

The maximum acceptable concentration for fluoride in Canada is 1.5 mg/L, based on its potential cosmetic concern in cases of moderate dental fluorosis in the segment of the population most at risk: children 1–4 years old. Short-term exceedances slightly above the guideline value are unlikely to have an effect on health. However, in the event that monitoring data show elevated levels of naturally occurring fluoride, it is suggested that a plan be developed and implemented to address these situations, with an emphasis on young children (Health Canada 2010).

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of fluoride in drinking water should not exceed 1.5 mg/L.

The Prescribed Concentration or Value (PCV) for fluoride in England and Wales is 1.5 mg/L. See Notes.

The DWSNZ state “for oral health reasons the Ministry of Health recommends that the fluoride content for drinking-water in New Zealand be in the range of 0.7–1.0 mg/L; this is *not* a MAV.”

The US Public Health Service (USPHS 2015) updated its earlier recommendation for fluoride concentrations which was based on outdoor air temperature of geographic areas and ranged from 0.7–1.2 mg/L; they now recommend an optimal fluoride concentration of 0.7 mg/L for dental caries prevention.

### Sources to drinking-water

#### 1 To source waters

Contrary to some people’s belief, fluorine does not occur naturally. Fluorine is a reactive gas, the end-product usually being the fluoride ion which forms salts with metals such as sodium. This is analogous with chlorine (a toxic gas that does not occur naturally) that forms salts such as sodium chloride (“common salt”). Fluoride and chloride do occur naturally.

Fluoride is present in trace amounts in soils and rocks, where it averages about 0.3 g/kg (0.03 percent) of the Earth’s crust, and some coals. It is most prevalent in active or inactive volcanic regions. The most common fluoride containing minerals include fluorite and apatite, while it is also present due to the replacement of the hydroxide in hornblende and some micas. The weathering of alkaline and siliceous, igneous and sedimentary rocks, especially shales, supplies fluoride to the aquatic environment. Major occurrences of fluorite in New Zealand are known near Nelson, in the Buller Gorge, at Greymouth and on Stewart Island. Fluoride compounds are also rich in volcanic and geothermal fluids.

The concentration of fluoride in seawater is about 1.3 mg/L.

Fluoride compounds are used in a variety of industrial processes, including metal plating, metal casting, welding, brazing and the manufacture of aluminium, steel, bricks, tiles, glass and ceramics, hydrofluoric acid and other fluorine chemicals, adhesives and metallurgical fluxes. They are also used in insecticides and herbicides. Sodium fluoride is used for commercial use only as a wood preservative for utility poles and railroad ties. Sodium fluoride products are used as supplemental wood treatments and are not intended for primary wood preservative or pressure treated wood preservation (USEPA 2007). Fluoride is added to toothpaste, tooth powders, mouth washes and vitamin supplements.

Concentrations in surface water are relatively low (usually <0.1 but occasionally up to 0.5 mg/L), while water from deeper wells may have quite high concentrations  
(1–10 mg/L) if the rock formations are fluoride-rich or volcanic.

#### 2 From the treatment processes

Fluoride is often added to drinking-water supplies to protect teeth against dental caries.

Fluoride was originally added to water as the fairly soluble salt, sodium fluoride (NaF), and is still the choice for many smaller supplies. Silicofluoride is a cheaper source; the acid or sodium salt is manufactured from a by-product of the fertiliser industry. The silicofluoride ion hydrolyses in water to release the fluoride ion.

Where fluoride is deliberately added to water, dose control is important to maintain the concentration within the allowable limit. The Victorian (2009), NSW (2011) and Queensland (2013) Governments have published codes of practice, as has the UK (DWI 2005). Some fluoridation chemicals contain impurities; the New Zealand standard addresses this (NZWWA 2014). DWI (2013) states that BS EN has established contamination limits as follows:

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | **BS EN** | **As** | **Cd** | **Cr** | **Hg** | **Ni** | **Pb** | **Sb** | **Se** |
| Hexafluorosilicic acid (mg/kg H2SiF6 max) | 12,175 | 400 | 40 | 400 | 10 | 400 | 400 | 80 | 80 |
| Sodium hexafluorosilicate (mg/kg max) | 12,174 | 400 | 40 | 400 | 10 | 400 | 400 | 80 | 80 |

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

Fluorine, being the most reactive element of the halogen series, does not occur freely in nature, but is present as fluoride ion. In general, most fluorides associated with monovalent cations are water soluble (eg, NaF, AgF, and KF) but those formed with divalent cations are usually insoluble, eg, calcium fluoride. Groundwater usually contains higher concentrations of fluoride than surface water.

### Typical concentrations in drinking-water

Fluoride was routinely measured in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. Of 1904 samples analysed between 1983 and 1989, 10 samples (0.5 percent) exceeded the (then) guideline range of 0.9–1.1 mg/L.

The P2 Chemical Determinand Identification Programme, sampled from 166 zones, has found naturally-occurring fluoride concentrations to range from “not detectable” (nd) to 1.8 mg/L, with the median concentration being “nd” (Limit of detection = 0.1 mg/L). The Priority 2 Identification Programme found 1 distribution zone supplying drinking-water to a total of 60 people with a natural fluoride content of greater than the MAV (ESR 2001).

### Removal methods

Defluoridation of waters with high natural levels of fluoride can be achieved through the passage of the water through bone char, granular tricalcium phosphate, ion exchange resins, bauxite or activated alumina. Activated alumina has several advantages: specificity for fluoride; a high exchange capacity not affected by other anions; low cost compared to synthetic anion exchange resins; and easily regenerated with sodium hydroxide and dilute acid. However, bauxite may well offer a cheaper option.

Lime-softening may remove fluoride by precipitation as calcium fluoride. Alum flocculation may remove some fluoride by adsorbing the fluoride on to the aluminium hydroxide floc.

Fawell et al (2007) discuss several removal processes.

### Analytical methods

#### Referee method

Ion Selective Electrode Method (APHA 4500-F C).

#### Some alternative methods

1. SPADNS Method (APHA 4500-F D).

2. Ion Chromatography Method (APHA 4500-C; USEPA method 300.1).

### Health considerations

Fluoride is probably an essential element for animals and humans although for humans this has not been demonstrated unequivocally. Soluble fluorides are rapidly absorbed in the gastrointestinal tract after uptake via drinking-water. Fluoride has an affinity for mineralising tissues of the body: in young people, bone and teeth; in older people, bone. Thus excretion is somewhat greater in adults because they have proportionally less mineralising tissue than do children. People with kidney impairment have a lower margin of safety for fluoride intake and limited data indicate that their fluoride retention may be up to three times normal.

Fluoride is often added to water as the silicofluoride ion, either as the acid (H2SiF6) or the sodium salt (Na2SiF6). When sodium fluorosilicate (Na2SiF6) or hydrofluorosilicic acid (H2SiF6) is added to the water fluorosilicate (also called silicofluoride) ions (SiF62-) are formed, dissociating to form silica and fluoride (Urbansky 2002), who stated “all the rate data suggest that equilibrium should have been achieved by the time the water reaches the consumer’s tap if not by the time it leaves the waterworks plant.” The dissociation process can be summarised:

H2SiF6 + 4H2O → 6HF + Si(OH)4

Silicofluoride can form a fairly stable complex with aluminium, the AlF63- ion, but at the pH of the stomach, the AlF63- ion dissociates, freeing up the fluoride ion.

Fluoride prevents tooth decay by strengthening the tooth surface and inhibiting growth of cariogenic bacteria. It also assists in repairing the early stages of tooth decay (from ESR 2007). Low concentrations of fluoride produce beneficial effects on the teeth, especially in children. In New Zealand the evidence is that populations receiving optimally fluoridated water benefit from decay levels approximately 30 percent below those not receiving optimally fluoridated water. An Australian survey concluded that children born after 1970, when fluoridated water became popular, have only half the level of decay the previous generation had developed by the time they were young adults (AIHW 2007). This protective effect for caries increases with concentration up to 2 mg fluoride/L drinking-water. The effectiveness of fluoride in dental disease prevention and health considerations has been reviewed extensively and fluoridation of public water supplies is recommended by many health authorities around the world, including WHO, as an important health measure.

Mild dental fluorosis (mottling of the teeth) may occur at a prevalence of  
12–33 percent at drinking-water concentrations between 0.9 and 1.2 mg/L. This may become manifest in areas with a temperate climate and with concentrations between 1.5 and 2 mg/L.

Skeletal fluorosis, characterised by hypermineralisation and consequently brittle bones, has occurred in association with high fluoride concentrations in drinking-water and also with occupational exposure to fluoride-containing dust. It generally occurs after prolonged exposure (several years) and is reversible. The US Environmental Protection Agency considers a concentration of 4 mg/L to be protective against crippling skeletal fluorosis. WHO (2011) states that crippling skeletal fluorosis usually develops only where drinking-water contains over 10 mg of fluoride per litre, and IPCS concluded that there is clear evidence from India and China that skeletal fluorosis and an increased risk of bone fractures occur at a total intake of 14 mg of fluoride per day.

The dental effects of fluorosis develop much earlier than the skeletal effects in people exposed to large amounts of fluoride. Clinical dental fluorosis is characterised by staining and pitting of the teeth. In more severe cases all the enamel may be damaged. However, fluoride may not be the only cause of dental enamel defects. Enamel opacities similar to dental fluorosis are associated with other conditions, such as malnutrition with deficiency of vitamins D and A or a low protein-energy diet. Ingestion of fluoride after six years of age will not cause dental fluorosis. People affected by fluorosis are often exposed to multiple sources of fluoride, such as in food, water, air (due to gaseous industrial waste), and excessive use of fluoridated toothpaste. However, drinking water is typically the most significant source. A person’s diet, general state of health as well as the body’s ability to dispose of fluoride all affect how the exposure to fluoride manifests itself (WHO 2001).

The results of several epidemiological studies, concerned with a possible adverse effect of fluoride in drinking-water on pregnancy, are inconclusive.

The Quebec Government reviewed the health benefits and risks of fluoridation (INSPQ 2007), and concluded: “Overall, the scientific data currently available does not show that water fluoridation at concentrations deemed beneficial to dental health is harmful to humans. Nor have environmental studies revealed any harmful ecosystemic effects of fluoridation. It is important to note, however, that the vast majority of scientific reviews on fluoride have found methodological weaknesses in the epidemiological studies published to date. Consequently, such research needs to continue and must be improved from a methodological standpoint.”

Virtually all foodstuffs contain traces of fluoride, in particular, high amounts can be found in some seafoods, and in tea leaves because of natural concentration process of the tea plant (which may contain 100 mg/kg of dry leaf). Total daily intake from all sources varies considerably, but has been estimated at 0.46 mg to 5.4 mg, with about 10 percent coming from unfluoridated drinking water.

Fawell et al (2007) discuss human health effects, guidelines, and reports of fluorosis from several countries that have some naturally high fluoride waters.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.05 mg/kg/day for chronic-duration oral exposure (>364 days) to sodium fluoride.

The reference dose or chronic RfD (USEPA 2009/2011) is 0.06 mg/kg/d, based on dental fluorosis in children, a cosmetic effect. Based on the available data, sodium fluoride has been classified as a “Group D” (inadequate evidence of carcinogenicity). This conclusion is consistent with the recent report by the National Academy of Sciences which concluded that “the evidence on the potential of fluoride to initiate or promote cancers, particularly of the bone, is tentative and mixed” (USEPA 2007).

The US Centers for Disease Control and Prevention cites community water fluoridation as one of 10 great public health achievements of the 20th century, and the American Dental Association (like New Zealand’s Dental Association) continues to endorse water fluoridation as a vital public health measure to prevent tooth decay.

EFSA (2013) describes the physiological and metabolic processes that occur upon ingestion of fluoride. They derive an Adequate Intake (AI) of fluoride from all sources (including non-dietary sources) of 0.05 mg/kg body weight per day for both children and adults, including pregnant and lactating women.

Yet another national study presents evidence that shows water fluoridation helps to reduce tooth decay in children and adults. There is no reliable evidence that water fluoridation at current Australian levels causes health problems (NHMRC 2017).

Tests for mutagenicity with strains of bacteria have been negative. Chromosome aberrations have been reported in tests with mammalian cells but only at extremely high fluoride concentrations. In 1987, the International Agency for Research on Cancer classified inorganic fluorides in Group 3 (not classifiable as to its carcinogenicity to humans). Fluoride has also not been carcinogenic in more recent studies in laboratory animals.

The livestock guideline value is 2 mg/L (ANZECC/ARMCANZ 2000). If livestock feed contains fluoride, the trigger value should be reduced to 1.0 mg/L. These guidelines were to have been updated in 2012.

### Derivation of Maximum Acceptable Value

WHO considered that there is no new evidence to suggest that the MAV of 1.5 mg/L, set in 1984 and reaffirmed in 1993/2011, needs to be revised. Concentrations above this value carry an increasing risk of dental fluorosis, and much higher concentrations lead to skeletal fluorosis. This value is higher than that recommended for artificial fluoridation of water supplies.

The latest study (National Research Council 2006) recommends a new maximum contaminant level in USA (2 mg/L) to help prevent fluorosis (mottling of the teeth) and the very small possibility of increased life-time risk of bone fracture with some at risk groups. Both risks are negligible in areas with fluoride levels lower than two parts per million. The report notes that the risk of bone fracture rates were lowest at one part per million (the recommended level for drinking water) which were lower than the category with the lowest fluoride intake – something the report highlighted as potentially important.

This latest review finds no new evidence of health effects below two parts per million reinforcing the findings of the recent York report’s conclusion on the overall safety of water fluoridation at the appropriate level.

The Ministry of Health recommends that the concentration of fluoride in fluoridated drinking-water supplies be between 0.7 and 1.0 mg/L. The minimum concentration of fluoride required for a protective effect against dental caries is about 0.5 mg/L and concentrations around 1 mg/L in temperate climates are optimal for the prevention of caries. Mottling of teeth due to dental fluorosis may occur at concentrations between 1.5 and 2 mg/L.

Recommendations for dietary intake of nutrients, including fluoride, are contained in the *Nutrient Reference Values for Australia and New Zealand* published in 2006 by the National Health and Medical Research Council of Australia and the New Zealand Ministry of Health.

The document sets recommended levels of fluoride intakes. The upper level of intake of fluoride for children 0–6 months is 0.7 mg/day, and for children aged 7–12 months 0.9 mg/day. This limit is set as the average safe upper level if consumed at that level daily. It is important to note that the upper limits are averages, and that the average applies over six-month periods when infants will be consuming varying volumes of breast milk, formula and/or food.

It is theoretically possible for young children to consume above this upper limit of dietary fluoride. However, this risk also requires an assumption that the level of consumption would occur for a sufficiently long period to pose a risk for enamel fluorosis to occur. Current information in Australia and New Zealand about actual levels of enamel fluorosis was considered.

When the Australia/New Zealand Food Standard Code was developed, consideration was given to the fact that fluoridated tap water would be used in the reconstitution of powdered infant formula.

#### Acute effects

A number of overdosing incidents have occurred, mostly in small water supplies, that practice artificial fluoridation. With well designed fail-safe equipment and working practices overdosing incidents can be avoided. Where incidents of acute intoxication have been reported following overdosing in water supplies, fluoride levels have ranged from 30 to 1,000 mg/L. To produce signs of acute fluoride intoxication, it is estimated that minimum oral doses of at least 1 mg fluoride per kg of body weight are required (WHO 1996). Indeed, such doses could be expected from water with a fluoride content of approximately 30 mg/L. Taken from WHO (2006).

The WHO World Health Assembly Resolution 2007 calls on Member States to consider development and implementation of systematic fluoridation programmes through drinking-water, salt or milk in countries that do not have access to optimal levels of fluoride. In order to prevent dental caries/tooth decay effectively, the WHO Oral Health Programme advocates for fluoridation and provides technical assistance to many countries worldwide to introduce or sustain systematic fluoridation programmes. The WHO Guidelines for Drinking-water Quality (WHO 2011) note that fluoride may be added to drinking-water to protect against dental caries. The amount added for such purposes is usually between 0.5 and 1 mg/L which is less than the guideline value, which is 1.5 mg/L. In setting national drinking-water standards, consideration should be given to total fluoride exposure. If the intake from other sources is likely to approach or be greater than 6 mg/day, setting a standard at concentrations lower than the Guideline Value should be considered. Furthermore, for planning of fluoridation programmes WHO recommends an assessment of population exposure to fluoride from food, drinking-water, air and dental preparations, since it is the total intake of fluoride that is important. Costs, technological development, trained water engineers and availability of municipal water supplies reaching large populations should also be considered in deciding to fluoridate drinking-water supplies. Ongoing monitoring of existing schemes is recommended to ensure that people are exposed to an optimal level of fluoride for prevention of dental caries and to prevent adverse health effects that could result from elevated fluoride intakes.

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# Iodine, Iodide and Iodate

CAS No. 7553-56-2 (iodine). Iodate: sodium or potassium iodate salts (CAS No. 7681‑55-2 and 7758-05-6, respectively).

### Maximum Acceptable Value

There are insufficient data to derive a MAV for iodine, iodide or iodate, and lifetime exposure to iodine through water disinfection is unlikely.

WHO (2004) states that because iodine is not recommended for long-term disinfection, lifetime exposure to iodine from water disinfection is unlikely. For these reasons, a guideline value for iodine has not been established at this time.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of iodide in drinking water should not exceed 0.5 mg/L (the earlier version stated 0.1 mg/L). No health-based guideline value was set for molecular iodine, which can be used as an emergency water disinfectant; the taste threshold is 0.15 mg/L.

### Sources to drinking-water

#### 1 To source waters

Iodine (as iodide) may be present in source waters at low concentration. Iodide is naturally present in nitrate minerals, and in sea water so any type of seafood provides a source, particularly seaweed (kelp), mostly in the form of iodide salts. Generally, the main source of iodide in New Zealand surface waters is from wind-blown seaspray or aerosols. This iodide can oxidise in water to form hypoiodous acid (HOI), which in turn can react with organics to adversely affect the taste and odour of drinking-water; this is somewhat analogous to HOCl and HOBr. Iodide may also be present in water due to leaching from salt and mineral deposits.

Geothermal waters in the Kawerau area can contain up to about 0.008 mg/L as I (GNS 2015). Iodide is more likely to be found in bore waters from shales (particularly organic-rich) and limestone strata rather than granite, basalt or volcanic material (DWI 2016).

The concentration of iodide in seawater is about 0.05 mg/L.

Iodine may enter source waters as a result of its use as an antiseptic, as a sanitising agent in hospitals and laboratories, in pharmaceuticals and in photographic developing materials.

Iodide is added to salt to ensure that people have enough iodine in their bodies to form essential thyroid hormones. In recent years people have been increasingly buying uniodised salt, so mandatory fortification of most bread with iodine (by replacement of non-iodised salt with iodised salt) was introduced into New Zealand and Australia in September and October 2009, respectively (NZSFA 2012). In general, foods from the sea contain the most iodine; of all foods seaweed is the most reliable source of natural iodine.

Because of its short half-life and useful beta emission, iodine-131 is used extensively in nuclear medicine. Iodine-123 is widely used in medical imaging, and iodine-124 is useful in immunotherapy. A less common isotope, iodine-125, is sometimes used to treat cancerous tissue (USEPA).

#### 2 From treatment processes

Ozonation oxidises any iodide through to iodate and therefore reduces the potential for iodinated-THM formation. Addition of chlorine to iodide-containing solutions results in the rapid and essentially complete oxidation of iodide to hypoiodous acid (HOI) (DWI 2009).

Iodine is occasionally used for the emergency disinfection of drinking-water for field use but it is not used for disinfecting larger drinking-water supplies. Iodine is used dosing a tincture (usually 2 percent), iodine solution (10 percent recommended – add eight drops per litre), ‘water purification’ tablets, or by passing the water through triiodide or pentaiodide resin. The contact time should exceed 30 minutes. Iodine is not recommended for long-term disinfection. See Chapter 15.

Excess iodine may interfere with the functioning of the thyroid gland. Therefore, the use of iodine as a disinfectant is not recommended for infants, pregnant women, those with a history of thyroid disease and those with known hypersensitivity to iodine, unless treatment includes an effective post-disinfection iodine removal device, such as activated carbon. Travellers intending to use iodine treatment daily for all water consumed for more than 3–4 weeks should consult their physician beforehand and not use it in excessive amounts. Iodine can inactivate bacteria, many viruses, *Giardia* if the contact time is long enough, but is not effective against *Cryptosporidium*.

Iodide is oxidised to iodine by strong disinfectants such as chlorine during water treatment. The chlorination of waters containing significant amounts of iodide would result in the formation of iodinated disinfection by-products; such waters are rare.

Iodinated trihalomethanes can form in the water treatment process. The commoner types include dichloroiodomethane, bromochloroiodomethane, chlorodiiodomethane, dibromoiodomethane and bromodiiodomethane – see Chapter 15, individual datasheets and DWI (2016).

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

Iodine occurs naturally in water in the form of iodide. Any iodide that is oxidised to iodine during water treatment will be incorporated into organic matter.

Elemental iodine is less soluble in water than chlorine or bromine. Water solubility depends on pH and temperature and is reported to be 0.03 mg/L at 20°C, 0.78 mg/L at 50°C and 4.45 mg/L at 100°C. A saturated aqueous solution of iodine can be produced by passing water through a column of crystalline iodine. The iodine concentration achieved will be approximately 200 mg/L at 10°C and 400 mg/L at 30°C. This concentrated solution can be diluted to achieve the desired concentration of iodine (WHO 2018).

### Typical concentrations in drinking-water

Iodine has not been monitored routinely in New Zealand drinking-water supplies. In 2013/14 Hamilton’s six-monthly analyses have found 0.002 to 0.003 mg/L iodide in the raw water, and <0.001 mg/L in the treated water.

The mean concentration of total iodine in USA drinking-water is 4 μg/L (0.004 mg/L) and the maximum concentration is 18 μg/L (0.018 mg/L), probably predominantly as iodide.

Two water utilities in the US reported detecting iodine-131 in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies; one was reported to contain 545 mg/L, the other 0.33 mg/L.

The maximum concentration of iodide found in 2113 samples from 774 groundwaters in the UK was 4.35 mg/L, mean 0.048 mg/L (DWI 2008).

The mean iodide concentration of tap water was calculated by the 22nd Australian Total Diet Study (ATGS) to be 11.3 μg/L (0.011 mg/L). Food Standards Australia and New Zealand (FSANZ) estimate that the Australian population would receive  
5–9 percent of its total dietary iodine supply from drinking water. The cut-off value for water iodide concentrations which is considered to be indicative of environmental deficiency is <2 μg/L (0.002 mg/L) (WQRA 2011).

DWI (2016) lists drinking water concentrations around the world. They also measured iodide in waters in England and Wales, measuring iodide, iodate and iodine; on average the sum of iodide and iodate comprised 98.4 percent of total iodine. Tap samples showed that on average iodate comprised 74.5 percent of the total iodine measured in treated water commensurate with the use of chlorine as a disinfectant and the use of other oxidants at some sites. Due to the fast oxidation of iodide to iodate by free chlorine, it is rational that iodate was the dominant iodine species in these samples.

### Removal methods

It is unlikely that the concentration of iodine in drinking-water would ever be high enough to justify treating to remove it. Chemical reducing agents, such as sodium thiosulphate, could be used to reduce the iodine to iodide, but disinfection of the water using oxidants (eg, chlorine) could not be used subsequently, as the iodide would be converted back to iodine again.

### Analytical methods

#### Referee method

A referee method cannot be selected for iodine 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 iodine for the above reason. However, the following information may be useful.

Iodine cannot be preserved in a sample. Analysis must be carried out as soon after sampling as possible, and if a delay between sampling and analysis is unavoidable the sample must be kept dark and chilled.

Iodine can be analysed using the Leuco Crystal Violet Method (APHA 4500-I B). The limit of determination of this method is approximately 0.01 mg/L.

A comparison of methods and LoDs appears in DWI (2016).

### Health considerations

Iodine (as iodide) is an essential micronutrient which plays a vital role in the normal development of most organs including the brain. Iodine deficiency is the single greatest cause of preventable brain damage and developmental delay in the world and can account for a loss of 10–15 IQ points even with moderate deficiency. Cretinism is due to iodine deficiency (WQRA 2011).

Iodine is an essential trace element for humans and is used in the synthesis of thyroid hormones which are essential because they regulate the body’s metabolism. The human body contains approximately 10–15 mg of iodine, of which about  
70–90 percent is in the thyroid gland, where it is involved in the synthesis of the thyroid hormones triiodothyronine, or T3, and tetraiodothyronine, or T4, also known as thyroxine. Muscle and the eye also contain relatively high iodide concentrations. The entry of the chemical element iodine through the oral route is typically in the monovalent anionic iodide form (I-) which is absorbed efficiently throughout the gastrointestinal tract. Once absorbed into the thyroid, two iodide ions are enzymatically converted to an iodine molecule before incorporation into the thyroid hormones. This iodine can be eliminated from the body either as iodine or as iodide, depending on the specific mechanism of elimination.

In children, especially young children, including the developing foetus, thyroid hormones help regulate physical and mental development. Most iodide enters the body with food. Although only small amounts are needed, iodine is needed regularly because it is not stored in the body. The WHO recommended intake (population requirement) of iodine is 0.15 mg/day for adults and adolescents 13 years of age and older, 0.20 mg/day for women during pregnancy and lactation, 0.12 mg/day for children 6–12 years of age, and 0.09 mg/day for children 0–59 months of age (WHO 2009).

Excessive iodine can lead to hyperthyroidism or, less commonly in adults, hypothyroidism. Babies appear more susceptible to hypothyroidism following excessive iodine exposure which can occur during pregnancy from the mother’s intake of iodine. The commonest symptoms of hyperthyroidism are palpitations, fatigue and weight loss. Hypothyroidism is often insidious and, in mild cases, may only be detected on routine screening but it can cause fatigue, weight gain and mental clouding. WHO (2009) report a TDI of 0.01 mg/kg body weight, based upon reversible subclinical hypothyroidism, can be established by dividing the NOAEL of 0.01 mg/kg body weight per day by an uncertainty factor of 1. An uncertainty factor of 1 was considered appropriate because the dose was a NOAEL, this was a human study, and the study cohort was considered to represent the most sensitive population. Supporting studies indicate that the NOAEL would be applicable to elderly adults, who may represent another sensitive population.

The lack of knowledge on long-term toxic effects of iodine consumption impedes the use of iodine for disinfection of municipal or community supplies. Considerable controversy exists about the maximal “safe” dietary dose of iodine (in the range of 0.5 to 1.0 mg/day in healthy adults) and the maximum “safe” period of consumption for iodine treated water. Although a number of studies have been carried out, the data are not adequate to establish a linear and temporal dose response between iodine intake and altered thyroid function (WHO 2018).

The effect of an iodine compound (tetraglycine hydroperiodide) used for water disinfection has been studied. Eight healthy euthyroid adults who were negative for thyroid antimicrosomal antibody ingested approximately 32 mg iodine/day as tetraglycine hydroperiodide dissolved in juice or water, for 90 days. The mean pretreatment 24-hour urinary iodide excretion rate was 0.276 mg/day. Thyroid gland volumes, as determined from ultrasound measurements, increased significantly during the treatment, with a peak volume 37 percent above the pretreatment volume and reverted to pretreatment volumes seven months after the iodine dosing was discontinued. Serum TSH concentrations increased significantly during treatment, with only one subject having a three-fold increase to a value above normal, 6.1 mU/L; this subject also had the highest thyroid volume during the treatment period. None of the subjects developed clinical hypothyroidism.

Chronic iodide exposure results in iodism. Symptoms resemble those of a sinus cold and may also include salivary gland swelling, gastrointestinal irritation, acneform skin, metallic or brassy taste, gingivitis, increased salivation, conjunctival irritation and oedema of the eyelids. Long-term consumption of iodinated drinking-water has not been associated with adverse health effects in humans. Prisoners’ drinking-water containing up to 1 mg/L iodine for five years showed no signs of iodism or hypothyroidism, but some changes in uptake of iodine by the thyroid gland were observed.

No data are available on the mutagenic activity of iodine. Recent data from studies in rats indicate that the effects of iodine in drinking-water on thyroid hormone concentrations in the blood differ from those of iodide. Available data therefore suggest that derivation of a guideline value for iodine on the basis of information on the effects of iodide is inappropriate, and there are few relevant data on the effects of iodine.

Using iodine for disinfection of drinking water is not suitable for all people. Backer and Hollowell (2000) noted the following people are at increased risk for iodine-induced hypothyroidism:

* those with underlying thyroid problems, including current or prior thyroiditis, previous treatment for Graves disease, previous subtotal thyroidectomy for benign nodules, and previous treatment with interferon-α
* fetuses, preterm neonates, and newborn infants who are at risk because of the placental transfer of iodide from mothers treated with iodides
* those with endemic goiter due to very high dietary iodine intake – mainly described in coastal areas of Japan
* those with other conditions, including:
* elderly people without clinical thyroid disease who may have subclinical hypothyroidism (ie, elevated TSH but normal free T4 levels) (subclinical hypothyroidism is very common, affecting 5–10 percent of adults over the age of 50)
* patients with certain non-thyroid diseases such as chronic dialysis and cystic fibrosis, especially those taking sulfasoxazole
* patients taking medications containing iodine, formerly iodide expectorants but, more recently, amiodarone
* patients taking lithium
* people with a family history of goiter or thyroiditis (suggested by a few case reports).

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes minimal risk levels (MRLs) for iodide of:

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

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

Iodate is rapidly absorbed and extensively distributed through the body. It is metabolised via a non-enzymatic process, with a proportion of the available iodate converted to iodine. Iodate is excreted via the kidneys. Iodate is of low acute oral toxicity in experimental animals. Data for longer term studies are limited; however, gastric, renal, and ophthalmological effects are manifested in the longer-term studies available. A NOAEL has been reported from guinea pig tests of 300 mg/kg/d, and from mouse tests of 120 mg/kg/d (DWI 2009).

There are both radioactive and non-radioactive (iodine-127) isotopes of iodine. Iodine-129 and iodine-131 are the most important radioactive isotopes in the environment. Some isotopes of iodine, such as I-123 and I-124 are used in medical imaging and treatment, but are generally not a problem in the environment because they have very short half-lives. Both iodine-129 and iodine-131 are produced by the fission of uranium atoms during operation of nuclear reactors and by plutonium (or uranium) in the detonation of nuclear weapons. Iodine-129 has a half-life of 15.7 million years; iodine‑131 has a half-life of about 8 days. Both emit beta particles upon radioactive decay. Isotopes of iodine are found as compounds rather than as a pure elemental nuclide. Thus, iodine-129 and -131 found in nuclear facilities and waste treatment plants quickly form iodide or compounds with the mixture of chemicals present. However, iodine released to the environment from nuclear power plants is usually a gas.

### Derivation of Maximum Acceptable Value

No MAV.

Iodine has a taste threshold in water of about 0.15 to 0.21 mg/L, unaffected by pH.

WHO (2003) states:

In 1988, JECFA set a PMTDI for iodine of 1 mg/day (17 μg/kg of body weight per day) from all sources, based mainly on data on the effects of iodide. However, recent data from studies in rats indicate that the effects of iodine in drinking-water on thyroid hormone concentrations in the blood differ from those of iodide.

Available data therefore suggest that derivation of a guideline value for iodine on the basis of information on effects of iodide is inappropriate, and there are few relevant data on the effects of iodine. Because iodine is not recommended for long-term disinfection, lifetime exposure to iodine from water disinfection is unlikely. For these reasons, a guideline value for iodine has not been established at this time.

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# Lanthanum

### Maximum Acceptable Value

There is no MAV for lanthanum in the DWSNZ, and lanthanum is not mentioned in the WHO Guidelines for Drinking-water Quality.

The draft version of the Australian Drinking Water Guidelines (NHMRC 2009) states that based on health considerations, and concentrations that are as low as reasonably practicable to achieve, the concentration of lanthanum in drinking water should not exceed 0.002 mg/L. Lanthanum is not mentioned in the 2011 Australian Drinking Water Guidelines.

### Sources to drinking-water

#### 1 To source waters

Lanthanum is a rare earth element (also known collectively as lanthanides) that can enter water via run-off from agricultural soil where it has been used in fertiliser, from the weathering of rock, from specific discharges, or use as a phosphate binder (which its usage is growing to prevent and manage algal blooms), and from leaching from the tailings of rare earth mining.

The concentration of lanthanum in seawater is about 0.0001 mg/L.

A commercial product, PhoslockTM (CAS No. 302346-65-2), has been developed in Australia to remove phosphorus from water. PhoslockTM is a reaction product of bentonite clay and lanthanum chloride in which the proportion of exchangeable cations (mainly sodium) is replaced by lanthanum cations through electrostatic binding. Phoslock™ is designed to adsorb oxyanions, predominantly phosphate, from a variety of natural aquatic environments notably in order to reduce the incidence of algal blooms. The recommended dosage is 100:1 Phoslock™ to filterable reactive phosphorus (FRP). NICNAS (2014) has assessed the use of PhoslockTM. The application in Deep Creek Reservoir (NSW) used approximately three times the recommended dose and a high level of peak dissolved lanthanum (0.22 mg/L) was observed. With a value of 27.6 mg/L CaCO3, the reservoir was of low hardness. Dissolved lanthanum levels stabilised rapidly after a few days of application.

NICNAS (2014) reports:

In New Zealand, the applicant provided the New Zealand Environmental Protection Authority (then ERMA – Environmental Risk Management Authority) a self-assessment report and approval has been granted since. Phoslock™ is not classified under the NZ EPA’s Hazardous Substances and New Organisms (HSNO) regulations. Phoslock™ has been repeatedly applied in Lake Okareka to reduce the phosphorus from the lake. Thorough monitoring of the lake was conducted by the regional authority responsible for the lake.

Lanthanum carbonate octahydrate is currently authorised as a zootechnical additive (decrease in phosphorus excretion via urine) for cats at the maximum authorised concentration of 7.5 g/kg of complete feed (EFSA 2019).

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

The lanthanum ion, La3+ in surface freshwaters could be expected to undergo hydrolysis reactions to form hydroxy species, react with phosphate and carbonate ions, and dissolved organics, and show a high propensity for adsorption to colloidal and suspended particles with negative surface charge (NICNAS 2014).

### Typical concentrations in drinking-water

The concentration of total lanthanum in raw drinking water sources in the Netherlands was reported to range between 0.00005 to 0.013 mg/L, but concentrations in surface waters within rare earth mining areas may be much higher.

### Removal methods

Treatment processes that remove particulate matter should remove insoluble forms of lanthanum and any lanthanum adsorbed to the particulate matter.

### Analytical methods

#### Referee method

No MAV.

#### Some alternative methods

The concentration of lanthanum in water samples can be determined by inductively coupled plasma mass spectroscopy.

### Health considerations

NHMRC (2009) states:

Lanthanum salts have very low oral bioavailability. The absorption and kinetics of lanthanum from lanthanum carbonate (a relatively insoluble salt) has been reasonably well-studied in humans; it has an oral bioavailability of 0.00015–0.02 percent, but with a terminal half life of 1.5–7 hours and only 1.7 percent of the absorbed dose excreted in urine. There is potential for lanthanum to accumulate. The oral bioavailability of soluble forms of lanthanum may be one or two orders of magnitude higher than that of lanthanum carbonate (Pennick et al 2006, He et al 2007).

Lanthanum has a low order of acute toxicity for both soluble and insoluble forms, with oral LD50 values ranging predominantly between 2 and 10 g/kg (Bruce et al 1963, Pattys 2001, NICNAS 2001). Lanthanum carbonate is approved for medical use in non-pregnant adults with end-stage renal failure to prevent dietary phosphate from being absorbed (Health Canada 2005, USFDA 2008, Swedish MPA 2006). A number of oral repeat dose studies with this compound in a variety of species show no systemic toxicity; the observed effect is gastric irritation due to high doses precipitating in the rodent stomach. The no‑observed-adverse-effect level (NOAEL) is 100 mg/kg bw/day and the effect is not observed in dogs (Swedish MPA 2006).

Chinese environmental epidemiological studies have tentatively linked high environmental exposures of lanthanum with delayed intellectual development in children in areas of rare earth mining. Rodent studies of up to six months with oral soluble lanthanum chloride have demonstrated the agent can cause histopathological neurotoxicity, learning deficiency in animals, measurable but nonetheless small increases in brain lanthanum after high doses, and various changes in brain biochemistry. The NOAEL, based on neurotoxicity (decreased numbers of brain cells) and learning decrements is 0.1 mg/kg bw/day of lanthanum chloride (Briner et al 2000, Feng et al 2006, He et al 2008). The equivalent amount of lanthanum ion is 0.06 mg La3+/kg bw/day.

An assessment of total daily intake of lanthanum in humans was not available.

In the rat, two oral toxicity studies for the soluble lanthanum chloride and nitrate salts have commonly demonstrated liver effects and a trend for reduced body weight gain. Neurotoxicity, as demonstrated by changes in brain neurotransmitter levels, trace element distribution in the brain and impaired cognitive ability, was seen in a six-month rat study using lanthanum chloride. A LOAEL for these brain alterations and learning decrements was reported to be 40 mg/kg bw/day, and the NOAEL was 2 mg/kg bw/day (NICNAS 2014).

### Derivation of Maximum Acceptable Value

No MAV in the DWSNZ.

NICNAS recommended in its 2014 report that ‘risk to humans is considered acceptable if the lanthanum levels are maintained in accordance with a controlled concentration for lanthanum of no greater than 0.002 mg/L when present in drinking water’ (NHMRC 2015).

The Australian draft guideline value for lanthanum in drinking water was derived (NHMRC 2009) as follows:

0.002 mg/L = 0.06 mg/kg bw/day x 70 kg x 0.1

100 x 2 L/day

where:

* 0.06 mg/kg bw/day is the La3+ NOAEL for neurotoxicity and neurobehavioral effects in rats
* 70 kg is taken as the average weight of an adult
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* 100 is the safety factor to account for intraspecies variations and interspecies variations
* 2 L/day is the estimated maximum amount of water consumed by an adult.

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# Lead

CAS No. 7439-92-1 (metal).

### Maximum Acceptable Value

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

In 2011/2017, WHO designated their guideline value as provisional on the basis of treatment performance and analytical achievability. As this is no longer a health-based guideline value, concentrations should be maintained as low as reasonably practical. New sources of lead, such as service connections and lead solder, should not be introduced into any system, and low lead alloy fittings should be used in repairs and new installations. The primary source of lead is from service connections and plumbing in buildings; therefore, lead should be measured at the tap.

The action level (USEPA 2009/2011) is 0.015 mg/L at the tap.

The maximum acceptable concentration in Canada is 0.01 mg/L. Because the MAC is based on chronic effects, it is intended to apply to average concentrations in water consumed for extended periods. Exposure to lead should nevertheless be kept to a minimum; plumbing should be thoroughly flushed before water is used for consumption; most significant contribution is generally from lead service line entering the building.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) states that based on health considerations, the concentration of lead in drinking water should not exceed 0.01 mg/L.

The Prescribed Concentration or Value (PCV) for lead in England and Wales is 0.01 mg/L since 2014. See Notes.

Lead is one of the “priority pollutants” under the US Clean Water Act. Tetraethyl and tetramethyl lead 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>.

Lead (inorganic) 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

The principal natural pathway by which lead is released into the environment is weathering of sulphide ores, especially galena (PbS). In New Zealand, occurrences of galena are generally in intermediate volcanic rocks or associated with granitic rocks. Examples of major occurrences include on the Coromandel Peninsula; at Broadlands, Taupo; and around Nelson, Fiordland and Otago. Several stable isotopes exist in nature, 208Pb being the most abundant.

The concentration of lead in seawater is about 0.001 mg/L.

NZFSA reported lead levels of 0.012 to 0.015 mg/kg in wheat in their 2010 season 1 food residues surveillance programme.

Anthropogenic input of lead to the environment outweighs all natural sources. Lead reaches the aquatic environment through precipitation, fallout of lead dust, street run-off and industrial and municipal wastewater discharges. It has been estimated that in New Zealand, 99 percent of the lead emitted to the atmosphere comes from petrol combustion in vehicle engines (written in 1995 – this use has reduced greatly since – according to the USEPA, atmospheric emissions of lead decreased 93 percent over the 21-year period of 1982–2002). The Ministry of Economic Development stated in 2005 “that New Zealand has used lead-free petrol since 1996; only a contamination level of 13 mg/L is allowed, and this is proposed to be reduced to 5 mg/L”. Lead is also a component of diesel exhaust.

Lead is used in the manufacture of acid-storage batteries, an anti-knocking additive for petrol (as tetraethyl and tetramethyl lead), construction materials, coatings and dyes, electronic equipment, plastics, veterinary medicines, fuels, radiation shielding, ammunition, corrosive-liquid containers, paints, glassware, fabricating storage tank liners, transporting radioactive materials, solder, piping, cable sheathing, roofing, and sound attenuators. It is also used in electroplating and the metallurgy industries. The lead content of paint used on roofs used for drinking-water collection should not exceed 0.1 percent and 0.2 percent (percentage based on the non-volatile content of the paint) for lead and lead compounds and lead and lead compounds occurring as an impurity in zinc based paint, respectively (MoH 2012).

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; inorganic lead levels range from 160 to 3,300 mg/kg depending on land use.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

Water is not normally considered the major source of exposure to lead. Lead is rarely present in tap water as a result of its dissolution from natural sources; rather, its presence is primarily from household plumbing systems and fittings.

Lead may be released into drinking-water from the dissolution of lead pipes (not used now in New Zealand) and solders (use greatly reduced), and brass fittings in homes. The weathering of lead-based paints and fittings may also be a concern in rain-water supplies. It has also been found that the lead used as a stabiliser in uPVC pipes will leach from these pipes for several months after the pipes are installed. This has led to high elevated levels of lead in the water at the dead-ends of new subdivisions, until the leaching process has run its course.

The amount of lead dissolved from plumbing systems depends, amongst other things, on pH, temperature, alkalinity, water hardness and standing time of the water, with soft, acidic water being the most plumbosolvent. Free chlorine residuals in drinking-water tend to form more insoluble lead-containing sediments, whereas chloramine residuals may form more soluble sediments in lead pipe. It is recommended that water be flushed from the tap before using to run to waste that water that has been in contact with fittings containing lead.

The following has been taken from the Conclusions of the Executive Summary of DWI (2014):

* Brass fittings typically found in plumbing systems between the water main and the kitchen tap comprise on the public side ferrule, meter, stoptap and compression joints, and on the private side compression joints, stoptap and kitchen tap. The literature review, stagnation tests and field trials indicate that all brass fittings, including those manufactured with low lead grades, can be expected to leach lead throughout their lifetimes.
* The literature review and testimony from manufacturers shows that the highest concentration of lead leaches from new fittings immediately after installation and that the concentration falls off with length of time in service levelling off over a period of a few weeks to a few months. This was confirmed by the results of field trials at hard and soft water sites. The age of an installation is therefore a key issue affecting observed leaching. The literature review indicates this levelled-off value is around one third of the concentration observed in the new product; but the field trials indicated that even lower levelled-off concentrations can be found.
* The literature review indicates that there is a strong effect caused by the characteristics of the water in supply, in particular hardness; lead leaching tends to be higher in soft waters. The field trial results found that leaching was higher from most fittings in non-phosphate dosed soft water than hard water.
* Lead concentrations increase with increasing stagnation time but the most substantial increase generally occurs during the first few hours.
* The stagnations tests show that some models of unused ferrules, meters, stopcocks and taps can, following stagnation of 16 hours, individually yield lead concentrations of >10 μg/L and even >25 μg/L in a one litre sample in the absence of any other lead leaching pipework or fittings. This was confirmed by field tests where, after four months, water meters in soft non-phosphate dosed water could still exceeded >10 μg/L after overnight stagnation and that certain combinations of other fittings could approach this concentration.
* The amount of lead leached from the high lead fittings and some low lead fittings in both soft and hard water fell rapidly from the start of the field trials, within the first two weeks, and generally reached a stable rate of release after 20 to 30 days. However, in non-phosphate dosed soft water leaching from water meters and a stoptap were found to increase following this initial reduction before levelling/falling slowly.
* Phosphate dosing exerts a strong, rapid effect on lead leaching from fittings, reducing both the time taken to reach a stable leaching concentration and reducing this concentration compared with non-phosphate dosed water.
* The laboratory and field trials confirmed that low-lead brass fittings give substantially lower leaching of lead compared to conventional brass fittings.
* The literature search and stagnation tests show large differences in the concentration of lead that leaches from different models of the same type of fitting and smaller differences between samples of the same model (the latter also verified by the long term testing). The leaching characteristics of brass fittings are influenced by the characteristics of the individual fitting and water quality.
* Predicting the contribution of any fitting without undertaking some form of testing on the finished product is very difficult since information on the grades of alloy, surface areas in contact with water, manufacturing technique and so on are very hard to obtain. With the substantial range of fittings/models that are available and the fact that information on leaching characteristics is limited to very few individual models that have been studied it is not possible to predict the leaching characteristics of any particular installation within a building, save that the concentrations will be highest immediately following installation and will reduce over time.
* As regulatory sampling uses one-litre samples, taps, isolation valves and other fittings in contact with that one litre of water are likely to have the greatest influence on lead concentrations. However, overall any fitting containing lead can cause elevated lead concentrations – for example a meter could be the biggest contributor to lead levels at the tap.
* The field trials indicate that in a worst case scenario (where the RDS were taken from a non-phosphate dosed supply after an overnight stagnation, where the fittings had been installed for four month and the kitchen tap was located close to the road, so the draw would include the contents of brass fittings between the supply pipe and the tap) then 10 μg/L could be exceeded, but not 25 μg/L Pb.
* At the national level the UK has to take into consideration international developments relating to the regulation of the lead content of brass. Specifically: low-lead brass requirements in the USA, implementation of the 4MS scheme within Europe which may also influence the international fittings supply market and the UK designated grades of brass, bronze and gunmetal with different compositions for different uses in fittings.
* Advising customers to replace brass taps/fittings with other brass taps/fittings following a PCV exceedance is no guarantee of reducing observed lead concentrations.
* Guidance on flushing could be used as part of an exposure reduction strategy. Suitable advice might be to flush frequently when brass fittings are new and to flush after prolonged stagnation when fittings are older.
* There is no regime for routine inspection or approval of the brass alloys used in fittings installed in the UK so there is no guarantee that the current norms are adhered to across the supply chain.

DWI (2018) reported a study of 104 UK properties grouped as follows:

* leaded properties and non-phosphate (non-P) dosed
* leaded properties and phosphate (P) dosed
* unleaded properties with leaded fixtures and fittings and non-P dosed
* unleaded properties with leaded fixtures and fittings and P dosed.

The study has shown that lead consumption from drinking water is generally low and is unlikely to have a significant impact on human health when considered in isolation. Only six out of 104 individuals were consuming higher lead intakes than the European Food Standards Agency (EFSA) benchmark dose lower confidence limit values (BMDL). These individuals all lived in properties with lead pipes and no P dosing. Low alkalinity and high natural organic matter source waters had the highest potential for plumbosolvency. There was no discernible link between high lead levels and the first drink of the day.

A manufacturer of stainless steel and galvanised iron rainwater tanks in Tasmania had used a 50:50 lead:tin solder. Concentrations of lead from the 64 stainless steel tanks were significantly higher than from the galvanised iron tanks, with median concentrations of 121 μg/L compared with 1 μg/L respectively. The maximum lead concentration found in water from a stainless steel tank was 2030 μg/L. All stainless steel tanks had water lead concentrations above the ADWG health-related limit of 10 μg/L, only four of the 60 galvanised tanks exceed this limit; all of these four had lead flashing on roofing or lead in plumbing materials (Lodo et al 2018).

### Forms and fate in the environment

The principal dissolved inorganic forms of lead are the free ion, and probably the carbonate and sulphate ion pairs. The importance of organic complexes is uncertain, but they may constitute a significant part of the dissolved lead in some waters. Soluble lead is removed from solution by association with sediments and suspended particulates, such as organic matter, hydrous oxides and clays.

### Typical concentrations in drinking-water

Lead was routinely measured in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. Of 1,900 samples analysed between 1983 and 1989, 15 samples (0.8 percent) had concentrations equal to or exceeding the MAV of 0.05 mg/L.

Of the 511 samples taken by the Ministry of Health from school water sources from 1995 to 2004, 126 (25 percent) exceeded 50 percent of the MAV and 84 (16 percent) exceeded the MAV. Sixty-six of the 158 school supplies sampled during this period exceeded 50 percent of the MAV, and 38 of these supplies had two or more samples which exceeded 50 percent of the MAV (MoH 2012).

The P2 Chemical Determinand Identification Programme, sampled from 921 zones, found lead concentrations to range from “not detectable” (nd) to 0.98 mg/L, with the median concentration being 0.001 mg/L (Limit of detection = 0.001 mg/L). The Priority 2 Identification Programme found 195 distribution zones supplying drinking-water to a total of 477,314 people with lead at greater than the MAV (ESR 2001).

In a study in Ontario, the concentration of lead in water actually consumed was determined using a composite sampler in 40 homes at seven locations. The average concentration of lead over a one-week sampling period ranged from 1.1 to 30.7 µg/L, with a median level of 4.8 µg/L. The results of this study are considered to be the most realistic estimate of the intake of lead from drinking water. Using the median concentration of 4.8 µg/L and daily drinking water consumption of 1.5 L for an adult and 0.6 L for a child, the average daily consumption of lead from drinking water is 7.2 µg for an adult and 2.9 µg for a child (Health Canada 1992).

WHO (2004) states that concentrations in drinking-water are generally below 0.005 mg/L, although much higher concentrations (above 0.1 mg/L) have been measured where lead fittings are present. For example, boiled water from a lead-soldered electric jug, or jugs with lead in their element, contained an average of about 1 mg/L of lead (quoted in IARC 2006).

15,750 water utilities in the US reported detecting lead (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.13 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found <0.00011 to 0.000118 mg/L lead in the raw water, and 0.00033 to 0.00048 mg/L in the treated water.

### Removal methods

Lead is not expected to be a raw water contaminant. However, coagulation/flocculation with iron or aluminium salts has been shown to be effective in removing lead, especially where the lead is sorbed on to particles. Lime-softening removes lead in the form of Pb(OH)2. PAC and GAC have also been reported to be effective in removing lead.

The principal problem with lead in drinking-waters comes from corrosion of the distribution system (lead solder, pipes, weathering of lead paints). Corrosion can be minimised by:

* avoiding the use of lead plumbing
* calcium carbonate saturation, to seal lead surfaces with a non-toxic barrier of calcium carbonate
* pH and carbonate adjustment, to form a lead hydroxy-carbonate compound with minimum lead solubility. Correct pH and carbonate concentration are essential
* orthophosphate addition, to form a low solubility lead hydroxy-phosphate compound.

MoH (2012) provides a list of abatement options for controlling lead.

WRF (2015) is dedicated to procedures for controlling the amount of lead in drinking water.

### Analytical methods

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Inductively Coupled Plasma Method (APHA 3120).

2. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

### Health considerations

Lead is a metal with no known biological benefit to humans (WHO 2001).

Lead can be absorbed by the body through inhalation, ingestion or placental transfer. In adults, approximately 10 percent of ingested lead is absorbed. However, young children absorb 4–5 times as much and the biological half-life is considerably longer in children than in adults. After absorption, the lead is distributed in soft tissue such as the kidney, liver and bone marrow where it has a biological half-life in adults of less than 40 days, and in skeletal bone where it can persist for 20 to 30 years.

Lead is a cumulative general poison. Infants, children up to six years of age, the foetus and pregnant women are the most susceptible to adverse health effects. Placental transfer of lead occurs in humans as early as the 12th week of gestation and continues throughout development.

Lead can severely affect the central nervous system. Signs of acute intoxication include dullness, restlessness, irritability, poor attention span, headaches, muscle tremor, abdominal cramps, kidney damage, hallucinations, and loss of memory. Encephalopathy occurs at blood lead levels of 100–120 g/dL in adults and  
80–100 μg/dL in children. Signs of chronic toxicity may appear in adults with blood lead levels of 50–80 g/dL. Symptoms include tiredness, sleeplessness, irritability, headaches, joint pains and gastro-intestinal symptoms.

Many epidemiological studies have been carried out on the effects of lead exposure on the intellectual development of children. Although there are some conflicting results, on balance the studies demonstrate that exposure to lead can adversely affect intelligence.

Research on primates has supported the results of the epidemiological studies, in that significant behavioural and cognitive effects have been observed following postnatal exposure.

Other adverse effects associated with exposure to high amounts of lead include kidney damage, interference with the production of red blood cells, and interference with the metabolism of calcium needed for bone formation.

Renal tumours have been induced in experimental animals exposed to high concentrations of lead compounds in the diet, and the International Agency for Research on Cancer (IARC) has classified lead and inorganic lead compounds in Group 2B (possibly carcinogenic to humans). However, there is evidence from studies in humans that adverse neurotoxic effects other than cancer may occur at very low concentrations of lead and that a MAV derived on this basis would also be protective for carcinogenic effects. Lead compounds appear on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008. USEPA (2004) states the lead classification is B2: probable human carcinogen on the basis of sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumours with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumour sites. Short term studies show that lead affects gene expression. Human evidence is inadequate.

Owing to the decreasing use of lead-containing additives in petrol and of lead-containing solder in the food processing industry, concentrations in air and food are declining, and intake from drinking-water constitutes a greater proportion of total intake. From 1976 to 1991, the mean lead content in the blood of the US population aged from 1 to 74 years dropped 78 percent.

MfE (2011) states:

The most significant critical effect of low concentrations of lead is considered to be reduced cognitive development and intellectual performance in children. The JECFA was the only authoritative body that had previously derived a tolerable intake for lead; the PTWI of 25 μg/kg bw/week, and the TDI derived from this, has been the value most widely used by different international agencies. However, this value has been recently withdrawn. A toxicological intake of 1.9 μg/kg bw/day is instead recommended to be used in the derivation of soil contaminant standards in New Zealand. This intake is based on dose-response modelling by JECFA and is the dietary intake at which the IQ decreases 3 points in the population. This general shift in distribution was deemed to be of concern by JECFA, although the effects were considered to be insignificant at an individual level. Exposures of individuals are more relevant in the context of contaminated sites. Inhalation exposure and dermal absorption are expected to be negligible, and could be ignored in the derivation of soil contaminant standards for contaminated land in New Zealand, as has been done by other jurisdictions. Dietary intake is the primary source of background exposure to lead and was estimated to be 0.97 μg/kg bw/day for a child and 0.41 μg/kg bw/day for an adult.

The livestock guideline value is 0.1 mg/L (ANZECC/ARMCANZ 2000). These guidelines were to have been updated in 2012.

### Derivation of Maximum Acceptable Value

WHO (2011/2017) states that PTWI has since been withdrawn, and no new PTWI has been established, on the basis that there does not appear to be a threshold for the key effects of lead. However, substantial efforts have been made to reduce lead exposure from a range of sources, including drinking-water. Because it is extremely difficult to achieve a lower concentration by central conditioning, such as phosphate dosing, the guideline value is maintained at 0.01 mg/L but is designated as provisional on the basis of treatment performance and analytical achievability.

Prior to 2011 WHO considered there was sufficient evidence from human studies that adverse effects other than cancer may occur at very low levels of lead, and that a guideline thus derived would also be protective for carcinogenic effects, it is considered appropriate to derive the MAV using a tolerable daily intake approach.

In 1986, the Joint FAO/WHO Expert Committee on Food Additives had established a provisional tolerable weekly intake (PTWI) of 25 g lead/kg body weight (equivalent to 3.5 g/kg body weight per day) for infants and children on the basis that lead is a cumulative poison and that there should be no accumulation of body burden of lead. The PTWI was based on metabolic studies in infants showing that a mean daily intake of 3–4 g/kg body weight was not associated with an increase in blood lead levels or in the body burden of lead, whereas an intake of 5 g/kg body weight or more resulted in lead retention.

Up to and including the 2008 DWSNZ, the MAV for lead in drinking-water had been derived using the case of a bottle fed baby. As infants are considered to be the most sensitive subgroup of the population, this MAV will also be protective for other age groups. The MAV had been derived as follows:

0.0035 mg/kg body weight per day x 5 kg x 0.5 = 0.012 mg/L (rounded to 0.01 mg/L)

0.75 L per day

where:

* - Tolerable Daily Intake = 0.0035 mg/kg body weight per day
* average weight of a bottle fed baby = 5 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.5
* average amount of water consumed by a bottle fed baby = 0.75 L per day.

WHO (2003) stated that most lead in drinking-water arises from plumbing in buildings and the remedy consists principally of removing plumbing and fittings containing it. This requires time and money, and it is recognised that not all water will meet the guideline immediately. Meanwhile, all other practical measures to reduce total exposure to lead, including corrosion control, should be implemented.

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# Lithium

CAS No. 7439-93-2 (the metal).

### Maximum Acceptable Value

There are insufficient data to set a MAV for lithium.

In DWSNZ 2000 the provisional MAV was 0.9 mg/L (900 μg/L). In DWSNZ 2005, this was rounded up to 1 mg/L.

WHO (2004) and (2011) do not discuss lithium.

The use of lithium salts for meter calibration in water mains is acceptable providing that the maximum concentration of lithium in water supplied to consumers does not exceed 0.10 mg/L at any time (the analytical method used for lithium determination should have an accuracy better than ±0.01 mg/L at 0.10 mg/L). DWI (2015 and regularly updated).

EPA established an environmental exposure limit of 0.0001 mg/L (0.1 µg/L) for lithium hydroxide in fresh water (<http://www.epa.govt.nz/search-databases/Pages/substance-exposure-limit-register.aspx>).

### Sources to drinking-water

#### 1 To source waters

Lithium is an alkali metal that has properties similar to potassium and sodium. It is found in nature in nearly all igneous rocks, and in geothermally influenced water and natural brines. Lithium can enter water naturally through its dissolution from rocks and also through its disposal in wastewater at sites mining spodumene, lepidolite, petalite, amblygonite or triphylite (Merck & Co 1996).

The concentration of lithium in seawater is about 0.15 mg/L. Geothermal waters in the Taupo Volcanic Zone can contain 10 to 30 mg/L Li (GNS 2015).

Lithium can also enter drinking-water through its use in a variety of industries including alloys for the aircraft industry, lithium carbonate and borate as ceramics, air-conditioning, welding and brazing fluxes (as lithium chloride and fluoride) and lubrication grease (as organo-lithium compounds). It is used in long-life batteries (including pacemakers and lifebuoys). In countries other than New Zealand (which is nuclear-free) it is used in some nuclear applications as a coolant. Lithium salts an be used as a tracer.

Lithium metal and lithium compounds are used by drug manufacturers in the synthesis of intermediates for important pharmaceuticals and in the production of lithium carbonate, which is widely used for treatment of clinical depression. Lithium metal is a key reagent in the production of Vitamin A and the hormones beta-methasone and ethinylestradiol. Lithium bromide and lithium carbonate are used in the process of steroidal synthesis.

Lithium also occurs in meat, poultry, dairy products and some vegetables. Therapeutic doses of lithium for the control of bipolar disorder are 900 to 1,200 mg/day for adults. Health authorities have not endorsed a recommended dietary allowance for lithium, although an intake of 1 mg/day for a 70 kg adult has been suggested by some advocates.

### Forms and fate in the environment

Lithium metal does not occur in free form in nature and is highly soluble in water (it becomes hydrolysed to the lithium ion. The metal presents a fire and explosion risk when exposed to water); the reaction forms lithium hydroxide and hydrogen gas.

Lithium is a common element and is present in many plant and animal tissues. Daily intake of lithium is about 2 mg (Klaassen 1995).

### Typical concentrations in drinking-water

Lithium was included in Ministry of Health surveillance programmes until 1995, when the *Drinking-water Standards for New Zealand 1995* (MoH 1995) were introduced. At this stage, analysis was halted because lithium was not listed as a health significant determinand in the 1995 DWSNZ.

7490 samples were collected as part of the Ministry of Health’s five yearly surveillance programmes (reference: WINZ and STANLEY databases). The results ranged from nd (not detected) to 0.43 mg/L, with a median concentration of nd. Geothermal and volcanic areas, including the Waikato River, produce the highest concentrations.

In 2013/14 Hamilton’s six-monthly analyses have found 0.08 to 0.09 mg/L lithium in the raw water, and 0.08 to 0.10 mg/L in the treated water.

9 water utilities in the US reported detecting lithium in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.05 mg/L.

Harari *et al* (2012) report that in San Antonio de los Cobres (Northwestern Argentina) the public drinking water, supplied from a natural spring outside the village, has about 1 mg/L of lithium and 6 mg/L of boron. The authors note that at present there are no specific data to suggest adverse effects in children born in the San Antonio de los Cobres area compared to other areas in the study.

### Removal methods

No information is available on methods for removal of lithium in drinking-water.

### Analytical methods

#### Referee method

Flame emission (APHA 3500-Li B).

#### Some alternative methods

Inductively coupled plasma – mass spectrometer (EPA 200.8).

### Health considerations

Lithium is readily absorbed from the gastrointestinal tract. Distribution in human organs is almost uniform. Excretion is chiefly through the kidneys, but some is eliminated in the faeces. In general the distribution of lithium in the body is quite similar to that of sodium and potassium, and it may compete with these elements at the renal tubular level (VDH 1997).

Concentrations of arsenic in groundwater samples in the Waikato region showed a highly (p<0.001) significant positive correlation to concentrations of lithium, and to ammonia, dissolved Fe and Mn (see arsenic datasheet). Strong correlations were also shown with caesium, antimony, boron and bromine, and good correlation with rubidium (Waikato Regional Council 2010).

#### Acute poisoning

The oral toxicity of most lithium compounds is relatively low, oral LD50 values for several compounds and animal species range from 422–1165 mg/kg (Opresko 1995).

#### Chronic exposure

There is ample evidence that long-term exposure to lithium at low levels causes no serious adverse health effects. Because lithium is used routinely as a drug for the treatment of clinical depression, there is a large body of data on the human health effects of long term exposure to lithium. Lithium is administered therapeutically as the carbonate salt (lithium carbonate) in daily oral doses of 100–1800 mg/day for the treatment of manic and endogenous depression. The active principle in these salts is the lithium ion Li+, which having a smaller diameter, can easily displace K+ and Na+ and even Ca2+, in spite of its greater charge, occupying their sites in several critical neuronal enzymes and neurotransmitter receptors. Therapeutically useful amounts of lithium are only slightly lower than toxic amounts, so the blood levels of lithium must be carefully monitored during treatment to avoid toxicity.

The therapeutic use of lithium carbonate may produce unusual toxic responses. These include neuromuscular changes, eg, tremor, muscle hyperirritability, and ataxia (muscle incoordination), central nervous system changes (blackout spells, epileptic seizures, slurred speech, coma, psychosomatic retardation), and increased thirst, cardiovascular changes (cardiac arrhythmia, hypertension, and circulatory collapse), gastrointestinal changes (anorexia, nausea, and vomiting), and renal damage (albuminuria and glycosuria) (Klaassen 1995, Merck & Co 1996 and VDH 1997).

Lithium treatment is not recommended for patients with significant renal or cardiovascular disease, severe debilitation or dehydration, or sodium depilation, or to patients receiving other medications (eg, diuretics), because the risk of lithium toxicity is high in such patients. Data from lithium birth registries suggest an increase in cardiac and other abnormalities in infants born to women who have been prescribed treatment with lithium carbonate. Animal studies have shown lithium carbonate is a weak teratogen (causes birth defects). Increased frequency of cleft palate and faetal loss have been observed among the offspring of mice treated chronically during pregnancy with lithium carbonate. However, studies in rats, rabbits and monkeys gave no evidence of lithium-induced birth defects (VDH 1997).

A Japanese study found that lithium levels in tap water had a small but significant inverse association with depressive symptoms and interpersonal violence in a general population of adolescents using a large individual-level data set. The mean (SD) lithium concentration in tap water was 0.48 (0.52) μg/L; range 0.01 to 2.10 µg/L (Ando et al 2017). However, Knudsen et al (2017) found no benefits in a 22 year study in Denmark, concluding there does not seem to be a protective effect of exposure to lithium on the incidence of suicide at levels below 31 μg/L in drinking water.

There are no current US federal standards for lithium in drinking-water. The USEPA derived a provisional chronic oral reference dose (RfD) for lithium of 0.02 mg/kg/day. This reference dose is based on a Lowest Observable Adverse Effect Level (LOAEL) of 20 mg/kg/day for nephrotoxicity in humans receiving long-term lithium therapy for the treatment of manic depressive disorders. These studies examined 21 cases of intoxication in patients receiving daily doses of lithium for months or years. Also, to protect human health, the USEPA estimated that a lithium concentration in a potable water supply should not exceed 0.7 mg/L (VDH 1997).

The International Agency for Research on Cancer (IARC) has not classified lithium for its ability to cause cancer. Lithium carbonate and citrate appear 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 basis for the PMAV in the DWSNZ (2000) was:

A tolerable daily intake approach has been used for the derivation of the PMAV for lithium in drinking-water, as follows:

NOAEL mg/kg body weight per day x 70 kg x proportion from d/w = 0.9 mg/L

2 L x uncertainty factor

(rounded up to 1 mg/L in DWSNZ (2005).

where:

* No-Observable-Adverse-Effect Level = X mg /kg body weight per day
* 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 = X percent
* uncertainty factor = X.

The above was derived in-house by the MoH – references and calculation lost, but presumed to have been based on Canadian data.

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# Manganese

### Maximum Acceptable Value

WHO (2011/2017) states that manganese is not of health concern at levels found in drinking-water.

Previously, based on health considerations, WHO stated that the concentration of manganese in drinking-water should not exceed 0.4 mg/L. DWSNZ (2008) had adopted that value as the MAV.

The USEPA (2009/2011) established a lifetime health advisory of 0.3 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.

Based on aesthetic considerations, the concentration of manganese in drinking-water should not exceed 0.04 mg/L (the GV in the DWSNZ). The aesthetic objective in Canada is not greater than 0.05 mg/L. The USEPA has a secondary drinking water regulation of 0.05 mg/L for manganese, based on staining and taste considerations; however, WRF (2015a) recommends utilities maintain treated water in the range of 0.015–0.020 mg/L to avoid consumer complaints.

The Prescribed Concentration or Value (PCV) for manganese in England and Wales is 0.05 mg/L. See Notes.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on aesthetic considerations, the concentration of manganese in drinking water should not exceed 0.1 mg/L measured at the consumer’s tap, and manganese would not be a health consideration unless the concentration exceeded 0.5 mg/L.

### Sources to drinking-water

#### 1 To source waters

Manganese can reach the aquatic environment from the weathering of rocks and minerals and runoff from soils. Manganese is not an essential constituent of any of the more common silicate rock minerals, but it can substitute for iron, magnesium or calcium in silicate structures. Many igneous and metamorphic minerals contain manganese as a minor constituent. It is a significant constituent of basalt and many olivines and of pyroxene and amphibole. Small amounts are present in dolomite and limestone, substituting for calcium. It is ubiquitous in the environment and comprises about 0.1 percent of the Earth’s crust.

The concentration of manganese in seawater is about 0.001 mg/L.

Manganese may enter water from industrial discharges and agricultural runoff. Manganese is a component of the pesticide mancozeb, and in some formulations of prochloraz. Manganese and its compounds are used in the steel industry in the manufacture of metal alloys, in the manufacture of dry cell batteries, paints, varnishes, inks, dyes, glass, ceramics, matches, fire works and fertilisers. Manganese is also used in animal feeds. Methylcyclopentadienyl manganese tricarbonyl (MMT) has been used as an octane enhancer in petrol, eg, in USA and Canada.

Bottom waters in lakes and reservoirs can become very low in dissolved oxygen. Under these conditions iron and manganese leach out from the sediments (manganese can exceed 2 mg/L in bottom anoxic water) and begin to circulate through the water column, resulting in raw water concentrations that may be too high for the treatment process to handle. Uncontaminated rivers and streams generally have low concentrations of manganese, ranging from 0.001 mg/L to 0.1 mg/L.

Similarly, manganese concentrations can be elevated in groundwaters, particularly when the dissolved oxygen content is low; usually the iron concentration is elevated too.

Manganese is an essential nutrient for micro-organisms, plants, and animals. The concentrations of manganese in food can vary considerably. The highest concentrations have been reported in grains, nuts and vegetables, while tea leaves can have very high concentrations. It has been estimated that the average dietary intake of manganese is 2 to 4 mg per day.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

There are situations in which manganese concentrations in the water at the consumers’ taps can be higher than those entering the distribution system. This is not the result of manganese being dissolved from reticulation materials. It arises from manganese, in either soluble or insoluble form, passing into the distribution system, insoluble manganese may settle out in pipes, and soluble manganese may be oxidised to insoluble forms, by oxygen or other chemical oxidants, such as chlorine (particularly at elevated pH as occurs in concrete lined mains and fibolite pipes), and also settle. Changes in water flows through the system may then resuspend the particulate manganese, which may lead to “black” water at the consumers’ taps. This is discussed fully in WRF (2015b).

Certain nuisance organisms (usually bacteria) can concentrate manganese and give rise to taste and odour, and colour and turbidity problems in distributed water.

### Forms and fate in the environment

Manganese has three valence states in natural environments (2+, 3+, 4+). In the absence of dissolved oxygen the Mn(II) form predominates, otherwise it is readily oxidised to Mn(IV). In natural oxygenated waters, a substantial fraction of manganese is present in suspended form. In surface waters, divalent manganese will be oxidised to manganese dioxide which will undergo sedimentation. In the presence of complex-forming inorganic and organic compounds, the colloidal stability of manganese oxides will be enhanced. Alternatively, in areas of low dissolved oxygen or in anaerobic areas at low pH, soluble manganese forms may persist. Many groundwaters reported to carry large manganese concentrations are from thermal springs.

### Typical concentrations in drinking-water

Manganese was measured routinely in New Zealand drinking-water supplies as an aesthetic parameter as part of the Department of Health three yearly surveillance programme. Since 1995 it has been classified as an inorganic parameter of health significance. Of 1,143 samples analysed from 913 supplies between 1983 and 1989, 91 samples (9.3 percent of supplies) were equal to or exceeded the highest desirable level (ie, GV) of 0.05 mg/L.

The P2 Chemical Determinand Identification Programme, sampled from 400 zones, found manganese concentrations to range from “not detectable” (nd) to 1.7 mg/L, with the median concentration being 0.002 mg/L (Limit of detection = 0.001 mg/L). The Priority 2 Identification Programme found 6 distribution zones supplying drinking-water to a total of 1,551 people with manganese at greater than the GV (ESR 2001).

9,913 water utilities in the US reported detecting nanganese in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 3.7 mg/L.

### Removal methods

Oxidation of Mn(II) to insoluble Mn(IV) compounds is a commonly employed technique. Aeration uses oxygen from the air to achieve oxidation. This precipitates the manganese, which is either allowed to settle or is removed by filtration. The rate at which oxidation occurs is pH dependent, becoming faster as the pH is increased. Some Mn(II) is adsorbed on to higher oxidation states of Mn in slightly alkaline solution. A coating of higher oxides of manganese on filter granules acts to catalyse the removal of lower oxidation states. Organically-bound manganese is not removed very well by aeration.

Other oxidising agents such as chlorine, ozone and potassium permanganate may be used for the removal of manganese. A pH of 8 is required for chlorine oxidation, and pH 7 to 8 is optimum for permanganate oxidation.

Sand or anthracite filters can be used to filter the precipitated manganese from the water. Before the removal process becomes efficient, a coating of iron and manganese oxides must develop on the grains of the filter medium. Until this coating develops, removal may be poor. Natural zeolites (ion exchange materials) treated with manganese can be used as the filter medium. This medium is known as greensand, and requires periodic regeneration of the iron and manganese oxide coating. Mn(II) ions come into contact with the zeolite and are converted to the insoluble oxide which is filtered out by the filter bed.

Natural zeolites or synthetic resins can also be used to remove manganese by a true ion-exchange process. Sodium attached to the zeolite is exchanged for Mn(II) ions in the incoming water. It is important that the water is free of oxygen that might lead to oxidation and precipitation of the manganese, as this will foul the zeolite.

Manganese, in the absence of oxygen, can be removed effectively by raising the pH above approximately 10 to precipitate manganese hydroxide. This can be exploited if the lime-soda ash process is being used for hardness reduction.

Refer also to Chapter 18: Aesthetic considerations.

### Analytical methods

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Flame Atomic Absorption Spectrometric Method (APHA 3111).

2. Inductively Coupled Plasma Method (APHA 3120B).

3. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

### Health considerations

The greatest exposure to manganese is usually from food. Adults consume between 0.7 and 10.9 mg/day in their diet, with even higher intakes being associated with vegetarian diets high in grains, beans and nuts. Heavy tea drinkers may also have a higher intake of manganese than the average person.

Nuts, chocolate, cereal-based products, crustaceans and molluscs, pulses, and fruits and fruit products are rich sources of manganese. The main contributors to the manganese intake of adults are cereal-based products, vegetables, fruits and fruit products and beverages. In the EU, estimated mean manganese intakes of adults range from 2 to 6 mg/day, with a majority of values around 3 mg/day. Estimated mean manganese intakes range from 1.5 to 3.5 mg/day in children, and from 2 to 6 mg/day in adolescents (EFSA 2013a).

Manganese is an essential trace element for mammals, is a component of a number of metalloenzymes involved in amino acid, lipid and carbohydrate metabolism, with an estimated daily nutritional requirement of 30–50 g/kg body weight. Manganese functions as an enzyme activator and is a constituent of several enzymes (eg, glycosyl transferases, pyruvate carboxylase, manganese superoxide dismutase). Primary signs of manganese deficiency are impaired growth, skeletal abnormalities, depressed reproductive function, ataxia of the newborn and faults in lipid and carbohydrate metabolism (EFSA 2013). The body is able to adapt to a wide range of manganese intakes by regulating both efficiency of absorption in the intestine and the quantity excreted via bile (EFSA 2013a).

Its absorption rate can vary considerably according to actual intake, chemical form, and presence of other metals such as iron and copper. Typically, only about 3 to 8 percent of ingested manganese is absorbed by the gastro-intestinal tract. In infants and young animals, very high absorption rates of manganese have been observed. After absorption it is concentrated in the liver and eventually excreted in faeces. It has a relatively short biological half-life of 13 to 37 days in humans. Manganese deficiency affects bone, the brain and reproduction in a number of animal species.

The health effects from over-exposure of manganese are dependent on the route of exposure, the chemical form, the age at exposure, and an individual’s nutritional status. Irrespective of the exposure route, the nervous system has been determined to be the primary target with neurological effects generally observed.

Most human studies reporting adverse effects are of exposure via inhalation. There is conclusive evidence from occupational studies in humans that inhalation exposure to high levels of manganese compounds can lead to a disabling syndrome of neurological effects referred to as “manganism” and is characterised by a “Parkinson-like syndrome”. Although it is typical for symptoms to occur after several years of exposure, some individuals may begin to show signs after 1–3 months of exposure.

There is no convincing evidence of toxicity in humans associated with the consumption of manganese in drinking-water, but there are only limited studies available. By the oral route, manganese is often regarded as one of the least toxic metals.

Experiments with animals have shown no adverse effects, other than a change in appetite and a reduction in the metabolism of iron in haemoglobin synthesis. Some *in vitro* studies have reported mutagenic activity for manganese on mammalian cells and bacteria.

Oral doses ranging from 1 to 150 mg per kg of body weight per day produced a number of neurological effects in rats and mice, mainly involving alterations in neurotransmitter and enzyme levels in the brain. These changes were sometimes accompanied by clinical signs, such as changes in coordination and activity level (ATSDR 2008).

In a recent study (DWI 2014) the authors concluded that the experimental studies that have been conducted do suggest that there is a biologically plausible hypothesis for an adverse effect on neurological development of Mn taken in orally via drinking water. The human epidemiological studies particularly on children are suggestive of an effect on intellectual and cognitive development; however, the type of studies conducted are not the most appropriate for measuring the longer-term effect such as those which may occur after accumulation of Mn in the brain. Mn(II) is the oxidation form of Mn that is absorbed by the gastrointestinal tract when consumed orally. Mn(IV) is generally insoluble and so less likely to be absorbed to any extent. In adults, oral absorption is low (3–5 percent), but in infants and children, this is higher (40 percent) and excretion, which is via the biliary system, is lower. Therefore, the potential for uptake into the body is higher in children. In the body there is some exchange of oxidative states between Mn(II) and Mn(III), with Mn(II) more likely to be excreted.

WRF (2015b) reports that manganese oxides and oxyhydroxides on pipe linings have been shown to be important in scavenging dissolved trace metals. It appears possible that during releases of legacy Mn, drinking water customers could be exposed to other contaminants at levels greater than maximum contaminant levels for contaminants such as lead, arsenic, barium, nickel and radium.

The reference dose or RfD (USEPA 2009/2011) is 0.14 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 1.6 mg/L.

As there is insufficient evidence available to derive an average requirement or a population reference intake, an Adequate Intake (AI) is proposed. Mean intakes of manganese in adults in the EU are around 3 mg/day. In addition, null or positive balances have consistently been observed with intakes of manganese above 2.5 mg/day. An AI of 3 mg/day is proposed for adults, including pregnant and lactating women. For infants aged from 7 to 11 months, an AI of 0.02–0.5 mg/day is proposed, which reflects the wide range of manganese intakes that appear to be adequate for this age group (EFSA 2013a).

There is no firm evidence that manganese is carcinogenic. Some studies indicate that it may, in fact, have an anti-carcinogenic effect.

### Derivation of Maximum Acceptable Value

WHO (2011a and 2017) state that because the health-based value is well above concentrations of manganese normally found in drinking-water, it is not considered necessary to derive a formal guideline value. Their health-based value is calculated on the same basis as the MAV in the 2008 DWSNZ.

The WHO GV for manganese in drinking-water is based on the upper range value of manganese intake of 11 mg/day, identified using dietary surveys, at which there are no observed adverse effects (ie, considered a NOAEL), using an uncertainty factor of 3 to take into consideration the possible increased bioavailability of manganese from water. This results in a TDI of 0.06 mg/kg of body weight. The 2008 DWSNZ MAV was derived as follows:

0.06 mg/kg body weight per day x 70 kg x 0.2 = 0.42 mg/L (rounded to 0.4 mg/L)

2 L per day

where:

* Tolerable Daily Intake = 0.06 mg/kg body weight per day
* average adult weight = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.2
* average amount of water consumed per day = 2 L per day.

The aesthetic guideline value in the DWSNZ for manganese is 0.04 mg/L due to the fact that it deposits in water mains and causes discoloration of porcelain and clothing when scoured out. Some water supplies may need to aim for a lower concentration to prevent the build-up of manganese in the distribution system. At concentrations exceeding 0.1 mg/L, manganese can impart an undesirable taste to water.

Health Canada (1987) states that the aesthetic objective for manganese in drinking water is 0.05 mg/L. The presence of manganese in drinking water supplies may be objectionable for a number of reasons. At concentrations above 0.15 mg/L, manganese stains plumbing fixtures and laundry and produces undesirable tastes in beverages. As with iron, the presence of manganese in water may lead to the accumulation of microbial growths in the distribution system. Even at concentrations below 0.05 mg/L, manganese may form coatings on water distribution pipes that may slough off as black precipitates.

The USEPA has a secondary MCL of 0.05 mg/L reportedly due to both a bitter taste and brown-black colour and staining of plumbing fixtures and laundry. Manganese in tap water can cause peculiar tasting tea, coffee, and other heated aqueous beverages. However, recent research indicates that Mn(II) and Mn(IV) have very little taste at concentrations found in water. A concentration of 0.05 mg/L Mn may not control customer complaints; 97 percent of consumers who viewed MnO2 in a glass of tap water visually detected 0.005 mg/L in the form of MnO2 (from WRF 2015).

The lifetime health advisory value of 0.3 mg/L will protect against concerns of potential neurological effects (USEPA 2004). In addition, this document provides a one-day and 10-day HA of 1 mg/L for acute exposure. However, it is advised that for infants younger than six months, the lifetime HA of 0.3 mg/L be used even for an acute exposure of 10 days, because of the concerns for differences in manganese content in human milk and formula and the possibility of a higher absorption and lower excretion in young infants.

The MAV for manganese in the DWSNZ in 1995 and 2000 had been 0.5 mg/L. The 1995 datasheet stated: no single study is suitable to calculate a MAV. However, the weight of evidence from actual daily intake and studies in laboratory animals give manganese in drinking-water in which neurotoxic and other toxic effects were observed, support the view that a provisional health-based MAV of 0.5 mg/L would be adequate to protect public health.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The risk assessment advice level for chronic effects (exposure greater than 10 percent of a lifetime) for manganese is 0.3 mg/L.

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# Mercury

CAS No. (metallic mercury) 7439-97-6.

### Maximum Acceptable Value

Based on health considerations, the concentration of inorganic mercury in drinking-water should not exceed 0.007 mg/L (7 g/L) as Hg.

In DWSNZ 2005, the MAV was 0.002 mg/L, as total mercury.

The maximum contaminant level or MCL for inorganic mercury (USEPA 2009/2011) is 0.002 mg/L. The USEPA also established a lifetime health advisory of 0.002 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 Health Advisory for Group C carcinogens includes an adjustment for possible carcinogenicity.

The maximum acceptable concentration in Canada is 0.001 mg/L; this applies to all forms of mercury.

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

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of total mercury in drinking water should not exceed 0.001 mg/L.

Mercury is one of the “priority pollutants” under the US Clean Water Act. Mercury compounds (inorganic, alkyl, alkyloxyalkyl and aryl) 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>.

Mercury (inorganic) 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

Mercury can enter the aquatic environment from the weathering of rocks and minerals and run-off from soils and due to geothermal activity. Cinnabar (HgS) is the most common mercury containing mineral. In New Zealand, mercury occurs in Permian-Triassic marine volcanic associations at Moumoukai (Hunua Ranges), at Kakariki, Waiohine River (Wairarapa) and Greenvale (Southland); in late Cenozoic Hydrothermal deposits at Puhipuhi and Ngawha Springs (Northland), and Lake Waikare in the lower Waikato; and in minor alluvial occurrences in Southland, Westland, Northland and the Hauraki Goldfields. High levels of mercury may also be present in geothermal fluids and natural springs. Mercury is released to the environment during volcanic eruptions, and by coal burning which is one of the principle routes to the environment today.

The concentration of mercury in seawater is about 0.0001 mg/L.

The mining and refining of metals (eg, gold) may be a major source of mercury in the environment, once when used in the amalgam extraction process, but now more as an impurity in the ore. Industrial discharges can also contribute to the mercury in water. Mercury is or was used in the paint industry in paint pigments and preservatives, including anti-fouling paints, in manufacturing chlorine and sodium hydroxide, and in the production of thermometers, electrical equipment (eg, mercury switches, batteries and fluorescent and mercury vapour lamps), dental amalgam (which is approximately 50 percent metallic mercury, 35 percent silver, 9 percent tin, 6 percent copper, and trace amounts of zinc), and in therapeutic medicinal compounds. Environment Canada estimates that more than one-third of the mercury load in sewage systems is due to dental practices (WHO 2005a). It is also a component of diesel exhaust. Of approximately 6,000 tonnes of mercury which enters the environment annually, about 2,000 tonnes comes from power stations and related industrial uses.

The WHO is engaged in a global mercury-free health-care initiative. One such initiative is the production of the technical guide “Replacement of mercury thermometers and sphygmomanometers in health care” (WHO 2011a). Each sphygmomanometer contains 80–100 g of mercury. Approximately two-thirds of the mercury circulating in the global environment is the result of industrial and other human activities.

The total annual mercury emissions for New Zealand in 2008 have been estimated using the 2005 draft of the UNEP Mercury Toolkit to be 3,000 kg Hg/year, with roughly equal contributions from natural and anthropogenic sources (MfE 2009). The most significant natural sources of mercury were volcanic emissions, comprising 54 percent of natural emissions and 28 percent of total emissions. The most significant anthropogenic sources were combustion of fossil fuels (440 kg Hg/year) and geothermal power generation (350 kg Hg/year), representing 29 percent and 23 percent of the anthropogenic emissions, respectively. Other significant anthropogenic mercury sources included wastewater biosolids (180 kg Hg/year) and mercury-containing batteries (170 kg Hg/year). Mercury-containing lamps are currently a small potential source of emissions, contributing approximately 50 kg/year which is only 3 percent of total anthropogenic emissions and less than 2 percent of overall mercury emissions from New Zealand. Compact fluorescent lamps currently make up about a third of potential emissions from mercury-containing lamps. MfE (2013) reported an inventory of the annual distribution of mercury and mercury-containing goods and materials in New Zealand from anthropogenic (man-made) sources in accordance with the guidance provided in the UNEP *Toolkit for identification and quantification of mercury releases*. The outputs to water are dominated by fuel/energy use and, to a lesser extent, primary metal production.

MfE (2012) developed a national set of soil contaminant standards for 12 priority contaminants and five common land uses; inorganic mercury levels range from 200 to 4,200 mg/kg depending on land use.

#### 2 From treatment processes

Mercury is used in the electrolytic production of chlorine; minute traces used to be able to be found in chlorine gas. Some mercury may enter the water as an impurity in low-iron content aluminium sulphate, polyaluminium chloride, titanium dioxide-derived ferric chloride and potassium permanganate (DWI 2013).

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

Mercury compounds can exist in three oxidation states in the natural environment: 0, +1 and +2. The stable form of mercury in most natural water systems is the free metal. However, mercury metal in surface water open to the atmosphere is liable to escape by vaporisation. In fresh waters, it is common for the mercury to be adsorbed to suspended particulate matter and to the sediments.

Methylation of inorganic mercury is an important process in water and occurs in both fresh water and seawater. Bacteria (*Pseudomonas* spp.) isolated from mucous material on the surface of fish and soil were able to methylate mercury under aerobic conditions. Some anaerobic bacteria that possess methane synthetase are also capable of mercury methylation. Once methylmercury is released from microbes, it enters the food-chain as a consequence of rapid diffusion and tight binding to proteins in aquatic biota. The enzymology of CH3Hg+ hydrolysis and mercury(II) ion reduction is now understood in some detail. Environmental levels of methylmercury depend on the balance between bacterial methylation and demethylation.

### Typical concentrations in drinking-water

Mercury was not measured routinely in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. Highest levels of mercury in water are found in some thermal groundwaters or waters receiving them, volcanic areas, and in water associated with mercury ores or in mining areas.

The P2 Chemical Determinand Identification Programme, sampled from 114 zones, found mercury concentrations to range from “not detectable” (nd) to 0.0011 mg/L, with the median concentration being “nd” (Limit of detection = 0.0005 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water mercury at greater than the MAV, but one DZ supplying 100 people was >50 percent MAV (ESR 2001).

Concentrations of total mercury in natural water are generally so low that accurate analysis is difficult. Studies overseas have reported concentrations of less than 0.0005 mg/L, with some sources less than 0.00003 mg/L (30 ng/L). The highest value was 0.0055 mg/L from some wells in Japan, where volcanic activity is frequent. In major Australian reticulated supplies, the concentrations of total mercury range up to 0.001 mg/L, with typical concentrations usually less than 0.0001 mg/L.

958 water utilities in the US reported detecting mercury (total inorganic) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.005 mg/L.

In 2013/14 Hamilton’s six-monthly analyses have found <0.00008 mg/L mercury in the raw water and the treated water.

Almost all mercury in uncontaminated drinking-water is thought to be in the form of Hg2+. There have been no reports of methylmercury being found in drinking-water.

### Removal methods

Inorganic and methyl mercury can be removed by filtration through GAC. Removal is better in the presence of ferric sulphate than in its absence, and removal is pH dependent with efficiency decreasing with increasing pH over the range 7 to 9. Co‑precipitation of Hg(OH)2 with Fe(III) hydroxide is effective.

Coagulation/flocculation with iron or aluminium salts removes mercury (about 80 percent of inorganic and 20–40 percent organic mercury), to an extent determined by the turbidity. Powdered activated carbon is effective for the removal of inorganic and organic mercury and can be used to enhance removal during coagulation.

### Analytical methods

#### Referee method

Cold-Vapour Atomic Absorption Method (APHA 3112B).

#### Some alternative methods

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

### Health considerations

The principal routes of human health impacts from mercury exposure are through direct inhalation of mercury vapour, and through ingestion of methylmercury. The former is the primary route for those working in the small-scale gold mining sector. Studies of the economic effects of these health impacts are few. The main source of ingestion of methylmercury (the second route) is the consumption of fish. Methylmercury is a form of mercury which is created in the environment from other types of mercury, and which bioaccumulates in fish, particularly in longer-lived species near the top of the food chain (OECD 2018).

Food is the main source of mercury in non-occupationally exposed populations; the mean dietary intake of mercury in various countries ranges from 0.002 to 0.02 mg/day per person. Kjellström et al (1989) reported an important study of the effects on IQ from ingesting methyl mercury in fish in New Zealand.

#### Inorganic mercury

Absorption of inorganic mercury from drinking-water by the gastro-intestinal tract may be less than 15 percent. Inorganic mercury compounds are rapidly accumulated in the kidney, the main target organ for mercury toxicity. The biological half-life of mercury is very long, probably years.

Chronic exposure of workers to mercury has resulted in reported health effects including tremors, mental disturbances and gingivitis (inflammation of the mucous membrane surrounding the teeth). The main toxic effects are to the kidney, leading to kidney failure.

In general, acute lethal toxic doses by ingestion of any form of mercury will result in symptoms including shock, cardiovascular collapse, acute renal failure and severe gastrointestinal damage.

Various reports indicate that inorganic mercury binds to, and damages, mammalian DNA. Some evidence of carcinogenicity in rats has been reported. The overall weight of evidence is that mercury(II) chloride possesses weak genotoxic activity but does not cause point mutations. IARC considers that methylmercury compounds are possibly carcinogenic to humans (Group 2B); metallic mercury and inorganic mercury compounds are not classifiable as to their carcinogenicity to humans (Group 3). Mercury compounds appear on the State of California EPA list of chemicals known to cause cancer or reproductive toxicity as at December 2008.

Mercury in drinking-water is considered to be a minor source of exposure to mercury except in circumstances of significant pollution. Almost all mercury in uncontaminated drinking-water is thought to be in the form of Hg2+. Thus, intake of organic mercury compounds, especially the alkyl mercurials, is unlikely to pose a direct risk through ingestion of drinking-water. However, the conversion of methylmercury to inorganic mercury may occur.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes minimal risk levels (MRLs) for mercuric chloride of:

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

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

The reference dose or RfD for inorganic mercury (USEPA 2009/2011) is 0.0003 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.01 mg/L.

#### Organic mercury

Organic mercury compounds are unlikely to be found in uncontaminated drinking-water. However, the toxic effects are more severe than those of inorganic mercury.

Methylmercury compounds are almost completely absorbed by the gastro-intestinal tract. Methylmercury has greater lipid solubility than inorganic mercury which permits it to cross biological membranes more easily, especially in the brain, spinal cord, peripheral nerves and the placenta.

Methylmercury affects the central nervous system and the main effects of poisoning are irreversible neurological disorder and mental disability.

Two major epidemics of methylmercury poisoning in Japan, known as Minimata disease, were caused by the industrial release of methyl mercury and other mercury compounds. The mercury was accumulated by edible fish, which were then consumed by humans. A number of other countries have suffered epidemics associated with the consumption of bread prepared from cereals treated with mercury contaminated fungicide.

MfE (2011) states:

Inorganic mercury is considered to be a threshold contaminant, with renal effects in rats considered the most sensitive endpoint. A tolerable daily intake of 2 μg/kg bw/day is recommended as this is the value most widely used by different international agencies. Inhalation exposure is expected to be negligible on contaminated sites due to limited volatility of the forms of mercury likely to be present (mercury II). Dermal absorption is also expected to be negligible. Dietary intake, in particular seafood, and dental amalgam are the primary sources of background exposure to mercury. Dietary intakes of inorganic mercury were estimated to be 0.05 μg/kg bw/day for a child and 0.025 μg/kg bw/day for adults. Intake from dental amalgam was considered to be negligible for children and 0.04 μg/kg bw/day for adults, giving rise to a total inorganic mercury intake of 0.065 μg/kg bw/day for adults.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.0003 mg/kg/day for chronic-duration oral exposure (>364 days) to methyl mercury.

The livestock guideline value is 0.002 mg/L (ANZECC/ARMCANZ 2000). These guidelines were to have been updated in 2012.

WHO (2011b) includes an 80-page paper on the health effects of mercury in food.

### Derivation of Maximum Acceptable Value

The MAV for inorganic mercury (measured as Hg) in drinking-water has been derived from the following two studies (WHO 2005b). WHO (2017) now considers the first calculation to be the more important, although they are very similar.

1. Based on observed kidney effects in a NTP 26-week study in rats:

0.23 mg/kg body weight per day x 70 kg x 0.1 = 0.00805 mg/L

2 L per day x 100

where:

* NOAEL = 0.23 mg/kg body weight per day and applying an uncertainty factor of 100 (for inter- and intraspecies variation) after adjusting for five days per week dosing
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* average amount of water consumed by an adult = 2 L per day.

2. Based on observed renal effects in a NTP two-year study in rats:

1.9 mg/kg body weight per day x 70 kg x 0.1 = 0.0067 mg/L

2 L per day x 1000

where:

* LOAEL = 1.9 mg/kg body weight per day and applying an uncertainty factor of 1000 (10 for inter- and intraspecies variation, plus 10 for adjustment from a LOAEL to a NOAEL)
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* average amount of water consumed by an adult = 2 L per day.

WHO 2005 averaged the two results in deriving their guideline value, so the MAV based on a 70 kg body weight will be 0.007 mg/L.

The 0.002 mg/L MAV for total mercury in the DWSNZ (1995, 2000 and 2005) had been based on the following, taken from the 1995 datasheet:

In 1972, the Joint FAO/WHO Expert Committee on Food Additives established a provisional tolerable weekly intake of 5 g/kg bw of total mercury, of which no more than 3.3 g/kg body weight should be present as methylmercury. In 1988, JECFA reassessed methylmercury, as new data were only then available on this compound, and confirmed the previously recommended provisional tolerable weekly intake (PTWI) of 3.3 g/kg of body weight for the general population. It should be noted that pregnant women and nursing mothers are likely to be at a greater risk from the adverse effects of methylmercury. The available data were considered insufficient to recommend a specific methylmercury intake for this population group. To be on the conservative side, WHO has used the intake for methylmercury to derive a MAV for inorganic mercury in drinking-water.

The MAV for mercury in drinking-water had been derived as follows:

0.00047 mg/kg body weight per day x 70 kg x 0.1 = 0.00165 mg/L (rounded to 0.002)

2 L per day

where:

* Tolerable Daily Intake = (0.003.3/7) = 0.00047 mg/kg body weight per day
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* average amount of water consumed by an adult = 2 L per day.

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# Molybdenum

### Maximum Acceptable Value

WHO (2011/2017) states that molybdenum is not of health concern at levels found in drinking-water.

Previously, based on health considerations, the concentration of molybdenum in drinking-water should not exceed 0.07 mg/L. Molybdenum 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.

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

The USEPA (2009/2011) established a lifetime health advisory of 0.04 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 health considerations, the concentration of molybdenum in drinking water should not exceed 0.05 mg/L.

### Sources to drinking-water

#### 1 To source waters

Molybdenum is widely distributed in nature, occurring chiefly as molybdenite and molybdates. The weathering of igneous and sedimentary rocks (especially shales) and run-off from soils constitutes an important natural source of molybdenum to the aquatic environment. In New Zealand disseminated molybdenite occurs in granitic rocks around Nelson and in quartz and pegmatite veins with molybdenite and chalcopyrite close to granitic contacts in the South Island, eg, Mt Radiant. Minor molybdenite-chalcopyrite mineralisation has also been identified in Westland, Southland, Fiordland and near Reefton.

Geothermal waters in the Kawerau area contain about 0.002 µg/L Mo (GNS 2015).

The concentration of molybdenum in seawater is about 0.008 mg/L.

Molybdenum can also enter water via the discharge of industrial wastes from industries in which it is used, and from agricultural run-off. Molybdenum and its compounds are used in the manufacture of special steel alloys, electronic apparatus, glass, ceramics, pigments, as lubricants in oils and greases (as molybdenum disulphide), and in some fertilisers to overcome molybdenum deficiency in soils. It is found in fly-ash deposits from coal-fired power stations.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources. Results from a British study suggest that water distribution pipework has a negligible effect on supplied tapwater molybdenum concentrations (NERC 2008).

### Forms and fate in the environment

In natural waters molybdenum occurs in the +4 and +6 oxidation states as molybdenum sulphide (disulphide) (MoS2) and molybdate anion (MoO42-) respectively. In aerobic waters it is the soluble molybdates which are stable. Adsorption and co‑precipitation of the molybdate anion by hydrous oxides of iron and aluminium play primary roles in determining the aquatic fate of molybdenum. In oxic waters at pH>5, molybdenum occurs principally as the molybdate oxyanion (MoO42-). This means that molybdenum can be present as a stable soluble species under the conditions of many natural waters.

### Typical concentrations in drinking-water

Molybdenum was not measured routinely in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme.

The P2 Chemical Determinand Identification Programme, sampled from 831 zones, found molybdenum concentrations to range from “not detectable” (nd) to 0.003 mg/L, with the median concentration being “nd” (Limit of detection = 0.005 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with molybdenum at greater than the MAV (ESR 2001).

In 2013/14 Hamilton’s six-monthly analyses have found 0.0004 mg/L molybdenum in the raw water and the treated water.

WHO (2003/2011) states that molybdenum does not normally exceed 0.01 mg/L in drinking-water although concentrations as high as 0.2 mg/L have been reported in areas near mining sites. Molybdenum was present in 32.7 percent of surface water samples from 15 major river basins in the US at concentrations ranging from 2 to 1,500 μg/L (mean 60 μg/L). Levels in groundwater ranged from undetectable to 270 μg/L in another survey (from WHO 2011).

Some water supplies in the USA have occasionally recorded molybdenum levels around 0.05 mg/L.

299 water utilities in the US reported detecting molybdenum in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.13 mg/L.

A survey has been carried out of molybdenum concentrations in drinking water from 12 public-supply sources distributed across England and Wales, monitored up to four times over an 18-month period, together with domestic taps from three of their supply areas. All analysed concentrations were more than an order of magnitude lower than the earlier WHO guideline value for Mo of 0.07 mg/L (DWI 2008). The variability was very small between houses (old and new), between pre- and post-flush samples, and between the tapwater and respective source-water samples.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 62,986 drinking water samples for molybdenum between 2013 and 2015, and found 25,377 samples exceeded the minimum reporting level (MRL) of 0.001 mg/L, and 40 contained >0.04 mg/L.

### Removal methods

There are no published methods for the removal of molybdenum from drinking-water. Sorption reactions have a strong control on molybdenum mobility. The molybdate ion adsorbs readily to iron oxides (eg, hydrous ferric oxide) at low to neutral pH as well as to aluminium oxides. It also adsorbs strongly to manganese oxides and some clays under acidic conditions (DWI 2008). Therefore the concentration of molybdenum should be reduced in most coagulation and filtration systems.

### Analytical techniques

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Inductively Coupled Plasma Method (APHA 3120B).

2. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

### Health considerations

Molybdenum is considered to be an essential element, with an estimated daily requirement of about 0.1–0.3 mg for adults. Many foods contain significant amounts of molybdenum. Legumes, grains and liver have the highest concentrations and food is a significant source of intake.

In humans, 30–70 percent of dietary molybdenum is absorbed from the gastro-intestinal tract and appears rapidly in the blood and most organs, and is excreted primarily in the urine. Molybdenum crosses the placental barrier. There is no apparent bioaccumulation of molybdenum in human tissues.

Few data are available on the long- and short-term toxicity of molybdenum in humans. The results from a cross-sectional study of 400 persons in two settlements of a molybdenum-rich province of the former Soviet Union suggested that the high incidence (18–31 percent) of a gout-like disease was associated with high intake of molybdenum (10–15 mg/day). The disease was characterised by joint pains of the legs and hands, enlargement of the liver, disorders of the gastrointestinal tract, liver and kidney, increased blood levels of molybdenum and uric acid, increased xanthine oxidase activity, decreased blood levels of copper and increased urinary copper (WHO 2011).

Following a request from the European Commission, the Panel on Dietetic Products, Nutrition and Allergies (NDA) derived Dietary Reference Values (DRVs) for molybdenum. Molybdenum is efficiently and rapidly absorbed at a wide range of intakes, and the body is able to maintain homeostasis through the regulation of excretion via the urine. Molybdenum deficiency in otherwise healthy humans has not been observed and there are no biomarkers of molybdenum status. Various metabolic balance studies have been performed to establish molybdenum requirements. However, only one balance study, which was performed with a constant diet and under controlled conditions in adult men, was considered to be of sufficient duration. In this small study, balance was reported to be near zero when molybdenum intakes were 22 µg/day. Biochemical changes or symptoms suggestive of molybdenum deficiency were not observed, and it is possible that humans may be able to achieve molybdenum balance at even lower intakes. Data on molybdenum intakes and health outcomes were unavailable for the setting of DRVs for molybdenum. As the evidence required to derive an Average Requirement and a Population Reference Intake was considered insufficient, an Adequate Intake (AI) is proposed. Observed molybdenum intakes from mixed diets in Europe were taken into consideration in setting this value. An AI of 65 µg/day is proposed for adults; a figure that is based on molybdenum intakes at the lower end of the wide range of observed intakes. It is suggested that the adult AI also applies to pregnant and lactating women. An AI is also proposed for infants from seven months and for children based on extrapolation from the adult AI using isometric scaling and the reference body weights of the respective age groups (EFSA 2013).

The reference dose or RfD (USEPA 2009/2011) is 0.005 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.2 mg/L.

No data are available on the carcinogenicity of molybdenum by the oral route. Tests for mutagenicity with bacteria have been inconclusive.

In April 2017 ATSDR established minimal risk levels (MRL) for molybdenum:

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

0.008 mg/kg/day for intermediate-duration oral exposure (15–365 days)

The livestock guideline value is 0.15 mg/L depending on total dietary intakes of molybdenum, copper, iron and sulfur. At molybdenum concentrations greater than 0.15 mg/L, the animal diet should be investigated to ensure that copper levels are sufficient to account for the total dietary intake of molybdenum (ANZECC/ARMCANZ 2000). These guidelines were to have been updated in 2012.

### Derivation of Maximum Acceptable Value

The 0.07 mg/L MAV in the 2008 DWSNZ was based on the next paragraph. WHO (2011 and 2017) has changed this from a guideline value to a health-based value, using the same data.

In a two-year study of humans exposed through their drinking-water, a no-observable-adverse-effects level (NOAEL) of 0.2 mg/L was identified for molybdenum. There are some concerns about the quality of this study although it has been used for the derivation of the MAV. An uncertainty factor of 10 would normally be applied to reflect intraspecies variation. However, it is recognised that molybdenum is an essential element, and therefore a factor of 3 is considered to be adequate. This gives a MAV of 0.07 mg/L, which is in the same range as a value derived on the basis of the results of toxicological studies in animal species and is consistent with the essential daily requirement for molybdenum.

Ammonium molybdate imparts a slightly astringent taste to water at concentrations above about 10 mg of molybdenum per litre.

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# Monochloramine

CAS No. 10599-90-3. Sometimes called monochloroamine or chloramide. When the word ‘chloramine’ is used, it usually refers to monochloramine. When the word ‘chloramines’ is used it usually refers to all of monochloramine, dichloramine and nitrogen trichloride (trichloramine), and often the organic chloramines as well.

Monochloramine is not the same as chloramine-T or other similarly named products. Those are sodium salts of chlorinated arylsulphonamides which are used as antibiotics or germicides in human medicine or veterinary topical antiseptics and disinfectants. They are not used in water supply, nor are they disinfection by-products. Chloramine T has a datasheet in the organics section.

### Maximum Acceptable Value

Based on health considerations, the concentration of monochloramine in drinking-water should not exceed 3.0 mg/L. WHO (2004) talks of mg/L monochloramine. Analytical techniques usually report chloramines as mg Cl as Cl2/L. The monochloramine MAV of 3.0 mg/L can be expressed as 4.14 mg/L Cl as Cl2.

**Note:** To convert monochloramine to chlorine use the factor 71/51.5. Before rounding, the monochloroamine MAV is 3.29 mg/L so the conversion to the chlorine equivalent is actually 4.54 mg/L.

The maximum residual disinfectant level for monochloramine (USEPA 2009/2011) is 4 mg/L measured as free chlorine. The USEPA also established a lifetime health advisory of 3 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 2 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 2016) state that based on health considerations, the concentration of monochloramine in drinking water should not exceed 3 mg/L (equivalent to 5 mg Cl as Cl2/L in chloraminated systems).

### Sources to drinking-water

#### 1 To source waters

Monochloramine may be present in source waters as a result of discharge from industries in which it is used (rarely). Its principal use is as intermediates in the manufacture of hydrazine.

#### 2 From treatment processes

Monochloramine is formed in chlorinated water that contains ammonia, and some organic nitrogen compounds. The concentration of monochloramine depends upon the pH and chlorine to nitrogen ratio – see Chapter 15.

Ammonia (and chlorine) may be added intentionally to the water to produce monochloramine as a disinfectant, particularly where the water has a high chlorine demand due to organic matter and/or high temperatures. As at 2017, no New Zealand water supplies are chloraminated.

#### 3 From the distribution system

It is possible that reactions of chlorine with nitrogenous material in the distribution system may produce monochloramine. Most individuals are able to taste chloramines at concentrations below 5 mg/L, and some at levels as low as 0.3 mg/L. The odour threshold has been reported at 0.5 mg/L (NHMRC, NRMMC 2016), which is often about the level aimed for when using chloramine for disinfection.

### Forms and fate in the environment

Monochloramine is persistent in the environment and its rate of disappearance is a function of pH and salinity: its half-life increases with increasing pH and decreases with increasing salinity. Monochloramine decomposes faster if discharged into receiving waters containing bromide, presumably by the formation of bromochloramine and decomposition of the dihalamine. At pH <5.5, monochloramine slowly converts to form dichloramine.

### Typical concentrations in drinking-water

No typical value data are available for New Zealand. Monochloramine is not intentionally used at present as a disinfectant in New Zealand, and the concentrations of inorganic chloramines present in waters depends upon the concentrations of inorganic and some organic nitrogen compounds present in the raw water, and control of the chlorination process.

NHMRC, NRMMC (2016) reports that monochloramine is used as a disinfectant in some Australian reticulated supplies, and concentrations up to 4 to 5 mg/L (as total chlorine) have been applied at the start of long distribution systems to achieve concentrations ranging from 0.5 to 1.5 mg/L at the ends of distribution systems.

### Removal methods

It would not be normal practice to remove chloramine. Chemical reducing agents, including sodium thiosulphate, sulphur dioxide, and sodium bisulphite can be used to remove monochloramine. Activated carbon adsorbs monochloramine. Aeration and boiling are not effective for the removal of monochloramine; a minimal aeration loss of 10–15 percent has been reported with monochloramine. Ultraviolet light depletes only free chlorine, whereas chloramines seem to be quite stable in sunlight.

### Analytical methods

#### Referee method

DPD Ferrous Titrimetric Method (APHA 4500-Cl F).

The limit of detection for this method is approximately 0.2 mg/L for field use, although lower levels can be determined under laboratory conditions and with care. Analytical texts indicate that by manipulation of the conditions of the analysis measurement of monochloramine, dichloramine and trichloramine can be made. These methods are of use when ammonia only is in the water being chlorinated. In most natural waters nitrogen-containing organic compounds are also present. Organic chloramines are formed from these compounds when chlorine reacts with them. Organic chloramines also produce colour during the DPD test and make attempts to differentiate between the different inorganic chloramines of little value. Unless investigating taste and odour problems, it is recommended that only the total combined chlorine, ie, total chloramine concentration, is reported.

These methods measure monochloramine in terms of mg Cl as Cl2/L. The MAV of 3.0 mg/L monochloramine can be converted to mg Cl as Cl2/L based on molecular weights as follows:

to convert 3.0 mg/L monochloramine: 71/51.5 x 3 = 4.14 mg Cl as Cl2/L

where:

* 71 is the molecular weight of chlorine (Cl2)
* 51.5 is the molecular weight of monochloramine (NH2Cl).

#### Some alternative methods

1. Amperometric Titration Method (APHA 4500-Cl D and E)

While more accurate than the DPD methods, expensive equipment and a high degree of skill and care are required for this method. The limit of detection is better than 0.1 mg/L (or 0.01 mg/L for APHA 4500-Cl E). The APHA method describes variations that will allow the determination of mono- and di-chloramine. Interferences due to organic chloramines may also cause interferences with these methods.

2. DPD Colorimetric Method (APHA 4500-Cl G)

This method requires a spectrophotometer for the colorimetric measurements, although hand-held comparators do offer a cheaper, though less reliable variation for field use. The limit of detection (LOD), with instrumental assistance, is approximately 0.1 mg/L.

The LOD for the comparator depends on the colour disc in use. Chloramine concentrations as low as 0.2 mg/L approximately should be detectable, but the accuracy of the method depends upon use of the correct lighting (natural lighting should be used with the sun behind the viewer), the individual’s ability to match colours and judge their intensity, and ensuing that readings are taken as soon after colour development as possible. The LOD may be about 0.10 mg/L when using a Nessleriser.

The same comment on the usefulness of trying to determine the individual chloramine concentrations made for the referee method, also applies to this method.

### Health considerations

Monochloramine is readily absorbed by the gastro-intestinal tract following oral administration in the rat; about 25 percent is excreted in urine over 120 hours. Following administration, the highest concentration was contained in the plasma. Monochloramine is metabolised rapidly by gastric juices to the chloride ion which is excreted mainly in the urine.

Monochloramine has been reported to be weakly mutagenic with some bacteria but did not increase bone marrow chromosomal aberrations. Although monochloramine has been shown to be mutagenic in some *in vitro* studies, it has not been found to be genotoxic *in vivo*.

Studies have revealed equivocal evidence of carcinogenic activity of chloraminated drinking-water in female rats, as indicated by an increase in incidence of mononuclear cell leukaemia. The IARC (2004) states that chloramine is not classifiable as to its carcinogenicity to humans (Group 3). USEPA (1994) classified monochloramine as D: not classifiable as to human carcinogenicity, based on inadequate human data and equivocal evidence of carcinogenicity from animal bioassays. A two-year bioassay showed a marginal increase in mononuclear cell leukemia in female F344/N rats. No evidence of carcinogenic activity was reported in male rats or in male or female B6C3F1 mice. Genotoxicity studies, both *in vitro* and *in vivo*, gave negative results.

In humans, short-term exposure to concentrations of up to 24 mg/L of monochloramine in drinking-water did not produce adverse effects. Similarly, volunteers given water containing up to 5 mg/L of monochloramine for 12 weeks did not exhibit adverse effects.

Epidemiological studies did not report an association between ingestion of chloraminated drinking-water and increased urinary bladder mortality rates in humans.

When tap-water containing chloramines was used for dialysis, acute haemolytic anaemia, characterised by denaturation of haemoglobin and lysis of red blood cells, was reported in haemodialysis patients.

The reference dose or RfD (USEPA 2009/2011) is 0.1 mg/kg/d. The NOAEL of 9.5 mg chloramine/kg/day in rats was chosen as the basis for the chronic oral RfD. Earlier, USEPA (1994a) had suggested a NOEL of 8.3 mg/kg/day monochloramine based on chronic liver inflammatory changes in mice. Although a higher NOAEL in the study of 17.2 mg/kg-day was found for mice, rats may be the more sensitive species since doses between 9.5 and 17.2 mg/kg-day were not tested in rats. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 3.5 mg/L.

There has been some public concern in the US about possible links between the use of chloramine as a residual disinfectant in drinking water and skin, gastric and lung irritation. Despite many people in the US claiming to be susceptible to dermatitis from exposure to chloraminated water, there is said to be no substantive evidence to support these concerns and the USEPA has concluded that monochloramine has not been shown to be a cause or a factor in the reported skin problems and that the standard for monochloramine is set at a level where no digestive problems are expected to occur (DWA 2011).

Monochloramine has been reported to cause taste/odour problems at low concentrations (for some people as low as 0.5 mg/L; most people can taste it at <5 mg/L). However, some water supplies intentionally maintain a chloramine residual without problems. It is possible that where taste/odour problems have been reported, the chloramine may involve a more complex root. This will be particularly so in swimming pools.

In the Stage 1 D/DBPR, USEPA established a MRDLG of 4 mg/L for chloramine based on a weight of evidence evaluation of both the cancer and non-cancer effects and classified chloramines as “not classifiable as to human carcinogenicity.” USEPA has not set an RfD for chloramines. The MRDLG was based on a NOAEL of 9.5 mg/kg/day for no treatment-related effects for monochloramine from a two-year drinking water study in rats and mice, an uncertainty factor of 100, adult tap water consumption of two litres/day for a 70 kg adult and an assumed drinking water contribution of 80 percent of total exposure. Due to the lack of significant new health effects data available for the Stage 2 D/DBPR, USEPA did not revise the MRDLG for chloramines at that time (USEPA 2016).

### Derivation of Maximum Acceptable Value

The MAV for monochloramine has been derived using a tolerable daily intake approach as follows:

9.4 mg/kg body weight per day x 70 kg x 1 = 3.3 mg/L (rounded to 3.0)

2 L per day x 100

where:

* No-observable-adverse-effects level = 9.4 mg/kg body weight per day. This is the highest dose administered to male rats in a two-year drinking-water study. It was chosen because of the probability that the lower body weights were caused by the unpalatability of the drinking-water
* average adult weight = 70 kg
* the proportion of the tolerable daily intake attributable to monochloramine = 1
* the average quantity of water consumed by an adult = 2 L per day
* uncertainty factor = 100 for intra- and interspecies variation. An additional uncertainty factor for possible carcinogenicity was not applied because equivocal cancer effects reported in the NTP study in only one species and in only one sex were within the range observed in historical controls.

An additional uncertainty factor for possible carcinogenicity was not applied because equivocal cancer effects reported in the NTP study in only one species and in only one sex were within the range observed in historical controls. Because higher chloramines are formed only occasionally and cause taste and odour problems at concentrations lower than those at which monochloramine causes taste and odour problems, only monochloramine has been considered for development of a health-based guideline value (WHO 2017).

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# Nanoparticles

Often referred to as engineered nanoparticles (ENPs), man-made nanoparticles, or engineered nanomaterials (ENMs). Refer also to the datasheet for silver. This datasheet also touches on the subject of microplastics.

### Maximum Acceptable Value

The WHO Guidelines for Drinking-water Quality do not have a Guideline Value and the DWSNZ do not have a MAV for nanoparticles.

### Sources to drinking-water

#### 1 To source waters

The chemical substance(s) that constitute an ENM can be classified into the following main categories:

* inorganic nanomaterials – these include metals (titanium, zinc, silver, calcium and magnesium), metal oxides and metal nitrides and non-metals such as selenium and silicates
* organic nanomaterials – these include nanopolymers and nanomedicines as well as nano-carrier systems (eg, encapsulates) containing antimicrobials, and nutritional and health supplements, etc
* surface functionalised nanomaterials – these may be inorganic materials that are surface functionalised with organic moieties, or vice versa. Examples include organically modified nanoclays for food packaging applications.

Applications for engineered nanoparticles (approximately 1 nm to 100 nm) are being found increasingly in almost every field.

Nano-silver is currently the most commonly used ENM in a wide range of consumer products. An increasing number of nano-silver containing products is available, including cosmetics and personal care products, food and health-food, antimicrobial paints and coatings, hygienic surfaces and packaging materials, and medical applications etc. Indeed, the number of products incorporating nano-silver as an antimicrobial, antiodorant and a (proclaimed) health supplement has surpassed all other ENMs currently in use in different consumer sectors.

Nano-sized cerium oxide is used as a secondary fuel catalyst in diesel. The application is claimed to reduce fuel consumption and particulate emissions. Typically added to diesel at a concentration of 5 to 10 ppm, nano-cerium oxide is claimed to increase fuel efficiency by ~10 percent. The catalyst is already in use on a large scale in bus fleets in a number of countries including the UK, Philippines and New Zealand.

A range of metal, metal oxide and organic-based ENPs have been identified that have the potential to contaminate drinking waters. Worst case predicted concentrations in drinking waters were in the low to sub-microgram/L range and more realistic estimates were tens of ng/L or less. From the available information on the scales of production/use, cost of materials, and the likely release patterns into the environment, the following application areas have been regarded the most relevant in relation to potential for contamination of drinking water sources:

* Paints, coatings, and adhesives: In most cases ENP will be fixed in, or bound to, the paint/coating matrix. However, environmental degradation over time may release ENPs into the aquatic environment. Overall environmental loading from this area of application may be high because of the shear high volumes of ENMs produced/used in these categories. The ENMs to consider include titanium dioxide, zinc oxide, silica (including organo-silica), alumina, and silver.
* Cosmetics and personal care products: This category is the most relevant in terms of potential for contamination of aquatic environments due to direct release of ENMs into waters both during use and on disposal of the products. The main ENMs to consider include titanium dioxide and zinc oxide, silver, hydroxyapatite, and fullerenes.
* Cleaning products: This small-scale use category is relevant in terms of potential for contamination of aquatic environments due to direct release of ENMs into wastewaters during use and on disposal of the products. The main ENMs to consider include alumina and alumina-polyurethane nanocomposites, silica, titanium, zirconium and silicon carbide.
* Eyeglass/lens coating: This is a small scale use category, but the ENMs used may enter the aquatic environment. The main ENM to consider include nano polymer thin film coating.
* Water treatment/filtration system: Currently a small use category, but likely to increase in the future (growing use in developing countries). The ENMs used in this category may end up in water (dependent on solubility and stability of the materials in water). The main ENMs used include alumina, zero-valent iron, and titanium dioxide.
* Fuel additives: This small scale use category is important because it will lead to direct release of ENMs into the air, expected to end up in the aquatic environment. The main ENM to consider is cerium oxide (CeO).
* Algae preventers: This product category will lead to direct release of ENMs into the aquatic environment. The main ENM to consider is lanthanum. The current production/use scales are unknown.

Microplastics (MPs) are plastic particles under 5 mm in size (but seldom <0.3 mm). They enter the environment through human use. Some plastics are manufactured as MPs; however, larger plastic debris can degrade into micro-sized particles over time with exposure to sun and water. Microplastics have been found to adsorb and transport ambient pollutants such as persistent organic pollutants (WRF 2018).

There are 13 types of MPs – polyethylene, polypropylene, and polystyrene are the most common. There are three primary categories of MPs:

* microfibers, usually the most common type of microplastics; derived from synthetic textiles and slough off during daily use and machine washing of clothing. Most microfibers released into water are between 0.1–0.8 mm in size
* fragments forming as a result of physical breakage of macroplastics
* microbeads, which are common in personal care products; these are being increasingly banned around the world.

#### 2 From treatment processes

No known sources.

### Forms and fate in the environment

The stability of nanoparticles in water depends upon their chemical structure, but also on other particle properties (eg, size and surface coating) as well as on environmental conditions (eg, water pH, presence of organic matter, temperature, ionic background and strength). For example, carbon-based nanoparticles such as C-60 have been found to form negatively charged colloids that are dispersible in water.

### Typical concentrations in drinking-water

It is believed that wastewater treatment plants should remove at least 90 percent of microplastics. Removal in water treatment plants has not been studied but the traditional size class of 300–500 μm would be expected to make it through a modern-day drinking water treatment plant that has just filtration. One study found microfibres in 83 percent of 159 tap water samples from around the world and in 94 percent of the US tap waters sampled (WRF 2018).

### Removal methods

Research with ENPs has shown that metallic, metal oxide and carbonaceous ENPs can be removed during coagulation, sedimentation and filtration.

Microfiltration and ultrafiltration membranes have nominal pore sizes around 0.1 µm (100 nm). Therefore ENPs should be removed in conjunction with particle neutralisation (ie, coagulation and flocculation).

Activated carbon is used to sorb dissolved pollutants such as pesticides. It likely improves removal of particles, including ENPs, but no direct evidence of this has been shown.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Humans can be exposed to these particles directly during product use or indirectly following release to the natural environment. One potential indirect exposure route is through the consumption of contaminated drinking waters.

For the majority of product types, human exposure via drinking water is predicted to be less important than exposure via other routes. The exceptions were some clothing materials, paints and coatings and cleaning products. The particles contained in these products include Ag, Al, TiO2, Fe2O3 and carbon-based materials. Although predicted concentrations of these materials in UK drinking water are low, any future work on risks of ENPs to drinking waters should probably focus on these materials and the development of the UK market for products containing these materials.

On the macro scale, the substances from which nanomaterials are made may be of low toxicity, but when formed into nanomaterials their toxicological properties change. The large surface area per unit mass of nano-sized particles plays an important part in their toxicity. Toxic substances, such as heavy metals, adsorb to the particles’ surfaces, and the surfaces also generate free radicals, which may trigger the body’s immune system, causing inflammation.

Assessments of the health risks presented by nanomaterials are hindered by several factors. It is technically difficult to measure their concentrations in the environment, with the result that limited data exists that allows an assessment of environmental exposure. Modelling approaches to the estimation of environmental exposures also encounter difficulties because of limited information about the amounts of the materials produced, the extent to which they are released into the environment during the lifecycle of a product, and the form they are in when they are released into the environment (they may agglomerate after manufacture). Attempts to estimate toxicity based on the structure of the nanomaterials are confounded by the very rapid development of new nanomaterials, which outstrips the rate at which structure-toxicity relationships are determined.

### Derivation of Maximum Acceptable Value

No MAV.

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# Nickel

CAS No. 7440-02-0 (the metal).

### Maximum Acceptable Value

Based on health considerations, the concentration of nickel in drinking-water should not exceed 0.08 mg/L. Nickel 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.

In DWSNZ 2005, the provisional MAV was 0.02 mg/L; the WHO guideline value had been considered provisional owing to uncertainties about the effect level for perinatal mortality.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of nickel in drinking water should not exceed 0.02 mg/L.

The USEPA (2009/2011) has a lifetime health advisory of 0.1 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 Prescribed Concentration or Value (PCV) for nickel in England and Wales is 0.02 mg/L. See Notes.

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

### Sources to drinking-water

#### 1 To source waters

Nickel can enter the aquatic environment naturally from the weathering of rocks and minerals and run-off from soils. Nickel is mined from sulfide or oxide ores (laterites). Nickel substitutes for iron in ferrous sulphides and also occurs in nickel-bearing laterites in ultramafic bedrock terranes, so may appear in groundwater. It is generally distributed uniformly through the soil profile, with the highest concentrations found in igneous rocks, and much lower levels found in sedimentary rocks (shales, clays, limestone, and sandstones). Atmospheric deposition of Ni has occurred as a result of the burning of oil and coal. Agricultural fertilisers, particularly phosphates, are also a significant source of Ni in soil but it is unlikely to build-up in soil in the long term from their use.

The average concentration in groundwater in the Netherlands ranges from 0.008 mg/L (urban areas) to 0.017 mg/L (rural areas).

The concentration of nickel in seawater is about 0.001 mg/L.

Industrial discharges can also contribute to the nickel in water. Nickel is used mainly in the production of stainless steel (major use) and other corrosion-resistant alloys, colouring glass, nickel-cadmium batteries, coins, plating metals, as a catalyst in industrial processes and in oil refining and in the manufacture of foods, baked goods, soft drinks, flavouring syrups and ice cream. Main releases to the environment are from the burning of fossil fuels and in waste discharges from electroplating industries.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

Elevated nickel levels can arise in reticulated waters from the corrosion of nickel-plated fittings. Drinking-water generally contains less than 0.01 mg/L, but, occasionally, nickel may be released from the plumbing fittings resulting in concentrations of up to 0.5 mg/L (IPCS 1991).

Leaching of nickel from kettles with nickel-plated elements was reported by DWI. 2009. In re-boiling tests, prolonged standing of water for 8 and 16 hours caused large increases in nickel concentrations. Test results when using drinking water were generally less than our MAV. Tests using deionised water resulted in significantly higher nickel concentrations than the drinking water used. Nickel concentrations fell over the 12-week period of the test. Nickel leaching was very low from a control kettle with a stainless steel element.

### Forms and fate in the environment

In aqueous solution nickel occurs mainly in the +2 oxidation state. It is present as relatively soluble salts in association with suspended solids and in combination with organic matter. Nickel co-precipitates with iron and manganese oxides and adsorbs to suspended organic matter.

Terrestrial plants take up nickel from soil, primarily via the roots. The amount of nickel uptake from soil depends on various geochemical and physical parameters including the type of soil, soil pH, humidity, the organic matter content of the soil, and the concentration of extractable nickel.

### Typical concentrations in drinking-water

Nickel was not measured routinely in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme.

The P2 Chemical Determinand Identification Programme, sampled from 900 zones, found nickel concentrations to range from “not detectable” (nd) to 0.70 mg/L, with the median concentration being “nd” (Limit of detection = 0.001 mg/L). The Priority 2 Identification Programme found 83 distribution zones supplying drinking-water to a total of 286,742 people with nickel at greater than the MAV, which then was 0.02 mg/L (ESR 2001).

In major Australian reticulated supplies, concentrations of nickel range up to 0.03 mg/L, with typical concentrations less than 0.01 mg/L.

In Canada, the median nickel level in drinking-water supplies was below the detection limit of 0.002 mg/L; the maximum level observed was 0.069 mg/L. In drinking-water in the USA, 90 percent of all samples (*n* = 2503) contained ≤0.01 mg/L, and 97 percent had nickel concentrations of ≤0.02 mg/L.

Certain stainless steel well materials were identified as the source of increased nickel concentrations in groundwaters in Arizona, USA. Mean nickel levels were  
0.008–0.395 mg/L; in some cases nickel levels were in the range 1–5 mg/L (WHO 2005).

Concentrations of nickel in water boiled in electric kettles may, depending on the material of the heating element, be increased markedly, especially in the case of new or newly decalcified kettles. The greatest concentrations are associated with nickel-plated elements; however, leaching decreases over time. Nickel concentrations in the range 0.1–0.4 mg/L, with extreme values over 1 mg/L, have been reported (WHO 2005).

Leaching of nickel from chromium–nickel stainless steel pipework into drinking-water diminished after a few weeks. As chromium was rarely found at any time in the water, this indicates that the leakage of nickel is not of corrosive origin, but rather attributable to passive leaching of nickel ions from the surface of the pipes. Concentrations of nickel leaching from new stainless steel pipes used for drinking-water were up to 0.006 mg/L. This maximum concentration can be increased when the pipes are assembled with tinned copper and gunmetal fittings. Fittings such as taps, which are chromium-plated, release much higher concentrations, but these decrease significantly with time (WHO 2005).

### Removal methods

Nickel is rarely found in natural waters, partly because of the low solubility of some of its compounds. Nickel can be co-precipitated with iron and manganese oxides. Conventional full water treatment can remove 35–80 percent of nickel, down to a concentration of about 0.02 mg/L.

Resins with chelating functional groups such as phosphoric acid and EDTA have a very high affinity for nickel.

To avoid corrosion of nickel-plated fittings, the corrosiveness of the water should be minimised. Taps should be flushed before using the water for drinking. Or nickel-plated taps should be avoided.

### Analytical methods

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Inductively Coupled Plasma Method (APHA 3120B).

2. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

### Health considerations

Food is the dominant source of nickel exposure in the non-smoking, non-occupationally exposed population; water is generally a minor contributor to the total daily oral intake. Nickel is present in many foods. Highest concentrations occur in cocoa, soy beans and some cereals. It has been estimated that the average daily dietary intake is between 0.1 mg/day and 0.3 mg/day. Pulmonary intake of  
0.002–0.023 mg nickel/day can result from smoking 40 cigarettes a day.

In humans, absorption of soluble nickel from drinking-water may be 40 times higher than absorption of nickel from food. Nickel appears to be distributed to all organs, with primary accumulation in the kidneys, lungs and liver, with excretion occurring mainly through urine. Long-term exposure may result in toxic effects to the kidney. Nickel is able to pass through the human placenta.

Thirty-two industrial workers accidentally drank water contaminated with nickel sulphate and nickel chloride (1,630 mg/L as Ni). The nickel doses in persons who developed symptoms were estimated to range from 7 to 35 mg/kg of body weight. Twenty workers developed symptoms, including nausea, vomiting, diarrhoea, giddiness, lassitude, headache, and shortness of breath. In most cases, these symptoms lasted for a few hours, but they persisted for 1–2 days in seven cases (WHO 2005).

Individuals may be sensitised to nickel via direct and prolonged dermal contact to this metal from the wearing of jewellery. In addition, traces of nickel in tap water may occur if plastic tap fittings are coated with nickel so that chrome plating is able to adhere to the brass or plastic fitting. Sale and installation of such taps is increasing in the UK, and nickel at levels approaching, or slightly exceeding the drinking water standards are being reported in water company random tap sample monitoring programmes. It is not known if these levels are high enough to trigger skin disease in already sensitised individuals (DWI 2011).

The reference dose or RfD for soluble nickel salts (USEPA 1996/2009/2011) is 0.02 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.7 mg/L.

Nickel is a common skin allergen and can cause dermatitis, particularly in adult women.

Tests for mutagenicity with strains of bacteria have mostly been negative but gene mutations and chromosome aberrations have been reported in mammalian cells.

Several epidemiological studies have suggested a risk of nasal, sinus and lung cancer by inhalation of nickel. The International Agency for Research on Cancer concluded that inhaled nickel sulphate is carcinogenic to humans. There is no evidence that nickel is carcinogenic when ingested. However, in 2009 IARC stated that “The Working Group reaffirmed the classification of beryllium and its compounds, cadmium and its compounds, chromium (VI) compounds, and nickel compounds as “carcinogenic to humans” (Group 1). Studies involved complex occupational exposures to a metal and its compounds, making it impossible to separately assess their carcinogenicity.”

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

The livestock guideline value is 1 mg/L (ANZECC/ARMCANZ 2000). These guidelines were to have been updated in 2012.

### Derivation of Maximum Acceptable Value

The MAV for nickel in drinking-water was derived as follows:

0.012 mg/kg body weight per day x 70 kg x 0.2 = 0.084 mg/L (rounded to 0.08 mg/L)

2 L per day

where:

* lowest-observable-adverse-effect level = 0.012 mg/kg body weight per day established after oral provocation of fasted patients with an empty stomach. In this study, nickel was administered as a single dose at a level that is much higher than would normally be possible through drinking-water and/or with the presence of food in the stomach, which would significantly reduce the absorption
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.2
* uncertainty factor: because this LOAEL is based on a highly sensitive human population, it is not necessary to include an uncertainty factor
* average amount of water consumed by an adult = 2 L per day.

See WHO (2007/2017) for a summary of a well-conducted two-generation study on rats, using a NOAEL of 1.1 mg of nickel per kg of body weight per day, also resulting in a guideline value of 0.07 mg/L (for a 60 kg person), ie, equivalent to a MAV of 0.08 mg/L.

A general toxicity value of 0.13 mg/L can be derived, but this may not be sufficiently protective of individuals sensitised to nickel, for whom a sufficiently high oral challenge has been shown to elicit an eczematous reaction (WHO 2005/2011/2017).

The basis for the MAV in the 1995 and 2000 DWSNZ, and the PMAV in the 2005 DWSNZ had been:

A tolerable daily intake approach has been used for the derivation of the PMAV. The relevant database for deriving a no-observable-adverse-effects level is limited. On the basis of a dietary study in rats in which altered organ to body weight ratios were observed, a NOAEL of 5 mg/kg body weight per day has been determined. An uncertainty factor of 1000 had been used.

5 mg/kg body weight per day x 70 kg x 0.1 = 0.02 mg/L

2 L per day x 1000

where:

* no-observable-adverse-effect level = 5 mg/kg body weight per day from a dietary study in rats in which altered organ to body weight ratios were observed
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* uncertainty factor: 1000 (100 for inter- and intraspecies variation and an additional factor of 10 to compensate for the lack of adequate studies on long-term exposure and reproductive effects, the lack of data on carcinogenicity by the oral route (although nickel, as both soluble and sparingly soluble compounds, is now considered as a human carcinogen in relation to pulmonary exposure), and a much higher intestinal absorption when taken on an empty stomach in drinking-water than when taken together with food
* average amount of water consumed by an adult = 2 L per day.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for soluble nickel salts is 0.1 mg/L.

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# Nitrate and Nitrite

### Maximum Acceptable Value for nitrate (short-term)

Based on health considerations, the concentration of nitrate (as NO3-) in drinking-water should not exceed 50 mg/L. This is protective against methaemoglobinaemia and thyroid effects in the most sensitive subpopulation, bottle-fed infants, and, consequently, other population subgroups.

### Maximum Acceptable Value for nitrite (short-term)

Based on health considerations, the short-term concentration of nitrite (as NO2-) in drinking-water should not exceed 3 mg/L. This is protective against methaemoglobinaemia induced by nitrite from both endogenous and exogenous sources in bottle-fed infants, the most sensitive subpopulation, and, consequently, the general population.

### Maximum Acceptable Value for nitrate plus nitrite

The sum of the ratios of the concentrations of each to its Maximum Acceptable Value (short-term) should not exceed 1.

### DWSNZ (2008): Maximum Acceptable Value for nitrite (long-term and provisional)

Based on health considerations expressed in WHO (2004), the long-term concentration of nitrite (as NO2-) in drinking-water should not exceed 0.2 mg/L. This WHO guideline value for chronic (long-term) effects of nitrite was considered provisional owing to uncertainty surrounding the relevance of the observed adverse health effects for humans and the susceptibility of humans compared with animals.

Note that WHO (2011/2017) no longer includes a long-term (chronic) guideline value for nitrite.

The Prescribed Concentration or Value (PCV) for nitrate in England and Wales is 50 mg/L as nitrate. The Prescribed Concentration or Value (PCV) for nitrite in England and Wales is 0.5 mg/L as nitrite at the consumers’ taps and 0.1 mg/L at the WTP. See Notes.

The maximum contaminant level or MCL for nitrate (USEPA 2009/2011) is 10 mg/L as N, and 1 mg/L for nitrite as N, or a total of 10 mg/L. The maximum acceptable concentration in Canada is 10 mg/L for nitrate as N, and 1 mg/L for nitrite as N*. In cases where nitrite is measured separately from nitrate, the concentration of nitrite should not exceed 3.2 mg/L as NO2-.*

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the guideline value of 50 mg-NO3/L (as nitrate) has been set to protect bottle-fed infants under three months of age. Up to 100 mg-NO3/L can be safely consumed by adults, and by children over 3 months of age. Where a water supply has between 50 and 100 mg-NO3/L nitrate, active measures are required to ensure that those caring for infants are aware of the need to use alternative water sources in making up bottle feeds for babies under three months of age. Based on health considerations, the concentration of nitrite in drinking water should not exceed 3 mg-NO2/L (as nitrite).

Note that 50 mg/L nitrate as NO3- is equivalent to 11.3 mg/L as N, 3 mg/L nitrite as NO2- is equivalent to 0.9 mg/L as N, and 0.2 mg/L nitrite as NO2- is equivalent to 0.06 mg/L as N.

### Sources to drinking-water

#### 1 To source waters

Nitrate and nitrite can enter the aquatic environment from the oxidation of vegetable and animal debris and animal excrement.

Nitrate and nitrite can also enter water from agricultural, domestic and industrial discharges. Nitrate is used in chemical fertilisers, oxidising agents in the chemical industry, in the manufacture of glass, enamels for pottery, matches, pickling meat and in the production of explosives. A major source of nitrate is from municipal wastewaters and septic tanks. Nitrite is also used as a corrosion inhibitor in industry, and as a food preservative, especially for curing meats.

An example of the long-term effect of land use on the nitrate content in groundwater was presented in Figure 17 of Auckland City (2016). Prior to 1977 Pukekohe Borough water was supplied from a shallow spring, Hickey Spring. The nitrate level of the spring progressively increased from 6.5 mg/L as N in 1959 to 20 mg/L in 2015. Other bores in the area are also trending upwards.

#### 2. From the Treatment Processes

The chlorination of raw waters containing significant amounts of ammonia or nitrite may lead to increases in nitrate through their oxidation. As 70 percent or more of the chlorine consumed during the oxidation of ammonia leads to nitrogen (the gas) production, the increase in nitrate concentrations is likely to be small unless ammonia concentrations are very high.

#### 3 From the distribution system

Nitrite can be formed chemically in distribution pipes by *Nitrosomonas* bacteria during stagnation of nitrate-containing and oxygen-poor drinking-water in galvanised steel pipes, or if chloramination is used to provide a residual disinfectant but its occurrence is almost invariably sporadic. Nitrification in distribution systems can increase nitrite levels, usually by 0.2–1.5 mg/L.

### Forms and fate in the environment

Nitrate and nitrite are naturally occurring ions which make up part of the nitrogen cycle. Nitrate is the oxidised form of combined nitrogen found in natural waters and in dilute aqueous solutions is chemically unreactive. Under anaerobic conditions nitrate may be reduced to nitrite and ammonia. Nitrite is seldom present in surface waters at significant concentrations but may be present in groundwaters. High nitrite concentrations are generally indicative of contamination. Incomplete nitrification of ammonia and denitrification of nitrate result in the biochemical production of nitrite which is generally present only under anaerobic conditions.

### Typical concentrations in drinking-water

#### Nitrate

Nitrate was routinely measured in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. Of 1908 samples analysed between 1983 and 1989, 14 samples (0.7 percent) contained concentrations equal to or exceeding the 1984 MAV of 10 mg/L (N).

The P2 Chemical Determinand Identification Programme, sampled from 673 zones, found nitrate concentrations to range from “not detectable” (nd) to 30 mg/L as NO3-N, with the median concentration being 0.2 mg/L (Limit of detection = 0.1 mg NO3-N/L). The Priority 2 Identification Programme found six distribution zones supplying drinking-water to a total of 1017 people with nitrate at greater than the MAV (ESR 2001).

In 2012 the Canterbury District Health Board stated that 33 of 289 wells tested in Canterbury exceeded the MAV for nitrate, the majority being around Ashburton.

26,177 water utilities in the US reported detecting nitrate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 30 mg/L as N.

Results of a survey of 210 waters (both source and zone) were reported by ESR (2016). Source waters sampled were groundwaters, mainly from dairying districts. Nitrate concentrations were below the limit of detection of the method used in 88 percent of samples. In only 2 percent (3) of source waters, and in none of the zone waters, did the nitrate concentration exceed 50 percent of the MAV. The MAV was not exceeded in any water sampled in this study. The highest reported nitrate concentrations in source waters were found in supplies in the Waikato (27 mg NO3/L) and Canterbury (27 mg NO3/L) districts. The highest nitrate concentration found in a zone water was in Canterbury (25 mg NO3/L) in the Darfield zone.

#### Nitrite

Nitrite was not measured routinely in New Zealand drinking-water supplies as part of the Department of Health three-yearly surveillance programme.

The P2 Chemical Determinand Identification Programme, sampled from 227 zones, found nitrite concentrations to range from “not detectable” (nd) to 0.088 mg/L, with the median concentration being “nd” (Limit of detection = 0.005 NO2- -N mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with nitrite at greater than the MAV (ESR 2001).

2,719 water utilities in the US reported detecting nitrite in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 2.78 mg/L as N.

### Removal methods

#### Nitrate

Nitrate is not removed from water by classical methods of treatment. Ion exchange systems have been developed for removing nitrate, but dilution with water of lower nitrate concentration from another source, where one is available, is commonly used. Reverse osmosis is also effective.

WRF (2016) evaluated the performance of four emerging treatment technologies: (1) a proprietary biological denitrification system (MB-N2), (2) standard ion exchange (IX) resin without brine reuse, (3) nitrate selective IX resin without brine reuse (NSR), and (4) nitrate selective IX resin with an electrochemical denitrification system (IX with ECD). For each alternative, capital, annual operation and maintenance (O&M), and 20-year present worth costs were calculated. When non-cost factors, such as overall safety, process reliability, and treatment history were evaluated, the conventional IX system was ranked higher than the MB-N2 and IX with ECD systems. However, the MB-N2 and IX with ECD systems were more favourable when considering waste disposal and environmental sustainability factors.

#### Nitrite

Treatment of the water with an oxidising agent such as chlorine will convert the nitrite to nitrate. The nitrate can then be treated as explained for nitrate. The USEPA Maximum Concentration Level (MCL) for nitrite indicates that the concentration at which it might be of concern is ten times less than the MCL for nitrate. The oxidation of high nitrite levels to nitrate therefore will not create an unacceptably high nitrate concentration in the water, unless the nitrate level is already high, or the nitrite level is extremely high.

#### Household treatment (WHO 2016)

Point-of-use reverse osmosis and point-of-entry anion exchange technologies are available for home or small-volume applications. Certified units are available, but they are costly, require good maintenance and are not readily available in many locations. Point-of-entry anion exchange uses strong base anion exchange resins that are regenerated with sodium chloride. Concurrent sulfate presence will significantly affect nitrate removal performance, as sulfate is preferentially retained and will displace nitrate back into the water if the system is operated beyond its nitrate capacity. Point-of-use reverse osmosis systems are potentially useful, but they operate at low line pressures, so they are much less efficient than high-pressure central desalination systems. Water reject levels can be of the order of up to 80 percent. Point-of-entry reverse osmosis is not used because of the corrosivity of the treated water to pipe and plumbing components (for more information, refer to the WHO website on household water treatment and safe storage at <http://www.who.int/water_sanitation_health/water-quality/household/en/>).

For individual households that obtain their drinking-water from private wells, residential drinking-water treatment devices are an option for reducing nitrate and nitrite concentrations in drinking-water. Before a treatment device is installed, the water should be tested to determine its general water chemistry and verify the presence and concentrations of nitrate and nitrite in the source water. As bacterial contamination of a well water supply frequently occurs in conjunction with nitrate contamination, the bacterial and chemical aspects of the water quality should be considered prior to selecting a water treatment device, and disinfection is essential.

### Analytical methods

#### Nitrate

##### Referee method

Cadmium Reduction Method (APHA 4500-NO3-E).

##### Some alternative methods

1. Ion Chromatography Method (APHA 4110B; USEPA 300.1).

2 Nitrate Electrode Method (APHA 4500-NO3 D).

#### Nitrite

##### Referee method

Colorimetric Method (APHA 4500-NO2 B).

##### Some alternative methods

1. Ion Chromatography Method (APHA 4110 B; USEPA 300.1).

### Health considerations

For nitrate, the main sources of exposure are vegetables, especially leafy vegetables. Other food sources include baked and processed cereal products and cured meat. For an average adult consumer who lives in an area with low drinking-water contamination, total exposure to nitrate from food and water is estimated to be about 60 to 90 mg per person per day, of which at least 90 percent is from food. For high consumers of vegetables, the intake of nitrate may reach 200 mg per person per day. Similar intakes could result from high consumption of water contaminated with more than 50 mg/L nitrate (as NO3-).

For nitrite, the main source of exogenous human exposure is also food. Important sources include cereal products, vegetables and cured meat. Over the last 30 years, the relative contribution of cured meat to dietary exposure to nitrite for an average consumer has decreased from about 40 percent to about 20 percent. For high consumers of cured meat, the relative contribution may have reached 90 percent. The total intake of exogenous nitrite is estimated to be about 0.75 to 2.2 mg per day for an adult with an average food consumption pattern.

Ingested nitrate is absorbed readily and completely from the upper small intestine. Nitrite may be absorbed directly from the stomach as well as from the small intestine. When nitrate levels in drinking-water exceed 50 mg/L as NO3-, drinking-water may become the major source of total nitrate intake, especially for bottle-fed infants.

The toxicity of nitrate in humans is thought to be due solely to its reduction to nitrite. Bacteria are responsible for most of the conversion of nitrate to nitrite in the gastrointestinal system. Consequently, the risk of methemoglobinemia from ingestion of nitrate depends not only on the dose of nitrate, but also on the number and type of enteric bacteria. In healthy adults, available data suggest about 5 percent of a dose of nitrate is reduced to nitrite by bacteria in the mouth. Conversion of nitrate to nitrite may also occur in the stomach if the pH of the gastric fluid is sufficiently high (above pH 5) to permit bacterial growth. This is of concern in adults with diseases such as achlorhydria or atrophic gastritis. It is commonly accepted that infants younger than 3 months may be highly susceptible to gastric bacterial nitrate reduction, as their stomach pH is generally higher than in adults.

The primary health concern regarding nitrate and nitrite is the formation of methaemoglobinaemia, so-called blue-baby syndrome. Nitrate is reduced to nitrite in the stomach of infants, and nitrite is able to oxidise haemoglobin (Hb) to methaemoglobin (metHb), which is then unable to transport oxygen around the body. The reduced oxygen transport becomes clinically manifest when metHb concentrations reach 10 percent or more of normal Hb concentrations; the condition, called methaemoglobinaemia, causes cyanosis and, at higher concentrations, asphyxia. The normal metHb level in infants under 3 months of age is less than 3 percent. Other susceptible groups include pregnant women and people with a deficiency of glucose-6-phosphate dehydrogenase or methaemoglobin reductase. Methaemoglobinaemia in infants also appears to be associated with simultaneous exposure to microbial contaminants, eg, Addison and Benjamin (2004).

Walton (1951) described a survey performed by the American Public Health Association to identify clinical cases of infantile methemoglobinemia that were associated with ingestion of nitrate-contaminated water. A total of 278 cases of methemoglobinemia were reported. Of 214 cases for which data were available on nitrate levels in water, none occurred in infants consuming water containing <10 mg nitrate-nitrogen/L (1.6 mg nitrate-nitrogen/kg/day). There were 5 cases (2 percent) in infants exposed to 11–20 mg nitrate-nitrogen/L (1.8–3.2 mg/kg/day), 36 cases (17 percent) in infants exposed to 21–50 mg/L (3.4–8.0 mg/kg/day), and 173 (81 percent) in infants exposed to >50 mg/L (>8 mg/kg/day). Based on these studies of nitrate contamination and occurrence of methemoglobin the USEPA set the maximum contaminant level or reference dose for oral intake of 10 mg/L nitrate as N (USEPA 1987, revised 1991).

In Hungary in 1975 to 1977, 190 cases of methaemoglobinaemia were reported, 94 percent in infants less than three months of age. The nitrate level in drinking water was more than 100 mg/L in 92 percent of cases and between 40 and 100 mg/L in the remaining 8 percent. In 1982, 96 cases of methaemoglobinaemia were reported. All cases were associated with privately dug wells, and 92 percent of the patients were three months of age or younger. Nitrate levels in drinking water were above 100 mg/L in 93 percent of cases and between 40 and 100 mg/L in the remaining 7 percent (WHO 1985).

For over 50 years, there has existed a widespread belief that nitrate in drinking water is a primary cause of infantile methemoglobinemia. Hunter Comly originally proposed this theory in 1945 in a report in the *Journal of the American Medical Association* after treating several infantile methemoglobinemia victims. Comly proposed that because nitrite is known to react directly with hemoglobin to form methemoglobin, nitrate from drinking water must be converted to nitrite within the gastrointestinal tract of infants. Because many infants did not appear susceptible to methemoglobinemia from nitrate-contaminated water, Comly suggested that the nitrate-to-nitrite conversion might only occur in the presence of a bacterial infection of the upper gastrointestinal tract, where such reactions could occur before nitrate is absorbed. The nitrate-derived nitrite could then react with haemoglobin to form methemoglobin and, in sufficient quantities, lead to the cyanosis of methemoglobinemia. This theory was reinforced by the fact that cyanosis typically subsided once an infant was switched to an uncontaminated water supply. Comly’s hypothesis became widely accepted as further research revealed a consistent pattern of elevated well water nitrate levels in infantile methemoglobinemia cases. Limiting infant exposure to nitrate was thus decided to be the most prudent approach to protecting infant health, and a committee from the American Public Health Association conducted a nationwide survey to determine a safe level of nitrate in water. A total of 278 cases with 39 deaths were compiled. The results showed that methemoglobinemia incidence correlated with increasing nitrate levels. Because no infantile methemoglobinemia cases were observed with concentrations <10 ppm nitrate-nitrogen, the United States and the World Health Organization established a maximum contaminant level (MCL) of 10 ppm nitrate-N for nitrate in drinking water. Over the last 20 years, however, a more complex picture of infantile methemoglobinemia causation has emerged which indicates that current limits on drinking water nitrate may be unnecessarily strict. It is now well established that diarrhoeal illness and some gastrointestinal disturbances, typically accompanied by diarrhoea and/or vomiting, can lead to methemoglobinemia in young infants without exposure to high-nitrate drinking water or exposure to abnormal levels of nitrate through food. There many reported infantile methemoglobinemia cases associated with diarrhoea without exposure to nitrate-contaminated water. Because diarrhoea was a prominent symptom in the majority of drinking water linked methemoglobinemia cases, the evidence suggests that diarrhoea and/or gastrointestinal infection/ inflammation, are the principle causative factors in infantile methemoglobinemia. A survey in Germany found that 53 percent of 306 infantile methemoglobinemia cases reported diarrhoea. Contrary to some reports, diarrhoea and vomiting are not symptoms that typically accompany cyanosis, methemoglobinemia due to oxidant drug exposure, or genetic abnormalities in haemoglobin (Avery 1999, and discussed in WHO 2011).

Methaemoglobinaemia in infants appears to be associated with simultaneous diarrhoeal disease. Authorities should therefore be all the more vigilant that water to be used for bottle-fed infants is microbiologically safe when nitrate is present at concentrations near the guideline value or in the presence of endemic infantile diarrhoea. Water should not be used for bottle-fed infants if the concentration of nitrate is above 100 mg/L as NO3 but can be used if the concentration is between 50 and 100 mg/L if the water is microbiologically safe and there is increased vigilance by medical authorities (WHO 2011).

Current evidence suggests that exposure to nitrate in drinking-water may alter human thyroid gland function by competitively inhibiting thyroidal iodide uptake, leading to altered thyroid hormone concentrations and functions. Although studies found that exposure to nitrate concentrations above 50 mg/L are weakly associated with altered thyroid function, the evidence is limited, conflicting and based on studies with important methodological limitations (from WHO 2016).

The reference dose or RfD (USEPA 1991/2009/2011) for nitrate as N is 1.6 mg/kg/d, and for nitrite as N it is 0.16 mg/kg/d (USEPA 1997/2009/2011).

As at October 2015 and July 2017 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for nitrate of:

4 mg/kg/d for acute-duration oral exposure (1–14 days)

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

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

As at July 2017 ATSDR (<http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf>) quotes a minimal risk level (MRL) for nitrite of:

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

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).

Nitrate is not mutagenic in bacteria and mammalian cells *in vitro*. Chromosomal aberrations were observed in the bone marrow of rats after oral nitrate uptake, but this could have been due to exogenous N-nitroso compound formation. Nitrite is mutagenic, causing morphological transformations in in vitro systems.

IARC (2005) stated that “Ingested nitrate or nitrite under conditions that result in endogenous nitrosation is *probably carcinogenic to humans (Group 2A)*. The underlying mechanism is endogenous nitrosation, which in the case of nitrate must be preceded by reduction to nitrite. Nitrate and nitrite are interconvertible *in vivo*. Nitrosating agents that arise from nitrite under acidic gastric conditions react readily with nitrosatable compounds, especially secondary amines and alkyl amides, to generate *N‑*nitroso compounds. Many *N*-nitroso compounds are carcinogenic.” However, the weight of evidence indicates that there is unlikely to be a causal association between gastric cancer and nitrate in drinking-water.

The livestock guideline value for nitrite (as NO2-) is 30 mg/L. Nitrate (as NO3-) concentrations less than 400 mg/L in livestock drinking water should not be harmful to animal health; stock may tolerate higher nitrate concentrations in drinking water provided nitrate concentrations in feed are not high. Water containing more than 1,500 mg/L nitrate (as NO3-) is likely to be toxic to animals and should be avoided (ANZECC/ARMCANZ 2000). These guidelines were to have been updated in 2012.

### Derivation of Maximum Acceptable Values

#### Nitrate (short-term)

The MAV of 50 mg/L (as NO3-) is to protect against methaemoglobinaemia in bottle-fed infants (short-term exposure). In epidemiological studies, methaemoglobinaemia was not reported in infants in areas where drinking-water consistently contained less than 50 mg of nitrate per litre.

The epidemiological evidence for an association between dietary nitrate and cancer is insufficient, and the MAV for nitrate in drinking-water is established solely to prevent methaemoglobinaemia, which depends upon the conversion of nitrate to nitrite. Although bottle-fed babies are the most susceptible, occasional cases have been reported in some adult populations.

#### Nitrite (short-term)

The short-term MAV of 3 mg/L nitrite (as NO2-) is to protect against methaemoglobinaemia in bottle-fed infants. WHO (2017) guideline value for nitrite of 3 mg/L as nitrite (or 0.9 mg/L if reported as nitrite-nitrogen) is based on:

1. no incidence of methaemoglobinaemia at nitrate concentrations below 50 mg/L (as nitrate ion) in drinking-water for bottle-fed infants less than six months of age (assuming body weight of 2 kg)

2. converting 50 mg/L as nitrate to corresponding molar concentration for nitrite

3. multiplying by a factor of 0.1 to account for the estimated conversion rate of nitrate to nitrite in infants where nitrite is formed endogenously from nitrate at a rate of 5–10 percent

4. multiplying by a source allocation factor for drinking-water of 100 percent or 1, as a bottle-fed infant’s primary exposure to nitrite is through consumption of formula reconstituted with drinking-water that contains nitrate or nitrite.

As the guideline value is based on the most sensitive subgroup of the population bottle-fed infants less than six months of age), application of an uncertainty factor is not deemed necessary.

Earlier, WHO had stated that animal studies were inappropriate to establish a firm No-Observable-Adverse-Effect Level (NOAEL) for methaemoglobinaemia in rats. Therefore, a pragmatic approach was followed, accepting a relative potency for nitrite and nitrate with respect to methaemoglobin formation of 10:1 (on a molar basis), and a provisional MAV of 3 mg/L had been adopted for nitrite.

#### Nitrite (long-term)

The long-term MAV had been based on the next paragraph. WHO (2011/2017) now states: “However, owing to the uncertainty surrounding the susceptibility of humans compared with experimental animals, this value which was considered provisional has now been suspended and is being subjected to review in light of evidence on the differences in nitrite metabolism between laboratory rodents and humans”.

The 0.2 mg/L (as NO2-) MAV in the 2008 DWSNZ (based on WHO 2004) for long-term exposure for chronic effects of nitrite was considered provisional owing to uncertainty surrounding the relevance of the observed adverse health effects for humans and the susceptibility of humans compared with animals. The occurrence of nitrite in the distribution system as a consequence of chloramine use will be intermittent, and average exposures over time should not exceed the provisional MAV. The nitrite MAV (long-term exposure) is based on allocation to drinking-water of 10 percent of JECFA ADI of 0.06 mg/kg of body weight per day, based on nitrite-induced morphological changes in the adrenals, heart and lungs in laboratory animal studies.

#### Nitrate:Nitrite ratio

Because of the possibility of simultaneous occurrence of nitrite and nitrate in drinking-water, the sum of the ratio of the concentration of each to their short-term MAVs, as shown in the following formula, should not exceed 1:

where C = concentration, and MAV = Maximum Acceptable Value.

##### Note related to short-term MAVs

The short-term MAVs for nitrite and nitrate have been established to protect the health of infants, particularly those that are bottle-fed. Community water suppliers providing drinking-water that exceeds the short-term MAVs will need to find a procedure for advising parents of new-born babies. The WHO (2007) states that in areas where household wells are common, health authorities may wish to take a number of steps to ensure that nitrate contamination is not or does not become a problem. Such steps could include targeting mothers, particularly expectant mothers, with appropriate information about water safety, assisting with visual inspection of wells to determine whether a problem may exist, providing testing facilities where a problem is suspected, providing guidance on disinfecting water or where nitrate levels are particularly high, providing bottled water from safe sources or providing advice as to where such water can be obtained.

The MAV for nitrate in the 1995 and 2000 DWSNZ was 50 mg/L as NO3-, and the MAV for nitrite was 3 mg/L as NO2-. The 1995 datasheet stated:

The epidemiological evidence for an association between dietary nitrate and cancer is insufficient, and the MAV for nitrate in drinking-water is established solely to prevent methaemoglobinaemia, which depends on the conversion of nitrate to nitrite. Although bottle-fed babies are the most susceptible, occasional cases have been reported in some adult populations.

As a result of recent evidence of the presence of nitrite in some water supplies, it was concluded that a MAV for nitrite should be proposed. However, the animal studies were inappropriate to establish a firm NOAEL for methaemoglobinaemia in rats. Therefore a pragmatic approach was followed, accepting a relative potency for nitrate and nitrite with respect to methaemoglobin formation of 10:1 (on a molar basis), and a PMAV for nitrite of 3 mg/L is proposed. (The paragraph and formula related to the ratios of each appeared in the 1995 Guidelines as well.)

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

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# Ozone

### Maximum Acceptable Value

The DWSNZ do not include a MAV for ozone, and WHO does not have a Guideline Value.

In November 1982, the US FDA granted ozone disinfection for bottled water a ‘Generally Recognised as Safe’ classification for a residual dissolved ozone concentration of 0.4 mg/L.

The Natural Mineral Water, Spring Water and Bottled Drinking Water (Amendment) (England) Regulations 2004 (<http://www.opsi.gov.uk/si/si2004/20040656.htm>) state that treatment of natural mineral waters and spring waters with ozone-enriched air shall not leave an ozone residue in the water above 0.05 mg/L.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that because ozone used for disinfection leaves no residual, no health-based guideline value is needed.

### Sources to drinking-water

#### 1 To source waters

None.

#### 2 From treatment processes

Ozone is a powerful oxidant and has many uses in water treatment, including oxidation of organic chemicals and as a primary disinfectant. Ozone gas (O3) is formed by passing dry air or oxygen through a high-voltage electric field. The resultant ozone-enriched air is dosed directly into the water by means of porous diffusers at the base of baffled contactor tanks. The contactor tanks, typically about 5 m deep, provide 10–20 minutes of contact time. Dissolution of at least 80 percent of the applied ozone should be possible, with the remainder contained in the off-gas, which is passed through an ozone destructor and vented to the atmosphere. The performance of ozonation relies on achieving the desired ozone concentration after a given contact period. For oxidation of organic chemicals, such as a few oxidisable pesticides, a residual of about 0.5 mg/L after a contact time of up to 20 minutes is typically used. The doses required to achieve this vary with the type of water but are typically in the range 2–5 mg/L. Higher doses are needed for untreated waters because of the ozone demand of the natural background organics. Ozone reacts with natural organics to increase their biodegradability, measured as assimilable organic carbon. To avoid undesirable bacterial growth in distribution, ozonation is normally used with subsequent treatment, such as filtration or GAC, to remove biodegradable organics, followed by a chlorine residual, since it does not provide a disinfectant residual (WHO 2004).

UV radiation and hydrogen peroxide can act as a strong catalyst in oxidation reactions when used in conjunction with ozone. Processes aimed at generating hydroxyl radicals are known collectively as advanced oxidation processes and can be effective for the destruction of chemicals that are difficult to treat using other methods, such as ozone alone. Chemicals can react either directly with molecular ozone or with the hydroxyl radical, which is a product of the decomposition of ozone in water and is an exceedingly powerful indiscriminate oxidant that reacts readily with a wide range of organic chemicals. The formation of hydroxyl radicals can be encouraged by using ozone at high pH. One advanced oxidation process using ozone plus hydrogen peroxide involves dosing hydrogen peroxide simultaneously with ozone at a rate of approximately 0.4 mg/L of hydrogen peroxide per mg of ozone dosed per litre (the theoretical optimum ratio for hydroxyl radical production) and bicarbonate (WHO 2004).

Although ozonation is a fairly complex system, it has a long history of use for disinfection and for the control of taste, odour and colour in Europe. It has not been used in Australia to date for the disinfection of sizeable potable water supplies; it is used at Timaru.

The following chemicals may be present in ozone (NHMRC, NRMMC 2011):

|  |  |
| --- | --- |
| * acetylene | * carbon monoxide |
| * argon | * hydrocarbons |
| * carbon dioxide | * nitrous oxide |

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

Ozone converts to oxygen in air and in water. It is more soluble in water than oxygen.

### Typical concentrations in drinking-water

Ozone has an approximate half-life of 20 minutes in water, so very few consumers will receive ozone in their drinking-water supply.

### Analytical methods

Ozone is usually measured online. If the analyser is checked using a field test method, the field test method must be standardised against the indigo method, Standard Methods 4500-ozone (APHA 2005), at least once every six months by a Ministry of Health recognised laboratory. The preferred method for standardising the online ozone analyser is described in the Guidelines, section 15.5.4.

### Health considerations

Ozone in the air has toxic effects when breathed in, but due to its high reactivity, ozone levels in drinking-water are not likely to have health effects.

Ozone can cause drinking-water health effects indirectly. Ozone oxidises bromide to produce hypohalous acids, which react with precursors to form brominated THMs. A range of other DBPs, including aldehydes and carboxylic acids, may also be formed. Of particular concern is bromate, formed by oxidation of bromide. Bromate may also be present in some sources of hypochlorite, but usually at concentrations that will give rise to levels in final water that are below the guideline value (WHO 2004). See individual datasheets for further information.

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# Perchlorate

### Maximum Acceptable Value

The 2008 DWSNZ do not include a MAV for perchlorate. The WHO Guidelines (2017) adopted a guideline value of 0.07 mg/L for perchlorate.

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

The USEPA (2011) has a lifetime health advisory of 0.015 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.

Perchlorate is a regulated drinking-water contaminant in California, with a [maximum contaminant level](http://ww2.cdph.ca.gov/services/DPOPP/regs/Pages/R-16-04-PerchlorateinDrinkingWater.aspx) (MCL) of 0.006 mg/L (CDPH 2008). Where a contamination of drinking water supplies in Canada has occurred, Health Canada recommends a drinking water guidance value of 0.006 mg/L, based on a review of existing health risk assessments from other agencies.

### Sources to drinking-water

#### 1 To source waters

Perchlorate is both a naturally occurring and man-made chemical. Perchlorate is known to form in the atmosphere and is transferred through rainfall. Rainwater in China was recently reported to contain perchlorate at concentrations between 0.35 and 27.3 μg/L, with a mean concentration of 6.37 μg/L. Concentrations can be influenced by fireworks displays (WHO 2016).

Perchlorate is a chemical used to facilitate combustion of rocket and missile fuel, explosives, fireworks, and road flares. It may also be used in air-bag inflation systems, lubricating oils, and the manufacture of paints and many industrial goods. In the past, physicians used perchlorate to treat Graves’ Disease, a disorder that causes an overproduction of thyroid hormones. Perchlorate has been detected in certain fertilisers, particularly those derived from the Chilean ores. Wastes from the manufacture and improper disposal of perchlorate-containing chemicals are increasingly being discovered in soil and water in the US. Perchlorate can form naturally in the atmosphere; traces have been detected in rainwater.

#### 2 From treatment processes

The Massachusetts Department of Environmental Protection reported that significant levels of perchlorate could be detected in sodium hypochlorite samples that are aged for a few weeks. A new delivery of sodium hypochlorite (NaOCl) at one utility tested at 0.2 ppb perchlorate, but when the sample was aged 26 days, the perchlorate level in the product rose to 6750 ppb (6.75 mg/L).

In an NSF study of the perchlorate content in 82 different sodium hypochlorite samples (Purkiss, unpublished):

* seven samples were non-detectable, and others ranged up to 300 mg/L in the product, which could result in concentrations of perchlorate up to 0.03 mg/L in the final drinking-water if the NaOCl was dosed at its certified maximum use level
* all NaOCl samples less than 30 days old contributed less than 1.0 mg/L to drinking water when used at the maximum use level
* 83 percent of samples less than 90 days old contributed less than 1.0 mg/L to drinking water
* detectable levels of perchlorate were found in some sodium hydroxide (NaOH) samples, from 0.11 mg/L to 0.90 mg/L in 5 of 22 samples tested, which is far below the levels in sodium hypochlorite
* 35 other chemical types tested did not detect perchlorate in any other water treatment chemical.

The levels of perchlorate are most likely not a concern in water supplies where sodium hypochlorite is used within a few weeks of delivery. However, the levels in sodium hypochlorite may be a concern in water supplies that store sodium hypochlorite bleach for longer than three months or have aged chemical in storage tanks that could contaminate new shipments.

DWI (2013) recommends dilution of hypochlorite solutions upon delivery. A four-fold dilution will decrease the rate of formation of perchlorate by a factor of 36, a 10-fold dilution by a factor of 270. A 5°C reduction in storage temperature will reduce the rate of perchlorate formation by a factor of approximately 2. Store in the dark. Control the pH of stored hypochlorite solutions at pH 11–13 (even after dilution). Also, given the typical pH range of on-site-generated hypochlorite (pH 9-10), such solutions should be used as soon as possible after manufacture and should not be stored for more than  
1–2 days. Control concentrations of transition metal ions by purchasing filtered hypochlorite solutions and by using low-metal ion concentration feed water for onsite-generated systems. The presence of transition metal ions increases the degradation rate of hypochlorite and reduces free available chlorine, thus increasing the volume of hypochlorite required and increasing the amounts of contaminants dosed. Use low-bromide salt to produce on-site-generated hypochlorite.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

The perchlorate anion is highly soluble in water and very mobile in soil, so can find its way to groundwater. Neither photolysis nor vaporisation is known or expected to significantly affect perchlorate concentrations in water. In certain arid climates, sporadic and insufficient rainwater results in an accumulation of perchlorate in soils. The half lifes in soil and water are long, but ultimately certain anaerobic micro-organisms may degrade perchlorate.

### Typical concentrations in drinking-water

Dosing old sodium hypochlorite could result in a concentration of 0.03 mg/L perchlorate. In the US perchlorate has been identified at least once in approximately 4 percent of community water systems from 26 different states and two territories, with detectable levels averaging 0.01 mg/L and ranging from the method detection limit of 0.004 mg/L to a maximum at 0.42 mg/L.

279 water utilities in the US reported detecting perchlorate in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.068 mg/L.

The USEPA (2011a) reports the average concentration of perchlorate in samples with positive detections for perchlorate was 0.01 mg/L, median 0.0064 mg/L. Between 2000 and 2005, the USEPA conducted a survey of 3870 public water systems and found that 160 of the systems (4.1 percent) contained perchlorate at concentrations above the method reporting limit of 4 μg/L, and 13 of the systems surveyed had concentrations above 40 μg/L; from WHO (2016).

### Removal methods

Due to the high solubility and stability of the perchlorate ion, the conventional drinking water treatment processes involved do not show any signs of being able to remove perchlorate, though there is evidence that ion exchange (as used for nitrate removal) can be effective (DWI 2009). Biological treatment and anion exchange processes are the commonest removal systems used in the US. Reverse osmosis is reasonably effective.

Prevention is better than cure – see above (2. From treatment processes).

### Analytical methods

The following method has been promulgated (by USEPA) as the approved method for assessment monitoring of perchlorate under the Unregulated Contaminant Monitoring Regulation:

EPA Method 314.0: Determination of Perchlorate in Drinking Water by Ion Chromatography.

See also DWI (2009).

### Health considerations

Perchlorate has been shown to accumulate in the leaves of some food crops, tobacco plants, and more generally in broad leaf plants. Therefore food is likely to be the main source of perchlorate. WHO (2011) includes a 78-page paper on the health effects of perchlorate in food.

JECFA estimated that international dietary exposure to perchlorate (using the 13 consumption cluster diets of the Food Contamination Monitoring and Assessment Programme of the Global Environment Monitoring System) was 0.03–0.22 μg/kg bw per day. In a study from Japan, the actual relative source contribution of perchlorate from water ranged from 0.5 percent to 22 percent of the total daily perchlorate intake; from WHO (2016).

Relatively high mean middle bound occurrence values were found in dried products, like ‘Tea and herbs for infusion’ (324 μg/kg) and ‘Herbs, spices and condiments’ (63 μg/kg), and in some fresh vegetables, like ‘Radishes’ (117 μg/kg), ‘Rocket salad, rucola’ (75 μg/kg) and ‘Spinach (fresh)’ (132 μg/kg). The mean short-term exposure of infants, toddlers and other children was in the range of 0.40–2.3 μg/kg bw per day, while in the older population groups, the range was 0.26–1.3 μg/kg bw per day (EFSA 2017).

One of the more serious human health effects observed in scientific studies is perchlorate’s disruption of thyroid hormone production by blocking the transport of iodide to the thyroid gland. Poor iodide uptake and subsequent impairment of the thyroid function in pregnant and lactating women have been linked to delayed development and decreased learning capability in their infants and children. A few internal organs (for example, the thyroid, breast tissue, and salivary glands) can take up relatively large amounts of perchlorate from the bloodstream. Perchlorate generally leaves these organs in a few hours.

Given the mode of action of perchlorate (see section 3.4), the key vulnerable groups are likely to be pregnant women, fetuses, newborns, young infants, those with hypothyroidism and possibly those with iodine-deficient diets.

The USEPA (2009) established an official reference dose (RfD) of 0.007 milligrams per kilogram of body weight per day (mg/kg/day) of perchlorate. This level is consistent with the recommended reference dose included in the National Academy of Science’s January 2005 report. A reference dose is a scientific estimate of a daily exposure level that is not expected to cause adverse health effects in humans. Work on appropriate levels in drinking-water is proceeding.

The RfD of 0.007 mg/kg/day leads to a Drinking Water Equivalent Level (DWEL) of 0.025 mg/L. The USEPA calculates the DWEL using the RfD, multiplied by an adult body weight of 70 kg, and divided by a conservative tap water consumption value of 2 L/day. These values are now listed in USEPA. 2011. The RfD includes a ten-fold intraspecies uncertainty factor to protect the most sensitive population, the foetuses of pregnant women who might have hypothyroidism or iodide deficiency; it is also protective of other sensitive populations such as neonates and developing children.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.0007 mg/kg/day for chronic-duration oral exposure (>364 days) to perchlorate.

### Derivation of Maximum Acceptable Value

Using the unrounded provisional maximum tolerable daily intake (PMTDI) of 0.011 mg/kg bw, a typical adult body weight of 60 kg, the assumption that drinking-water contributes 20 percent of the total exposure to perchlorate and a typical consumption of two litres of water per day, a health-based value of 0.07 mg/L (rounded figure) can be calculated. The allocation factor (floor value) of 20 percent was determined to be appropriate considering the large number of occurrence studies indicating that perchlorate exposure from food exceeded the exposure from water.

WHO (2016) states that a guideline value of 0.07 mg/L for perchlorate is therefore established.

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# Potassium

### Maximum Acceptable Value

Potassium occurs in drinking-water at concentrations well below those of health concern.

### Sources to drinking-water

#### 1 To source waters

Potassium is an essential element in humans and is seldom, if ever, found in drinking-water at levels that could be a concern for healthy humans. It occurs widely in the environment, including all natural waters, existing in the ionic form, K+.

Geothermal waters in the Taupo Volcanic Zone can contain 100 to 200 mg/L K (GNS 2015).

#### 2 From treatment processes

No known sources. However, there are accounts from overseas where some water softeners are being recharged with potassium salts instead of sodium chloride, in an effort to increase potassium intake.

#### 3 From the distribution system

Some potassium leaches from new concrete pipes, pipe linings and tanks.

### Forms and fate in the environment

Potassium ions are inert and entirely soluble.

### Typical concentrations in drinking-water

New Zealand waters tend to contain less potassium than most of those overseas. Many contain less than 1 mg/L. Some (more likely to be groundwaters) may contain up to 10 mg/L; water produced by desalination may contain more.

The concentration of potassium in seawater is about 390 mg/L. Rainwater concentrations may be as low as 0.1 mg/L.

In the UK a survey carried out for the Regional Heart Study (WRc 1987) found a mean concentration of 2.5 mg/L potassium with an upper 90 percentile of 5.2 mg/L. Data from Canada indicate that average concentrations in treated and raw drinking water in different areas vary between <1 to 8 mg/L. However, concentrations ranged up to 51 mg/L in Saskatchewan, which is the largest commercial production area for potassium chloride in Canada.

Just 0.0118 percent of all potassium is the radioactive isotope potassium-40. Five water utilities in the US reported detecting potassium-40 (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 152 pCi/L.

### Removal methods

Not usually removed, but deionisation would be effective. Potassium will pass through nanofilters.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Potassium is an essential element in humans and is seldom, if ever, found in drinking-water at levels that could be a concern for healthy humans. The recommended daily requirement for humans is greater than 3000 mg.

WHO (2012) recommends an increase in potassium intake from food for reduction of blood pressure and risk of cardiovascular disease, stroke and coronary heart disease in adults. WHO suggests a potassium intake of at least 3510 mg/day for adults. A New Zealander would be lucky to ingest more than 3 mg per day from their drinking water.

The primary source of potassium for the general population is the diet, as potassium is found in all foods, particularly vegetables and fruits, and marine fish. Some food additives are also potassium salts (eg, potassium iodide). Some individuals require potassium supplements, which are given under medical supervision; others take potassium supplements without supervision, although this is not generally recommended.

Potassium water softeners are being used as an alternative to sodium water softeners in response to a perception that potassium is better for health. This is not a concern for the general population. However, some people with specific diseases or on certain medications are susceptible to hyperkalaemia. It is recommended that susceptible individuals with kidney dysfunction or taking medications that interfere with normal potassium-dependent functions in the body avoid the consumption of water treated by water softeners using potassium chloride; Health Canada. 2008. The intake of potassium from the consumption of drinking water treated with a water softener using potassium chloride will vary depending on the level of hardness. Assuming that a potassium chloride water softener emits 14 mg/L of K in water with a hardness of 17 mg/L CaCO3 , the amount of potassium emitted in 1 L of drinking water can be calculated for different hardness levels. The table shows that a water softener using potassium chloride can add significantly to the intake of potassium when compared to the amount that Canadian adults typically consume in drinking water, even when the water treated had water hardness levels considered to be acceptable.

Intake of potassium as a result of water softener use, by hardness level

|  |  |  |
| --- | --- | --- |
| **Drinking water hardness (mg/L CaCO3)** | **mg/L K** | **Intake \* (mg/kg bw per day)** |
| treated tap water | 8.0 | 0.2 |
| 100 (acceptable) | 82 | 1.8 |
| 200 (poor) | 164 | 3.5 |
| 500 (unacceptable) | 411 | 8.8 |

Source: Health Canada 2008

\* Based on consumption of 1.5 L of water per day by a 70-kg adult.

Potassium is an essential mineral [micronutrient](http://en.wikipedia.org/wiki/Micronutrient) in human nutrition; it is the major cation (positive ion) inside animal cells, and it is thus important in maintaining fluid and [electrolyte](http://en.wikipedia.org/wiki/Electrolyte) balance in the body. Potassium helps the kidneys function normally. Potassium is crucial to heart function and plays a key role in skeletal and smooth muscle contraction, making it important for normal digestive and muscular function. A potassium intake sufficient to support life can generally be guaranteed by eating a variety of foods, especially plant foods. Clear cases of potassium deficiency are rare in healthy individuals eating a balanced diet. Foods with high sources of potassium include [orange juice](http://en.wikipedia.org/wiki/Orange_juice), [potatoes](http://en.wikipedia.org/wiki/Potato), [bananas](http://en.wikipedia.org/wiki/Banana), [avocados](http://en.wikipedia.org/wiki/Avocado), [tomatoes](http://en.wikipedia.org/wiki/Tomato), [broccoli](http://en.wikipedia.org/wiki/Broccoli), [soybeans](http://en.wikipedia.org/wiki/Soybeans), [brown rice](http://en.wikipedia.org/wiki/Brown_rice), [garlic](http://en.wikipedia.org/wiki/Garlic) and [apricots](http://en.wikipedia.org/wiki/Apricot), although it is also common in most [fruits](http://en.wikipedia.org/wiki/Fruit), [vegetables](http://en.wikipedia.org/wiki/Vegetable) and [meats](http://en.wikipedia.org/wiki/Meat). Drinking-water is not normally an important source of potassium in the human diet.

### Derivation of Maximum Acceptable Value

It is not considered necessary to establish a health-based guideline value for potassium in drinking-water.

See Chapter 11 of the Guidelines for a discussion on the relevance of radioactive potassium, potassium-40. It includes the following:

Only a very small percentage (0.0118 percent) of all potassium is the radioactive isotope potassium-40. The gross beta measurement includes a contribution from potassium-40, a beta emitter that occurs naturally in a fixed ratio to stable potassium. Potassium is an essential metabolic element for humans and is absorbed mainly from ingested food. Potassium-40 does not accumulate in the body but is maintained at a constant level independent of intake. The contribution of potassium-40 to beta activity should therefore be subtracted following a separate determination of total potassium.The specific activity of potassium-40 is 30.7 Bq/g of potassium. However, not all the radiation from potassium-40 appears as beta activity. The beta activity of potassium-40 is 27.6 Bq/g of stable potassium, which is the factor that should be used to calculate the beta activity due to potassium-40 (WHO 2004).

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# Potassium permanganate

### Maximum Acceptable Value

There are insufficient data to set a MAV for potassium permanganate.

### Sources to drinking-water

#### 1 To source waters

Potassium permanganate (KMnO4, where the manganese is in the +7 oxidation state, MnVII) may enter source waters in discharges from industries in which it is used. It is used as an oxidising agent, in pharmaceutical manufacture (deodorant, disinfectant, detergent, gargling), as a bleaching agent, analytical reagent, dye and catalyst.

USEPA (1985) states: application rates for cooling towers, air washers, and evaporative condensers, use 3.6 ounces of a.i./500 to 1,000 gal. of water, or 27 to 62 ppm a.i. For aquaria and ornamental ponds, use 4 ppm a.i. to control algae growth. For human drinking water treatment, use 1.1 mg of product per gal. of water (ie, 4.4 to 4.5 ppm). For poultry drinking water, use 0.648 to 1.296 grains of product per quart of water (ie, 272 to 543 ppm a.i.).

#### 2 From treatment processes

Potassium permanganate, as a disinfectant, should only be employed in emergencies, and then only on a small scale. However, it is more widely used as a preoxidant to help in the elimination of tastes and odours, or the oxidation of iron and manganese for their subsequent removal by physical processes.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

Although it is a strong oxidising agent at the pH values typical of environmental waters, the removal of potassium permanganate by oxidation-reduction reactions is likely to be relatively slow in comparison with the reactions of chlorine. Water solubility is about 60 grams per litre (6 percent).

As the result of its oxidation reactions, the permanganate ion is usually converted into highly insoluble, brown or brown-black manganese oxides.

### Typical concentrations in drinking-water

No information about the concentrations of potassium permanganate in drinking-water is available. The permanganate ion is highly unlikely to persist into the distribution system, unless as a result of a serious dosage error.

### Removal methods

Reaction with reducing reagents, such as sodium thiosulphate, sulphur dioxide, and sodium bisulphite, can be used to remove permanganate from water. However, because of the likely formation of insoluble products from these reactions, some means of physical removal of precipitated material will probably have to be used downstream of the reducing reagent addition.

### Analytical methods

#### Referee method

A referee method cannot be selected for potassium permanganate 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 potassium permanganate for the above reason. However, the following information may be useful:

Potassium permanganate cannot be preserved in a sample. Analysis should be carried out as soon after sampling as possible, and if a delay between sampling and analysis is unavoidable the sample should be kept chilled.

No method for determining potassium permanganate appears in any of the standard texts for the analysis of water. However, by modification of APHA 3500-Mn D – Persulphate Method, satisfactory measurements probably could be made. This method is intended to allow the manganous ion concentration in the water to be measured by oxidising these ions to permanganate. If the oxidation step is omitted and the remainder of the method, using spectrophotometric absorption measurement at 525 nm, is used, permanganate concentrations as low as 0.05 mg/L as Mn, should be detectable.

### Health considerations

No information is available on the chronic health effects of ingestion of potassium permanganate.

Most acute effects of KMnO4 solutions seem to be reported when its strength is measured in percent. Symptoms of acute poisoning following ingestion of potassium permanganate include nausea, vomiting of a brownish coloured material, corrosion, oedema, liver and kidney damage and cardiovascular depression. The fatal dose is probably about 10 g and death may occur up to one month from the time of poisoning.

### Derivation of Maximum Acceptable Value

There are insufficient data to set a MAV for potassium permanganate.

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# Radium

### Maximum Acceptable Value

There is no MAV in the DWSNZ for radium, and it is not mentioned in the WHO Guidelines. The radium level in drinking-water is controlled via the radiochemical MAVs.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that radium‑226 and radium-228 should be determined if the gross alpha radioactivity in drinking-water exceeds 0.5 Bq/L, or the gross beta activity (with the contribution of potassium-40 subtracted) exceeds 0.5 Bq/L.

### Sources to drinking-water

#### 1 To source waters

Radium is a naturally-occurring radioactive metal that can exist as several isotopes. It is formed when uranium and thorium (two other natural radioactive substances) decay in the environment. Radium has been found at very low levels in soil, water, rocks, coal, plants, and food. For example, a typical amount might be one picogram of radium per gram of soil or rock. This would be about one part of radium in one trillion parts of soil or rock. These levels are not expected to change with time.

When radium decays it divides into two parts; one part is called radiation, and the other is called a daughter. The daughter, like radium, also divides into radiation and another daughter. The dividing continues until a stable, non-radioactive daughter is formed. During the decay process, alpha, beta, and gamma radiations are released. Alpha particles can travel only a short distance and cannot travel through the skin. Beta particles can penetrate through skin, but they cannot go all the way through the body. Gamma radiation, however, can go all the way through the body. Thus, there are several types of decay products that result from radium decay. The half-life of radium‑226 (the most stable isotope) is 1,620 years. The half-life of radium-228 is 5.8 years.

Radium-226, an alpha emitter, has been used, separated from its parent uranium, in cancer therapy.

Radium is generally very dispersed in the environment and is expected to occur at extremely low levels in surface waters. It is more likely to be detected in groundwater.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

The bromide, chloride and nitrate salts of radium are described as soluble. The solubility of radium salts in water generally increases with increased pH levels. The radium content of surface water is usually very low; radium-226 generally ranges from 0.1 to 0.5 pCi/L (0.004 to 0.019 Bq/L).

### Typical concentrations in drinking-water

Data on the presence of radon in groundwater can be used as a guide to the presumably corresponding presence of radium in the same source. Based on 990 random samples of drinking water from groundwater sources, the average population-weighted concentrations of radium-226 and radium-228 in the United States (excluding Hawaii) were about 0.91 pCi/L (0.034 Bq/L) and 1.41 pCi/L (0.052 Bq/L), respectively.

7,935 water utilities in the US reported detecting combined radium-226 in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 14.9 pCi/L.

8,701 water utilities in the US reported detecting combined radium-228 in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 14.9 pCi/L.

### Removal methods

Radium in water may be readily adsorbed by sediments, soils, and aquifer components, so water treatment by adsorption and water-softening techniques are thought to be effective in reducing radium in untreated drinking water. Limited field data also support the generalisation that radium is not mobile in groundwater.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Because radium is usually naturally present at very low levels in the surrounding environment, humans are always exposed to it and to the small amounts of radiation that it releases to its surroundings. Major man-made sources include where it is released into the air from the burning of coal or other fuels, leaching from coal ash lagoons and mine tailings, or if drinking-water is taken from a source that is high in natural radium, such as a deep well.

If radium is swallowed in water or with food, most of it (about 80 percent) will promptly leave the body in the faeces. The other 20 percent will enter the blood stream and be carried to all parts of the body, especially the bones where it is retained for a long time. Some of this radium will then be excreted gradually in the faeces and urine on a daily basis.

There is no clear evidence that long-term exposure to radium at the levels that are normally present in the environment (for example, 1 pCi of radium per gram of soil) is likely to result in harmful health effects. However, exposure to higher levels of radium over a long period of time may result in harmful effects including anemia, cataracts, fractured teeth, cancer (especially bone cancer).

In rats, the absorption of orally administered radium may be quite low. At 400 to 500 days after administration, they retained 1 to 7 percent of the ingested radium, primarily in the skeleton. The affinity for bone is assumed to be related to its chemical similarity to calcium, barium and strontium.

The USEPA has classified radium as Class A: a human carcinogen, and has a maximum contaminant level (MCL) of 5 pCi/L or 0.2 Bq/L.

### Derivation of Maximum Acceptable Value

No MAV.

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# Radon

CAS No. 14859-67-7 (222Rn); or 22481-48-7 (220Rn); or 14835-02-0 (219Rn).

### **Maximum Acceptable Value**

Note this is a radiological, not chemical, MAV.

Based on health considerations, the concentration of radon in drinking-water should not exceed 100 Bq/L, which is also the WHO Guideline Value. Radon 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.

The USEPA (2009) maximum contaminant level or MCL (proposed) is 300 pCi/L (= 11 Bq/L); note that 1 pCi/L corresponds to 37 Bq/m3, or 0.037 Bq/L.

Following common usage, the term radon will in some cases refer simply to radon-222, but sometimes to radon-222 plus its progeny. For example, one often talks about “radon risk” when most of that risk is actually conferred by inhaled decay products (USEPA 2003).

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on a consideration of the potential health impact from radon released from tap water to the air inside a dwelling, the activity concentration of radon-222 in drinking water should not exceed 100 Bq/L. The guideline value applies to the concentration of radon at the point of use of the water, not at the source, because of the significant decrease in concentration which can occur due to radioactive decay during storage, treatment and reticulation.

The European Commission has agreed a new Directive (2013/51/Euratom), which lays down requirements for the protection of the health of the general public with regard to radioactive substances in water intended for human consumption. A parametric value is introduced for radon of 100 Bq/L. However, Member States may set a level higher than 100 Bq/L but lower than 1,000 Bq/L which is judged inappropriate to be exceeded and remedial action must be taken where radon concentrations exceed 1,000 Bq/L. The 1,000 Bq/L corresponds to an indoor air concentration of radon that is similar to, but not much lower than, that used to manage radon in indoor air in the UK (DWI 2015).

### Sources to drinking-water

#### 1 To source waters

The most stable and environmentally relevant isotope of radon (Rn-222) forms from the alpha decay of Ra-226 (radium), which itself is ultimately a decay product of U-238 (uranium). DWI (2015) details the geology and sources of radon and subsequent hygrogeological factors.

Radon is a naturally occurring colourless, odourless, tasteless, dense and unreactive radioactive gas. Radon has no commercial uses. The largest source of radon in the environment is widely distributed uranium and its decay products in the soil. Radon has a half-life of 3.82 days.

Groundwater that is in contact with radium-containing rock and soil will be a receptor of radon emanating from the surroundings. High radon concentrations are associated with groundwater running over granitic rock or through alluvial soils originating from granite. When the groundwater reaches the surface by natural or mechanical means, this radon will start to be released to air. Although most of the radon present in groundwater will decay before reaching the surface, groundwater is considered to be the second largest source of environmental radon and is estimated to contribute 5 x 108 Ci (1.85 x 1019 Bq)/year to the global atmosphere.

If drinking water is derived from groundwater, the concentration of radon in the water will depend on the concentration of radon in the source aquifer, which in turn is controlled by uranium distribution, and to a limited extent by the aquifer’s physical properties, especially pore size. The pathway from aquifer to consumer, and especially the extent to which water is stored and aerated, will affect the ultimate radon concentration.

Approximately 1–5 percent of the radon in indoor air is estimated to originate from water.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

The solubility of radon in water is about 230 cm3/L. DWI (2015) expresses this as 2200 mg/L at 25°C, implying a maximum nominal activity of 1.26 x 1016 Bq/L (of radon‑222) in a water sample saturated with radon gas.

The ultimate fate of radon is transformation through radioactive decay. Radon decays only by normal radioactive processes, ie, an atom of radon emits an alpha particle resulting in an atom of polonium, which itself undergoes radioactive decay to other radon daughters or progeny. There are no sinks for radon, since its radioactive half-life is so short (3.8 days).

### Typical concentrations in drinking-water

In groundwater, radon moves by diffusion and, primarily, by the mechanical flow of the water. Radon solubility in water is relatively low and, with its short radioactive half-life of 3.825 days, much of it will decay before it can be released from groundwater. Groundwater supplies in the US have been surveyed for radon levels. In larger aquifers, average radon concentrations were reported to be 240 pCi/L (8.8 Bq/L), while in smaller aquifers and wells, average levels were considerably higher: 780 pCi/L of water (28.9 Bq/L). The highest levels reported were in smaller groundwater systems in Maine that averaged 10,000 pCi/L (370 Bq/L); lowest average levels were found in larger systems in Tennessee with levels of 24 pCi/L (0.9 Bq/L). These differences in radon levels between large and small groundwater supplies are a reflection of the type of rock that surrounds them. For public groundwater-derived water supplies, the average radon concentration is estimated at 540 pCi/L (20 Bq/L), although some wells have been found to have radon concentrations up to 400 times the average concentration.

Water supplies in New Zealand serving population groups of 5,000 or more were surveyed for radioactivity levels in 1980. Samples representing 102 water sources were analysed for total alpha and beta radioactivity and radon concentration; the results are summarised in Table 11.2 in Chapter 11 of the *Guidelines*. Results for radon ranged from 2 to 54 Bq/L, mean 16 Bq/L.

1,300 water utilities in the US reported detecting radon in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 32,400 pCi/L (= 1,200 Bq/L).

### Removal methods

The following has been copied from Section 11.8 of Chapter 11: Radiological compliance.

The most effective treatment device to remove radon from drinking-water is a point-of-entry (POE) device. A POE device removes contaminants immediately before they enter the home, USEPA. 1999. There are two types of point-of-entry devices that remove radon from water:

* granular activated carbon (GAC) filters which use activated carbon to remove the radon
* aeration devices which bubble air through the water and carry radon gas out into the atmosphere through an exhaust fan.

GAC filters tend to cost less than aeration devices; however, radioactivity collects on the filter, which may cause a handling hazard and require special disposal methods for the filter.

The USEPA formulated a proposed Radon in Drinking Water Bill in 1996 and updated it in April 2000 (USEPA 2000). This reference leads the reader to several other links. The Fact Sheet states:

**Best Available Technology (BAT) for Radon in Drinking Water Removal**

High-performance aeration is the proposed BAT for all systems. High Performance Aeration is defined as the group of aeration technologies that are capable of being designed for high radon removal efficiencies (up to 99.9 percent removal), ie, Packed Tower Aeration, Multi-Stage Bubble Aeration and other suitable diffused bubble aeration technologies, Shallow Tray and other suitable Tray Aeration technologies, and any other aeration technologies that are capable of similar high performance. In addition to listing BAT, which is based on technology evaluations for large systems, the SDWA directs EPA to list “Small Systems Compliance Technologies” (SSCTs): affordable and technically feasible technologies based upon technology evaluations for small systems. EPA is proposing that high performance aeration, granular activated carbon (GAC), and point-of-entry GAC be listed as SSCTs. Issues relevant to safe operation procedures and safe and legal disposal of spent GAC material are addressed in the preamble to the proposed radon rule.

Radon is on the WHO plan of work of the “Rolling Revision” of their Guidelines. This is what appears in the draft as at September 2008:

Radon, being a gas, is relatively easy to remove by air stripping. Removal efficiencies of >99 percent were obtained with diffuse bubble and packed tower aeration at air:water ratios of 15:1 and 5:1, respectively. Other investigations focusing on aeration at public waterworks have given similar results, with  
67–99 percent efficiencies. This is the preferred method of treatment.

GAC is also effective in removing radon from water, with removals of  
70–100 percent. The amount of radon removed by activated carbon is effectively unlimited because the adsorbed radon decays into other radioactive products, such as 210Pb. As the adsorbed radon decays, radioactive progeny emitting gamma radiation is produced, possibly creating a disposal problem. Elevated gamma dose rates (up to 120 μSv/h) near the filter have been recorded. Screening of the GAC filter could be required. In some circumstances, a twin tank system, which introduces a time delay that allows the radon to decay to a significant extent, may be a low-cost option.

### Analytical methods

#### Referee method

Rather than measuring radon in mg/L units, it is measured in terms of its radioactivity. The short half life of radon means that great care must be taken when collecting and transporting samples; refer to the National Radiation Laboratory. Similar care is needed when collecting a sample for carbon dioxide analysis; the procedure is described in Chapter 4: Selection of Water Source and Treatment, Section 4.4.1.

DWI (2015) states that regardless of which method is selected, all collection and measurement systems must be included within an ISO 17025 accredited system which also conforms to the Drinking Water Testing Specification. Whilst these requirements are already well known to most laboratories, there are problems in applying it to the determination of radon in water due to the lack of suitable reference standards, particularly for field based methods. DWI (2015) thoroughly discusses methods of sampling and analysis.

### Health considerations

Radon is odourless, colourless and tasteless, thereby making it difficult to detect its presence in water with the human senses.

As radon undergoes radioactive decay, radiation is released predominantly by high-energy alpha particle emissions, which are the source of health concerns. The health hazard from radon does not come primarily from radon itself, but rather from its radioactive progeny. Radon is the largest fraction of natural radiation exposure.

Radon in water can become airborne; it is estimated that 1/1,000th of the radon in water may become airborne during indoor activities that use water. It can then be inhaled, for example while showering.

Most of the inhaled radon gas is breathed out again. Some of the radon progeny, both unattached and attached to particles, may remain in the lungs and undergo radioactive decay. The radiation released during this process passes into lung tissue and can cause lung damage.

Since it is chemically inert, most inhaled radon-222 is rapidly exhaled, whereas inhaled progeny readily deposit in the airways of the lung. Two of these daughters, polonium-218 and polonium-214, emit alpha-particles. When this happens in the lung, the radiation can damage the cells lining the airways, leading ultimately to cancer. (Nuclear decay of radon decay products also releases energy in the form of beta particles and high energy photons, but the biological damage resulting from these emissions is believed to be small compared to that from alpha particles.) There is also evidence that ingestion of radon can cause stomach cancer.

Two other radon isotopes, radon-219 (actinon) and radon-220 (thoron), occur in nature and produce radioactive radon daughters. Because of its very short half-life (3.9 seconds), environmental concentrations of actinon and its daughters are extremely low, so their contribution to human exposure is negligible. The half-life of thoron is also relatively short (56 seconds), and a lower fraction of released alpha-particle energy is absorbed within target cells in the bronchial epithelium than in the case of radon‑222. As a result, thoron is thought to pose less of a problem than radon-222, but we have rather limited information on human exposure to thoron, and no direct information on its carcinogenicity in humans (USEPA 2003).

Some of the radon that is swallowed with drinking-water passes through the walls of the stomach and intestine. After radon enters the blood stream most of it quickly moves to the lungs where it is expelled. Radon that is not expelled goes to other organs and fat tissue where it may remain and undergo decay.

Smoking cigarettes greatly increases the chance of developing lung cancer when exposed to radon and radon progeny at the same levels as people who do not smoke. WHO (2017) states that radon is the most important cause of lung cancer after smoking; smokers are estimated to be 25 times more at risk from radon than non-smokers. To date, no other cancer risks have been established. To date, epidemiological studies have not found an association between consumption of drinking-water containing radon and an increased risk of stomach cancer.

Large pooled studies of indoor radon and lung cancer risk have recently become available. The European pooled analysis of 13 indoor radon studies estimated a 16 percent risk increase per 100 Bq/m3. Based on these data, radon accounts for about 9 percent of all lung cancer deaths and 2 percent of total cancer deaths in Europe. Similar results were obtained from the joint analysis of North American radon studies. For the USA, the USEPA has estimated that radon causes about 21,000 lung cancer deaths per year (with an uncertainty range of 8,000–45,000), out of about 160,000 annual lung cancer deaths. Radon is the second leading cause of lung cancer, after smoking (WHO 2008). The US National Research Council (NRC) estimated that 89 percent of the estimated cancer risk results from inhalation of the radon emitted from water, and 11 percent is caused by ingestion.

The IARC considers radon and its decay products are carcinogenic to humans (Group 1); note that the conclusion was based on inhalation studies.

### Derivation of Maximum Acceptable Value

The WHO Guidelines (2004) are based on:

* a recommended reference dose level (RDL) of the committed effective dose, equal to 0.1 mSv from one year’s consumption of drinking-water (from the possible total radioactive contamination of the annual drinking-water consumption). This comprises 10 percent of the intervention exemption level recommended by the ICRP for dominant commodities (eg, food and drinking-water) for prolonged exposure situations, which is most relevant to long-term consumption of drinking-water by the public (ICRP 2000). The RDL of 0.1 mSv is also equal to 10 percent of the dose limit for members of the population, recommended by both the ICRP (1991) and the International Basic Safety Standards (IAEA 1996). These are accepted by most WHO Member States, the European Commission, FAO and WHO
* dose coefficients for adults, provided by the ICRP.

Note however that WHO (2011) states that “As the dose from radon present in drinking-water is normally received from inhalation rather than ingestion, it is more appropriate to measure the radon concentration in air than in drinking-water. The World Health Organization reference level for radon concentration in indoor air is 100 Bq/m3 in dwellings”.

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# Rubidium

### Maximum Acceptable Value

There is no MAV in the DWSNZ for rubidium, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1 To source waters

Rubidium is about the 20th most abundant element in the Earth’s crust (average 310 mg/kg), roughly as abundant as zinc and rather more common than copper, but does not appear in mineral-rich deposits.

Natural rubidium comprises two isotopes: at 72 percent is the stable isotope 85Rb; at 28 percent is the slightly radioactive 87Rb, with a half-life of 49 billion years, more than three times longer than the estimated [age of the universe](https://en.wikipedia.org/wiki/Age_of_the_universe).

Geothermal waters in the Taupo Volcanic Zone can contain up to 2.5 mg/L Rb (GNS 2015), hence it is detected in the Waikato River. The concentration of rubidium in seawater is about 0.125 mg/L.

Granite and gneiss weathering soils produce by far the highest concentrations in food and water. Water at gneiss sites contained 0.018 mg/L Rb, cf from diluvial sands at 0.003 mg/L. Fruit and vegetables accumulate more rubidium than cereals but it tends to leach out on boiling (Anke and Angelow 1995).

Rubidium compounds have been known to be used in fireworks to give a purple colour. Most uses are extremely specialised, such as in atomic clocks.

#### 2 From treatment processes

No known sources.

### Forms and fate in the environment

Rubidium occurs in water as the rubidium ion, Rb+.

### Typical concentrations in drinking-water

Drinking water in Germany contained a mean concentration of 11 ± 8.8 µg/L Rb, a median of 8.1 µg/L, and a range 1.9 to 38 µg/L (Anke and Angelow 1995).

### Health considerations

Rubidium does not appear to have any function in living organisms (disputed by Anke and Angelow). However, being similar to potassium ions, rubidium is actively taken up and behaves rather like potassium. This property leads to its use as a biomarker in research.

Rubidium salts have been used to treat depression. A 70 kg person contains on average 0.36 g of rubidium, and an increase in this value by 50 to 100 times did not show negative effects in test persons.

### Derivation of Maximum Acceptable Value

No MAV.

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# Selenium

CAS No. 7782-49-2.

### Maximum Acceptable Value (provisional)

Based on health considerations, the concentration of selenium in drinking-water should not exceed 0.04 mg/L.

The WHO (2011/2017) guideline value is designated as provisional because of the uncertainties inherent in the scientific database.

In the 2008 DWSNZ the MAV for selenium in drinking-water had been 0.01 mg/L.

The maximum contaminant level or MCL (USEPA 2009/2011) is 0.05 mg/L. The USEPA also established a lifetime health advisory of 0.05 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 0.01 mg/L.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of selenium in drinking water should not exceed 0.01 mg/L.

The Prescribed Concentration or Value (PCV) for selenium in England and Wales is 0.01 mg/L. See Notes.

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

### Sources to drinking-water

#### 1 To source waters

Selenium enters the aquatic environment naturally via the weathering of rocks and minerals, soil runoff and volcanic activity. Selenium occurs essentially in sulphide ores of heavy metals, and is usually produced as a by-product of copper refining.

Geothermal waters in the Kawerau area can contain about 0.01 µg/L Se; GNS (2015).

The concentration of selenium in seawater is about 0.002 mg/L.

Selenium is used in a wide variety of manufacturing, including electronic, metallurgic, glass and ceramic, pigment, chemical and pharmaceutical industries. It is also used in plastics, paints, anti-dandruff shampoos, vitamin and mineral supplements, fungicides, and certain types of glass. Selenium is also used to prepare drugs and as a nutritional feed supplement for poultry and livestock. It may also be present in effluents from copper and lead refineries and in municipal sewage. Selenium is released from natural and human-made sources, with the main source being the burning of coal. Selenium is added as a trace element to some soils, and to some stainless steels.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources. Some lead based brass fittings are being replaced with bismuth based fittings; some of these contain 0.7 percent selenium. So far, reports suggest that selenium does not leach from these new brasses.

### Forms and fate in the environment

Selenium exists in four oxidation states in the aquatic environment: 0, -2, +4 and +6. Dissolved selenium species are mainly in the +4 and +6 oxidation states while those in the -2 state decompose under aerobic conditions to form elemental selenium which is insoluble. Selenium (IV) is also reduced to insoluble elemental selenium under acidic and reducing conditions, whereas alkaline and oxidising conditions favour the formation of the stable Se(VI) compounds.

Both selenites and selenates are taken up by plants (the major point of entry of selenium into the human food chain), where they are converted to protein-bound selenomethionine, soluble inorganic forms, several free amino acids, and volatile organoselenium compounds. The selenates, however, are considered the most readily available to plants.

### Typical concentrations in drinking-water

Selenium was measured routinely in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. Of 1779 samples analysed between 1983 and 1989, 21 (1.2 percent) had concentrations equal to or exceeding the MAV of 0.01 mg/L.

The P2 Chemical Determinand Identification Programme, sampled from 285 zones, found selenium concentrations ranged from “not detectable” (nd) to 0.009 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 selenium at greater than the MAV, but two distribution zones supplying 502 people had >50 percent MAV (ESR 2001).

A 1982 survey of drinking water supplies from 122 municipalities across Canada (representing 36 percent of the Canadian population) showed that selenium was present at or below the detection limit of 0.0005 mg/L (raw, treated, and distributed water samples were analysed) (Health Canada 1992).

In major Australian reticulated water supplies, selenium concentrations are less than 0.005 mg/L. Concentrations in drinking-water supplies overseas are generally below 0.01 mg/L but groundwater concentrations as high as 6 mg/L have been reported in the United States.

3,935 water utilities in the US reported detecting selenium (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.15 mg/L.

### Removal methods

The efficiency with which selenium can be removed from waters by conventional coagulation depends on the oxidation state of the selenium (IV and VI). Virtually no removal of selenium(VI) is achieved using either ferric salts or alum. Some studies have shown significant removal of selenium(IV) with ferric sulphate at pH values in the 6 to 7 region. However, contradictory data were obtained when the effect of coagulant dose was studied.

Adsorption or ion-exchange on alumina columns has been shown to be effective in removing both these oxidation states, but removal efficiency depends on the concentrations of other anions that are preferentially taken up by alumina.

Reverse osmosis can remove both oxidation states of selenium. Lime-softening and PAC or GAC adsorption have little effect on the concentration of either form of selenium.

### **Analytical methods**

#### Referee method

Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

#### Some alternative methods

1. Hydride Generation / Atomic Absorption Spectrometric Method (APHA 3114B).

2. Inductively Coupled Plasma (ICP) Method (APHA 3120).

3. Inductively Coupled Plasma – Mass Spectrometry (EPA Method 200.8).

### Health considerations

Selenium is an essential element for humans, and forms an integral part of the enzyme glutathione peroxidase and probably other proteins as well. Foodstuffs such as cereals, meat and fish are the principal source of selenium in the general population. Levels in food also vary greatly according to geographical area of production. However, even in high-selenium areas, the relative contribution of selenium from drinking-water is likely to be small in comparison with that from locally produced food.

As selenium is an essential element, various national and international organisations have established recommended daily intakes of selenium. A joint FAO/WHO consultation recommended intakes of 6–21 μg of selenium per day for infants and children, according to age, 26 and 30 μg of selenium per day for adolescent females and males, respectively, and 26 and 35 μg of selenium per day for adult females and males, respectively. FAO/WHO established an upper tolerable limit for selenium of 400 μg/day (WHO 2011).

Most selenium compounds are water soluble and are efficiently absorbed from the intestine. Selenium compounds appear to be of the same order of toxicity in humans and laboratory animals. There have been a number of reports of adverse effects caused by short- and long-term exposure to selenium, most of which have resulted from occupational exposure or accidental poisoning. The occurrence of acute or chronic nutritional toxicity is comparatively rare. Intakes above about 1 mg/day over prolonged periods may produce nail deformities characteristic of selenosis. Other characteristics of excess selenium intake include non-specific symptoms such as gastro-intestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath.

Long term toxicity in rats is characterised by depression of growth and liver pathology at levels of 0.03–0.4 mg selenium/kg body weight per day given in food.

Tests for mutagenic activity using bacteria have reported both positive and negative results. Studies indicate that selenite can cause chromosome damage to mammalian cells.

Except for selenium sulphide, which does not occur in drinking-water, experimental data do not indicate that selenium is carcinogenic. The International Agency for Research on Cancer has placed selenium and selenium compounds in Group 3 (not classifiable as to its carcinogenicity to humans). Selenium sulphide 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 0.005 mg/kg/day for chronic-duration oral exposure (>364 days) to selenium.

The reference dose or RfD (USEPA 2009/2011) is 0.005 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.2 mg/L.

EFSA (2014) quote a value of 300 μg/day as a Tolerable Upper Intake Level (UL) for adults including pregnant and lactating women, on the basis of a No Observed Adverse Effect Level (NOAEL) of 850 μg/day for clinical selenosis and applying an uncertainty factor of 3, supported by three studies reporting no adverse effects for selenium intake between about 200 and 500 μg/day.

The livestock guideline value is 0.02 mg/L (ANZECC/ARMCANZ 2000). These guidelines were to be updated in 2012.

### Derivation of Maximum Acceptable Value

WHO (2011/2017) states that their guideline value of 0.04 mg/L, which is based on an allocation of 20 percent of the upper tolerable intake of 400 μg/day to drinking-water (2 litres per day), provides a sensible balance that will assist regulators and suppliers in making decisions about whether further action is needed. WHO (2017) adds:

For most Member States, a drinking-water guideline for selenium is unnecessary. Where there are regions of high intake from a number of sources, of which drinking-water may be one, then Member States should take into consideration exposure from all sources in determining actions to reduce exposure. For drinking-water, this may include using alternative sources, blending low-selenium sources with high-selenium sources as well as considering selenium removal.

The 2008 DWSNZ MAV had been based on the toxic effects of long-term selenium exposure manifested in nails, hair and liver. Based on Chinese data, clinical signs seemed to occur at a daily intake above 0.8 mg. Daily intakes by Venezuelan children with clinical signs were estimated at about 0.7 mg, on the basis of their blood levels and Chinese data on blood level/intake relationships. Effects on synthesis of a liver protein were also seen in a group of patients with rheumatoid arthritis given 0.25 mg selenium/day. No clinical or biochemical signs of selenium toxicity had been reported in a group of 142 persons with a mean daily intake of 0.24 mg (maximum 0.72 mg).

Based on these data, a No-observable-adverse-effects level (NOAEL) in humans was estimated to be about 4 g/kg body weight per day. The recommended daily intake of selenium was 1 g/kg of body weight for adults.

Up to 2008 the MAV for selenium in drinking-water had been derived as follows:

0.004 mg/kg body weight per day x 70 kg x 0.1 = 0.014 mg/L (rounded to 0.01 mg/L)

2 L per day

where:

* No Observable Adverse Effect Level = 0.004 mg/kg body weight per day
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.1
* average amount of water consumed by an adult = 2 L per day.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for selenium is 0.03 mg/L.

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# Silver

CAS No. 7440-22-4.

### Maximum Acceptable Value

Available data are inadequate to permit derivation of a health-based guideline value (WHO 2017).

WHO (2004/17) considered that there are no adequate data with which to derive a health-based guideline value for silver in drinking-water, and that where silver salts are used to maintain the bacteriological quality of drinking-water, levels of silver up to 0.1 mg/L can be tolerated without risk to health.

In DWSNZ 2005, the provisional MAV had been 0.1 mg/L.

In the 2000 DWSNZ, the provisional MAV of 0.02 mg/L had been based on the guideline value in the 1996 Australian Drinking Water Guidelines.

The 1995 DWSNZ and datasheet stated that no health-based MAV is proposed for silver in drinking-water because it is not hazardous to human health at concentrations normally found in drinking-water.

Health Canada (1986) states that it is not considered necessary to specify a maximum acceptable concentration for silver in drinking water.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of silver in drinking water should not exceed 0.1 mg/L.

The USEPA (2009/2011) has 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. However, in the case of silver, the lifetime health advisory isbased on cosmetic effects.The USEPA also has a secondary drinking water regulation of 0.1 mg/L for silver; in 1991 this replaced their MCL of 0.05 mg/L (USEPA 1992).

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

### Sources to drinking-water

#### 1 To source waters

Silver enters the aquatic environment naturally via the weathering of rocks and minerals and soil runoff, mainly in the form of its very insoluble and immobile oxides, sulfides and some salts. In New Zealand, silver occurs almost always in association with gold. Typical geological settings include: associated with intermediate volcanics (eg, Hauraki Goldfield), associated with metamorphic rocks (eg, with most South Island gold-bearing reefs), in base-metal mineralisation near granite contacts (eg, at Richmond Hill, Copperstain Creek and Mt Radiant in Nelson), in some South Island ultrabasics around Nelson and in silver mineralisation at Puhipuhi, Northland.

Geothermal waters in the Taupo Volcanic Zone can contain up to 0.03 mg/L Ag (GNS 2015).

Silver also may be present in water from industrial discharges. Silver is used in photographic materials, in the manufacture of sterling and plated ware, jewellery, coins, electrical and electronic products, eg, batteries, contacts and conductors, brazing alloys and solders, catalysts, mirrors, fungicides, and in dental (about 35 percent of amalgam) and medical supplies.

Various silver compounds have been used as antibiotics and disinfectants. For example, silver sulfadiazine 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). Silver sodium hydrogen zirconium phosphate (Antimicrobial AlphaSan® RC 5000) is incorporated in plastic, polymeric and related materials, in non-food and food contact uses as an antimicrobial agent (New York State 2001). This product may be used at up to 2 percent by weight in finished manufactured products to suppress the growth of bacteria, algae, fungus, mould, and mildew.

Silver nanoparticles are approved by the USEPA for use in consumer products and are increasingly being used as biocides in products such as soaps and laundry detergents, laundry machines, cooking utensils, water purifiers, clothing, personal care products (eg, toothpaste, shampoo, cosmetics), and humidifiers. Silver from these products could become dissolved or particulate silver in wastewaters or source waters (WRF 2015).

Silver concentrations in natural source waters are very low, generally less than 0.0002 mg/L. Measurements of silver in Canadian surface waters (measured at NAQUADAT stations) indicated that the silver concentrations in 90 percent of the water samples were below detection limits (which ranged from 0.004 to 0.01 mg/L), and none was greater than 0.01 mg/L. Measurements at seven points along the length of the Ottawa River, Ontario, found concentrations of silver that ranged between 0.00001 and 0.00006 mg/L.

NICNAS (2013) states: “According to the Silver Institute, approximately 5 percent of the global silver production is available for production of silver nanoparticles, therefore global production of silver nanoparticles is estimated to be somewhere between  
400–500 tonnes a year”. Note: there is also a datasheet for nanoparticles.

#### 2 From the treatment processes

Soluble silver compounds may be used to disinfect drinking-water. At present, this method of disinfection is not known to be employed in any New Zealand drinking-water supplies. Also refer to Chapter 15, and the datasheet for nanoparticles.

Situations exist where silver salts may be used to maintain the bacteriological quality of drinking-water. Higher levels of silver, up to 0.1 mg/L (this concentration gives a total dose over 70 years of half the human NOAEL of 10 g), could be tolerated in such cases without risk to health (WHO 2017).

#### 3 From the distribution system

Some point-of-use water filters contain activated carbon impregnated with silver.

### Forms and fate in the environment

In aqueous systems silver is predominantly present in the univalent state Ag(I). Under aerobic conditions Ag(I) is soluble and mobile but as the pH increases the silver tends to precipitate. Silver occurs naturally, mainly in the form of its very insoluble and immobile oxides, sulfides and some salts. Sorption and precipitation are the dominant mechanisms for controlling the transport and removal of silver in the aquatic environment. In sewage, any soluble silver tends to be precipitated as silver sulphide.

### Typical concentrations in drinking-water

Silver was not routinely monitored in New Zealand drinking-water supplies and therefore no information is available about its concentration.

The P2 Chemical Determinand Identification Programme, sampled from 831 zones, found silver concentrations to range from “not detectable” (nd) to 0.0032 mg/L, with the median concentration being “nd” (Limit of detection = 0.0005 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with silver at greater than 50 percent of the MAV (ESR 2001).

In a survey of Canadian tap water, silver was found in only 0.1 percent of 239 sampled waters; the detection limit for silver in this study, which used neutron activation analysis, was 0.000001 to 0.000005 mg/L.

250 water utilities in the US reported detecting silver (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.29 mg/L.

The maximum concentration found in 19,402 samples from 2,929 groundwaters in the UK was 0.080 mg/L, mean 0.0005 mg/L (DWI 2008).

Silver concentrations in excess of 0.05 mg/L may occur when silver is used as an antimicrobial agent in water treatment devices (Smith et al 1977). Silver is used to treat potable water for Swiss ski resorts, German breweries, soft drink bottlers, British ships, Shell Oil Company tankers, drilling rigs, and over half the world’s airlines (USEPA 1980). Concentrations of 0.1 to 0.2 mg/L of silver are sufficient to ensure antimicrobial action.

### Removal methods

Natural turbidity influences the effectiveness of coagulation/flocculation with iron or aluminium salts to remove silver. Ferric sulphate is more effective than alum, and removal with alum is pH-dependent.

Lime-softening is useful for the removal of silver when operating at a pH greater than 9.

Soluble silver has been removed effectively (86–92 percent) from secondary and industrial wastewaters by cation exchange.

### Analytical methods

#### Referee method

Atomic Absorption Spectrometric Method (APHA 3111B).

#### Some alternative methods

1. Electrothermal Atomic Absorption Spectrometric Method (APHA 3113).

The detection limits for silver by Flame AA and Furnace AA are 0.01 mg/L and 0.0002 mg/L (0.2 g/L) respectively.

2. Inductively Coupled Plasma Method (APHA 3120B).

The upper limit for silver by ICP is 50 mg/L with an estimated detection limit of 0.007 mg/L (7 g/L).

### Health considerations

Traces of silver can be found in most foods but it is not considered an essential trace element for mammals; estimates of daily intake are about 7 μg per person.. Only a small percentage of silver is absorbed by the gastro-intestinal tract. Retention rates in humans and laboratory animals range between 0 and 10 percent. It is stored mainly in the liver and skin and is capable of binding to amino acids and proteins.

The only obvious sign of silver intoxication is argyria, a condition is which skin and hair are heavily discoloured by silver in the tissues. An oral No-Observed-Adverse-Effect Level (NOAEL) for argyria in humans for a total lifetime intake of 10 g silver can be estimated from human case reports and long-term animal experiments. Excessive industrial or medical exposures to silver have been associated with arteriosclerosis and lesions of the lungs and/or kidneys (USEPA 1992).

The USEPA secondary MCL of 0.1 mg/L is to prevent argyria, which is a blue-grey discoloration of the skin or whites of the eyes (WRF 2015).

Special situations exist where silver salts may be used to maintain the bacteriological quality of drinking-water. Higher levels of silver, up to 0.1 mg/L (this concentration gives a total dose over 70 years of half the human NOAEL of 10 g), could be tolerated in such cases without risk to health. Normally the contribution of drinking-water to this NOAEL would be negligible, therefore the establishment of a health-based guideline value is not deemed necessary.

The USEPA (1992) classified silver as a Group D carcinogen (not classifiable as to human carcinogenicity). Silver salts are not mutagenic in tests with bacteria but can induce damage in mammalian DNA.

The estimated acute lethal dose of silver nitrate is at least 10 g.

The reference dose or RfD (USEPA 1992/2009/2011) is 0.005 mg/kg/d (note that this isbased on cosmetic effects). The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.2 mg/L.

Physico-chemical properties that contribute to the toxicity of silver nanoparticles include size, shape, surface coating, and surface charge. Factors that affect silver ion release may contribute to the indirect toxicity of silver nanoparticles. As the high surface area per mass of nanoparticles provides the opportunity for closer interaction with the surrounding materials and environment, nanoparticles may have different properties and biological activity compared with regular/bulk silver. In a study with 42 nm silver particles administered in the diet of mice for 28 days at three different doses (0.25, 0.50 or 1 mg/kg bw/day), hepatotoxicity and significant increases in alkaline phosphatase (ALP), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were seen in the highest dose group. Some histopathological changes were also seen in the kidneys at the highest dose. Based on the effects reported at the highest dose, a no observed adverse effect level (NOAEL) of 0.50 mg/kg bw/day can be determined (NICNAS 2013).

The body of evidence on safety seems to suggest that silver (in ionic form or as silver nanoparticles) is toxic to mammalian cells, although the sensitivity of the cells varies according to the cell type and the type of silver to which it is exposed. Most of the evidence on the toxicity of silver comes from in vitro studies. However, there is accumulating evidence from mammalian in vivo data, especially with silver nanoparticles, that suggest that exposure to silver may result in toxic effects in exposed subjects, given sufficient dosage and lengths of exposure. In particular, available data indicate that silver nanoparticles have potential to damage DNA, although the potential for genotoxicity or DNA damage with silver nanoparticles requires further investigation as to its significance for humans (WHO 2018).

### Derivation of Maximum Acceptable Value

No MAV.

The low levels of silver in drinking-water, generally below 0.005 mg/L (5 g/L), are not relevant to human health with respect to argyria. On the other hand, special situations exist where silver salts may be used to maintain the bacterial quality of drinking-water. Higher levels of silver of up to 0.1 mg/L (this concentration gives a total dose over 70 years of half the human NOAEL of 10 g) could be tolerated in such cases without risk to health.

The guideline value for silver in drinking water in the DWSNZ 2005 had been derived as follows:

0.4 mg per day x 0.5 = 0.1 mg/L

2 L per day

where:

* 0.4 mg/day is derived from a lifetime no effect level of 10 g (Hill and Pillsbury 1939)
* 0.5 is the proportion of total daily intake attributable to the consumption of drinking water
* 2 L/day is the average amount of water consumed by an adult.

No additional safety factors were used, as the calculation was based on a human no effect level. It is unlikely that silver concentrations in drinking water would ever reach a concentration that could cause adverse effects. Silver or silver salts should not be used as antimicrobial agents unless no other disinfectants are available.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for silver is 0.03 mg/L.

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# Strontium

CAS No. 7440-24-6 (the metal).

### Maximum Acceptable Value

No MAV in the DWSNZ; radioactive strontium is covered generally by the radiochemical determinands. The only reference to strontium in the WHO Guidelines is in relation to radiochemistry.

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

The USEPA (2009/2011) has a lifetime health advisory of 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. In 2014, the USEPA announced a preliminary determination to regulate Sr in drinking water. This action followed a revision of the Health Reference Level (HRL) for Sr in drinking water from 4.2 mg/L to 1.5 mg/L (WRF 2016).

### Sources to drinking-water

#### 1 To source waters

Strontium commonly occurs in nature, the 15th most abundant element on earth, averaging 0.034 percent of all igneous rock and is found chiefly as the form of the [sulfate](http://en.wikipedia.org/wiki/Sulfate) [mineral](http://en.wikipedia.org/wiki/Mineral) [celestite](http://en.wikipedia.org/wiki/Celestite) (SrSO4) and the [carbonate](http://en.wikipedia.org/wiki/Carbonate) [strontianite](http://en.wikipedia.org/wiki/Strontianite) (SrCO3). Strontium metal reacts rapidly with water and oxygen and is thus found in nature only in the 2+ oxidation state.

The concentration of strontium in seawater is about 10 mg/L.

Volatile strontium salts impart a [crimson](http://en.wikipedia.org/wiki/Crimson) colour to [flames](http://en.wikipedia.org/wiki/Fire), and these salts are used in [pyrotechnics](http://en.wikipedia.org/wiki/Pyrotechnic) and in the production of [flares](http://en.wikipedia.org/wiki/Flare_(pyrotechnic)). Natural strontium is not radioactive and exists in four stable isotopic forms: 88Sr (82.6 percent), 86Sr (9.9 percent), 87Sr (7.0 percent) and 84Sr (0.6 percent). The primary use for strontium compounds is for [colour](http://en.wikipedia.org/wiki/Colour) television [cathode ray tubes](http://en.wikipedia.org/wiki/Cathode_ray_tube) and in faceplate [glass](http://en.wikipedia.org/wiki/Glass) to prevent [X-ray](http://en.wikipedia.org/wiki/X-ray) emission. Strontium-89 is a short-lived radioactive isotope of strontium used as a radiopharmaceutical for the treatment of metastatic bone cancer. Strontium-90 is a radioactive pollutant from nuclear fallout and possibly weapons and power production.

The average concentration of strontium in seawater is approximately 8 mg/L. Strontium is present in nearly all fresh surface waters across the USA; average concentrations being between 0.3 and 1.5 mg/L. Strontium concentrations in European stream waters range from 0.001 to 13.6 mg/L, with a median value of 0.11 mg/L. The median strontium concentration in European stream sediments is 126 mg/kg. Mean strontium levels of up to 225 mg/kg dry weight have been reported for river sediments contaminated by old mine workings.

Strontium is released to the environment by seaspray, burning coal and with superphosphate. Atmospheric strontium is returned to the ground by deposition. Strontium is found naturally in soil in amounts that vary over a wide range, but the typical concentration is 0.2 mg/kg. Plants readily absorb strontium via their normal calcium uptake pathway. Foods containing strontium range from very low, eg, in corn (0.4 ppm) and oranges (0.5 ppm), to high, eg, in cabbage (45 ppm), onions (50 ppm) and lettuce (74 ppm).

#### 2 From the treatment processes

No known sources.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

Like calcium, strontium has moderate mobility in soils and sediments and sorbs moderately to metal oxides and clays.

### Typical concentrations in drinking-water

Typically, the amount of strontium that has been measured in drinking-water in different parts of the United States by the USEPA is less than 1 mg/L, although many supplies contain >4 mg/L.

366 water utilities in the US reported detecting strontium in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 33 mg/L.

Two water utilities in the US reported detecting strontium-89 in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 250 pCi/L.

Fifty-one water utilities in the US reported detecting strontium-90 in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 3 pCi/L.

The Indiana Department of Natural Resources tested 1,839 samples collected from 866 groundwater sources; there was no correlation between Sr and Ca levels (WRF 2016).

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 62,799 drinking water samples for strontium between 2013 and 2015, and found 1,739 samples exceeded the minimum reporting level (MRL) of 0.0003 mg/L, and 286 samples contained >1.5 mg/L.

### Removal methods

Precipitation of SrCO3 is attainable under the same conditions commonly used to precipitate CaCO3 (e.g., lime softening processes). However, the analysis also showed that using precipitation chemistry for targeted removal of Sr from water without removing other constituents (primarily Ba and Ca) is not technically feasible. Research is needed to find (WRF 2016):

1. chemical precipitation conditions that could maximise Sr removal and minimise Ca removal

2. cation exchange resin regeneration conditions that minimise waste brine production when operated for Sr removal.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

For adult humans, the total daily intake of strontium in many parts of the world is estimated to be up to about 4 mg/day. Drinking-water contributes about  
0.7–2 mg/day, and food (mainly leafy vegetables, grains and dairy products) another 1.2–2.3 mg/day. Intakes might be substantially higher in areas where strontium concentrations in the drinking-water are at the high end of the measured range.

Since strontium is so similar to calcium, it is incorporated in the bone; all four isotopes are incorporated, in roughly similar proportions as they are found in nature. It comprises about 4.6 ppm by weight of the human body, but does not have any recognised essential biological role. Strontium can be used for treating cancer and bone diseases.

The International Agency for Research on Cancer (IARC) has determined that radioactive strontium is a human carcinogen.

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 2 mg/kg/day for intermediate-duration oral exposure  
(15–364 days) to strontium.

The USEPA derived a chronic reference dose (RfD) of 0.6 mg/kg/day for strontium. The RfD is based on a NOAEL of 190 mg strontium/kg/day for skeletal toxicity in young rats. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 20 mg/L.

From a study in which no adverse effects (the examination included a microscopic evaluation of the bone) were seen in young rats ingesting strontium at a dose of 40 mg/kg body weight per day for 90 days, a tolerable daily intake (TDI) of 0.13 mg/kg body weight per day can be derived (WHO 2010).

Short-term, the USEPA (2009) recommends <25 mg/L for a 10 kg child for a one-day exposure.

### Derivation of Maximum Acceptable Value

No MAV.

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# Sulfuryl fluoride

CAS No. 2699-79-8. Sometimes called sulfonyl fluoride, sulfur dioxide difluoride, sulphuryl fluoride and sulfuryl difluoride.

### Maximum Acceptable Value

There is no MAV in the DWSNZ for sulfuryl fluoride, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1 To source waters

Sulfuryl fluoride is an inorganic colourless gas, SO2F2.

As the deadline for phasing out the use of methyl bromide as a fumigant approaches, alternative fumigants are being evaluated. Sulfuryl fluoride has emerged as a promising alternative and is gaining increasing acceptance in Europe, despite it also being a greenhouse gas. However, the USEPA may withdraw its approval for use on foods due to fluoride intake (USEPA 2002).

Sulfuryl fluoride has been used as an insecticide and rodenticide. Sulfuryl fluoride breaks down to fluoride and sulfate inside the insect’s body. Like methyl bromide, it is used under cover.

Becausesulfuryl fluoride is an odourless, colourless gas, applicators introduce trace amounts of a warning agent, chloropicrin (see datasheet), into the structure.

#### 2 From the treatment processes

No known sources.

### Form and fate in the environment

Sulfuryl fluoride has an atmospheric lifetime of 30–40 years, much longer than the five years earlier estimated. It is slowly lost from the atmosphere by dissolving in water (mainly the oceans) where it hydrolyses is broken down by hydrolysis into fluoride and sulfide and sulfate ions.

Groundwater contamination is unlikely based on the present use pattern and volatility of sulfuryl fluoride.

Water solubility is about 0.075 percent (750 mg/L) at 25°C.

### Typical concentrations in drinking water

Being a gas, sulfuryl fluoride is not expected to be found in drinking water.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Based on the current use pattern of sulfuryl fluoride, the USEPA did not require carcinogenicity tests. Therefore, the USEPA has not classified the potential for sulfuryl fluoride to cause cancer. Mammalian toxicity by inhalation is about equal to that of methyl bromide.

As at May 2014, <http://water.epa.gov/drink/standards/hascience.cfm> quotes a RfD of 0.08 mg/kg/d; there is no ARfD – these are from their Human Health Benchmarks for Pesticides.

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 chronic (lifetime) HHBP in drinking water for sulfuryl fluoride is 0.56 mg/L. (No acute one day value available.)

JMPR developed an ADI and ARfD of 0–0.01 mg/kg bw and 0.3 mg/kg bw respectively, both based on inhalation studies.

The Acceptable Daily Intake (ADI) adopted in Australia for sulfuryl fluoride is 0.01 mg/kg body weight and the ARfD is 0.3 mg/kg bw, based on inhalation studies.

### Derivation of Maximum Acceptable Value

No MAV.

### Bibliography

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# Sulphur hexafluoride

CAS No. 2551-62-4. Sometimes called sulphur fluoride.

### Maximum Acceptable Value

There is no MAV in the DWSNZ for sulphur hexafluoride, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1 To source waters

The electric power industry uses roughly 80 percent of all SF6 produced worldwide. Ideally, none of this gas would be emitted into the atmosphere. In reality significant leaks occur from aging equipment, and gas losses occur during equipment maintenance and servicing. With a global warming potential 23,900 times greater than CO2 and an atmospheric life of 3,200 years, one pound of SF6 has the same global warming impact of 11 tons of CO2.

Sulphur hexafluoride was one of the greenhouse gases (eight times denser than air) that many industrialised nations agreed (in 1997 at the Kyoto Protocol to the UN Framework Convention on Climate Change) to an overall emission reduction of 5.2 percent. A threshold for releases to air of 50 kg/year was established in 2009.

The main use of sulphur hexafluoride was in high voltage electrical switchgear, largely replacing PCBs. It had also been used as an [inert gas](http://en.wikipedia.org/wiki/Inert_gas) for the casting of magnesium, and as an inert filling for [insulated glazing](http://en.wikipedia.org/wiki/Insulated_glazing) windows. It has also been used as a tracer in air dispersion trials and in leak testing, including water supply. Tiny amounts of sulphur hexafluoride are introduced into the leaking main with compressed air and using a very sensitive detector the area can be surveyed in minutes to locate the leak. With the leak found, only one excavation needs to be made.

It is used in small quantities in echocardiography and Doppler diagnostic testing.

#### 2 From the treatment processes

No known sources.

### Form and fate in the environment

In its normal state, SF6 is chemically inert, non-toxic, non-flammable, non-explosive, and thermally stable (it does not decompose in the gas phase at temperatures less than 500°C).

Sulphur hexafluoride is extremely inert and extremely long-lived; it is inert in the troposphere and stratosphere where it has an estimated [atmospheric lifetime](http://en.wikipedia.org/wiki/Atmospheric_lifetime) of  
800–3,200 years. Average global SF6 concentrations increased by about 7 percent per year during the 1980s and 1990s, mostly as the result of its use in the magnesium production industry, and by electrical utilities and electronics manufacturers.

Water solubility is about 40 mg/L. It evaporates from water (half-life about 3–4 hours). Without evaporation, the half-life in water and soil is about 1,000 years.

### Typical concentrations in drinking water

Very low concentrations may be found in groundwater, where it can be used to help determine the age of the groundwater. See section 4.5 of the DWSNZ, and Chapter 3 of the *Guidelines for Drinking-water Quality Management in New Zealand*. The same principles allow an estimate of the groundwater component of stream flows (eg, Stewart et al 2007).

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Sulphur hexafluoride is described as being “non-toxic”.

### Derivation of Maximum Acceptable Value

No MAV.

### Bibliography

Stewart MK, Mehlhorn J, Elliot S. 2007. Hydrometric and natural tracer (oxygen-18, silica, tritium and sulphur hexafluoride) evidence for a dominant groundwater contribution to Pukemanga Stream, New Zealand. *Hydrol Process* 21: 3340–56. <http://www.google.co.nz/url?q=http://www.aquiferdynamics.com/downloads/Pukemanga %2520HP07.pdf&sa=U&ei=xfqLTbexOI3UtQPAhZn0CA&ved=0CBYQFjABOFo&usg=AFQjCNHcw72Mn0SmLN0ricpQGCqXG-XNcg>

# Thallium

### Maximum Acceptable Value

There is no MAV in the DWSNZ for thallium, and it is not mentioned in the WHO Guidelines.

The USEPA (2009a/2011) has a maximum contaminant level (MCL) for thallium of 0.002 mg/L, based on hair loss, due to changes in blood chemistry, and to kidney, liver and testicular problems. The 2011 Australian Drinking Water Guidelines do not have a guideline value for thallium.

Thallium is on the USEPA List of Priority Pollutants.

### Sources to drinking-water

#### 1 To source waters

Thallium is present in the environment as a result of natural processes and from man-made sources. It is ubiquitous in nature (mostly in association with [potassium](http://en.wikipedia.org/wiki/Potassium)-based [minerals](http://en.wikipedia.org/wiki/Mineral) in [clays](http://en.wikipedia.org/wiki/Clay), [soils](http://en.wikipedia.org/wiki/Soil), and [granites](http://en.wikipedia.org/wiki/Granite)), but also occurs in a more concentrated form in sulfide ores of various heavy metals (eg, zinc, copper, iron, arsenic and lead (IPCS 1996) (USGS).

There are only a few areas with a naturally very high thallium concentration. Several thallium minerals, containing 16 percent to 60 percent thallium, occur in nature as sulfide or selenide complexes with antimony, arsenic, copper, lead, and silver but are rare and have no commercial importance as sources of this element.

Thallium forms compounds in both the monovalent (thallous) and trivalent (thallic) states; however, the monovalent state is the more stable in aqueous solutions. Many of the thallium salts are soluble in water with the exception of thallium (III) oxide, which is insoluble. Thallium occurs naturally in the earth’s crust, with a crustal abundance of approximately 1 mg/kg. In soil, thallium concentrations are on the order of 0.1 to 1 mg/kg; higher concentrations occur in the vicinity of metallic ore deposits. Measureable concentrations of thallium are also found in marine water, freshwater, and air. Thallium is taken up by vegetation, with the extent of uptake determined by soil acidity and plant species.

The greatest use of thallium is in specialised electronic research equipment. It may leach to water from ore-processing sites, or discharges from the electronics industry (primarily for the semiconductor industry), specialist glasses and drug factories. It can enter the atmosphere from smelters, brickworks, cement factories and coal burning (associated with the fly ash). Traces have been associated with copper, gold, zinc, and cadmium mining/refining. Thallium radioisotopes are used in medicine for scintigraphy of certain tissues and the diagnosis of melanoma.

Up until 1972 thallium sulphate was used as a rat poison, but was then banned because of its potential harm to man; grain baits contained from 1 to 2 percent. Due to its ability to remove hair, thallium (I) sulfate was used in the past as a depilatory agent. Thallium (I) sulfate was once used in medicine to treat infections, such as venereal diseases, ringworm of the scalp, typhus, tuberculosis, and malaria. WHO (1975) describes thallium sulphate (also called thallous sulphate), Tl2SO4, as a rodenticide, with insecticide as a secondary use (mainly ants and cockroaches).

In areas not contaminated by thallium, concentrations in water are usually <0.001 mg/L, and those in water sediments <1 mg/kg.

Geothermal waters in the Kawerau area can contain up to 0.005 µg/L Tl (GNS 2015).

Pewter hip flasks have been observed to release thallium to fluids; WQRA (2009) reports 0.022 mg/L being found in water.

#### 2 From the treatment processes

No known sources.

### Form and fate in the environment

Thallium tends to bind to alkaline soils, clay soils and soils rich in organic matter, but may otherwise migrate to groundwater; in some strongly acid soils significant amounts of thallium can be leached to local ground and surface water. Thallium sulphate is a cumulative soil sterilant; it is extremely phytotoxic and inhibits germination of seeds.

Apart from the oxide, thallium compounds are very soluble, the least soluble being thallium iodide at 6 mg/L; others are 100s of mg/L; for example the solubility of thallium sulphate is about 4.8 percent.

Most dissolved thallium in freshwater is expected to be in the monovalent form. However, in strongly oxidised fresh water and most seawater trivalent thallium may predominate. Thallium can be removed from the water column and accumulate in sediment by various exchange, complexation or precipitation reactions.

### Typical concentrations in drinking water

A survey of tap water from 3,834 homes in the United States detected thallium in 0.68 percent of samples at an average thallium concentration of 0.89 μg/L, or 0.0009 mg/L (ATSDR 1992).

782 water utilities in the US reported detecting thallium (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.005 mg/L.

The maximum concentration found in 7451 samples from 2413 groundwaters in the UK was 0.024 mg/L, mean 0.0007 mg/L (DWI 2008).

Because thallium can occur naturally in conjunction with arsenic, copper and lead, four water samples were collected in the Coromandel area in 2012; they contained less than the detection limit of 0.0005 mg/L.

### Removal methods

The following treatment methods have been approved by the USEPA for removing thallium from drinking-water: activated alumina and ion exchange.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Thallium builds up in fish and shellfish, so food is the commonest route for exposure. It has been estimated that the average person eats, on a daily basis, two parts thallium per billion parts (ppb) of food. Thallium is readily absorbed from the gastrointestinal tract and is also absorbed through the intact skin. ICPS (1990) reports the normal level in urine is 1.3 mg/L. People who smoke have twice as much thallium in their bodies as do non-smokers (ATSDR 1992).

Birth defects were not reported in the children of mothers exposed to low levels from eating vegetables and fruits contaminated with thallium. Studies in rats, however, exposed to high levels of thallium, showed adverse developmental effects. It is not known if breathing or ingesting thallium affects human reproduction. Studies showed that rats that ingested thallium for several weeks had some adverse reproductive effects. Animal data suggest that the male reproductive system may be susceptible to damage by low levels of thallium.

Both potassium and thallium are monovalent cations with similar atomic radii; thallium has been shown to mimic the biological actions of potassium (USEPA 2009). Toxic effects of thallium in test animals include degenerative changes in mitochondria of the kidneys, liver, brain and intestines. A NOAEL of 0.2 mg/kg/day was identified from a 13‑week dietary study in which rats were given approximately 0.008 to 0.20 mg thallium/kg/day. Significant increases in blood levels of glutamic-oxaloacetic transaminase (SGOT), lactic dehydrogenase (LDH), and sodium levels were reported.

USEPA (2009) states that there is “inadequate information to assess the carcinogenic potential” for thallium and thallium compounds.

The reference dose or RfD (USEPA 2006) was 0.00007 mg/kg/d and the Drinking Water Equivalent Level or DWEL (USEPA 2006) was 0.002 mg/L. USEPA (2009a) states that because the available toxicity database for thallium studies are generally of poor quality, an RfD for soluble thallium salts is not derived in this specific case. No RfD or DWEL are listed in USEPA (2011).

### 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 groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for thallium salts is 0.0006 mg/L.

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WQRA. 2009. *HealthStream*, March issue. <http://www.wqra.com.au>.

# Thorium

### Maximum Acceptable Value

There is no MAV in the DWSNZ for thorium, and it is not mentioned in the WHO Guidelines. The thorium level in drinking-water is controlled via the radiochemical MAVs.

### Sources to drinking-water

#### 1 To source waters

Thorium is a naturally-occurring, radioactive metal. Small amounts of thorium are present in all rocks, volcanic discharges, soil, above-ground and underground water, plants, and animals. These small amounts of thorium contribute to the weak background radiation for such substances. Soil commonly contains an average of about 6 mg/kg.

More than 99.9 percent of natural thorium exists in the form (isotope) thorium-232. Besides this natural thorium isotope, there are more than 10 other different isotopes that can be artificially produced. In the environment, thorium-232 exists in various combinations with other minerals, such as silica.

The decay of thorium-232 produces a small part called “alpha” radiation and a large part called the decay product. The decay product breaks down to an unstable isotope and the process continues until a stable product is formed. During these decay processes, the parent thorium-232, its decay products, and their next decay products produce a series of new substances (including radium and radon), alpha and beta particles, and gamma radiation. The alpha particles can travel only very short distances through most materials and cannot go through human skin. The gamma radiation can travel farther and can easily go through human skin. The decay of thorium-232 into its decay products happens very slowly; it takes about 14 billion years for half the thorium-232 to change into new forms.

Thorium is used to make ceramics, lantern mantles, and metals used in the aerospace industry and in nuclear reactions. Thorium can also be used as a fuel for generating nuclear energy. More than 30 years ago thorium oxides were used in hospitals to make certain kinds of diagnostic x-ray photographs.

#### 2 From the treatment processes

No known sources.

### Form and fate in the environment

Water soluble thorium compounds include the chloride, fluoride, nitrate, and sulfate salts. These compounds dissolve fairly readily in water.

### Removal methods

The concentrations of dissolved thorium in water with high pH (more than 8) are expected to be very low; the concentration may increase with decreasing pH. Thorium-232 concentrations rarely exceed 0.1 pCi/L in natural waters. In a natural surface water in Austria, the concentration of thorium was reported to be 1.24 to 2.90 μg/L.

The transport of thorium in water is principally controlled by the particle flux in the water, ie, most of the thorium will be carried in the particle-sorbed state; hence treatment processes that remove particulate matter will reduce its concentration.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Since thorium is found almost everywhere, it can be detected in food and water. The main way thorium will enter the body is by breathing dust contaminated with thorium.

Thorium is radioactive and may be stored in bone for a long time, so bone cancer is a potential concern for people exposed to thorium. Liver diseases and effects on the blood have been found in people injected with thorium in order to take special x-rays.

ATSDR (1990) reports a study which estimated that the daily intake of thorium through food, water, and inhalation was 2.29 μg /day, with the majority from food and water ingestion (2.27 μg/kg). However, it was determined that, since absorption through the gastrointestinal tract is so low (0.02 percent), two-thirds of the body burden of thorium results from inhalation exposure.

### Derivation of Maximum Acceptable Value

No MAV.

### Bibliography

ATSDR. 1990. *Toxicological Profile for Thorium*. Agency for Toxic Substances and Disease Registry, Division of Toxicology. <http://www.atsdr.cdc.gov/toxprofiles/tp147.html>.

# Tin

### Maximum Acceptable Value

No health based MAV is proposed for inorganic tin in drinking-water because it is not hazardous to human health at concentrations normally found in drinking-water.

The 2000 DWSNZ stated: based on health considerations, the concentration of tin in drinking water should not exceed 1 mg/L; this was a provisional MAV, and related to just inorganic tin.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that no guideline value is considered necessary for tin in drinking water, as concentrations are likely to be considerably lower than the level that can cause health effects; tin is one of the least toxic metals.

Refer also to datasheets for dialkyl tin and tributyltin oxide (organic chemical listings).

### Sources to drinking-water

#### 1 To source waters

Tin may enter the aquatic environment via the weathering of igneous rocks that contain the mineral cassiterite or tinstone. The major anthropogenic sources of inorganic tin to the environment are discharges from mining, refineries, food processing and packaging plants, steel manufacture and construction.

The concentration of tin in seawater is about 0.001 mg/L.

Most tin is used for metal plating and in alloys. Metallic tin appears in brass, bronze, pewter, and some soldering materials. Inorganic tin can be found in toothpaste (stannous fluoride), perfumes, soaps, colouring agents, food additives, and dyes. Organic tin can be found in or on plastics, food packages, plastic pipes, pesticides, paints, wood preservatives, and rodent (rats and mice) repellants. Municipal sewage, containing tin from discarded household products, may discharge tin into the environment. Tin(II) fluoride is broadly used in preventive dentistry.

Tin occurs naturally as the stable isotopes 112Sn (0.97 percent), 114Sn (0.65 percent), 115Sn (0.36 percent), 116Sn (14.5 percent), 117Sn (7.7 percent), 118Sn (24.2 percent), 119Sn (8.6 percent), 120Sn (32.6 percent), 122Sn (4.6 percent), and 124Sn (5.8 percent); quoted in WHO (2005).

#### 2 From the treatment processes

No known sources.

#### 3 From the distribution system

Tin may enter drinking-water through the dissolution of (mainly) antimony-tin solder, and also copper-tin or silver-tin solders. These replaced the previously common lead-tin solder (50:50) during the 1980s. Some water tanks are tinned (on the water side).

### Form and fate in the environment

Tin can exist in several oxidation states, but in the aquatic environment will usually exist in the +4 (stannic) oxidation state. Divalent tin (stannous) may occur in anaerobic sediments and waters of low redox potential. Divalent tin compounds are soluble in water, but tin is usually oxidised to the less soluble +4 state. The most likely forms of +4 tin in natural water include the hydroxides and hydroxyoxides. In general, tin concentrations increase as follows: water < surface microlayer < sediments, ie, tin compounds are likely to partition to soils and sediments. Tin may be methylated in aqueous in the presence of some micro-organisms. The concentration of tin in rivers, estuaries and oceans is generally less than 5 ng/L (0.000005 mg/L).

### Typical concentrations in drinking water

Tin was not routinely monitored in New Zealand drinking-water supplies and therefore no information is available about its concentration. WHO (2011) states that for the general population, drinking-water is not a significant source of tin, and levels in drinking-water greater than 0.001 to 0.002 mg/L are exceptional.

The P2 Chemical Determinand Identification Programme, sampled from 897 zones, found tin concentrations to range from “not detectable” (nd) to 0.048 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 tin at greater than 50 percent of the MAV (ESR 2001).

A mean range of 0.001–0.002 mg/L (maximum 0.03 mg/L) was found in a survey of water supplies in the USA. Values greater than 0.002 mg/L are exceptional.

Twenty-four water utilities in the US reported detecting tin (total) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.075 mg/L.

WHO (2017) states that levels in drinking-water greater than 1–2 μg/L are exceptional.

### Removal methods

Treatment of drinking-water to reduce the concentration of inorganic tin is unlikely to be required.

### Analytical methods

#### Referee method

A referee method cannot be selected for tin 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 tin for the above reason. However, the following methods are used to analyse for tin:

1. Flame Atomic Absorption Spectrometric Method (APHA 3111B)

The detection limit for tin by flame AA is 0.8 mg/L.

2. Furnace Atomic Absorption Spectrometric Method (APHA 3113B).

The detection limit for tin by furnace AA is 0.005 mg/L.

### Health considerations

There is no evidence that tin is an essential element for humans although it is thought to be an essential element in animals.

Food, particularly canned food, represents the major route of human exposure to tin. Intake from this source varies widely and for some segments of the population can reach several milligrams per kilogram of body weight. Food from unlacquered tin-lined cans contains up to 100 ppm of tin because the reaction of the food with the can causes some of the tin to dissolve in the contents of the can. Greater than 90 percent of tin-lined cans used for food today are lacquered. Only light coloured fruit and fruit juices are packed in unlacquered tin-lined cans, because tin helps maintain the colour of the fruit. Tin concentrations in food also increase if food is stored in opened cans. The main adverse effect on humans of excessive levels of tin in canned beverages (above 150 mg/kg) or other canned foods (above 250 mg/kg) has been acute gastric irritation. There is no evidence of adverse effects in humans associated with chronic exposure to tin.

The low toxicity of tin and inorganic tin compounds is largely the result of its low absorption, low tissue accumulation and rapid excretion, primarily in the faeces. JECFA (1989) established a PTWI of 14 mg/kg of body weight from a TDI of 2 mg/kg of body weight on the basis that the problem with tin is associated with acute gastrointestinal irritancy, the threshold for which is about 200 mg/kg in food. This was reaffirmed by JECFA in 2000 (WHO 2004a).

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) to inorganic tin.

As at July 2013 ATSDR quotes a minimal risk level (MRL) of 0.005 mg/kg/day for intermediate-duration oral exposure (15–364 days) to dibutyltin dichloride.

For tributyltin oxide they developed:

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).

Tin and inorganic tin compounds are poorly absorbed from the gastrointestinal tract, do not accumulate in tissues, and are rapidly excreted, primarily in the faeces. No increased incidence of tumours was observed in long-term carcinogenicity studies conducted in mice and rats fed tin(II) chloride. Tin has not been shown to be teratogenic or fetotoxic in mice, rats or hamsters. In rats, the NOAEL in a long-term feeding study was 20 mg/kg body weight per day (WHO 2011/2017).

### Interpretation of Maximum Acceptable Value

Because of the low toxicity of inorganic tin, a tentative guideline value could be derived three orders of magnitude higher than the normal tin concentration in drinking-water. Therefore, the presence of tin in drinking-water does not represent a hazard to human health. For this reason, the establishment of a numerical guideline value for inorganic tin is not deemed necessary.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for tin is 4 mg/L.

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# Titanium

### Maximum Acceptable Value

There is no MAV in the DWSNZ for titanium, and it is not mentioned in the WHO Guidelines.

### Sources to drinking-water

#### 1 To source waters

Titanium dioxide, including TiO2 nanoparticles, is used extensively in paints and paper as a pigment, in cosmetics, suncreams and surface coatings. TiO2 is used in some foods as a whitener or filler. Its wide usage and insolubility result in titanium dioxide being one of the major components of sewage sludge.

Being a photocatalyst, it can also be used in conjunction with UV to disinfect water. Titanium dioxide (TiO2) nanoparticles, like the regular/bulk form of TiO2, exist in three crystalline structures or polymorphs: rutile, anatase and brookite. They can vary in size and shape, can form agglomerates or aggregates and be coated with other materials. Brookite TiO2 is less common than rutile or anatase TiO2. Samples of TiO2 nanoparticles may contain more than one polymorphic form. All polymorphs of TiO2 have low water solubility. Hazards and biological activity of TiO2 nanoparticles may vary with its crystalline form (eg, anatase TiO2 is considered more reactive than rutile TiO2); particle size; particle shape; agglomerate/aggregate status; surface properties; and type of coating (NICNAS 2013).

The concentration of titanium in seawater is about 0.001 mg/L.

#### 2 From the treatment processes

Titanium, usually as titanium dioxide, is often an impurity in iron salts used for coagulation.

### Form and fate in the environment

Titanium dioxide is very insoluble so will be a very minor component of water.

### Typical concentrations in drinking water

Titanium was not routinely monitored in New Zealand drinking-water supplies and therefore no information is available about its concentration. WHO (2011) states that for the general population, drinking-water is not a significant source of titanium, and levels in drinking-water greater than 0.001 to 0.002 mg/L are exceptional.

### Removal methods

Any treatment process that removes particulate matter should reduce the concentration of titanium dioxide.

### Analytical methods

#### Referee method

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

### Health considerations

Titanium is not a dietary requirement; our daily intake is approximately 0.8 mg, and only a small part of the total daily intake is absorbed by the body.

Studies in rodents showing systemic absorption of TiO2 nanoparticles through the gastrointestinal tract, based on doses ~5000 mg/kg bw, are not likely to reflect realistic human exposures. Repeated dose oral studies indicated bioavailability through the gastrointestinal tract, although the extent of absorption/bioavailability was not quantified. Studies on rats reported accumulation in the liver, spleen, lung and peritoneal tissues for 500 nm particles. For particles <50 nm, accumulation occurred in the liver and heart. Information on the adverse effects of this accumulation in repeated dose oral studies was limited.

Accumulation of anatase TiO2 nanoparticles (~5 nm) in the liver of mice was reported following intragastric administration of 10 and 50 mg/kg bw doses for 60 days. The accumulated particles in the liver induced fatty degeneration, necrosis and some inflammatory cell infiltration and hepatocyte apoptosis. The liver toxicity in mice was triggered by altering the expression levels of genes and their proteins involved in the signalling pathway and reduction of immune capacity (NICNAS 2013).

### Interpretation of Maximum Acceptable Value

No MAV.

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# Trichloramine

CAS No. 10025-85-1. Also called nitrogen trichloride and chlorine nitride.

There are insufficient data to establish a health-based MAV for trichloramine. Taste and/or odour will be objectionable to most people at a much lower concentration than any health-based value.

Trichloramine has not been extensively studied, and available data are inadequate to permit derivation of health-based guideline value (WHO 2017).

### Sources to drinking-water

#### 1 To source waters

Chloramines may be present in source waters as a result of their discharge from industries in which they are used (rarely). Their principal use is as intermediates in the manufacture of hydrazine.

#### 2 From treatment processes

Trichloramine can be formed in chlorinated water that contains ammonia and some organic nitrogen compounds. The concentration depends upon the pH and chlorine to nitrogen ratio. Ammonia may be intentionally added to the water to produce the chloramines as disinfectants. See Chapter 15.

Trichloramine is formed in acid solutions where chlorine concentrations are much greater than those of ammonia. At these high chlorine concentrations and at pH values <3, trichloramine is the only chloramine present. Nitrogen trichloride occurs in diminishing proportions at chlorine-to-ammonia mole ratios >2 and pH values of <7.5. At pH >7.5, no trichloramine is found, regardless of the ratio of chlorine to ammonia. In aqueous solutions at neutral pH it decomposes slowly to ammonia and hypochlorous acid by an autocatalytic pathway (USEPA 1994).

#### 3 From the distribution system

It is possible that reactions of chlorine with nitrogenous material in the distribution system may produce chloramines. Trichloramine is more likely to form at low pH.

### Typical concentrations in drinking-water

No typical value data are available for New Zealand. Chloramination is not used intentionally at present as a disinfectant in New Zealand, and the concentrations of inorganic chloramines present in waters depends upon the concentrations of inorganic and some organic nitrogen compounds present in the raw water, and control of the chlorination process.

### Removal methods

Chemical reducing agents, including sodium thiosulphate, sulphur dioxide, and sodium bisulphite can be used to remove trichloramine. Activated carbon adsorbs trichloramine.

Trichloramine in water will not be present intentionally because of its irritant properties. Although mono- and dichloramine are volatile to some extent, trichloramine is very much more so and may be readily stripped from water by aeration. As dichloramine also has some undesirable properties, any loss of it during the process may also be welcomed.

Loss of trichloramine can be expected from waters exposed to sunlight.

### Analytical methods

#### Referee method

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

#### Some alternative methods

1. DPD Ferrous Titrimetric Method (APHA 4500-Cl F).

The limit of detection for this method is approximately 0.2 mg/L for field use, although lower levels can be determined under laboratory conditions and with care. Analytical texts indicate that by manipulation of the conditions of the analysis measurement of monochloramine, dichloramine and trichloramine can be made.

2. Amperometric Titration Method (APHA 4500-Cl D and E).

While more accurate than the DPD methods, expensive equipment and a high degree of skill and care are required for this method. The limit of detection is better than 0.1 mg/L, or less if using APHA 4500-Cl E. The APHA method describes variations that will allow the determination of monochloramine and dichloramine. Interferences due to organic chloramines may also cause interferences with these methods.

3. DPD Colorimetric Method (APHA 4500-Cl G)

This method requires a spectrophotometer for the colorimetric measurements, although hand-held comparators do offer a cheaper, though less reliable variation for field use. The limit of detection (LOD), with instrumental assistance, is approximately 0.1 mg/L.

The LOD for the comparator depends on the colour disc in use. Chloramine concentrations as low as 0.2 mg/L approximately should be detectable, but the accuracy of the method depends upon use of the correct lighting (natural lighting should be used with the sun behind the viewer), the individual’s ability to match colours and judge their intensity, and ensuing that readings are taken as soon after colour development as possible. The LOD may be about 0.10 mg/L when using a Nessleriser.

Note: These methods are of use when ammonia only is in the water being chlorinated. In most natural waters nitrogen-containing organic compounds are also present. Organic chloramines are formed from these compounds when chlorine reacts with them. Organic chloramines also produce colour during the DPD test and make attempts to differentiate between the different inorganic chloramines of little value. Unless investigating taste and odour problems, it is recommended that only the total combined chlorine, ie, total chloramine concentration, is reported.

These methods measure trichloramine in terms of mg Cl as Cl2/L. See monochloramine sheet for conversion.

### Health considerations

Studies have revealed equivocal evidence of carcinogenic activity of chloraminated drinking-water in female rats, as indicated by an increase in incidence of mononuclear cell leukaemia.

Epidemiological studies did not report an association between ingestion of chloraminated drinking-water and increased urinary bladder mortality rates in humans.

When tap-water containing chloramines was used for dialysis, acute haemolytic anaemia, characterised by denaturation of haemoglobin and lysis of red blood cells, was reported in haemodialysis patients.

Trichloramine is an eye irritant, and will also irritate breathing passages. It is believed to be responsible for breathing difficulties experienced by asthmatics in some swimming pools. Its odour has been reported to be detectable in water as low as 0.02 mg/L, and it has been described as “geranium”.

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# Uranium

### Maximum Acceptable Value (Provisional)

The guideline value in WHO (2011/2017) is 0.03 mg/L. This guideline value is designated as provisional because of scientific uncertainties surrounding uranium toxicity. A MAV based on a 70 kg body weight would also be 0.03 mg/L.

The 2008 DWSNZ PMAV stated: Based on health considerations, the concentration of uranium in drinking water should not exceed 0.02 mg/L.

The new WHO provisional guideline value of 0.03 mg/L, which is derived from new epidemiological studies on populations exposed to high uranium concentrations, replaces the previous value derived from experimental animal studies and designated as provisional on the basis of uncertainties regarding the toxicology and epidemiology of uranium as well as difficulties concerning its technical achievability in smaller supplies. It is noted that studies on human populations, when available and of good quality, are the preferred source of health-related information to be used in deriving guideline values (WHO 2011).

In the 2000 DWSNZ, the PMAV for uranium was 0.002 mg/L. The 1995 DWSNZ had no MAV, the datasheet saying that there were insufficient data to set a health-based MAV for uranium in drinking-water.

The WHO considers a guideline value of up to 0.03 mg/L may be protective of kidney toxicity because of uncertainty regarding the clinical significance of changes observed in epidemiological studies. The MAV derived from chemical toxicity data is also protective of radiological effects. This is because it gives off very small amounts of radiation.

The maximum contaminant level (USEPA 2009/2011) is 0.03 mg/L. The maximum acceptable concentration in Canada is 0.02 mg/L.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the concentration of uranium in drinking water should not exceed 0.017 mg/L.

### Sources to drinking-water

#### 1 To source waters

Uranium occurs naturally in the +2, +3, +4, +5 and +6 valence states, but it is most commonly found in the hexavalent form. In nature, hexavalent uranium is commonly associated with oxygen as the uranyl ion, UO22+. Naturally occurring uranium is a mixture of three radionuclides (234U, 235U and 238U), all of which decay by both alpha and gamma emissions. Natural uranium consists almost entirely of the 238U isotope (by weight, natural uranium is about 0.01 percent 234U, 0.72 percent 235U, and 99.27 percent 238U). Uranium is widespread in nature, occurring in granites and various other mineral deposits.

Uranium can enter water naturally from the weathering of rocks, minerals and phosphate fertilisers. Geologically, uranium is a very mobile element. In New Zealand, uranium occurs in the non-marine Ohika Beds and Hawks Crag Breccia adjacent to the Paparoa Range of southwest Nelson. Uranium is also present in several granites and diorites on the West Coast.

The concentration of uranium in seawater is about 0.003 mg/L.

Groundwaters often have higher concentrations of uranium than surface waters because of the large solid/solution ratios in aquifers and the greater influence of water-rock interactions. Uranium occurs as a major constituent of minerals such as uraninite, coffinite and autunite. These can be significant localised sources of U in some groundwaters, especially those in mineralised areas and some granitic terrains. Uranium is also closely associated with iron oxides, phosphates, clays and organic matter and these minerals can be important sources, as well as sinks, of uranium. The concentrations of uranium in abundant silicate minerals such as quartz and feldspar and carbonate minerals are usually low.

DWI (2006) states that:

As the oxygenated groundwater permeates the aquifer, a redox front is created between the oxidising and reducing zones. Uranium dissolves in the oxidising zone but is immobilised at the redox front and so precipitates as uraninite (or in some cases coffinite). The uranium front moves in the direction of groundwater flow, but at a much slower rate than the water itself. The accumulation of uranium at the front can lead to the development of economic deposits. High concentrations of dissolved V, Mo and Se can accompany uranium at the roll front.

Uranium may also be discharged to water in wastes from industries in which it is used. Uranium is used in radio emissions shielding material, photographic emulsions, porcelains used in dentistry and optical lenses. In addition, alloys may contain uranium, and certain uranium compounds are used as catalysts in the chemical industry. Uranium is used as a radioactive tracer. Uranium is present in the environment as a result of leaching from natural deposits and release from mill or mine tailings, and can result from combustion of coal and phosphatic fertilisers.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

The mobility of uranium in water is controlled by a number of factors, among the most important being pH, redox status and concentrations of coexisting dissolved ions. Uranium is a redox-sensitive heavy metal that occurs in water principally under oxic conditions in its hexavalent U(VI) or uranyl form. It is usually complexed in solution, especially with carbonate ligands, but also less significantly with phosphate, fluoride or sulphate depending on their respective dissolved concentrations and ambient pH. Under anoxic conditions, uranium is reduced to its tetravalent U(IV) uranous state and its concentration in water is usually low as a result of stabilisation of the sparingly soluble mineral, uraninite.

Under conditions typical of natural waters, adsorption to hydrous ferric oxide in aerobic waters plays an important role in removing uranium from the aquatic environment.

### Typical concentrations in drinking-water

Uranium was not routinely monitored in New Zealand drinking-water supplies as part of the Department of Health three-yearly survey.

The P2 Chemical Determinand Identification Programme, sampled from 828 zones, did not find uranium at detectable concentrations (Limit of detection = 0.0005 mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with uranium at greater than 50 percent of the MAV, which at the time was 0.002 mg/L (ESR 2001).

WHO (2004) states that levels in drinking-water are generally less than 0.001 mg/L; concentrations as high as 0.7 mg/L have been measured in private supplies in Canada. Concentrations in excess of 0.02 mg/L have been reported in groundwater from parts of New Mexico and central Australia.

2,830 water utilities in the US reported detecting combined uranium (in mg/L) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.089 mg/L.

3,122 water utilities in the US reported detecting combined uranium (as pCi/L) in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 60 pCi/L.

The maximum concentration found in 8062 samples from 2,484 groundwaters in the UK was 0.07 mg/L, mean 0.0014 mg/L (DWI 2008).

USEPA (2018) states that in most areas of the United States, low levels of uranium are found in drinking water, with a population mean concentration of about 1 μg U/L. Higher levels of uranium are seen in water from wells in uranium-rich rock. Approximately 4 percent of reporting US drinking water systems (serving 8 million people in total) reported some exceedance of the EPA maximum contaminant limit (MCL) for uranium of 30 μg/L.

### Removal methods (ex WHO 2005)

A review of treatment technologies for uranium removal reported the following removals:

coagulation/filtration at high pH (10+): >95 percent

lime softening: 85 – 99 percent

anion exchange: 99 percent

reverse osmosis: >95 percent

Coagulation at pH 10 using aluminium salts would not be feasible owing to the high solubility of aluminium, but 80 – 89 percent removal of uranium can be achieved by aluminium coagulation at pH 6.

Another review gave the following removals:

coagulation/filtration: 80 – 89 percent

lime softening: 85 – 99 percent

anion exchange: 90 – 100 percent

reverse osmosis: 90 – 99 percent

activated alumina: 90 percent

### Analytical methods

#### Referee method

Inductively coupled plasma – mass spectrometry (APHA 3125).

#### Some alternative methods

1. Fluorimetry (ASTM D2907-91).

These test methods are applicable to the determination of uranium in water in the concentration range 0.005 to 50 mg/L. If interfering ions are present (small quantities of cadmium, chromium, cobalt, copper, iron, manganese, nickel, lead, platinum, silicon, thorium and zinc), an extraction method must be used. The direct method has a concentration range 0.005 to 2 mg/L, and the extraction method has a concentration range 0.04 to 50 mg/L. A fluorimeter is required having an excitation wavelength from 320 to 370 nm and measuring emission at 530 to 570 nm.

2. Colorimetric methods are available, but tend to be prone to interferences.

### Health considerations

#### Chemical toxic effects

Intake of uranium through air is low, and it appears that intake through food (highest concentrations are found in shellfish) is between 1 and 4 μg/day. For naturally occurring uranium, chemical toxicity effects become a potential health concern at lower concentrations than radiation related effects.

Intake through drinking-water is normally extremely low. Absorption of dietary uranium by the gastro-intestinal tract has been estimated to be less than 1 percent. Highest uranium concentrations occur in the kidney and bone, with little in the liver. The overall biological half-life of uranium has been estimated at 6–12 months.

There is no evidence that uranium is essential to man. In humans, the main toxic chemical effect of short-term exposure to high concentrations of uranium is inflammation of the kidney (nephritis). Little information is available on the effects of long-term exposure. One study (Health Canada 2001), where 324 people drank contaminated water from wells with uranium concentrations up to 0.7 mg/L, reported no increase in the incidence of kidney disease or any other symptomatic complaint.

In a number of studies carried out in rats, rabbits and dogs, most report that uranium has an effect on the kidney, although there are significant differences between species.

Depending on the chemical form of uranium and circumstances of intake, about 0.1−6 percent of ingested uranium is absorbed by the gastrointestinal tract and enters the systemic circulation in humans, with soluble uranium compounds being more readily absorbed. Urinary excretion is the principal elimination pathway for absorbed uranium. Absorbed uranium is retained in many organ systems, with the highest levels found in the bones, liver, and kidneys. It is estimated that 66 percent of the typical human body burden of uranium is found in the skeleton. Uranium in the skeleton is retained for a longer period, with a half-life on the order of 70−200 days; most of the uranium in other tissues leaves the body in 1−2 weeks following exposure (USEPA 2018).

No data are available on mutagenic effects in relation to uranium.

The oral reference dose or RfD for soluble uranium (USEPA 2009/2011) is 0.0006 mg/kg/d. The Drinking Water Equivalent Level or DWEL (USEPA 2009/2011) is 0.02 mg/L. The oral RfD had earlier been 0.003 mg/kg/d (USEPA 1989).

As at July 2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quotes a minimal risk level (MRL) of 0.002 mg/kg/day for acute-duration oral exposure (1–14 days), and 0.0002 mg/kg/d for intermediate-duration oral exposure (15–364 days) to soluble uranium salts.

The livestock guideline value is 0.2 mg/L (ANZECC/ARMCANZ 2000). These guidelines were to be updated in 2012.

#### Radiological effects

In natural uranium, about 48.9 percent of the radioactivity is associated with 234U, about 2.2 percent is associated with 235U, and 48.9 percent is associated with 238U.

Studies have shown high specific activity uranium isotopes to be carcinogenic in animals, causing malignant tumours in mice and bone sarcomas in rats. Similar studies using natural uranium (U-238) have not shown similar effects, possibly due to the lower radiation doses involved. Epidemiological data are inadequate to show whether exposure to uranium in drinking-water will lead to an increased risk of cancer.

### Derivation of Maximum Acceptable Value

WHO (2011) states that the provisional guideline value of 0.03 mg/L is based on a TDI of 60 μg, derived from the lower 95 percent confidence limit on the 95th percentile uranium exposure distribution in a study from Finland, using an uncertainty factor of 10 for intraspecies variation.

WHO (2012) included: Based on developmental effects in pups, a no-observed-effect level (NOEL) for uranium of 2.8 mg/kg body weight per day was established.

The MAV in the 2008 DWSNZ for uranium in drinking-water had been derived as follows:

There are insufficient data regarding the carcinogenicity of uranium in humans and experimental animals. Therefore a TDI of 0.06 mg/kg of body weight per day was used, based on the application of an uncertainty factor of 100 (for inter- and intraspecies variation) to a LOAEL (equivalent to 0.06 mg of uranium per kg of body weight per day) for degenerative lesions in the proximal convoluted tubule of the kidney in male rats in a 91-day study in which uranyl nitrate hexahydrate was administered in drinking-water. It was considered unnecessary to apply an additional uncertainty factor for the use of a LOAEL instead of a NOAEL and the short length of the study because of the minimal degree of severity of the lesions and the short half-life of uranium in the kidney, with no indication that the severity of the renal lesions will be exacerbated following continued exposure. This is supported by data from epidemiological studies.

0.06 mg/kg body weight per day x 70 kg x 0.8 = 0.0168 mg/L (rounded to 0.02 mg/L)

100 x 2 L per day

where:

* Lowest Observable Adverse Effect Level = 0.06 mg/kg body weight per day
* average weight of an adult = 70 kg
* the proportion of tolerable daily intake assigned to the consumption of water = 0.8
* average amount of water consumed by an adult = 2 L per day.

The 0.002 mg/L PMAV in the 2000 DWSNZ was based on WHO (1998).

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# Vanadium

CAS No. 7440-62-2.

### Maximum Acceptable Value

There is no MAV for vanadium in the DWSNZ, and vanadium is not mentioned in the WHO Guidelines.

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

The legal limit for total vanadium in Italian drinking water is 0.14 mg/L.

### Sources to drinking-water

#### 1 To source waters

Vanadium is widely distributed in the earth’s crust (around 0.05 g/kg). Vanadium is released naturally to air through the formation of continental dust, marine aerosols, and volcanic emissions. Anthropogenic sources include the combustion of fossil fuels, particularly coal, and residual fuel oils which constitute the single largest overall release of vanadium to the atmosphere. These releases are generally in the form of vanadium oxides and contribute approximately two-thirds of atmospheric vanadium. The natural release of vanadium to water and soils occurs primarily as a result of weathering of rocks and soil erosion. This process usually involves the conversion of the less-soluble trivalent form to the more soluble pentavalent form. Deposition of atmospheric vanadium is also an important source both near and far from industrial plants burning residual fuel oils rich in vanadium.

The concentration of vanadium in seawater is about 0.001 mg/L.

Vanadium can exist in a number of different oxidation states: -1, 0, +2, +3, +4, and +5. The most common commercial form is vanadium pentoxide (V2O5). Vanadium occurs in two natural stable isotopes, 50V and 51V.

Three quarters of all vanadium produced is used in the metallurgical industry for the production of special steels up to a concentration of 5 percent. Vanadium has many metallurgical uses; the pentoxide is used as a catalyst in many manufacturing processes, eg, in the production of sulphuric acid by the contact process, in petroleum cracking, and as a catalytic converters for the exhaust gases of internal combustion engines. Vanadium compounds are also used in glass, in glazes and enamels for porcelain and pottery, in lacquers and paints, and as mordants in the dyeing of fabrics. Vanadium compounds can also be used as synthetic rubber additives.

Measurements of vanadium in natural fresh waters such as the Animas, Colorado, Green, Sacramento, San Joaquin, and San Juan Rivers in the US, as well as some fresh water supplies in Wyoming, ranged from 0.0003 to 0.2 mg/L.

#### 2 From treatment processes

No known sources.

#### 3 From the distribution system

No known sources.

### Forms and fate in the environment

The transport and partitioning of vanadium in water and soil is influenced by pH, redox potential, and the presence of particulate matter. In fresh water, vanadium generally exists in solution as the vanadyl ion (V4+) under reducing conditions, and the vanadate ion (V5+) under oxidising conditions, or as an integral part of, or adsorbed on to, particulate matter. There is very limited leaching of vanadium through soil profiles. Most vanadium in surface waters is suspended and ultimately becomes incorporated in sedimentary deposits in oceans.

### Typical concentrations in drinking-water

Several supplies in the US exceed 0.05 mg/L. 670 water utilities in the US reported detecting vanadium in tap water since 2004, according to EWG’s analysis of water quality data supplied by state water agencies, with the highest concentration being 0.096 mg/L.

Arena et al (2015) found the annual mean concentration of total V in groundwater samples from each town around Mt Etna ranged significantly from 0.0156 mg/L to 0.182 mg/L. The pentavalent form ranged from 62.8 percent to 98.9 percent of total V.

As part of the Third Unregulated Contaminant Monitoring Rule (UCMR 3) USEPA tested 62,981 drinking water samples for vanadium between 2013 and 2015, and found 37,954 samples exceeded the minimum reporting level (MRL) of 0.002 mg/L, and 163 samples contained >0.021 mg/L.

### Analytical methods

#### Referee method

No MAV.

### Health considerations

Vanadium compounds have been used therapeutically in human beings for the treatment of various diseases. Vanadium has been given orally in doses of 21–30 mg vanadium/day, as diammonium oxytartarovanadate, for six weeks, in a study on its cholesterol-reducing effects. Vanadium compounds have been applied to the teeth to study their effectiveness in preventing dental caries (IPCS 1990).

V(V) exists as the VO43− ion, which is chemically identical and biologically indistinguishable from the PO43− ion. Thus, in biological systems it can accumulate in bones and teeth, and it inhibits enzymes that process phosphate (PO43−) (Arena 2015).

Pre-2013 ATSDR (<http://www.atsdr.cdc.gov/mrls/mrls_list.html>) quoted a minimal risk level (MRL) of 0.01 mg/kg/day for intermediate-duration oral exposure (15–364 days) to vanadium. This was based on a study that administered 0, 5, 10, or 50 ppm of sodium metavanadate in the drinking water of rats for 3 months. All treated groups showed mild histological changes in kidneys, lungs, and spleen, and the changes became progressively more severe with increased dosages. The 2010 list (and subsequently) reports the MRL as 0.01 mg/kg/day for intermediate-duration oral exposure.

Vanadium is probably essential to enzyme systems that fix nitrogen from the atmosphere (bacteria) and is concentrated by some organisms (tunicates, some polychaete annelids, some microalgae), but its function in these organisms is uncertain.

Estimates of total dietary intake of humans range from 0.01 to 0.03 mg/day. Data are not available to determine target organs in humans from chronic oral or dermal exposure. Vanadium is very poorly absorbed from the gastrointestinal system so it is unlikely to result in a significant internal dose by this route.

Vanadium is not suspected of being a carcinogen.

### Derivation of Maximum Acceptable Value

No MAV.

OEHHA does not concur with the proposed USEPA Notification Level of 0.05 mg/L. OEHHA has determined that use of the 2.1 mg/kg-day LOAEL based on a developmental and reproductive rat study, and the use of a total uncertainty factor of 1,000 are appropriate for deriving a Notification Level for vanadium. Therefore, the public health protective concentration (C) for vanadium of 0.015 mg/L in drinking-water can be derived from the following equation:

C = LOAEL x BW x RSC / UF x DWC

2.1 mg/kg-day x 70 kg x 0.2 / 1,000 x 2 L/day = 0.0147 mg/L = 0.015 mg/L

where:

* LOAEL = lowest-observed-adverse-effect-level
* BW = adult human body weight
* RSC = relative source contribution
* UF = uncertainty factor
* DWC = adult daily drinking water consumption.

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10 percent of a lifetime) for vanadium is 0.05 mg/L.

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