

filtration) or dissolved air flotation—can remove cell-bound ATXs effectively. Soil, slow sand and riverbank filtration can also remove dissolved cyanotoxins. For all these processes, it is important that they are optimized to target the removal of cells and dissolved toxins. Chlorination is not reliably effective, whereas ozonation at sufficiently high doses and contact times is effective for degrading dissolved ATXs; however, elevated organic carbon in bloom situations will substantially increase the disinfectant demand. Chlorine dioxide and chloramine are ineffective for degrading ATXs. Both for pre-oxidation and conventional treatment, cell rupture and toxin release should be avoided. GAC and PAC can be effective for removing dissolved ATXs, with efficacy dependent on several factors, including the type of activated carbon, contact times (PAC), flow rates (GAC) and water quality. As the challenges that blooms present for treatment are complex, periodic validation of efficacy during bloom situations and under the specific local conditions is particularly important. Avoiding bloom occurrence and intake is therefore the preferred option.

Antimony

Elemental antimony forms very hard alloys with copper, lead and tin. Antimony compounds have various therapeutic uses. Antimony is used in solders as a replacement for lead, but there is little evidence of any significant contribution to drinking-water concentrations from this source. Total exposure from environmental sources, food and drinking-water is very low compared with occupational exposure.

Guideline value	0.02 mg/l (20 µg/l)
Occurrence	Concentrations in groundwater less than 0.001 µg/l; concentrations in surface water less than 0.2 µg/l; concentrations in drinking-water appear to be less than 5 µg/l
Tolerable daily intake (TDI)	6 µg/kg body weight, based on a NOAEL of 6.0 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, using an uncertainty factor of 1000 (100 for interspecies and intraspecies variation, 10 for the short duration of the study)
Limit of detection	0.01 µg/l by electrothermal atomic absorption spectrometry (AAS); 0.1–1 µg/l by inductively coupled plasma mass spectrometry (ICP-MS); 0.8 µg/l by graphite furnace AAS; 5 µg/l by hydride generation AAS
Treatment performance	Conventional treatment processes do not remove antimony. However, antimony is not normally a raw water contaminant. As the most common source of antimony in drinking-water appears to be dissolution from metal plumbing and fittings, control of antimony from such sources would be by product control.
Guideline value derivation	
• allocation to water	10% of TDI
• weight	60 kg adult
• consumption	2 litres/day
Assessment date	2003
Principal reference	WHO (2003) <i>Antimony in drinking-water</i>

There has been a significant increase in the toxicity data available since the previous review, although much of it pertains to the intraperitoneal route of exposure. 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. IARC has concluded that antimony trioxide is possibly carcinogenic to humans (Group 2B) on the basis of an inhalation study in rats, but that antimony trisulfide was not classifiable as to its carcinogenicity to humans (Group 3). However, chronic oral uptake of potassium antimony tartrate may not be associated with an additional carcinogenic risk, as antimony after inhalation exposure was carcinogenic only in the lung but not in other organs and is known to cause direct lung damage following chronic inhalation as a consequence of overload with insoluble particulates. 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.

Arsenic¹

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 1–2 µg/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.

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% 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 10 µg/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.

Provisional guideline value	0.01 mg/l (10 µg/l)
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The guideline value is designated as provisional on the basis of treatment performance and analytical achievability.

¹ As arsenic is one of the chemicals of greatest health concern in some natural waters, its chemical fact sheet has been expanded.