access to the expertise needed to solve specific operational problems. For these small facilities, 0.2 mg/litre or less is a practicable level for aluminium in finished water.

**History of guideline development**

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to aluminium. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 0.2 mg/litre was established for aluminium, based on aesthetic considerations (as a compromise between the use of aluminium compounds in water treatment and discoloration that may be observed if levels above 0.1 mg/litre remain in the distributed water). No health-based guideline value was recommended in the 1993 Guidelines, but the Guidelines confirmed that a concentration of 0.2 mg/litre in drinking-water provides a compromise between the practical use of aluminium salts in water treatment and discoloration of distributed water. No health-based guideline value was derived for aluminium in the addendum to the Guidelines published in 1998, owing to the limitations of the animal data as a model for humans and the uncertainty surrounding the human data. However, taking the beneficial effects of the use of aluminium as a coagulant in water treatment into account and considering the health concerns about aluminium (i.e., its potential neurotoxicity), a practicable level was derived based on optimization of the coagulation process in drinking-water plants using aluminium-based coagulants, to minimize aluminium levels in finished water. Under good operating conditions, concentrations of aluminium of 0.1 mg/litre or less are achievable in large water treatment facilities. For small facilities, 0.2 mg/litre or less is a practicable level for aluminium in finished water.

**Assessment date**

The risk assessment was originally conducted in 1998. The Final Task Force Meeting in 2003 agreed that this risk assessment be brought forward to this edition of the *Guidelines for Drinking-water Quality*.

**Principal reference**


**12.6 Ammonia**

The term ammonia includes the non-ionized (\(\text{NH}_3\)) and ionized (\(\text{NH}_4^+\)) species. Ammonia in the environment originates from metabolic, agricultural and industrial processes and from disinfection with chloramine. Natural levels in groundwater and surface water are usually below 0.2 mg/litre. Anaerobic groundwaters may contain up to 3 mg/litre. Intensive rearing of farm animals can give rise to much higher levels in surface water. Ammonia contamination can also arise from cement mortar pipe
linings. Ammonia in water is an indicator of possible bacterial, sewage and animal waste pollution.

Ammonia is a major component of the metabolism of mammals. Exposure from environmental sources is insignificant in comparison with endogenous synthesis of ammonia. Toxicological effects are observed only at exposures above about 200 mg/kg of body weight.

Ammonia in drinking-water is not of immediate health relevance, and therefore no health-based guideline value is proposed. However, ammonia can compromise disinfection efficiency, result in nitrite formation in distribution systems, cause the failure of filters for the removal of manganese and cause taste and odour problems (see also chapter 10).

**History of guideline development**

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* and the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, did not refer to ammonia. In the 1993 Guidelines, no health-based guideline value was recommended, but the Guidelines stated that ammonia could cause taste and odour problems at concentrations above 35 and 1.5 mg/litre, respectively.

**Assessment date**

The risk assessment was originally conducted in 1993. The Final Task Force Meeting in 2003 agreed that this risk assessment be brought forward to this edition of the *Guidelines for Drinking-water Quality*.

**Principal reference**


**12.7 Antimony**

Elemental antimony forms very hard alloys with copper, lead and tin. Antimony compounds have various therapeutic uses. Antimony was considered as a possible replacement for lead in solders, but there is no evidence of any significant contribution to drinking-water concentrations from this source. 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.
12. CHEMICAL FACT SHEETS

Guideline value 0.02 mg/litre

Occurrence Concentrations in groundwater and surface water normally range from 0.1 to 0.2 µg/litre; concentrations in drinking-water appear to be less than 5 µg/litre.

TDI 6 µg/kg of body weight, based on a NOAEL of 6.0 mg/kg of 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 inter- and intraspecies variation, 10 for the short duration of the study)

Limit of detection 0.01 µg/litre by EAAS; 0.1–1 µg/litre by ICP/MS; 0.8 µg/litre by graphite furnace atomic absorption spectrophotometry; 5 µg/litre by hydride generation AAS

Treatment achievability 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 derivation
- allocation to water 10% of TDI
- weight 60-kg adult
- consumption 2 litres/day

Toxicological review
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, due 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, since 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.
History of guideline development
The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to antimony. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, it was concluded that no action was required for antimony. A provisional guideline value for antimony was set at a practical quantification level of 0.005 mg/litre in the 1993 Guidelines, based on available toxicological data.

Assessment date
The risk assessment was conducted in 2003.

Principal reference

12.8 Arsenic
Arsenic is widely distributed throughout the Earth’s crust, most often as arsenic sulfide or as metal arsenates and arsenides. Arsenicals are used commercially and industrially, primarily as alloying agents in the manufacture of transistors, lasers and semiconductors. Arsenic is introduced into drinking-water sources primarily through the dissolution of naturally occurring minerals and ores. Except for individuals who are occupationally exposed to arsenic, the most important route of exposure is through the oral intake of food and beverages. There are a number of regions where arsenic may be present in drinking-water sources, particularly groundwater, at elevated concentrations. Arsenic in drinking-water is a significant cause of health effects in some areas, and arsenic is considered to be a high-priority substance for screening in drinking-water sources. Concentrations are often highly dependent on the depth to which the well is sunk.

<table>
<thead>
<tr>
<th>Provisional guideline value</th>
<th>0.01 mg/litre</th>
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<tbody>
<tr>
<td>The guideline value is designated as provisional in view of the scientific uncertainties.</td>
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<th>Occurrence</th>
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<td>Levels in natural waters generally range between 1 and 2 μg/litre, although concentrations may be elevated (up to 12 mg/litre) in areas containing natural sources.</td>
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<tr>
<th>Basis of guideline derivation</th>
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<tr>
<td>There remains considerable uncertainty over the actual risks at low concentrations, and available data on mode of action do 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 1–10 μg/litre and the practical difficulties in removing arsenic from drinking-water, the guideline value of 10 μg/litre is retained. In view of the scientific uncertainties, the guideline value is designated as provisional.</td>
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