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.
### 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**


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**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>
</tr>
</thead>
<tbody>
<tr>
<td>The guideline value is designated as provisional in view of the scientific uncertainties.</td>
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<table>
<thead>
<tr>
<th>Occurrence</th>
<th>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.</th>
</tr>
</thead>
</table>

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<tr>
<th>Basis of guideline derivation</th>
<th>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.</th>
</tr>
</thead>
</table>