The beneficial effects of the use of aluminium as a coagulant in water treatment are recognized. Taking this into account, and considering the health concerns about aluminium (i.e. its potential neurotoxicity), a practicable level is derived, based on optimization of the coagulation process in drinking-water plants using aluminium-based coagulants, to minimize aluminium levels in finished water.

Several approaches are available for minimizing residual aluminium concentrations in treated water. These include use of optimum pH in the coagulation process, avoiding excessive aluminium dosage, good mixing at the point of application of the coagulant, optimum paddle speeds for flocculation and efficient filtration of the aluminium floc. Under good operating conditions, concentrations of aluminium of 0.1 mg/l or less are achievable in large water treatment facilities. Small facilities (e.g. those serving fewer than 10 000 people) might experience some difficulties in attaining this level, because the small size of the plant provides little buffering for fluctuation in operation; moreover, such facilities often have limited resources and limited access to the expertise needed to solve specific operational problems. For these small facilities, 0.2 mg/l or less is a practicable level for aluminium in finished water.

As indicated above, a health-based value derived from the JECFA PTWI would be 0.9 mg/l (rounded value) based on an allocation of 20% of the PTWI to drinking-water and assuming a 60 kg adult drinking 2 litres of water per day. However, as also noted above, practicable levels based on optimization of the coagulation process in drinking-water plants using aluminium-based coagulants are less than 0.1 mg/l in large water treatment facilities and less than 0.2 mg/l in small facilities. In view of the importance of optimizing coagulation to prevent microbial contamination and the need to minimize deposition of aluminium floc in distribution systems, it is important to ensure that average residuals do not exceed these values.

Ammonia

The term ammonia includes the non-ionized (NH_3) and ionized (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/l. Anaerobic groundwaters may contain up to 3 mg/l. 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.

Reason for not establishing a guideline value	Occurs in drinking-water at concentrations well below those of health concern
Assessment date	1993
Principal reference	WHO (2003) *Ammonia in drinking-water*

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 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).

Anatoxins (cyanobacterial toxins)[1]

Anatoxin-a, homoanatoxin-a and their dihydro derivatives (ATXs) are naturally occurring alkaloids produced by strains of various species of cyanobacteria, primarily in freshwater environments.[2] ATXs have been found in many countries but generally have been reported less often than MCs or CYNs. They have been reported from a number of cyanobacterial genera, including *Anabaena*, *Dolichospermum*, *Aphanizomenon* and *Cuspidothrix*, many of which are primarily benthic (i.e. grow on sediments or other submerged surfaces) (see also section 11.5). ATXs, like MCs and STXs, usually occur bound to cyanobacterial cells.

Drinking-water is the most likely route of exposure to ATXs where surface water with cyanobacterial blooms is the drinking-water source. Recreational activities in lakes with cyanobacterial blooms may also be a relevant exposure pathway, potentially to high concentrations (see WHO *Guidelines on recreational water quality*, 2021).

Reason for not establishing a guideline value	Available data inadequate to permit derivation of health-based guideline value
Provisional reference value (short-term)*	*Total ATXs (sum of all congeners, free plus cell-bound)*: 0.03 mg/l The reference value is based on data for anatoxin-a only
Occurrence	Concentrations reported usually range well below 1 mg/l; outside of scum areas, they rarely exceed several µg/l. ATXs largely occur cell-bound unless cell damage causes release.
TDI	A formal TDI could not be derived because of database limitations. However, 98 µg/kg bw per day, based on a 28-day study in mice, was selected as a NOAEL. An uncertainty factor of 100 (10 each for inter- and intra-species variability) was applied. An uncertainty factor for database limitations was not applied because of the conservative assumptions used to select the NOAEL (see text following the table for further detail).
Limit of detection	0.05 µg/L by LC-MS/MS and <30 µg/L by HPLC coupled with post-derivatization fluorescence. LC-MS/MS requires quantitative reference standards, which are available for anatoxin-a and for dihydro-anatoxin-a. A receptor-binding assay that is commercially available circumvents this problem and provides more reliable results than HPLC. Prior extraction of cells with freeze–thaw cycles and acidified water or acidified mixtures of methanol/water is necessary for cell-bound ATX; neglecting extraction from cells will lead to dramatic underestimation of concentrations.

[1] As cyanobacteria and their toxins are a concern in many areas and considering the complexities in their management, this chemical fact sheet has been expanded.

[2] ATXs do not include anatoxin-a(S), a naturally occurring organophosphate with a different mode of neurotoxicity.