8. CHEMICALS ASPECTS

In general, approaches to the management of chemical hazards vary between those where the source water quality is a significant contributor (with control effected through source water inputs or through treatment or blending), and those from materials and chemicals used in the production and distribution of drinking-water, (controlled by process optimisation or product specification).

Most chemicals arising from source waters are of health concern only after extended exposure. The principal exception is nitrate. Typically changes in water quality occur progressively except for those substances that are discharged or leach intermittently to flowing surface waters or groundwater supplies, for example, from contaminated landfill sites

In some cases, there are groups of chemicals which arise from the same sources for example the disinfection by-products, and it may not be necessary to set standards for all of the substances for which there are guideline values. If chlorination is practised, the trihalomethanes, of which chloroform is the major component, are likely to be the main disinfection by-products, together with the chlorinated acetic acids in some instances. In some cases, control of chloroform levels and, where appropriate, trichloroacetic acid levels will also provide an adequate measure of control over other chlorination by-products.

Several of the inorganic elements for which guideline values have been recommended are recognized to be essential elements in human nutrition. No attempt has been made here to define a minimum desirable concentration of such substances in drinking-water.

8.1 Chemical hazards in drinking-water

A number of chemical contaminants have been shown to cause adverse health effects in humans as a consequence of prolonged exposure through drinking-water. However, this is only a very small proportion of the chemicals that may reach drinking-water from various sources.

> ***The lists of chemicals addressed in these Guidelines do not imply that all of these chemicals will always be present, or that other chemicals not addressed will not be present.***

The substances considered here have been assessed for possible health effects and guideline values have only been proposed on the basis of health concerns. Additional consideration of the potential effects of chemical contaminants on the acceptability of drinking-water to consumers is included in chapter 10: Acceptability Aspects. Some substances of health concern have aesthetic effects that would normally lead to rejection of water at concentrations significantly lower than those of health concern. For such substances, health-based reference values are needed, for instance, for use in interpreting data collected in response to customer complaints.

In section 1.2 it is indicated that "In developing national drinking-water standards based on these guideline values, it will be necessary to take into account a variety of geographical, socioeconomic, dietary and other conditions affecting potential exposure. This may lead to national standards that differ appreciably from the guideline values." This is particularly

applicable to chemical contaminants, for which there is a long list, and setting standards for, or including, all of them in monitoring programmes is neither feasible nor desirable.

It is, important that chemical contaminants are prioritised so that the most important are considered for inclusion in national standards or monitoring programmes.

The scientific basis for guideline values has been made as clear as possible in the summaries presented here. This is particularly important in helping to modify guideline values to suit national requirements or in assessing the significance for health of concentrations of a contaminant that are greater than the guideline value.

Chemical contaminants in drinking-water may be categorised in various ways, however, the most appropriate is to consider the primary source of the contaminant i.e. to group chemicals according to where control may be effectively exercised. This aids in the development of approaches that are designed to prevent or minimise contamination, rather than those that rely primarily on the measurement of contaminant levels in final waters.

Sources of chemicals in drinking-water related to health are therefore divided into six major source groups, see table 8.1

Table 8.1 Categorisation of source of chemical constituents

Source of chemical constituents	Examples of sources
Naturally occurring	Rocks, soils and the effects of the geological setting and climate
Industry and human settlements	Mining (extractive industries) and manufacturing and processing industries, sewage, solid wastes, urban runoff, fuel leakages
Agricultural activities	Manures, fertilisers, intensive animal practices and pesticides
Water treatment and distribution systems	Coagulants, disinfection by-products, and piping materials
Larvicides used in water for Public Health	Larvaecides used in the control of insect vectors
Cyanobacteria	Eutrophic lakes

Some contaminants may fall into more than one category but the primary category is the one under which the guideline summary evaluation is listed. The categories are not always clear cut. The group of naturally occurring contaminants includes many of the inorganic chemicals that are found in drinking-water as a consequence of release from rocks and soils by rainfall, some of which may become problematical only where there is environmental disturbance such as in mining areas.

The criteria use to decide whether a Guideline Value is established for a particular chemical constituent are:

i. the chemical is of significant international concern;
ii. there is credible evidence of occurrence of the chemical in drinking-water;
iii. there is evidence of actual toxicity combined with evidence of occurrence in drinking – water at concentrations close to, or above those of health concern; or

iv. the chemical is being considered, or is included in the WHO-PES programme (approval programme for direct application to drinking-water for control of insect vectors of disease).

8.2 Derivation of chemical guideline values

Guideline Values are derived for many chemical constituents of drinking-water. A Guideline Value represents the concentration of a constituent which does not result in any significant risk to health over a lifetime of consumption

There are two principal sources of information on health effects resulting from exposure to chemicals that can be used in deriving guideline values. The first is investigation in human populations. The value of such studies for many substances is limited, owing to lack of quantitative information on the concentration to which people are exposed or on simultaneous exposure to other agents. However, for some substances such studies are sometimes the primary basis on which guidelines are developed. The second, and most frequently used source of information is toxicity studies using laboratory animals. The limitations of toxicology studies include the relatively small number of animals used and the relatively high doses administered, which creates uncertainty as to the relevance of particular findings to human health. This is because there is a need to extrapolate the results to the low doses to which human populations are usually exposed. In most cases, the study used to derive the guideline value is supported by a range of other studies, including human data, and these are also considered in carrying out a health risk assessment.

In order to derive a guideline value to protect human health, it is necessary to select the most suitable study or studies. Data from well-conducted studies, where a clear dose-response relationship has been demonstrated, are preferred. Expert judgement was exercised in the selection of the most appropriate study from the range of information available.

8.2.1: **Approaches taken**

To approaches to the derivation of Guideline Values are used: one for “threshold chemicals” and the other for “non-threshold chemicals” (mostly genotoxic carcinogens). It is generally considered that the initiating event in the process of genotoxic chemical carcinogenesis is the induction of a mutation in the genetic materials (DNA) of somatic cells (i.e. cells other than ova or sperm), and that there is a possibility of risk at any exposure (i.e. no threshold). On the other hand, there are carcinogens that are capable of producing tumours in animals or humans without exerting a genotoxic activity, but acting through an indirect mechanism. It is generally believed that a threshold dose exists for non-genotoxic carcinogens.

In deriving Guideline Values for carcinogens, consideration was given to the potential mechanism/s by which the substance may cause cancer, in order to decide whether a threshold or non-threshold approach should be used (see 8.2.1 for Threshold chemical effect and 8.2.6 Non-threshold chemicals).

The evaluation of the potential carcinogenicity of chemical substances is usually based on long-term animal studies. Sometimes data are available on carcinogenicity in humans, however mostly from occupational exposure.

On the basis of the available evidence, IARC categorises chemical substances with respect to their potential carcinogenic risk into the following groups:

Group 1: the agent is carcinogenic to humans
Group 2A: the agent is probably carcinogenic to humans
Group 2B: the agent is possibly carcinogenic to humans
Group 3: the agent is not classifiable as to its carcinogenicity to humans
Group 4: the agent is probably not carcinogenic to humans

According to IARC, these classifications represent a first step in carcinogenic risk assessment, which leads to a second step of quantitative risk assessment where possible. In establishing guideline values for drinking-water, the IARC evaluation of carcinogenic compounds is taken into consideration where available.

8.2.2 Threshold chemicals

For most kinds of toxicity, it is generally believed that there is a dose below which no adverse effect will occur. For chemicals that give rise to such toxic effects, a tolerable daily intake (TDI) should be derived as follows, using the most sensitive endpoint in the most relevant study, preferably in drinking water:

$$TDI = (NOAEL \text{ or } LOAEL) / UF$$

Where: NOAEL = no-observed-adverse-effect-level
LOAEL = lowest-observed-adverse-effect-level
UF = Uncertainty factor

The guideline value (GV) is then derived from the TDI as follows:

$$GV = (TDI \times bw \times P) / C$$

Where:
bw = body weight (see Annex 2)
P = fraction of the TDI allocated to drinking-water
C = daily drinking-water consumption (see Annex 2)

8.2.1 Tolerable daily intake

The TDI is an estimate of the amount of a substance in food or drinking-water, expressed on a body weight basis (mg/kg or µg/kg of body weight), that can be ingested over a lifetime without appreciable health risk.

Over many years, JECFA and JMPR have developed certain principles in the derivation of acceptable daily intakes (ADIs). These principles have been adopted where appropriate in the derivation of TDIs used in developing guideline values for drinking-water quality.

ADIs are established for food additives and pesticide residues that occur in food for necessary technological purposes or plant protection reasons. For chemical contaminants, which usually have no intended function in drinking-water, the term “tolerable daily intake” is seen

as more appropriate than "acceptable daily intake", as it signifies permissibility rather than acceptability.

As TDIs are regarded as representing a tolerable intake for a lifetime, they are not so precise that they cannot be exceeded for short periods of time. Short-term exposure to levels exceeding the TDI is not a cause for concern, provided the individual's intake averaged over longer periods of time does not appreciably exceed the level set. The large uncertainty factors generally involved in establishing a TDI (see below) serve to provide assurance that exposure exceeding the TDI for short periods is unlikely to have any deleterious effects upon health. However, consideration should be given to any potential acute effects that may occur if the TDI is substantially exceeded for short periods of time.

No-observed-adverse-effect-level and lowest-observed-adverse-effect-level

The NOAEL is defined as the highest dose or concentration of a chemical in a single study, found by experiment or observation, that causes no detectable adverse health effect. Wherever possible, the NOAEL is based on long-term studies, preferably of ingestion in drinking-water. However, NOAELs obtained from short-term studies and studies using other sources of exposure (e.g., food, air) may also be used.

If a NOAEL is not available, a LOAEL may be used, which is the lowest observed dose or concentration of a substance at which there is a detectable adverse health effect. When a LOAEL is used instead of a NOAEL, an additional uncertainty factor is normally applied (see below).

Uncertainty factors

The application of uncertainty factors has been widely used in the derivation of ADIs for food additives, pesticides and environmental contaminants. The derivation of these factors requires expert judgement and careful consideration of the available scientific evidence.

In the derivation of guideline values, uncertainty factors are applied to the NOAEL or LOAEL for the response considered to be the most biologically significant.

In relation to exposure of the general population, the NOAEL for the critical effect in animals is normally divided by an uncertainty factor of 100. This comprises two 10-fold factors, one for interspecies differences and one for interindividual variability in humans (see Table 8.2). Extra uncertainty factors may be incorporated to allow for database deficiencies and for the severity and irreversibility of effects.

Table 8.2 Source of uncertainty in derivation of guideline values

Source of uncertainty	Factor
Interspecies variation (animals to humans)	1 - 10
Intraspecies variation (individual variations within species)	1 – 10
Adequacy of studies or database	1 – 10
Nature and severity of effect	1 – 10

Factors lower than 10 were used, for example, for interspecies variation when humans are known to be less sensitive than the animal species studied. Inadequate studies or databases include those where a LOAEL was used instead of a NOAEL and studies considered to be

shorter in duration than desirable. Situations in which the nature or severity of effect might warrant an additional uncertainty factor include studies in which the end-point was malformation of a foetus or in which the end-point determining the NOAEL was directly related to possible carcinogenicity. In the latter case, an additional uncertainty factor was usually applied for carcinogenic compounds for which the guideline value was derived using a TDI approach.

The total uncertainty factor should not exceed 10,000. If the risk assessment would lead to a higher uncertainty factor, then the resulting TDI would be so imprecise as to lack meaning. For substances for which the uncertainty factors were greater than 1,000, guideline values are designated as provisional in order to emphasise the higher level of uncertainty inherent in these values.

The selection and application of uncertainty factors are important in the derivation of guideline values for chemicals, as they can make a considerable difference to the values set. For contaminants for which there is sufficient confidence in the database, the guideline value was derived using a smaller uncertainty factor. For most contaminants, however, there is greater scientific uncertainty, and a relatively large uncertainty factor was used. Hence, there may be a large margin between the guideline value and the concentration of the substance which would actually cause adverse health effects. This flexible approach to the use of uncertainty factors enables the particular attributes of the chemical and the data available to be considered in the deriving the guidelines. Where uncertainty factors have been used on calculating the guideline value, it is presented as part of the rationale for guideline derivation.

Allocation of intake

Drinking-water is not usually the sole source of human exposure to the substances for which guideline values have been set. In many cases, the intake of chemical contaminants from drinking-water is small in comparison with that from other sources such as food and air. Guideline values derived using the TDI approach take into account exposures from all sources by apportioning a percentage of the TDI to drinking-water. This approach ensures that total daily intake from all sources (including drinking-water containing concentrations of the substance at or near the guideline value) does not exceed the TDI.

Wherever possible, data concerning the proportion of total intake normally ingested in drinking-water (based on mean levels in food, air and drinking-water) or intakes estimated on the basis of consideration of physical and chemical properties were used in the derivation of the guideline values. Where such information was not available, an arbitrary (default) value of 10 per cent for drinking-water was used. This default value is, in most cases, sufficient to account for additional routes of intakes (i.e., inhalation and dermal absorption) of contaminants in water. In some cases a specific discussion has been made of the potential for exposure from intake through inhalation and dermal uptake in bathing and showering where the allocation of the ADI to drinking water is greater than 10%.

It is recognised that exposure from various media may vary with local circumstances. It should be emphasised, therefore, that the derived guideline values apply to a typical exposure scenario or are based on default values that may nor be applicable for all areas. In those areas where relevant exposure data are available, authorities are encouraged to develop context-specific guideline values that are tailored to local circumstances and conditions. For example, in areas where the intake of a particular contaminant in drinking-water is known to

be much greater than that from other sources (i.e., air and food), it may be appropriate to allocate a greater proportion of the TDI to drinking-water to derive a Guideline Value more suited to the local conditions. In addition, in cases in which guideline values are exceeded, efforts should be made to assess the contribution of other sources to total intake, if practicable, exposure from these sources should be minimised.

Significant figures

The calculated TDI is used to derive the guideline value, which was then rounded to one significant figure. In some instances, ADI values with only one significant figure set by JECFA or JMPR were used to calculate the guideline value. The guideline value was generally rounded to one significant figure to reflect the uncertainty in animal toxicity data and exposure assumptions made.

8.2.3 Non-threshold chemicals

In the case of compounds considered to be genotoxic carcinogens, guideline values were normally determined using a mathematical model. Although several models exist, the linearised multistage model was generally adopted in the development of these guidelines. Other models were considered more appropriate in a few cases. Guideline values presented are the concentrations in drinking-water associated with an estimate upper bound excess lifetime risk of 10^{-5} (or one additional cancer per 100,000 of the population ingesting drinking-water containing the substance at the guideline value for 70 years).

It should be emphasized that guideline values for carcinogenic compounds computed using mathematical models must be considered at best as hypothetical and a rough estimate of the upper bound cancer risk. These models do not usually take into account a number of biologically important considerations, such as pharmacokinetics, DNA repair, or protection by the immune system. They also assume the validity of extrapolation of very high dose exposures in test animals to very low dose exposures in humans. As a consequence, the models used are conservative (i.e. err on the side of caution).

8.2.4 Data quality and peer review

The assessment of the toxicity of drinking-water contaminants has been primarily made on the basis of published reports from peer reviewed open literature, reviews by recognised international bodies or national reviews recognised to be of high quality, information submitted by governments and other interested parties, and to a limited extent, unpublished proprietary data. In the development of the guideline values, existing international approaches to developing guidelines were carefully considered. In particular, previous risk assessments developed by the International Programme on Chemical Safety (IPCS) in Environmental Health Criteria monographs, the International Agency for Research on Cancer (IARC), the Joint FAO/WHO Meetings on Pesticide Residues (JMPR), and the Joint FAO/WHO Expert Committee on Food Additives (JECFA) were reviewed. These assessments were relied upon except where new information justified a reassessment but the quality of new data was critically evaluated before it was used in any risk assessment. The primary use of unpublished proprietary data has been in the evaluation of pesticides. Current revisions and future assessments of pesticides will only take place through WHO/IPCS/JMPR/JECFA processes.

8.2.9 **Quality of data for derivation of guideline values**

WHO has endeavoured to provide as much assistance as possible to users of the Guidelines by developing guideline values wherever possible. On occasion this has required the use of very large uncertainty factors and guideline values that are designated as provisional. One situation where this occurs is when the quality of health-effects data relevant to guideline derivation is lower than what is generally available to other chemicals. It is suggested that a formal guideline value for a substance in such circumstances might not be appropriate or even necessary. Instead where data are considered to be highly uncertain, it would be appropriate to provide a health-based number in the summary for use in cases where needed, but not propose a formal health based guideline value. The substance would be listed as having been considered but would not have an associated guideline value. An example of the successful use of such an approach is iron.

In terms of the quality of data and peer review of such data required to develop a health based guideline value, the following are some general considerations. However, it is not expected that all these will be available:

- The studies have undergone international peer review (such as by IPCS, JECFA or JMPR) or high quality national peer review
- Oral studies are preferred (in particular, drinking-water studies), with the pure substance with appropriate dosing regime and a good quality pathology.
- A sufficiently broad database to be reasonably comfortable that potential toxicological end-points of concern have been identified.
- The quality of the studies is such that they are considered reliable, for example, there has been adequate consideration of confounding factors in epidemiological studies.
- There is reasonable consistency between studies; the endpoint and study used to derive a guideline value do not contradict the overall weight of evidence.
- For inorganic substances there is some consideration of speciation in drinking-water.
- There is appropriate consideration of multi-media exposure.

8.3 Analytical aspects

8.3.1 Analytical achievability

Various collections of “standard” or “recommended” methods for water analysis are published by a number of national and international agencies. It is often thought that adequate analytical accuracy can be achieved provided that all laboratories use the same standard method. Experience shows that this is not always the case, as a variety of extraneous factors may affect the accuracy of the results. Examples include reagent purity, apparatus type and performance, degree of modification of the method in a particular laboratory and the skill and care of the analyst. These factors are likely to vary both between the laboratories and over time in an individual laboratory. Moreover, the accuracy that can be achieved with a particular method frequently depends upon the nature and composition of the sample. It is not essential to use standard methods except in the case of “non-specific” variables such as taste and odour, colour, and turbidity. In these cases, the result is determined by the method employed, and it is necessary for all laboratories to use identical methods if comparable results are to be obtained.

A number of considerations are important in selecting methods:

- The overriding consideration is that the method chosen can result in the required accuracy. Other factors, such as speed and convenience, should be considered only in selecting among methods that meet this primary criterion.
- There are a number of markedly different procedures for measuring and reporting the errors to which methods are subject. This needlessly complicates and prejudices the effectiveness of method selection, and suggestions for standardizing such procedures have been made. It is desirable that details of all analytical methods are published together with performance characteristics that can be interpreted unambiguously.
- If the analytical results from one laboratory are to be compared with those from others and/or with a numerical standard, it is obviously preferable for them not to have any associated systematic error. In practice, this is not possible, but each laboratory should select methods whose systematic errors have been thoroughly evaluated and shown to be acceptably small.

A qualitative ranking of analytical methods based on their degree of technical complexity is given in table 8.3. for inorganic chemicals and in table 8.4 for organic chemicals. The higher the ranking the more complex the process is in terms of equipment and/or operation. In general, higher rankings are also associated with higher costs. Analytical achievability of the chemicals based on detection limits are given in tables 8.5-8.9. When available, field test methods are included as well as instrumental analytical methods used in a laboratory.

Table 8.3 Ranking of analytical complexity for inorganic chemicals

Ranking	Example of analytical methods
1	Volumetric method Colorimetric method
2	Electrode method
3	Ion Chromatography
4	High-Performance Liquid Chromatography
5	Flame Atomic Absorption Spectrometry(AAS) method
6	Electrothermal AAS method
7	Inductively Coupled Plasma(ICP)-Atomic Emission Spectrometry
8	ICP-Mass Spectrometry method

Table 8.4 Ranking of analytical complexity for organic chemicals

Ranking	Example of analytical methods
1	High-Performance Liquid Chromatography
2	Gas Chromatography(GC) method
3	GC-Mass Spectrometry method

4	Head Space GC-Mass Spectrometry method
5	Puge and Trap GC method Puge and Trap GC-Mass Spectrometry method

Table 8.5 Analytical achievability for inorganic chemicals

	Field Methods		Laboratory methods				
	Colorimetry	*Absorptiometry*	*Ion Chromatography*	*Flame Atomic Absorption Spectrometry*	*Electrothermal Atomic Absorption Spectrometry*	*Inductively couple plasma*	*Inductively charged plasma/Mass Spectrometry*
Naturally occurring chemicals							
Arsenic		#		+(H)	++, +++(H)	++(H)	+++
Barium				+	+++	+++	+++
Boron		++				++	+++
Chloride	○	○	○				
Chromium		#		+	+++	+++	+++
Fluoride	#	+	++				
Hardness				○			
Hydrogen sulfide		○					
Manganese	+	++		++	+++	+++	+++
Molybdenum					+	+++	+++
pH	○	○					
Selenium		#		#	+++(H)	++(H)	+
Sodium			○	○	○	○	
Sulfate			○			○	
Total dissolved solids							
Uranium						+	+++
Chemicals from industrial sources and human dwellings							
Beryllium					○	○	
Cadmium		#			++	++	+++
Cyanide	#	+	+				
Mercury					+		
Chemicals from agricultural activities							
Ammonia	○	○	○				
Nitrate/nitrite	+++	+++	#				
Chemicals used in water treatment or materials in contact with drinking-water							
Silver				○	○	○	
Aluminium	#	+			+++	+	+++

Iron	+++	+++		+++	+++	+++	
Antimony				#	++(H)	++(H)	+++
Asbestos							
Copper	#	+++		+++	+++	+++	+++
Lead		#			+	+	++
Nickel		+		#	+	+++	++
Inorganic tin		○		○	○	○	○
Zinc	○	○		○	○	○	○

+: The detection limit is between the guide line value and 1/10 of its value.
++: The detection limit is between 1/10 and 1/50 of the guide line value.
+++: The detection limit is under 1/100 of the guide line value.
#: The analytical method is available for detection of the concentration of the guide line value, but it is difficult to detect the concentration of 1/10 of the guide line value.
○: The detection method(s) is/are available for the item.
(H):This method is applicable to the determination by conversion to their hydrides by hydride generator.

Table 8.6 Analytical achievability for organic chemicals from industrial sources and human dwellings

	Colorimetry	*Gas chromatography*	*Gas Chromatography (Photoionisation detector)*	*Gas Chromatography (Electron Capture)*	*Gas Chromatography (Flame Ionisation detector)*	*Gas Chromatography (Flame photodiode detector)*	*Gas Chromatography (Thermal ionisation detector)*	*Gas Chromatography (Mass spectrometry)*	*Purge-and-Trap Gas Chromatography (Mass spectrometry*	*High-performance Liquid Chromatography*	*High-performance Liquid Chromatography (Fluorescence detector)*	*High-performance Liquid Chromatography (Ultra-violet photodiode Array detector)*	*Electrothermal Atomic Absorption Spectrometry*	*Ion Chromatography with fluorescence detector*
Carbon tetrachloride				+					+					
Dichloromethane				#	+				+++					
1,1-Dichloroethane				○	○				○					
1,2-Dichloroethane				+++					++					
1,1,1-Trichloroethane				○	○				○					
1,1-Dichloroethene				+++	+				+++					
1,2-Dichloroethene				++	++				+++					
Trichloroethene				+++	+				+++					+
Tetrachloroethene				+++	+				+++					
Benzene				++	+				+++					
Toluene					+++				+++					
Xylenes					+++				+++					
Ethylbenzene				+++	+++				+++					
Styrene				++	+				+++					
Monochlorobenzene				○					○					
1,2-Dichlorobenzene			+++	+++					+++					
1,3-Dichlorobenzene				○					○					
1,4-Dichlorobenzene			+++	+++					+++					
Trichlorobenzenes			○	○					○					

	Colorimetry	*Gas chromatography*	*Gas Chromatography (Photoionisation detector)*	*Gas Chromatography (Electron Capture)*	*Gas Chromatography (Flame Ionisation detector)*	*Gas Chromatography (Flame photodiode detector)*	*Gas Chromatography (Thermal ionisation detector)*	*Gas Chromatography (Mass spectrometry)*	*Purge-and-Trap Gas Chromatography (Mass spectrometry*	*High-performance Liquid Chromatography*	*High-performance Liquid Chromatography (Fluorescence detector)*	*High-performance Liquid Chromatography (Ultra-violet photodiode Array detector)*	*Electrothermal Atomic Absorption Spectrometry*	*Ion Chromatography with fluorescence detector*
Di(2-ethylhexyl)adipate								○						
Di(2-ethylhexyl)phthalate								++						
Hexachlorobutadiene									+					
Edetic acid (EDTA)								+++						
Nitrilotriacetic acid (NTA)		+++												
1,4-Dioxane								+++						
Pentachlorophenol														

+: The detection limit is between the guide line value and 1/10 of its value.

++: The detection limit is between 1/10 and 1/50 of the guide line value.

+++: The detection limit is under 1/100 of the guide line value.

#: The analytical method is available for detection of the concentration of the guide line value, but it is difficult to detect the concentration of 1/10 of the guide line value.

○: The detection method(s) is/are available for the item.

Table 8.7: Analytical achievability for organic chemicals from agricultural activities

	Colorimetry	*Gas chromatography*	*Gas Chromatography Photoionisation detector*	*Gas Chromatography Electron Capture*	*Gas Chromatography Flame Ionisation detector*	*Gas Chromatography Flame photodiode detector*	*Gas Chromatography Thermal ionisation detector*	*Gas Chromatography Mass spectrometry*	*Purge-and-Trap Gas Chromatography Mass spectrometr*	*High-performance Liquid Chromatography*	*High-performance Liquid Chromatography Fluorescence detector*	*High-performance Liquid Chromatography Ultra-violet photodiode*	*Electrothermal Atomic Absorption Spectrometry*	*Ion Chromatography with fluorescence detector*
Alachlor				○				+++						
Aldicarb												+		
Aldrin and dieldrin				+										
Amitraz								+++						
Atrazine								+++○						
Bentazone				○										
Carbofuran							++				++			
Chlordane				+										
Chlorotoluron				○										
Cyanazine								++				+		
2,4-D				++				+++						
2,4-DB														
1,2-Dibromo-3-chloropropane														
1,2-Dibromoethane								+	+					
1,2-Dichloropropane				+++					+++					
1,3-Dichloropropane														
1,3-Dichloropropene				+++					+++					
Dichlorprop 2,4-DP														
Dimethoate								+++						
Diquat										○				
Endosulfan				○				○						
Endrin				+				#						

	Colorimetry	*Gas chromatography*	*Gas Chromatography Photoionisation detector*	*Gas Chromatography Electron Capture*	*Gas Chromatography Flame Ionisation detector*	*Gas Chromatography Flame photodiode detector*	*Gas Chromatography Thermal ionisation detector*	*Gas Chromatography Mass spectrometry*	*Purge-and-Trap Gas Chromatography Mass spectrometr*	*High-performance Liquid Chromatography*	*High-performance Liquid Chromatography Fluorescence detector*	*High-performance Liquid Chromatography Ultra-violet photodiode*	*Electrothermal Atomic Absorption Spectrometry*	*Ion Chromatography with fluorescence detector*
Fenitrothion					○	○	○	○		○				
Fenoprop				+										
Gryphosate (and AMPA)										○				
Heptachlor and heptachlor epoxide				○				○						
Hexachlorobenzene				+++				+						
Isoproturon				+								+++		
Lindane				+										
Malathion								○						
MCPA				+++				+++				+++		
Mecoprop				++				++						
Methoxychlor		+++												
Metolachlor				+++										
Molinate								+++						
Parathion														
Parathion-methyl														
Pendimethalin				+++			++	+++						
Pentachlorophenol			+	+++				+++						
Permethrin			○											
2-Phenylphenol			○											
Propanil														
Simazine					+		+	+++						
2,4,5-T			+	+++										
Terbuthylazine								+++				++		

	Colorimetry	*Gas chromatography*	*Gas Chromatography Photoionisation detector*	*Gas Chromatography Electron Capture*	*Gas Chromatography Flame Ionisation detector*	*Gas Chromatography Flame photodiode detector*	*Gas Chromatography Thermal ionisation detector*	*Gas Chromatography Mass spectrometry*	*Purge-and-Trap Gas Chromatography Mass spectrometr*	*High-performance Liquid Chromatography*	*High-performance Liquid Chromatography Fluorescence detector*	*High-performance Liquid Chromatography Ultra-violet photodiode*	*Electrothermal Atomic Absorption Spectrometry*	*Ion Chromatography with fluorescence detector*
Trifluralin		+++						+++				+		

+: The detection limit is between the guide line value and 1/10 of its value.
++: The detection limit is between 1/10 and 1/50 of the guide line value.
+++: The detection limit is under 1/100 of the guide line value.
#: The analytical method is available for detection of the concentration of the guide line value, but it is difficult to detect the concentration of 1/10 of the guide line value.
○: The detection method(s) is/are available for the item.

Table 8.8: Analytical achievability for chemicals used in water treatment, or from materials in contact with water

	Colorimetry	*Gas chromatography*	*Gas Chromatography (Photoionisation detector)*	*Gas Chromatography (Electron Capture)*	*Gas Chromatography (Flame Ionisation detector)*	*Gas Chromatography (Flame photodiode detector)*	*Gas Chromatography (Thermal ionisation detector)*	*Gas Chromatography (Mass spectrometry)*	*Purge-and-Trap Gas Chromatography (Mass spectrometry*	*High-performance Liquid Chromatography*	*High-performance Liquid Chromatography (Fluorescence detector)*	*High-performance Liquid Chromatography (Ultra-violet photodiode Array detector)*	*Electrothermal Atomic Absorption Spectrometry*	*Ion Chromatography with fluorescence detector*
Disinfectants														
Monochloramine	+++													
Di- and trichloramines	○													
Chlorine	+++									+++				+++
Chlorine dioxide	+													+++
Chlorate														○
Chlorite	○													○
Iodine	○													
Silver														
Disinfection by-products														
Bromate														+
2-Chlorophenol				○				○						
2.4-Dichlorophenol				○				○						
2,4,6-Trichlorophenol				+++				+++						
Formaldehyde				○				○						
MX								○						
Bromoform				+++	+				+++					
Bromodichloromethane				+++	#				+++					
Dibromochloromethane				+++	+				+++					
Chloroform				+++	+				+++					
Dibromoacetate				○				○						

	Colorimetry	*Gas chromatography*	*Gas Chromatography (Photoionisation detector)*	*Gas Chromatography (Electron Capture)*	*Gas Chromatography (Flame Ionisation detector)*	*Gas Chromatography (Flame photodiode detector)*	*Gas Chromatography (Thermal ionisation detector)*	*Gas Chromatography (Mass spectrometry)*	*Purge-and-Trap Gas Chromatography (Mass spectrometry*	*High-performance Liquid Chromatography*	*High-performance Liquid Chromatography (Fluorescence detector)*	*High-performance Liquid Chromatography (Ultra-violet photodiode Array detector)*	*Electrothermal Atomic Absorption Spectrometry*	*Ion Chromatography with fluorescence detector*
Bromochloroacetate				○				○						
Monochloroacetate				○				○						
Dichloroacetate				○				○						
Trichloroacetate				○				○						
Chloral hydrate (Trichloroacetaldehyde)				+				+						
Chloroacetones														
Dichloroacetonitrile				+++				+						
Dibromoacetonitrile				○				○						
Bromochloroacetonitrile				○				○						
Trichloroacetonitrile				○				○						
Cyanogen chloride														○
Chloropicrin				○					○					
Organic contaminants from treatment chemicals														
Acrylamide		+						+				+		
Epichlorohydrin				+	+				+					
Organic contaminants from pipes and fittings														
Dialkyltins						○							○	
Tributyltin oxide								○					○	
PAHs								○			○			
Benzo(a)pyrene								++			++			

	Colorimetry	*Gas chromatography*	*Gas Chromatography (Photoionisation detector)*	*Gas Chromatography (Electron Capture)*	*Gas Chromatography (Flame Ionisation detector)*	*Gas Chromatography (Flame photodiode detector)*	*Gas Chromatography (Thermal ionisation detector)*	*Gas Chromatography (Mass spectrometry)*	*Purge-and-Trap Gas Chromatography (Mass spectrometry*	*High-performance Liquid Chromatography*	*High-performance Liquid Chromatography (Fluorescence detector)*	*High-performance Liquid Chromatography (Ultra-violet photodiode Array detector)*	*Electrothermal Atomic Absorption Spectrometry*	*Ion Chromatography with fluorescence detector*
Fluoranthene								○			○			
Vinyl chloride				+					+					

+: The detection limit is between the guide line value and 1/10 of its value.
++: The detection limit is between 1/10 and 1/50 of the guide line value.
+++: The detection limit is under 1/100 of the guide line value.
#: The analytical method is available for detection of the concentration of the guide line value, but it is difficult to detect the concentration of 1/10 of the guide line value.
○: The detection method(s) is/are available for the item.

Table 8.9. Analytical methods for Pesticides Used in Water for Public Health Purposes

	Colorimetry	*Gas chromatography*	*Gas Chromatography (Photoionisation detector)*	*Gas Chromatography (Electron Capture)*	*Gas Chromatography (Flame Ionisation detector)*	*Gas Chromatography (Flame photodiode detector)*	*Gas Chromatography (Thermal ionisation detector)*	*Gas Chromatography (Mass spectrometry)*	*Purge-and-Trap Gas Chromatography (Mass spectrometry*	*High-performance Liquid Chromatography*	*High-performance Liquid Chromatography (Fluorescence detector)*	*High-performance Liquid Chromatography (Ultra-violet photodiode Array detector)*	*Electrothermal Atomic Absorption Spectrometry*	*Ion Chromatography with fluorescence detector*
Chlorpyrifos				+++	+++	+	+++	+++						
DDT (and metabolites)				+++				+						
Pyriproxyphen														

+: The detection limit is between the guide line value and 1/10 of its value.
++: The detection limit is between 1/10 and 1/50 of the guide line value.
+++: The detection limit is under 1/100 of the guide line value.
#: The analytical method is available for detection of the concentration of the guide line value, but it is difficult to detect the concentration of 1/10 of the guide line value.
○: The detection method(s) is/are available for the item.

8.3.2 Analytical methods

Volumetric titration method
In this method chemicals are analyzed by a titration with standardized titrant. The titration endpoint is identified by the development of colour resulting from the reaction with an indicator, by the change of electrical potential or by the change of pH value.

Colorimetric method
Colorimetric methods are based on measuring the intensity of colour of a coloured target chemical or reaction product. The optical absorbance is measured using light of a suitable wavelength. The concentration is determined by means of a calibration curve obtained using known concentrations of the determinand. The UV method is similar to this method except that ultra violet light is used.

Electrode methods
For ionic materials the ion concentration can be measured using an ion-selective electrode. The measured potential is proportional to the logarithm of the ion concentration.

Ultraviolet (UV) Absorption Method
Some organic compounds absorb ultraviolet light (wavelength; 190–380nm) in proportion to their concentration. UV absorption is useful for qualitative estimation of organic substances, because a strong correlation may exist between UV absorption and organic carbon content.

Atomic Absorption Spectrometry (AAS)
The Atomic Absorption Spectrometry method is used for determination of metals. It is based on the phenomenon that the atom in the ground state absorbs the light of wavelengths that are characteristic to each element, when light is passed through the atoms in the vapour state. Because this absorption of light depends on the concentration of atoms in the vapour, the concentration of the target element in the water sample is determined from the measured absorbance. The Beer-Lambert law describes the relationship between concentration and absorbance.

- *Flame AAS Method*

In the Flame Atomic Absorption Spectrometry Method, a sample is aspirated into a flame and atomized. A light beam from a hollow cathode lamp of the same element as the target metal is radiated through the flame and the amount of absorbed light is measured by the detector.

This method is relatively highly sensitive and free from spectral or radiation interference by co-existing elements. Pretreatment is either unnecessary or straightforward. However, it is not suitable for simultaneous analysis for many elements, because the light source is different for each target element.

- *Electrothermal AAS Method*

Electrothermal Atomic Absorption Spectrometry Method is based on the same principle as Flame Atomic Absorption Spectrometry Method but an electrically heated atomizer or graphite furnace replaces the standard burner head for determination of metals.

In comparison with Flame Atomic Absorption Spectrometry, Electrothermal Atomic Absorption Spectrometry gives higher sensitivities and lower detection limits and a smaller sample volume is required. Electrothermal Atomic Absorption Spectrometry suffers from

more interference through light scattering by co-existing elements and requires a longer analysis time than Flame Atomic Absorption Spectrometry.

Inductively Coupled Plasma – Atomic Emission Spectrometry (ICP–AES) Method
The principle of this method for determination of metals is as follows. An ICP source consists of a flowing stream of argon gas ionized by an applied radio frequency. A sample aerosol is generated in a nebulizer and spray chamber and then carried into the plasma through an injector tube. A sample is heated and excited in the high-temperature plasma. The high temperature of the plasma causes the atoms to become excited. On returning to the ground state, the excited atoms produce ionic emission spectra. A monochromator is used to separate specific wavelengths corresponding to different elements, and a detector measures the intensity of radiation of each wavelength.

A significant reduction in chemical interference is achieved. In the case of water with low pollution, simultaneous or sequential analysis is possible without special pretreatment to achieve low detection limits for many elements. This, coupled with the extended dynamic range from three digits to five digits, means that multi-element determination of metals can be achieved. It can atomize, excite and measure even refractory elements such as Beryllium, Bismuth, Boron, Potassium, Titanium, Tungsten and Vanadium.

ICP-AES has similar sensitivity to Flame Atomic Absorption Spectrometry or Electrothermal Atomic Absorption Spectrometry.

Inductively Coupled Plasma – Mass Spectrometry (ICP–MS) Method
In ICP-MS, elements are atomized and excited as in ICP-AES, then passed to a mass spectrometer. Once inside the mass spectrometer, the ions are accelerated by high voltage and passed through a series of ion optics, an electrostatic analyzer (ESA), and finally a magnet. By varying the strength of the magnet, ions are separated according to mass/charge ratio and passed through a slit into the detector which records only a very small atomic mass range at a given time. By varying the magnet and ESA settings the entire mass range can be scanned within a relatively short period of time.

In the case of water with low pollution, simultaneous or sequential analysis is possible in without special pretreatment to achieve low detection limits for many elements. This, coupled with the extended dynamic range from three digits to five digits, means that multi-element determination of metals can be achieved.

Chromatography
Chromatography is a separation method based on the affinity difference between two phases, the stationary and mobile phases. A sample is injected into a column, either packed or coated with the stationary phase, and separated by the mobile phase based on the difference in interaction (distribution or adsorption) between compounds and the stationary phase. Compounds with a low affinity for the stationary phase move more quickly through the column and elute earlier. The compounds that elute from the end of the column are determined by a suitable detector.

- ***Chromatography Method: Ion Chromatography***

An ion exchanger is used as the stationary phase, and the eluent for determination of anions is typically a dilute solution of sodium hydrogen carbonate and sodium carbonate. Colorimetric, electrometric or titrimetric detectors can be used for determining individual anions.

In suppressed ion chromatography, anions are converted to their highly conductive acid forms and in the carbonate-bicarbonate eluent anions are converted to weakly conductive carbonic acid. The separated acid forms are measured by conductivity and identified on the basis of retention time as compared with their standards.

- ***Chromatography Method: High-Performance Liquid Chromatography (HPLC)***

HPLC is an analytical technique using a liquid mobile phase and a column containing a liquid stationary phase. Detection of the separated compounds is achieved through the use of absorbance detectors for organic compounds and through conductivity or electrochemical detectors for metallic and inorganic compounds.

- ***Chromatography Method: Gas Chromatography (GC)***

This method permits the identification and quantitation of trace organic compounds. In GC, gas is used as the mobile phase and the stationary phase is a liquid that is coated either on an inert granular solid or on the walls of a capillary column. When the sample is injected into the column, the organic compounds are vaporized and moved through the column by the carrier gas at different rates depending on differences in partition coefficients between the mobile and stationary phases. The gas exiting the column is passed to a suitable detector. A variety of detectors can be used including Flame Ionization (FID), Electron Capture (ECD) and Nitrogen-phosphorus.

Since separation ability is good in this method, mixtures of substances with similar structure are systematically separated, identified and determined quantitatively in a single operation.

- ***Chromatography Method: Gas Chromatography (GC) /Mass Spectrometric (MS)***

GC/MS method is based on the same principle as the GC method, using a mass spectrometer (MS) as the detector.

As the gas emerges from the end of the GC column opening it flows through a capillary column interface into the MS. The sample then enters the ionization chamber where a collimated beam of electrons impact the sample molecules causing ionization and fragmentation. The next component is a mass analyzer, which uses a magnetic field to separate the positively charged particles according to their mass. Several types of separating techniques exist, the most common are quadrupoles and ion traps. After the ions are separated according to their masses, they enter a detector.

Chromatography Method: Purge and Trap Packed-Column Gas Chromatography (GC) /Mass Spectrometric (MS) Method or Purge and Trap Packed-Column Gas Chromatography (GC)

This method is applicable to the determination of various purgeable organic compounds that are transferred from the aqueous to the vapor phase by bubbling purge gas through a water sample at ambient temperature. The vapor is trapped with a cooled trap. The trap is heated and backflushed with the same purge gas to desorb the compounds onto a gas chromatographic column. The principles of GC or GC/MS are as referred to above.

8.3.3 Analytical quality control

Whichever method is chosen, appropriate analytical quality control procedures must be implemented to ensure the results produced are of adequate accuracy. Because of the wide range of substances, methods, equipment, and accuracy requirements likely to be involved in

the monitoring of drinking-water, many detailed, practical aspects of analytical quality control are concerned. These are beyond the scope of this publication, which can only give an idea of the approach involved.

Before analysing samples by the chosen method, preliminary tests should be conducted by each laboratory to provide estimates of its precision (random error of the results). The routine analysis of samples (accompanied by regular checks of precision) can begin when the results from the preliminary tests have acceptably small errors. These preliminary tests can, and should, check certain sources of systematic error, but this is usually very difficult for a routine laboratory. This emphasizes the need for sound selection of methods initially, and also for another form of analytical quality control, namely, inter-laboratory testing. Such testing is usually the best single approach to checking systematic error but should be undertaken only after satisfactory completion of preliminary tests of precision. There may be some difficulty in implementing an analytical quality control programme if the coordinating laboratory has to deal with a large number of other laboratories, or if the laboratories are far apart. A hierarchical structure of coordinating and participating laboratories allows such difficulties to be overcome.

8.4 Treatment

8.4.1 Treatment achievability

The ability to achieve a guideline value within a drinking-water supply depends on a number of factors including:

- concentration of the chemical in the raw water;
- control measures employed through the drinking-water supply;
- nature of the raw water (ground or surface water, presence of natural background organics); and
- treatment processes already installed

If a guideline value cannot be met with the existing plant then additional treatment may need to be considered or water should be obtained from alternative sources.

The cost of achieving a guideline value will depend on the complexity of any additional treatment required. It is not possible to provide quantitative information on the cost of achieving individual guideline values. Treatment costs (capital and operating) will depend not only on the factors identified above but also upon issues such as plant throughput; local costs for labour, civil and mechanical works, chemicals and electricity; life expectancy of the plant and so on.

A qualitative ranking of treatment processes based on their degree of technical complexity is given in table 8.8. The higher the ranking the more complex the process is in terms of plant and/or operation. In general, higher rankings are also associated with higher costs.

Table 8.8 Ranking of technical complexity and cost of water treatment processes

Ranking	Examples of treatment processes
1	Simple chlorination Plain filtration
2	Pre-chlorination plus filtration

	Aeration
3	Chemical coagulation Process optimisation for control of disinfection by-products
4	Granular activated carbon treatment Ion-exchange
5	Ozonation
6	Advanced oxidation processes Membrane treatment

Tables 8.9 to 8.12 summarize the treatment processes that are capable of removing contaminants of health significance. These indicate the effectiveness of processes as follows:

+	**Limited removal**
++	**50 per cent or more removal**
+++	**80 per cent of more removal**

A blank entry in a table indicates that either the process is completely ineffective or there are no data on the effectiveness of the process. For the most effective process(es), the table indicate the concentration of the chemical, in mg/litre, that should be achievable. The tables include only those chemicals for which treatment data are available (based on information available at this time). These tables are provided to help inform decisions regarding the ability of existing treatment to meet guidelines and what additional treatment might need to be installed. They have been compiled on the basis of published literature that includes mostly laboratory experiments, some pilot plant investigations and relatively few full-scale studies of water treatment processes. Consequently, a number of points need to be borne in mind in relation to the information presented:

- many of the treatments outlined are designed for larger treatment plants, and may not necessarily be appropriate for smaller or individual type treatment. In these cases, the choice of technology must be done on a case-by-case basis;
- the information is probably "best case" since the data would have been obtained under laboratory conditions or with carefully controlled plant for the purposes of experimentation;
- actual process performance will depend on the concentration of the chemical in the raw water and on general raw water quality. For example the removal of organic chemicals and pesticides using activated carbon or ozonation will be impaired if there is a high concentration of natural organic matter;
- for many contaminants, potentially several different processes could be appropriate and the choice between processes should be made on the basis of technical complexity and cost, taking into account local circumstances. For example, membrane processes can remove a broad spectrum of chemicals but simpler and cheaper alternatives are effective for the removal of most chemicals;
- it is normal practice to use a series of unit processes to achieve desired water quality objectives; e.g. coagulation, sedimentation, filtration, GAC, chlorination. Each of these may contribute to the removal of chemicals. It may be technically and economically advantageous to use a combination of processes, e.g. ozonation plus GAC to remove particular chemicals;
- the effectiveness of potential processes should be assessed using laboratory or pilot plant tests on the actual raw water concerned. These test should be of sufficient duration to identify potential seasonal or other temporal variations in contaminant concentrations and process performance.

Table 8.9 Treatment achievability for naturally occurring chemicals that are of health significance in drinking-water

	Chlorination	Coagulation	Ion exchange	Precipitation softening	Activated Alumina	Activated Carbon	Ozonation	Membranes
Arsenic		+++ <0.005	+++ <0.005	+++ <0.005	+++ <0.005			+++ <0.005
Barium		+	+++ <0.1	+++ <0.1		+		+++ <0.1
Fluoride		++			+++ <1			+++ <1
Manganese	+++ <0.05	++					+++ <0.05	+++ <0.05
Selenium		++	+++ <0.01		+++ <0.01			+++ <0.01
Uranium		++	+++ <0.001	++	+++ <0.001			

Table 8.10 – Treatment achievability for chemicals from industrial sources and human dwellings that are of health significance in drinking-water

	Air stripping	Coagulation	Ion exchange	Precipitation softening	Activated Carbon	Ozonation	Advanced oxidation	Membranes
Cadmium		+++ <0.002	+++ <0.002	+++ <0.002				+++ <0.002
Mercury		+++ <0.0001		+++ <0.0001	+++ <0.0001			+++ <0.0001
Carbon tetrachloride	+++ <0.001	+			+++ <0.001			+++ <0.001
1,2-Dichloroethane	+				+++ <0.01	+	++	
1,1,1-Trichloroethane	+++ <0.01				+++ <0.01		+++ <0.01	
1,2-Dichloroethene	+++ <0.01				+++ <0.01	+++ <0.01		
Trichloroethene	+++ <0.02				+++ <0.02	+++ <0.02	+++ <0.02	
Tetrachloroethene	+++ <0.001				+++ <0.001			
Benzene	+++ <0.01				+++ <0.01	+++ <0.01		
Toluene	+++ <0.001				+++ <0.001	+++ <0.001	+++ <0.001	
Xylenes	+++ <0.005				+++ <0.005		+++ <0.005	
Ethylbenzene	+++ <0.001	+			+++ <0.001	+++ <0.001		
Styrene	+++ <0.02				+++ <0.02			
Monochlorobenzene	+++ <0.015				+++ <0.015	+++ <0.015	+++ <0.015	
1,2-Dichlorobenzene	+++ <0.01				+++ <0.01	+++ <0.01		
1,4-Dichlorobenzene	+++ <0.01				+++ <0.01	+++ <0.01		
Trichlorobenzenes (total)					+++ <0.01	+++ <0.01	+++ <0.01	
Hexachlorobutadiene					+++ <0.001			
Edetic acid (EDTA)					++ <0.01	++ <0.01		
Nitrilotriacetic acid						+++ No data		
1,4-Dioxane							+++ No data	

Table 8.11 – Treatment achievability for chemicals from agricultural activities that are of health significance in drinking-water

	Chlorination	Air stripping	Coagulation	Ion exchange	Activated Carbon	Ozonation	Advanced oxidation	Membranes	Biological treatment
Nitrate				+++ <5				+++ <5	+++ <5
Nitrite	+++ <0.1					+++ <0.1	+++ <0.1		
Alachlor					+++ <0.001	++	+++ <0.001	+++ <0.001	
Aldicarb	+++ <0.001				+++ <0.001	+++ <0.001		+++ <0.001	
Aldrin/dieldrin			++		+++ <0.00002	+++ <0.00002		+++ <0.00002	
Atrazine			+		+++ <0.0001	++	+++ <0.0001	+++ <0.0001	
Bentazone					++	+++ <0.0001	+++ <0.0001	+++ <0.0001	
Carbofuran	+				+++ <0.001			+++ <0.001	
Chlordane					+++ <0.0001	+++ <0.0001			
Chlorotoluron					+++ <0.0001	+++ <0.0001			
Cyanazine					+++ <0.0001	+		+++ <0.0001	
2,4-D (2,4-dichlorophenoxyacetic acid)			+		+++ <0.001	+++ <0.001			
1,2-Dibromo-3-chloropropane		++ <0.001			++ <0.001				
1,2-Dibromoethane		+++ <0.0001			+++ <0.0001				
1,2-Dichloropropane (1,2-DCP)					+++ <0.001	+		+++ <0.001	
Dichlorprop					+++ <0.001	+		+++ <0.001	
Dimethoate	+++ <0.001				++	++			
Diquat					+++ <0.02	+++ <0.02			
Endosulfan			+		+++ <0.0001	+			
Endrin			+		+++ <0.0002				
Fenitrothion						+++ <0.001			
Heptachlor and heptachlor epoxide	+				+++ <0.0001			+++ <0.0001	
Hexachlorobenzene			+		+++ <0.001	+++ <0.001	+++ <0.001		
Isoproturon	++				+++ <0.0001	+++ <0.0001	+++ <0.0001	+++ <0.0001	
Lindane					+++ <0.0001	++			
MCPA					+++ <0.0001	+++ <0.0001			
Mecoprop					+++ <0.0001	+++ <0.0001			
Methoxychlor			++		+++ <0.0001	+++ <0.0001			
Metolachlor					+++ <0.0001	++			
Pentachlorophenol					+++ <0.0004				
Simazine	+				+++ <0.0001	++	+++ <0.0001	+++ <0.0001	
2,4,5-T			++		+++ <0.001	+			
Terbuthylazine (TBA)			+		+++ <0.0001	++			
Trifluralin					+++ <0.0001			+++ <0.0001	

Table 8.12 – Treatment achievability for pesticides used in water for public health

	Chlorination	Coagulation	Activated Carbon	Ozonation	Advanced oxidation	Membranes
DDT and metabolites	+	+++ <0.0001	+++ <0.0001	+	+++ <0.0001	+++ <0.0001
Pyriproxyfen			+++ <0.001			

The following sections provide a brief review of the more commonly used water treatment processes.

8.4.2 Chlorination

Chlorination can be achieved by using liquefied chlorine gas, sodium hypochlorite solution or calcium hypochlorite granules. Liquefied chlorine gas is supplied in pressurised containers. The gas is withdrawn from the cylinder and is dosed into water by a chlorinator, which both controls and measures the gas flow rate. Sodium hypochlorite solution is dosed using a hypochlorinator, electric dosing pump or gravity feed system. Calcium hypochlorite has to be dissolved in water then mixed with the main supply. Chlorine, whether in the form of pure chlorine gas from a cylinder, sodium hypochlorite or calcium hypochlorite, dissolves in water to form hypochlorous acid (HOCl) and hypochlorite ion (OCl^-).

Several regimes of chlorination can be used, including simple chlorination, breakpoint chlorination, superchlorination/dechlorination and chloramination. Simple chlorination involves the dosing of 2 to 3 mg/litre chlorine to produce a suitable residual free chlorine concentration within the distribution system. In breakpoint chlorination, sufficient chlorine is added to exceed the demand for byproduct and chloramine production and to ensure a free available chlorine residual.

Chlorination is employed primarily for microbial disinfection. However it also acts as an oxidant and can remove or assist in the removal of chemicals, e.g.

- decomposition of easily-oxidised pesticides such as aldicarb;
- oxidation of dissolved species (e.g. manganese(II)) to form insoluble products that can be removed by subsequent filtration;
- oxidation of dissolved species to more easily removable forms (e.g. arsenite to arsenate).

A major disadvantage of chlorine is its ability to react with natural organics to produce trihalomethanes (THMs) and other disinfection by-products. However, by-product formation may be controlled by optimization of the treatment system.

8.4.3 Filtration

Particulate matter can be removed from raw waters by rapid gravity or slow sand filters. Slow sand filtration is essentially a biological process whereas rapid sand filtration is a physical treatment process.

Rapid gravity sand filters usually consist of open rectangular tanks (usually <100 m^2) containing silica sand (size range 0.5 to 1.0 mm) to a depth of between 0.6 and 2.0 m. The water flows downwards and solids become concentrated in the upper layers of the bed. The flowrate is generally in the range 4 to 20 $m^3/m^2.h$. Treated water is collected *via* nozzles in the floor of the filter. The accumulated solids are removed periodically by backwashing with treated water, sometimes preceded by scouring of the sand with air. A dilute sludge that requires disposal is produced.

Rapid gravity filters are most commonly used to remove floc from coagulated waters (see 8.4.5). They may also be used to reduce turbidity (including adsorbed chemicals) and oxidised iron and manganese from raw waters.

Slow sand filters usually consist of tanks containing sand (size range 0.15-0.30 mm) to a depth of between 0.5 to 1.5 m. The raw water flows downwards and turbidity and micro-organisms are removed primarily in the top few centimetres of the sand. A biological layer of sludge, known as the 'schmutzdecke', develops on the surface of the filter that can be effective in removing micro-organisms. Treated water is collected in underdrains or pipework at the bottom of the filter. The top few centimetres of sand containing the accumulated solids are removed and replaced periodically. Slow sand filters are operated a water flow rate of between 0.1 and 0.3 $m^3/m^2.h$.

Slow sand filters are used to remove algae and micro-organisms and if preceded by microstraining or coarse filtration, to reduce turbidity (including adsorbed chemicals). Slow sand filtration is effective for the removal of certain pesticides.

8.4.4 Aeration

Aeration processes are designed to achieve efficient removal of gases and volatile compounds by air stripping. Oxygen transfer can usually be achieved using a simple cascade or diffusion of air into water, without the need for elaborate equipment. Stripping of gases or volatile compounds, however, may require specialised plant that provides a high degree of mass transfer.

For oxygen transfer, cascade or step aerators are designed so that water flows in a thin film to achieve efficient mass transfer. Cascade aeration may introduce a significant headloss; design requirements are between 1 and 3 metres to provide a loading of 10 to 30 $m^3/m^2.h$. Alternatively, compressed air can be diffused through a system of submerged perforated pipes. These types of aerator are used for oxidation and precipitation of iron and manganese.

Aeration processes to achieve air stripping need to be much more elaborate to provide the necessary contact between the air and water. The most common technique is cascade aeration, usually in packed towers in which water is allowed to flow in thin films over plastic media with air blown counter-current. The required tower height and diameter are functions of the volatility and concentration of the compounds to be removed, and the flowrate.

Air stripping can be used for removal of volatile organics (e.g. solvents), some taste and odour causing compounds, and radon.

8.4.5 Chemical coagulation

Chemical coagulation based treatment is the most common approach for treatment of surface waters, and is almost always based on the following unit processes.

Chemical coagulants, usually salts of aluminium or iron, are dosed to the raw water under controlled conditions to form a solid flocculent metal hydroxide. Typical coagulant doses are 2 to 5 mg/litre as Al or 4 to 10 mg/litre as Fe. The precipitated floc removes suspended and dissolved contaminants by mechanisms of charge neutralisation, adsorption and entrapment. The efficiency of the coagulation process depends on raw water quality, the coagulant or coagulant aids used, and operational factors including mixing conditions, coagulation dose and pH value. The floc is removed from the treated water by subsequent solid-liquid separation processes such as sedimentation or flotation,
and/or rapid gravity filtration.

Powdered activated carbon (PAC) may be dosed during coagulation to adsorb organic chemicals such as some pesticides. The PAC will be removed as an integral fraction of the floc and disposed of with the waterworks sludge.

The floc may be removed by sedimentation to reduce the solids loading to the subsequent rapid gravity filters. Sedimentation is most commonly achieved in horizontal flow or floc blanket clarifiers. Alternatively, floc may be removed by dissolved air flotation, in which solids are contacted with fine bubbles of air which attach to the floc causing them to float to the surface of the tank where they are removed periodically as a layer of sludge. The treated water from either process is passed to rapid gravity filters, where remaining solids are removed.

Rapid gravity filtration (see 8.4.3) is used to remove floc from coagulated water (usually preceded by sedimentation). Filtered water may be passed to a further stage of treatment, such as additional oxidation and filtration (for removal of manganese), to ozonation and/or GAC adsorption (for removal of pesticides and other trace organics), prior to final disinfection before the treated water enters supply.

Coagulation is suitable for removal of certain heavy metals and low solubility organic chemicals such as certain organochlorine pesticides. For other organic chemicals, coagulation is generally ineffective, except where the chemical is bound to humic material or adsorbed onto particulates.

8.4.6 Activated carbon adsorption

Activated carbon is produced by the controlled combustion of carbonaceous material, normally wood, coal, coconut shells or peat. This activation produces a porous material with a large surface area (500-1500 m^2/g) and a high affinity for organic compounds. It is normally used either in powdered (PAC) or granular (GAC) form. When the adsorption capacity of the carbon is exhausted, it can be reactivated by burning off the organics in a controlled manner. However, PAC (and some GAC) is normally used only once before disposal.

PAC is dosed as a slurry into the water, and is removed by subsequent treatment processes together with the waterworks sludge. Its use is therefore restricted to surface water treatment works with existing filters. The choice between PAC and GAC will depend upon the

frequency and dose of PAC required. PAC would generally be preferred in the case of seasonal or intermittent contamination, or where low dosage rates are required.

GAC is normally used in fixed beds, either in purpose-built adsorbers or in existing filter shells by replacement of sand with GAC of a similar particle size. Although at most treatment works it would be cheaper to convert existing filters rather than build separate adsorbers, use of existing filters usually allows only short contact times. It is therefore normal practice to install additional GAC adsorbers (some preceded by ozonation) between the rapid gravity filters and final disinfection. Most groundwater sources do not have existing filters, and separate adsorbers would need to be installed.

GAC in fixed bed adsorbers is used much more efficiently than PAC dosed into the water, and the effective carbon dose would be much lower than the dose of PAC required to achieve the same removal.

The service life of the bed is dependent on the capacity of the carbon used and the contact time between the water and the carbon, the empty bed contact time (EBCT), controlled by the flowrate of the water. EBCTs are usually in the range 5 to 30 minutes. GACs vary considerably in their capacity for specific organic compounds, which can have a considerable effect upon their service life. A guide to capacity can be obtained from published isotherm data. Carbon capacity is strongly dependent on the water source and is greatly reduced by the presence of background organic compounds. The properties of a chemical that influence its adsorption onto activated carbon include the water solubility and octanol-water partition coefficient. As a general rule chemicals with low solubility and high $\log K_{ow}$ are well adsorbed.

Activated carbon is used for the removal of pesticides and other trace organic chemicals, taste and odour compounds, algal toxins, and total organic carbon (TOC).

8.4.7 Ion-exchange

Ion-exchange is a process in which ions of like charge are exchanged between the water phase and the solid resin phase. Water softening is achieved by cation exchange. Water is passed through a bed of cationic resin and the calcium ions and magnesium ions in the water are replaced by sodium ions. When the ion-exchange resin is exhausted, i.e. the sodium ions are depleted, it is regenerated using a solution of sodium chloride. The process of ‘de-alkalisation’ can also soften water. Water is passed through a bed of weakly acidic resin and the calcium ions and magnesium ions are replaced by hydrogen ions. The hydrogen ions react with the carbonate and bicarbonate ions to produce carbon dioxide. The hardness of the water is thus reduced without any increase in sodium levels. Anion exchange can be used to remove contaminants such as nitrate, which is exchanged for chloride. Nitrate-specific resins are available for this purpose.

Ion-exchange plant normally consists of two or more resin beds contained in pressure shells with appropriate pumps, pipework and ancillary equipment for regeneration. The pressure shells are typically up to 4 m in diameter, containing 0.6 to 1.5 m depth of resin.

Cation exchange can be used for removal of certain heavy metals. Potential applications of anionic resins, in addition to nitrate removal, are for removal of arsenic and selenium species.

8.4.8 Ozonation

Ozone is a powerful oxidant and has many uses in water treatment, including oxidation of organic chemicals. Ozonation is generally used as a primary disinfection. Ozone gas (O_3) 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 to 20 minutes contact time. Dissolution of at least 80% 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 concentration after a given contact period. For oxidation of organic chemicals, such as pesticides, a residual of about 0.5 mg/litre 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 to 5 mg/litre. 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.

Ozone is effective for the removal of a wide range of pesticides and other organic chemicals by degradation. However, since ozonation does not provide a disinfection residual with the distribution system, a secondary disinfection such as chlorine or chloramines must be added to control microbial growth.

8.4.9 Advanced oxidation processes

Chemicals can react either directly with molecular ozone or with the hydroxyl radical, which is a product of the decomposition of ozone in water. The hydroxyl radical (OH) is an exceedingly powerful indiscriminate oxidant that reacts readily with a wide range of target organic chemicals. The formation of hydroxyl radicals can be encouraged by using ozone at high pH.

Other processes have been developed that are aimed at generating hydroxyl radicals. These are known collectively as Advanced Oxidation Processes (AOPs). These include ozone plus hydrogen peroxide, ozone plus ultraviolet (UV) irradiation and hydrogen peroxide with UV irradiation. Of these, only ozone plus hydrogen peroxide is used at full-scale for drinking-water treatment. The process involves dosing hydrogen peroxide simultaneously with ozone at a rate of approximately 0.4 mg/l hydrogen peroxide per mg/l ozone dosed (the theoretical optimum ratio for hydroxyl radical production).

AOPs can be effective for the destruction of chemicals that are difficult to treat using other methods such as ozone alone.

8.4.10 Membrane processes

The membrane processes of most significance in water treatment are reverse osmosis, ultrafiltration, microfiltration and nanofiltration. These processes have traditionally been

applied to the production of water for industrial or pharmaceutical applications but are now being applied to the treatment of drinking-water.

If two solutions are separated by a semi-permeable membrane, i.e. a membrane that allows the passage of solvent but not of the solute, the solvent will pass from the lower concentration solution to the higher concentration solution. This process is known as osmosis. It is possible, however, to force the flow of solvent in the opposite direction, from the higher to the lower concentration, by increasing the pressure on the higher concentration solution. The required pressure differential is known as the osmotic pressure and the process as reverse osmosis.

Reverse osmosis results in the production of a treated water stream and a relatively concentrated waste stream. Typical operating pressures are in the range 15 to 50 bar depending on the application. Membrane pore sizes are less than 0.002 μm. The most common application of reverse osmosis is desalination of sea water although the use of reverse osmosis for nitrate removal has also been proposed.

Ultrafiltration is similar in principle to reverse osmosis, but the membranes have much larger pore sizes (typically 0.002 to 0.03 μm) and operate at lower pressures. Ultrafiltration membranes reject organic molecules of molecular weight above 800 and usually operate at pressures less than 5 bar.

Nanofiltration uses a membrane with properties between those of reverse osmosis and ultrafiltration membranes; pore sizes are typically 0.001 to 0.01 μm. Nanofiltration membranes allow monovalent ions such as sodium or potassium to pass but reject a high proportion of divalent ions such as calcium and magnesium and organic molecules of molecular weight greater than 200. Operating pressures are typically about 5 bar. Nanofiltration may be effective for the removal of colour and organic compounds.

Microfiltration is a direct extension of conventional filtration into the sub-micron range. Microfiltration membranes have pore sizes typically in the range 0.01 to 12 μm and do not separate molecules but reject colloidal and suspended material at operating pressures of 1 to 2 bar. Microfiltration is capable of sieving out particles greater than 0.05 μm. It has been used for water treatment in combination with coagulation or powdered activated carbon (PAC) to remove dissolved organic carbon and to improve permeate flux.

8.4.11 Other processes

Other treatment processes that can be used in certain applications include:

- Precipitation softening – addition of lime, lime plus sodium carbonate or sodium hydroxide to precipitate hardness at high pH;
- Biological denitrification for removal or nitrate from surface waters;
- Biological nitrification for removal of ammonia from surface waters;
- Activated alumina (or other adsorbents) for specialized applications such as removal of fluoride and arsenic.

8.4.12 Disinfection by-products – process control measures

All chemical disinfectants produce either or both inorganic and organic disinfection by-products (DBPs) that may be of concern.

The principal DBPs formed during chlorination are trihalomethanes (THMs) as a result of chlorination of naturally occurring organic precursors such as humic substances. Other DBPs found following chlorination include chlorinated acetic acids, chlorinated ketones and haloacetonitriles. Chloramine produces lower THM concentrations than chlorination but does produce other DBPs including cyanogen chloride.

Ozone can form brominated THMs through the oxidation of bromide to produce hypobromous acid, which brominates precursors. 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 is also a potential impurity in sodium hypochlorite. The main by-products from the use of chlorine dioxide are chlorite ion, which is an inevitable decomposition product, and chlorate ion.

The basic strategies that can be adopted for reducing the concentrations of DBPs are:

- changing process conditions (including removal of precursor compounds),
- using a different chemical disinfectant,
- using non-chemical disinfection, or
- removal of DBPs (prior to distribution).

These strategies are outlined below.

> ***In attempting to control DBP concentrations it is of paramount importance to ensure that the efficiency of disinfection is not compromised and that a residual level of disinfectant is maintained throughout the distribution system.***

Changes to process conditions

The formation of THMs during chlorination can be reduced by removing precursors prior to contact with chlorine, for example by installing or enhancing coagulation (this may involve using higher coagulant doses and/or lower coagulation pH than are applied conventionally). THM formation can also be reduced by lowering the applied chlorine dose but if this is done it must be ensured that disinfection is still effective.

The formation of bromate during ozonation depends on several factors including concentrations of bromide and ozone and the pH. It is not practicable to remove bromide from the 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 (e.g. 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.

Changing disinfectants
It may be feasible to change disinfectant in order to achieve guideline values for DBPs. The extent to which this is possible will be dependent on raw water quality and installed treatment, e.g. for precursor removal.

It may be effective to change from chlorine to chloramine, at least to provide a residual disinfectant within distribution, in order to reduce THM formation and subsequent development within the distribution system. Whilst chloramine provides a more stable residual within distribution, it is generally considered to be a less powerful disinfectant and should not be used as a primary disinfectant.

Chlorine dioxide can be considered as a potential alternative to both chlorine and ozone. The main concerns with chlorine dioxide are with the residual concentrations of chlorine dioxide, and the by-products chlorite and chlorate. These can be addressed by controlling the dose of chlorine dioxide at the treatment plant.

Non-chemical disinfection
Ultraviolet (UV) irradiation or membrane processes could be considered as alternatives to chemical disinfection. Neither of these provides any residual disinfection and it may be considered appropriate to add a small dose of a persistent disinfectant such as chloramine to act as a preservative during distribution.

8.4.13 Treatment for corrosion control

General
Corrosion is the partial dissolution of the materials constituting the treatment and supply systems, tanks, pipes, valves, and pumps. It may lead to structural failure, leaks, loss of capacity, and deterioration of chemical and microbiological water quality. The internal corrosion of pipes and fittings can have a direct impact on the concentration of some water constituents, including lead and copper. Corrosion control is therefore an important aspect of the management of a water supply system.

Corrosion control involves many parameters, including the concentrations of calcium, bicarbonate, carbonate, and dissolved oxygen, as well as pH. The detailed requirements differ depending on water quality and the materials used in the distribution system. The pH controls the solubility and rate of reaction of most of the metal species involved in corrosion reactions. It is particularly important in relation to the formation of a protective film at the metal surface. For particular metals, alkalinity (carbonate and bicarbonate) and calcium (hardness) also affect corrosion rates.

Iron
Iron is the material most commonly used in water distribution systems and its corrosion is of concern. Whilst structural failure as a result of iron corrosion is rare, water quality problems (e.g. “red water”) can arise as a result of excessive corrosion of iron pipes. The corrosion of

iron is a complex process that involves the oxidation of the metal, normally by dissolved oxygen, ultimately to form a precipitate of iron(III). This leads to the formation of tubercules on the pipe surface. The major water quality factors that determine whether the precipitate forms a protective scale are pH and alkalinity. The concentrations of calcium, chloride and sulfate also influence iron corrosion. Successful control of iron corrosion has been achieved by adjusting the pH to the range 6.8 to 7.3, hardness and alkalinity to at least 40 mg/l (as $CaCO_3$), over-saturation with $CaCO_3$ of 4 to 10 mg/l, and a ratio of alkalinity to $Cl^- + SO_4^{2-}$ of at least 5 (when both are expressed as $CaCO_3$).

Lead

Lead corrosion (plumbosolvency) is of particular concern. Lead piping is still common in old houses, and lead solders have been used widely for jointing copper tube. The solubility of lead is governed by the formation of lead carbonates as pipe deposits. The solubility of lead increases markedly as the pH is reduced below 8 because of the substantial decrease in the equilibrium carbonate concentration. Thus, plumbosolvency tends to be at a maximum in waters with a low pH and low alkalinity, and a useful interim control procedure pending pipe replacement is to maintain pH in the range 8.0 to 8.5 and possibly to dose orthophosphate.

Lead can corrode more rapidly when it is coupled to copper. The rate of such galvanic corrosion is faster than that of simple oxidative corrosion, and lead concentrations are not limited by the solubility of the corrosion products. The rate of corrosion is affected principally by chloride concentration. Galvanic corrosion is less easily controlled but can be reduced by dosing zinc in conjunction with orthophosphate, and by adjustment of pH.

Copper

The corrosion of copper pipework and hot water cylinders can cause blue water, blue or green staining of bathroom fittings and, occasionally, taste problems. Copper tubing may be subject to general corrosion, impingement attack and pitting corrosion. General corrosion is most often associated with soft, acid waters; waters with pH below 6.5 and hardness of less than 60 mg/l $CaCO_3$ are very aggressive to copper. Copper, like lead, can enter water by dissolution of the corrosion product, basic copper carbonate. The solubility is mainly a function of pH and total inorganic carbon. Solubility decreases with increase in pH, but increases with increase in concentrations of carbonate species. Raising the pH to between 8 and 8.5 is the usual procedure to overcome these difficulties.

Impingement attack is the result of excessive flow velocities and is aggravated in soft water at high temperature and low pH. The pitting of copper is commonly associated with hard groundwaters having a carbon dioxide concentration above 5 mg/l and high dissolved oxygen. Surface waters with organic colour may also be associated with pitting corrosion. A high proportion of general and pitting corrosion problems are associated with new pipe in which a protective oxide layer has not yet formed.

Copper pipes can fail by pitting corrosion which involves highly localized attacks leading to perforations with negligible loss of metal. Two main types of attack are recognized.
Type I pitting affects cold water systems (below 40°C) and is associated particularly with hard borehole waters and the presence of a carbon film in the bore of the pipe, derived from the manufacturing process. Tubes that have had the carbon removed by cleaning are immune from Type I pitting. Type II pitting occurs in hot water systems (above 60°C) and is associated with soft waters.

Zinc

The solubility of zinc in water is a function of pH and total inorganic carbon concentrations; the solubility of basic zinc carbonate decreases with increase in pH and carbonate species concentrations. For low alkalinity waters increase of pH to 8.5 should be sufficient to control the dissolution of zinc.

With galvanized steel the zinc layer initially protects the steel by corroding preferentially. In the long-term a protective deposit of basic zinc carbonate forms. Protective deposits do not form in soft waters where the alkalinity is less than 50 mg/l as $CaCO_3$ or waters containing high CO_2 concentrations (>25 mg/l as CO_2) and galvanized steel is unsuitable for these waters. The corrosion of galvanized steel can be increased by coupling with copper tubing.

Zinc also can be selectively removed from duplex brass by dezincification – selective dissolution of zinc leaving behind copper as a porous mass of low mechanical strength. Meringue dezincification, in which a voluminous corrosion product of basic zinc carbonate forms on the brass surface, largely depends on the ratio of chloride to alkalinity. Meringue dezincification can be controlled by maintaining a low ratio (1:3 or lower), and by keeping the pH below 8.3.

Nickel

Elevated concentrations of nickel may arise due to the leaching of nickel from new nickel-chromium plated taps and from stainless steel pipes and fittings. Nickel leaching falls off over time. Increase of pH to control corrosion of other materials should also help to reduce leaching of nickel.

Concrete and cement

Concrete is a composite material consisting of a cement binder in which an inert aggregate is embedded. Cement is primarily a mixture of calcium silicates and aluminates together with some free lime. Cement mortar, in which the aggregate is fine sand, is used as a protective lining in iron and steel water pipes. In asbestos–cement pipe, the aggregate is asbestos fibres. Cement is subject to deterioration on prolonged exposure to aggressive water – due either to the dissolution of lime and other soluble compounds or to chemical attack by aggressive ions such as chloride or sulfate – and this may result in structural failure. Aggressiveness to cement is related to the 'Aggressivity Index', which has been used specifically to assess the potential for the dissolution of concrete. A pH of 8.5 or higher may be necessary to control cement corrosion.

Characterizing corrosivity

Most of the indices that have been developed to characterize the corrosion potential of waters are based on the assumption that water with a tendency to deposit a calcium carbonate scale on metal surfaces will be less corrosive. The Langelier Index (LI) is the difference between the actual pH of a water and its 'saturation pH', this being the pH at which a water of the same alkalinity and calcium hardness would be at equilibrium with solid calcium carbonate. Waters with positive LI are capable of depositing calcium carbonate scale from solution.

There is no corrosion index that applies to all materials, and corrosion indices, particularly those related to calcium carbonate saturation, have given mixed results. The parameters related to calcium carbonate saturation status are, strictly speaking, indicators of the tendency to deposit or dissolve calcium carbonate (calcite) scale, not indicators of the 'corrosivity' of a water. For example there are many waters with negative Langelier Index that are non-

corrosive, and many with positive LI that are corrosive. Nevertheless there are many documented instances of the use of saturation indices for corrosion control based on the concept of laying down a protective 'eggshell' scale of calcite in iron pipes. In general waters with high pH, calcium and alkalinity are less corrosive and this tends to be correlated with a positive LI.

The ratio of the chloride and sulfate concentrations to the bicarbonate concentration (Larson ratio) has been shown to be helpful in assessing the corrosiveness of water to cast iron and steel. A similar approach has been used in studying zinc dissolution from brass fittings - the Turner diagram.

Water treatment for corrosion control

To control corrosion in water distribution networks the methods most commonly applied are adjusting pH, increasing the alkalinity and/or hardness, or adding corrosion inhibitors such as sodium polyphosphates or silicates and orthophosphate. The quality and maximum dose to be used should be in line with appropriate national specifications for such water treatment chemicals. Although pH adjustment is an important approach its possible impact on other aspects of water supply technology, including disinfection, must always be taken into account.

It is not always possible to achieve the desired values for all parameters. For example, the pH of hard waters cannot be increased too much or softening will occur. The application of lime and carbon dioxide to soft waters can be used to increase both the calcium concentration and the alkalinity to at least 40 mg/l as $CaCO_3$.

Silicates and polyphosphates are often described as corrosion inhibitors but there is no guarantee that they will inhibit corrosion in water distribution systems. However, they can complex dissolved iron (in the iron(II) state) and prevent its precipitation as visibly obvious red "rust". These compounds may act by masking the effects of corrosion rather than by preventing it. Orthophosphate is a possible corrosion inhibitor and is used to prevent "red water", like polyphosphates.

Treatment to reduce plumbosolvency usually involves pH adjustment. When the water is very soft (less than 50 mg/l $CaCO_3$), the optimum pH is about 8.0 to 8.5. Alternatively, dosing with orthophosphoric acid or sodium orthophosphate might be more effective particularly when plumbosolvency occurs in non-acidic waters. Wherever practicable, lead pipework should be replaced.

8.5 Provisional Guideline Values

When the health-based GV is less than the level that can be determined by a routine analytical method, the GV is set at the analytical level that can be reasonably achieved (practical quantitation limit). Such values are denoted with an "A" in the summary table (rather than a "P", as was done in previous editions).

If the health-based GV can not be achieved through realistic technical means such as treatment, then the GV is set at the practical treatment level. From the third edition of the GDWQ, these values are denoted with a "T" in the summary table, rather than a "P" as was done previously and an explanatory footnote added.

Table 8.13 Designation of provision Guideline Values

Situations where a provisional guideline applies	Designation	
Significant scientific uncertainties regarding derivation of health based levels	P	
Calculated GLV is below the practical quantification level	A	*(Guideline value is maintained at achievable quantification level)*
Calculated GLV is below the level that can be achieved through practical treatment methods	T	*(Guideline value is set at the practical treatment limit)*
Calculated GLV is exceeded as a result of disinfection procedures	D	*(Guideline value is set on the basis of health, but disinfection of drinking-water remains paramount)*

Where a proposed guideline value for disinfectants or disinfection by-products may discourage disinfection, guideline values are set at the health-based value and have been designated as provisional "D" with an explanatory footnote. This follows from the principles of protection of public health and giving priority to microbial contaminants.

Some substances of health concern have aesthetic effects that would normally lead to rejection of water at concentrations significantly lower than those of concern for health. Such substances are not normally appropriate for routine monitoring. Nevertheless, health-based reference values may be needed, for instance, for use in interpreting data collected in response to consumer complaints. In these circumstances, a health-based summary statement is prepared and guideline values derived in the usual way. In the summary statement, the relationship between concentrations relevant for health and aesthetic concern is explained. In tables of guideline values, the health-based guideline values are designated (C). In footnotes to tables it is explained that whilst of health significance, because water would normally be rejected by consumers they would not normally be considered appropriate for routine monitoring; but that they would be of importance in responding to consumer complaints and guideline values are included for this purpose.

Provisional guideline values where there are significant scientific uncertainties including the use of uncertainty factors greater than 1000 are designated as (P).

8.6 Mixtures

Chemical contaminants of drinking-water supplies are present with numerous other inorganic or organic constituents. The guideline values are calculated separately for individual substances, without specific consideration of the potential for interaction of each substance with other compounds present. However, the large margin of safety incorporated in the majority of the guideline values is considered to be sufficient to account for potential interactions. In addition, the majority of contaminants will not be present at concentrations at or near their guideline value.

There may, however, be occasions when a number of contaminants with similar toxicological effects are present at levels near their respective guideline value. In such cases, decisions concerning appropriate action should be made, taking into consideration local circumstances. Unless there is evidence to the contrary, it is appropriate to assume that the toxic effects of these compounds are additive.

8.7 CATEGORIES OF CHEMICALS

8.7.1 Naturally occurring Chemicals

There are a number of sources of naturally occurring chemicals in drinking-water. All natural water contains a range of inorganic and organic chemicals. The former derive from the rocks and soil through which water percolates or over which it flows. The latter derive from the breakdown of plant material or from algae and other microorganisms that grow in the water or on sediments. Most of the naturally occurring chemicals for which guidelines have been derived or that have been considered for guideline derivation are inorganic. Only one, microcystin-LR, a toxin produced by cyanobacteria, or blue-green algae, is organic, and it is discussed separately in section 8.7.6.

The approach to dealing with naturally occurring chemicals will vary according to the nature of the chemical and the source. For inorganic contaminants that arise from rocks and sediments, it is important to screen possible water sources to determine whether the source is suitable for use or whether it will be necessary to treat the water to remove the contaminants of concern along with microbial contaminants. In some cases, where a number of sources may be available, dilution or blending of the water containing high levels of a contaminant with a water containing much lower levels may achieve the desired result.

A number of the most important chemical contaminants — i.e., those that have been shown to cause adverse health effects as a consequence of exposure through drinking-water — fall into the category of naturally occurring chemicals. Some naturally occurring chemicals have other primary sources and are therefore discussed in other sections of this chapter.

Guideline values have not been established for the chemicals listed in Table 8.4 because:
evidence indicates that the chemical does not occur at concentrations at or near levels expected to cause a health concern, or
there is lack of evidence of health effects, or there are insufficient data to support the establishment of a guideline value.

Table 8.14. Naturally occurring chemicals for which guideline values have not been established

Chemical	Reason for exclusion	Remarks
Chloride	Not of health concern at levels found in drinking-water	May affect acceptability of drinking-water (see chapter 10)
Hardness	Not of health concern at levels found in drinking-water	May affect acceptability of drinking-water (see chapter 10)
Hydrogen sulfide	Not of health concern at levels found in drinking-water	May affect acceptability of drinking-water (see chapter 10)
PH	Not of health concern at levels found in drinking-water	An important operational water quality parameter
Sodium	Not of health concern at levels found in drinking-water	May affect acceptability of drinking-water (see chapter 10)
Sulfate	Not of health concern at levels found in drinking-water	May affect acceptability of drinking-water (see chapter 10)
Total dissolved solids (TDS)	Not of health concern at levels found in drinking-water	May affect acceptability of drinking-water (see chapter 10)

Guideline values have been established for the chemicals listed in Table 8.5, which meet all of the criteria for inclusion.

Table 8.15. Guideline values for naturally occurring chemicals that are of health significance in drinking-water

Chemical	Guideline value[a] (mg/litre)	Remarks
Arsenic	0.01 (P)	
Barium	0.7	
Boron	0.5 (T)	
Chromium	0.05 (P)	For total chromium
Fluoride	1.5	Volume of water consumed and intake from other sources should be considered when setting national standards
Manganese	0.4	C[b]
Molybdenum	0.07	
Selenium	0.01	
Uranium	0.009 (P, T)	Only chemical aspects of uranium addressed

[a] Abbreviations used for provisional guideline values are as follows: P = evidence of a potential hazard but the available information on health effects is limited; T = calculated guideline value is below the level that can be achieved through practical treatment methods, source protection, etc.

[b] C = concentrations of the substance at or below the health-based guideline value may affect the appearance, taste or odour of the water, resulting in consumer complaints.

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 from 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.

Provisional guideline value	0.01 mg/litre
Occurrence	Levels in natural waters generally vary between 1 and 2 µg/litre, although concentrations may be elevated (up to 12 mg/litre) in areas containing natural sources.
Basis of guideline derivation	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, a guideline value of 10 µg/litre is retained. In view of the scientific uncertainties, the guideline value is designated as provisional.
Limit of detection	0.1 µg/litre by inductively coupled plasma/mass spectrometry; 2 µg/litre by hydride generation or flame atomic absorption spectrometry
Treatment achievability	It is technically feasible to achieve arsenic concentrations of 5 µg/litre or lower using any of several possible treatment methods. However, this requires careful process optimization and control, and a more reasonable expectation is that 10 µg/litre should be achievable by conventional treatment, e.g., coagulation.
Additional comments	A management guidance document on arsenic is available. In many countries, this guideline value may not be attainable. Where this is the case, every effort should be made to keep concentrations as low as possible.

Toxicological Review

Arsenic has not been demonstrated to be essential in humans. It 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. 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 curves at low intakes. Inorganic arsenic compounds are classified by IARC in Group 1 (carcinogenic to humans) on the basis of sufficient evidence for carcinogenicity in humans and limited evidence for carcinogenicity in animals.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* recommended a maximum allowable concentration of 0.2 mg/litre for arsenic, based on health concerns. In the 1963 International Standards, this value was lowered to 0.05 mg/litre, which was retained as a tentative upper concentration limit in the 1971 International Standards. The guideline value of 0.05 mg/litre was also retained in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984. A provisional guideline value for arsenic was set at the practical quantification limit of 0.01 mg/litre in the 1993 Guidelines, based on concern regarding its carcinogenicity in humans.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Barium

Barium is present as a trace element in both igneous and sedimentary rocks, and barium compounds are used in a variety of industrial applications, but barium in water comes primarily from natural sources. Food is the primary source of intake for the non-occupationally exposed population. However, where barium levels in water are high, drinking-water may contribute significantly to total intake.

Guideline value	0.7 mg/litre
Occurrence	Concentrations in drinking-water are generally below 100 µg/litre, although concentrations above 1 mg/litre have been measured in drinking-water derived from groundwater.
NOAEL in humans	7.3 mg/litre in the most sensitive epidemiological study conducted to date, in which there were no significant differences in blood pressure or in the prevalence of cardiovascular disease between a population drinking water containing a mean barium concentration of 7.3 mg/litre and one whose water contained a concentration of 0.1 mg/litre
Limit of detection	0.1 µg/litre by inductively coupled plasma/mass spectrometry; 2 µg/litre by atomic absorption spectroscopy; 3 µg/litre by inductively coupled plasma/optical emission spectroscopy
Treatment achievability	0.1 mg/litre should be achievable using either ion exchange or precipitation softening; other conventional processes are ineffective

Toxicological Review

There is no evidence that barium is carcinogenic or mutagenic. 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.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* did not refer to barium. The 1963 International Standards recommended a maximum allowable concentration of 1.0 mg/litre, based on health concerns. The 1971 International Standards stated that barium should be controlled in drinking-water, but that insufficient information was available to enable a tentative limit to be established. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was concluded that it was not necessary to establish a guideline value for barium in drinking-water, as there was no firm evidence of any health effects associated with the normally low levels of barium in water. A health-based guideline value of 0.7 mg/litre was derived for barium in the 1993 Guidelines, based on concern regarding the potential of barium to cause hypertension.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Boron

Boron compounds are used in the manufacture of glass, soaps and detergents and as flame retardants. The general population obtains the greatest amount of boron through food intake. Boron is found naturally in groundwater, but its presence in surface water is frequently a consequence of the discharge of treated sewage effluent, in which it arises from use in some detergents, to surface waters.

Provisional guideline value	0.5 mg/litre
Occurrence	Concentrations vary widely and depend on the surrounding geology and wastewater discharges. For most of the world, the concentration range of boron in drinking-water is judged to be between 0.1 and 0.3 mg/litre.
TDI	0.16 mg/kg of body weight, based on a NOAEL of 9.6 mg/kg of body weight per day for developmental toxicity (decreased fetal body weight in rats) and an uncertainty factor of 60 (10 for interspecies variation and 6 for intraspecies variation)
Limit of detection	0.2 µg/litre by inductively coupled plasma/mass spectroscopy; 6–10 µg/litre by inductively coupled plasma/atomic emission spectroscopy
Treatment achievability	Conventional water treatment (coagulation, sedimentation, filtration) does not significantly remove boron, and special methods need to be installed in order to remove boron from waters with high boron concentrations. Ion exchange and reverse osmosis processes may enable substantial reduction but are likely to be prohibitively expensive. Blending with low-boron supplies may be the only economical method to reduce boron concentrations in waters where these concentrations are high.

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Additional comments	The guideline is designated as provisional because it will be difficult to achieve in areas with high natural boron levels with the treatment technology available. The supporting document on boron is to be revised in response to the recent EHC on boron as well as later emerging data.

Toxicological Review

Short- and long-term oral exposures to boric acid or borax in laboratory animals have demonstrated that the male reproductive tract is a consistent target of toxicity. Testicular lesions have been observed in rats, mice and dogs given boric acid or borax in food or drinking-water. Developmental toxicity has been demonstrated experimentally in rats, mice and rabbits. Negative results in a large number of mutagenicity assays indicate that boric acid and borax are not genotoxic. In long-term studies in mice and rats, boric acid and borax caused no increase in tumour incidence.

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to boron. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was concluded that no action was required for boron. A health-based guideline value of 0.3 mg/litre for boron was established in the 1993 Guidelines, while noting that boron's removal by drinking-water treatment appears to be poor. This guideline value was increased to 0.5 mg/litre in the addendum to the Guidelines published in 1998 and was designated as

provisional because, with the treatment technology available, the guideline value will be difficult to achieve in areas with high natural boron levels.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

Chloride

Chloride in drinking-water originates from natural sources, sewage and industrial effluents, urban runoff containing de-icing salt and saline intrusion.

The main source of human exposure to chloride is the addition of salt to food, and the intake from this source is usually greatly in excess of that from drinking-water.

Excessive chloride concentrations increase rates of corrosion of metals in the distribution system, depending on the alkalinity of the water. This can lead to increased concentrations of metals in the supply.
No health-based guideline value is proposed for chloride in drinking-water. However, chloride concentrations in excess of about 250 mg/litre can give rise to detectable taste in water (see chapter 10).

History of Guideline Development
The 1958 WHO *International Standards for Drinking-water* suggested that concentrations of chloride greater than 600 mg/litre would markedly impair the potability of the water. The 1963 and 1971 International Standards retained this value as a maximum allowable or permissible concentration. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 250 mg/litre was established for chloride, based on taste considerations. No health-based guideline value for chloride in drinking-water was proposed in the 1993 Guidelines, although it was confirmed that chloride concentrations in excess of about 250 mg/litre can give rise to detectable taste in water.

Primary Reference
WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Chromium

Chromium is widely distributed in the Earth's crust. It can exist in valences of +2 to +6. In general, food appears to be the major source of intake.

Provisional guideline value	0.05 mg/litre for total chromium
Occurrence	Total chromium concentrations in drinking-water are usually less than 2 µg/litre, although concentrations as high as 120 µg/litre have been reported.
Basis of guideline value derivation	There are no adequate toxicity studies available to provide a basis for 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.
Limit of detection	0.05–0.2 µg/litre for total chromium by atomic absorption spectroscopy
Treatment achievability	0.015 mg/litre should be achievable using coagulation
Additional comments	The guideline value is designated as provisional because of uncertainties in the toxicological database. The supporting document on chromium will be revised once the results of new toxicology studies by the NTP are made available.

Toxicological Review

In a long-term carcinogenicity study in rats given chromium(III) by the oral route, no increase in tumour incidence was observed. In rats, chromium(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 chromium(VI) by the inhalation route and lung cancer. IARC has classified chromium(VI) in Group 1 (human carcinogen) and chromium(III) in Group 3. Chromium(VI) compounds are active in a wide range of *in vitro* and *in vivo* genotoxicity tests, whereas chromium(III) compounds are not.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* recommended a maximum allowable concentration of 0.05 mg/litre for chromium (hexavalent), based on health concerns. This value was retained in the 1963 International Standards. Chromium was not evaluated in the 1971 International Standards. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, the guideline value of 0.05 mg/litre for total chromium was retained; total chromium was specified because of difficulties in analysing for the hexavalent form only. The 1993 Guidelines questioned the guideline value of 0.05 mg/litre because of the carcinogenicity of hexavalent chromium by the inhalation route and its genotoxicity, although the available toxicological data did not support the derivation of a new value. As a practical measure, 0.05 mg/litre, which is considered to be unlikely to give rise to significant health risks, was retained as the provisional guideline value until additional information becomes available and chromium can be re-evaluated.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.
Health criteria and other supporting information

Fluoride

Fluoride accounts for about 0.3 g/kg of the Earth's crust and exists in the form of fluorides in a number of minerals. The most important source of fluoride in drinking-water is naturally occurring. Inorganic fluoride-containing minerals are used widely in industry for a wide range of purposes, including aluminium production. Fluorides can be released to the environment from the phosphate-containing rock used to produce phosphate fertilizers; these phosphate deposits contain about 4% fluorine. Fluorosilicic acid, sodium hexafluorosilicate and sodium fluoride are used in municipal water fluoridation schemes. Daily exposure to fluoride depends mainly on the geographical area. In most circumstances, food seems to be the primary source of fluoride intake, with lesser contributions from drinking-water and from toothpaste. In areas with relatively high concentrations, particularly in groundwater, drinking-water becomes increasingly important as a source of fluoride. Intakes in areas where high-fluoride coal is used indoors may also be significant.

Guideline value	1.5 mg/litre
Occurrence	In groundwater, concentrations vary with the type of rock the water flows through but do not usually exceed 10 mg/litre; the highest natural level reported is 2800 mg/litre.
Basis of guideline derivation	Epidemiological evidence that concentrations above this value carry an increasing risk of dental fluorosis, and progressively higher concentrations lead to increasing risks of skeletal fluorosis. The value is higher than that recommended for artificial fluoridation of water supplies.
Limit of detection	0.01 mg/litre by ion chromatography; 0.1 mg/litre by ion-selective electrodes or the SPADNS (sulfo phenyl azo dihydroxy naphthalene disulfonic acid) colorimetric method
Treatment achievability	1 mg/litre should be achievable using activated alumina (not a "conventional" treatment process, but relatively simple to install filters)
Additional comments	A management guidance document on fluoride is available. In setting national standards for fluoride or in evaluating the possible health consequences of exposure to fluoride, it is essential to consider the intake of water by the population of interest and the intake of fluoride from other sources (e.g., from food and air). Where the intakes are likely to approach, or be greater than, 6 mg/day, it would be appropriate to consider setting standards at a lower concentration than the guideline value. In areas with high natural fluoride levels in drinking-water, the guideline value may be difficult to achieve, in some circumstances, with the treatment technology available.

Toxicological Review

Many epidemiological studies of possible adverse effects of the long-term ingestion of fluoride via drinking-water have been carried out. These studies clearly establish that fluoride primarily produces effects on skeletal tissues (bones and teeth). In many regions with high fluoride exposure, fluoride is a significant cause of morbidity. Low concentrations provide protection against dental caries, especially in children. The pre- and post-eruptive protective effects of fluoride (involving the incorporation of fluoride into the matrix of the tooth during its formation, the development of shallower tooth grooves, which are consequently less prone to decay, and surface contact with enamel) increase with fluoride concentration up to about 2 mg/litre of drinking-water; the minimum concentration of fluoride in drinking-water required to produce it is approximately 0.5 mg/litre. However, fluoride can also have an adverse effect on tooth enamel and may give rise to mild dental fluorosis at drinking-water concentrations between 0.9 and 1.2 mg/litre, depending on intake. Elevated fluoride intakes can also

have more serious effects on skeletal tissues. It has been concluded that there is a clear excess risk of adverse skeletal effects for a total intake of 14 mg/day and suggestive evidence of an increased risk of effects on the skeleton at total fluoride intakes above about 6 mg/day.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* referred to fluoride, stating that concentrations in drinking-water in excess of 1.0–1.5 mg of fluorine per litre may give rise to dental fluorosis in some children, and much higher concentrations may eventually result in skeletal damage in both children and adults. To prevent the development of dental caries in children, a number of communal water supplies are fluoridated to bring the fluorine concentration to 1.0 mg/litre. The 1971 International Standards recommended control limits for fluorides in drinking-water for various ranges of the annual average of maximum daily air temperatures; control limits ranged from 0.6–0.8 mg/litre for temperatures of 26.3–32.6 °C to 0.9–1.7 mg/litre for temperatures of 10–12 °C. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 1.5 mg/litre was established for fluoride, as mottling of teeth has been reported very occasionally at higher levels. It was also noted that local application of the guideline value must take into account climatic conditions and higher levels of water intake. The 1993 Guidelines concluded that there was no evidence to suggest that the guideline value of 1.5 mg/litre set in 1984 needed to be revised. It was also recognized that in areas with high natural fluoride levels, the guideline value may be difficult to achieve in some circumstances with the treatment technology available. It was also emphasized that in setting national standards for fluoride, it is particularly important to consider climatic conditions, volume of water intake and intake of fluoride from other sources.

Primary Reference

IPCS (2002) *Fluorides*. Geneva, World Health Organization (Environmental Health Criteria 227).

Health criteria and other supporting information

Hardness

Hardness in water is caused by dissolved calcium and, to a lesser extent, magnesium. It is usually expressed as the equivalent quantity of calcium carbonate.

Depending on pH and alkalinity, hardness of above about 200 mg/litre can result in scale deposition, particularly on heating. Soft waters with a hardness of less than about 100 mg/litre have a low buffering capacity and may be more corrosive to water pipes.

Although a number of ecological and analytical epidemiological studies have shown a statistically significant inverse relationship between hardness of drinking-water and cardiovascular disease, the available data are inadequate to permit a conclusion that the association is causal. There is some indication that very soft waters may have an adverse effect on mineral balance, but detailed studies were not available for evaluation.

No health-based guideline value is proposed for hardness. However, the degree of hardness in water may affect its acceptability to the consumer in terms of taste and scale deposition (see chapter 10).

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to hardness. The 1971 International Standards stated that the maximum permissible level of hardness in drinking-water was 10 mEq/litre (500 mg calcium carbonate/litre), based on the acceptability of water for domestic use. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was concluded that there was no firm evidence that drinking hard water causes any adverse effects on human health and that no recommendation on the restriction of municipal water softening or on the maintenance of a minimum residual calcium or magnesium level was warranted. A guideline value of 500 mg/litre (as calcium carbonate) was established for hardness, based on taste and household use considerations. No health-based guideline value for hardness was proposed in the 1993 Guidelines, although hardness above approximately 200 mg/litre may cause scale deposition in the distribution system. Public acceptability of the degree of hardness may vary considerably from one community to another, depending on local conditions, and the taste of water with hardness in excess of 500 mg/litre is tolerated by consumers in some instances.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Hydrogen sulfide

Hydrogen sulfide is a gas with an offensive "rotten eggs" odour that is detectable at very low concentrations, below 0.8 µg/m^3 in air. It is formed when sulfides are hydrolysed in water. However, the level of hydrogen sulfide found in drinking-water will usually be low, because sulfides are readily oxidized in well aerated water.

The acute toxicity to humans of hydrogen sulfide following inhalation of the gas is high; eye irritation can be observed at concentrations of 15–30 mg/m^3. Although oral toxicity data are lacking, it is unlikely that a person could consume a harmful dose of hydrogen sulfide from drinking-water. Consequently, no health-based guideline value is proposed. However, hydrogen sulfide should not be detectable in drinking-water by taste or odour (see chapter 10).

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to hydrogen sulfide. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was recommended that hydrogen sulfide should not be detectable by the consumer, based on aesthetic considerations. A guideline value was not needed, since any contamination can be easily detected by the consumer. The 1993 Guidelines did not propose a health-based guideline value, as oral toxicity data are lacking; nevertheless, it is unlikely that a person could consume a harmful dose of hydrogen sulfide from drinking-water. The taste and odour thresholds of hydrogen sulfide in water are estimated to be between 0.05 and 0.1 mg/litre.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Manganese

Manganese is one of the most abundant metals in the Earth's crust, usually occurring with iron. It is used principally in the manufacture of iron and steel alloys, as an oxidant for cleaning, bleaching and disinfection as potassium permanganate, and as an ingredient in various products. More recently, it has been used in an organic compound, MMT, as an octane enhancer in petrol in North America. Manganese greensands are used in some locations for potable water treatment. Manganese is an essential element for humans and other animals and occurs naturally in many food sources. The most important oxidative states for the environment and biology are Mn^{2+}, Mn^{4+} and Mn^{7+}. Manganese is naturally occurring in many surface water and groundwater sources, particularly in anaerobic or low oxidation conditions, and this is the most important source for drinking-water. The greatest exposure to manganese is usually from food.

Guideline value	0.4 mg/litre
Occurrence	Levels in fresh water typically range from 1 to 200 µg/litre, although levels as high as 10 mg/litre in acidic groundwater have been reported.
TDI	0.06 mg/kg of body weight, 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, taking into consideration the possible increased bioavailability of manganese from water
Limit of detection	0.05 µg/litre by inductively coupled plasma/mass spectrometry; 0.5 µg/litre by inductively coupled plasma/optical emission spectroscopy; 1 µg/litre by electrothermal atomic absorption spectrometry; 10 µg/litre by flame atomic absorption spectrometry
Treatment achievability	0.05 mg/litre should be achievable using oxidation and filtration
Guideline derivation	
• *allocation to water*	20% of TDI (because manganese is essential trace element)
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The presence of manganese in drinking-water will be objectionable to consumers if it is deposited in water mains and causes water discoloration. Concentrations below 0.05–0.1 mg/litre are usually acceptable to consumers but may sometimes still give rise to the deposition of black deposits in water mains over an extended period; this may vary with local circumstances.

Toxicological Review

Manganese is an essential element for humans and other animals. Adverse effects can result from both deficiency and overexposure. Manganese is known to cause neurological effects following inhalation exposure, particularly in occupational settings, and there have been epidemiological studies that report adverse neurological effects following extended exposure to very high levels in drinking-water. However, there are a number of significant potential confounding factors in these studies, and a number of other studies have failed to observe adverse effects following exposure through drinking-water.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* suggested that concentrations of manganese greater than 0.5 mg/litre would markedly impair the potability of the water. The 1963 and 1971 International Standards retained this value as a maximum allowable or permissible concentration. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 0.1 mg/litre was established for manganese, based on its staining properties. The 1993 Guidelines concluded that although no single study is suitable for use in calculating a guideline value, the weight of evidence from actual daily intake and toxicity studies in laboratory animals given manganese in drinking-water supports the view that a provisional health-based guideline value of 0.5 mg/litre should be adequate to protect public health. It was also noted that concentrations below 0.1 mg/litre are usually acceptable to consumers, although this may vary with local circumstances.

Primary Reference

New background document

Health criteria and other supporting information

Molybdenum

Molybdenum is used in the manufacture of special steels and in the production of tungsten and pigments, and molybdenum compounds are used as lubricant additives and in agriculture to prevent molybdenum deficiency in crops.

Guideline value	0.07 mg/litre
Occurrence	Concentrations in drinking-water are usually less than 0.01 mg/litre, although concentrations as high as 0.2 mg/litre have been reported in areas near mining sites.
NOAEL	0.2 mg/litre in a 2-year study of humans exposed through their drinking-water, using an uncertainty factor of 3 for intraspecies variation (because molybdenum is an essential element)
Limit of detection	0.25 µg/litre by graphite furnace atomic absorption spectroscopy; 2 µg/litre by inductively coupled plasma atomic emission spectroscopy
Treatment achievability	Molybdenum is not removed from drinking-water.
Additional comments	The guideline value is within the range of that derived on the basis of results of toxicological studies in animal species and is consistent with the essential daily requirement. The supporting document on molybdenum is a high priority for revision because of exposure of and potential impact on bottle-fed infants.

Toxicological Review

Molybdenum is considered to be an essential element, with an estimated daily requirement of 0.1–0.3 mg for adults. No data are available on the carcinogenicity of molybdenum by the oral route.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to molybdenum. The 1971 International Standards stated that molybdenum should be controlled in drinking-water, but that insufficient information was available to enable a tentative limit to be established. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was concluded that no action was required for molybdenum. The 1993 Guidelines proposed a health-based guideline value of 0.07 mg/litre for molybdenum based on a 2-year study of humans exposed through their drinking-water. This value is within the range of that derived on the basis of results of toxicological studies in animal species and is consistent with the essential daily requirement.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

pH

No health-based guideline value is proposed for pH, although eye irritation and exacerbation of skin disorders have been associated with pH values greater than 11. Although pH usually has no direct impact on consumers, it is one of the most important operational water quality parameters (see chapter 10).

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* suggested that pH less than 6.5 or greater than 9.2 would markedly impair the potability of the water. The 1963 and 1971 International Standards retained the pH range 6.5–9.2 as the allowable or permissible range. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value range of pH 6.5–8.5 was established for pH, based on aesthetic considerations. It was noted that the acceptable range of pH may be broader in the absence of a distribution system. No health-based guideline value was proposed for pH in the 1993 Guidelines. Although pH usually has no direct impact on consumers, it is one of the most important operational water quality parameters, the optimum pH required often being in the range 6.5–9.5.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

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Selenium

Selenium is present in the Earth's crust, often in association with sulfur-containing minerals. Selenium is an essential trace element, and 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. In some areas selenium occurs at elevated levels in drinking-water and is associated with human health effects. It is therefore one of the high priority contaminants for screening in water supplies.

Guideline value	0.01 mg/litre
Occurrence	Levels in drinking-water vary greatly in different geographical areas but are usually much less than 0.01 mg/litre.
NOAEL in humans	Estimated to be about 4 µg/kg of body weight per day, based on data in which a group of 142 persons with a mean daily intake of 4 µg/kg body weight showed no clinical or biochemical signs of selenium toxicity
Limit of detection	0.5 µg/litre by atomic absorption spectrometry with hydride generation
Treatment achievability	0.01 mg/litre should be achievable using coagulation for selenium(IV) removal; selenium(VI) is not removed by conventional treatment processes

Guideline derivation

• *allocation to water*	10% of NOAEL
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Selenium is an essential element for humans, with a recommended daily intake of about 1 µg/kg of body weight for adults. Selenium compounds have been shown to be genotoxic in *in vitro* systems with metabolic activation, but not in humans. There was no evidence of teratogenic effects in monkeys. Long-term toxicity in rats is characterized by depression of growth and liver pathology. In humans, the toxic effects of long-term selenium exposure are manifested in nails, hair and liver. Data from China indicate that clinical and biochemical signs occur at a daily intake above 0.8 mg. Daily intakes of Venezuelan children with clinical signs were estimated to be about 0.7 mg on the basis of their blood levels and the Chinese data on the relationship between blood level and intake. Effects on synthesis of a liver protein were also seen in a small group of patients with rheumatoid arthritis given selenium at a rate of 0.25 mg/day in addition to selenium from food. No clinical or biochemical signs of selenium toxicity were reported in a group of 142 persons with a mean daily intake of 0.24 mg (maximum 0.72 mg) from food.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* recommended a maximum allowable concentration of 0.05 mg/litre for selenium, based on health concerns. In the 1963 International Standards, this value was lowered to 0.01 mg/litre, which was retained in the 1971 International Standards as a tentative upper concentration limit, while recognizing that selenium is an essential trace element for some species. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, the guideline value of 0.01 mg/litre was again retained, although it was noted that in areas of relatively higher or lower selenium dietary intake, the guideline value may have to be modified accordingly. The 1993 Guidelines proposed a health-based guideline value of 0.01 mg/litre on the basis of human studies.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Sodium

Sodium salts (e.g., sodium chloride) are found in virtually all food (the main source of daily exposure) and drinking-water. Although concentrations of sodium in potable water are typically less than 20 mg/litre, they can greatly exceed this in some countries. The levels of sodium salts in air are normally low in relation to those in food or water. It should be noted that some water softeners can add significantly to the sodium content of drinking-water.

No firm conclusions can be drawn concerning the possible association between sodium in drinking-water and the occurrence of hypertension. Therefore, no health-based guideline value is proposed. However, concentrations in excess of 200 mg/litre may give rise to unacceptable taste (see chapter 10).

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to sodium. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was concluded that there was insufficient evidence to justify a guideline value for sodium in water based on health risk considerations, but it was noted that intake of sodium from drinking-water may be of greater significance in persons who require a sodium-restricted diet. A guideline value of 200 mg/litre was established for sodium based on taste considerations. No health-based guideline value was proposed for sodium in the 1993 Guidelines, as no firm conclusions can be drawn concerning the possible association between sodium in drinking-water and the occurrence of hypertension. However, concentrations in excess of 200 mg/litre may give rise to unacceptable taste.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Sulfate

Sulfates occur naturally in numerous minerals and are used commercially, principally in the chemical industry. They are discharged into water in industrial wastes and through atmospheric deposition; however, the highest levels usually occur in groundwater and are from natural sources. In general, food is the principal source of exposure to sulfate, although intake from drinking-water can exceed that from food in areas with high concentrations. The contribution of air to total intake is negligible.

Sulfate is one of the least toxic anions; however, catharsis, dehydration and gastrointestinal irritation have been observed at high concentrations. Magnesium sulfate, or Epsom salts, has been used as a cathartic for many years.

No health-based guideline is proposed for sulfate. However, because of the gastrointestinal effects resulting from ingestion of drinking-water containing high sulfate levels, it is recommended that health authorities be notified of sources of drinking-water that contain sulfate concentrations in excess of 500 mg/litre. The presence of sulfate in drinking-water may also cause noticeable taste (see chapter 10) and may contribute to the corrosion of distribution systems.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* suggested that concentrations of sulfate greater than 400 mg/litre would markedly impair the potability of the water. The 1963 and 1971 International Standards retained this value as a maximum allowable or permissible concentration. The first two editions of the International Standards also suggested that concentrations of magnesium + sodium sulfate in excess of 1000 mg/litre would markedly impair drinking-water potability. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 400 mg/litre for sulfate was established, based on taste considerations. No health-based guideline value for sulfate was proposed in the 1993 Guidelines. However, because of the gastrointestinal effects resulting from ingestion of drinking-water containing high sulfate levels, it was recommended that health authorities be notified of sources of drinking-water that contain sulfate concentrations in excess of 500 mg/litre. The presence of sulfate in drinking-water may also cause noticeable taste at concentrations above 250 mg/litre and may contribute to the corrosion of distribution systems.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Total Dissolved Solids (TDS)

Total dissolved solids (TDS) comprise inorganic salts (principally calcium, magnesium, potassium, sodium, bicarbonates, chlorides and sulfates) and small amounts of organic matter that are dissolved in water. TDS in drinking-water originate from natural sources, sewage, urban runoff and industrial wastewater. Salts used for road de-icing in some countries may also contribute to the TDS content of drinking-water. Concentrations of TDS in water vary considerably in different geological regions owing to differences in the solubilities of minerals.

Reliable data on possible health effects associated with the ingestion of TDS in drinking-water are not available, and no health-based guideline value is proposed. However, the presence of high levels of TDS in drinking-water may be objectionable to consumers (see chapter 10).

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* suggested that concentrations of total solids greater than 1500 mg/litre would markedly impair the potability of the water. The 1963 and 1971 International Standards retained this value as a maximum allowable or permissible concentration. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 1000 mg/litre was established for TDS, based on taste considerations. No health-based guideline value for TDS was proposed in the 1993 Guidelines, as reliable data on possible health effects associated with the ingestion of TDS in drinking-water were not available. However, the presence of high levels of TDS in drinking-water (greater than 1200 mg/litre) may be objectionable to consumers. Water with extremely low concentrations of TDS may also be unacceptable because of its flat, insipid taste.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Uranium

Uranium is widespread in nature, occurring in granites and various other mineral deposits. Uranium is used mainly as fuel in nuclear power stations. Uranium is present in the environment as a result of leaching from natural deposits, release in mill tailings, emissions from the nuclear industry, the combustion of coal and other fuels, and the use of phosphate fertilizers that contain uranium. Intake of uranium through air is low, and it appears that intake through food is between 1 and 3 μg/day. Occurrence in drinking-water is primarily as a consequence of natural sources. Intake through drinking-water is normally extremely low; however, in circumstances in which uranium is present in a drinking-water source, the majority of intake can be through drinking-water.

Provisional guideline value	0.009 mg/litre
Occurrence	Levels in drinking-water are generally less than 1 μg/litre, although concentrations as high as 700 μg/litre have been measured in some private supplies and concentrations up to 100 μg/litre have been observed in some small municipal supplies.
TDI	0.6 μg/kg of body weight per day, based on the application of an uncertainty factor of 100 to a LOAEL 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 because of the minimal degree of severity of the lesions and the short half-life 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.
Limit of detection	0.01 μg/litre by inductively coupled plasma/mass spectrometry; 0.1 μg/litre by solid fluorimetry with either laser excitation or ultraviolet light; 0.2 μg/litre by inductively coupled plasma using adsorption with chelating resin
Treatment achievability	1 μg/litre should be achievable using conventional treatment, e.g., coagulation or ion exchange
Guideline derivation	
• *allocation to water*	50% of TDI (because intake from other sources is low in most areas)
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The data on intake from food in most areas suggest that intake from food is low and support the higher allocation to drinking-water. In some regions, exposure from sources such as soil may be higher and should be taken into account in setting national or local standards. The guideline value is designated as provisional because of outstanding uncertainties regarding the toxicology and epidemiology of uranium as well as difficulties concerning its technical achievability in smaller supplies. The concentration of uranium in drinking-water associated with the onset of measurable tubular dysfunction remains uncertain, as does the clinical significance of the observed changes at low exposure levels. A guideline value of up to 30 μg/litre may be protective of kidney toxicity, because the markers of tubular function observed at concentrations below this are still within the normal range.

Only chemical, not radiological, aspects of uranium toxicity have been addressed here.
A document on depleted uranium, which is a by-product of natural uranium, is available.

Toxicological Review

There are insufficient data regarding the carcinogenicity of uranium in humans and experimental animals. Data from high levels of exposure to uranium indicate that nephritis is the primary adverse effect in humans. A number of epidemiological studies of populations exposed to uranium in drinking-water have shown a correlation with alkaline phosphatase and β-microglobulin in urine along with modest alterations in proximal tubular function. However, the actual measurements were still within the normal range.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to uranium. The 1971 International Standards stated that uranium should be controlled in drinking-water, but that insufficient information was available to enable a tentative limit to be established. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was concluded that no action was required for uranium. A health-based guideline value for uranium was not derived in the 1993 Guidelines, as adequate short- and long-term studies on the chemical toxicity of uranium were not available. Until such information became available, it was recommended that the limits for radiological characteristics of uranium be used. The equivalent for natural uranium, based on these limits, is approximately 0.14 mg/litre. In the addendum to the Guidelines, published in 1998, a health-based guideline value of 0.002 mg/litre was established. This guideline value was designated as provisional, because it may be difficult to achieve in areas with high natural uranium levels with the treatment technology available and because of limitations in the key study. It was noted that several human studies are under way that may provide helpful additional data.

Primary Reference

New background document

Health criteria and other supporting information

8.7.2 Chemicals from Industrial Sources and Human Dwellings

Chemicals from industrial sources can reach drinking-water directly from discharges or indirectly from diffuse sources arising from the use and disposal of materials and products containing the chemical. In some cases, inappropriate handling and disposal may lead to contamination, e.g., degreasing agents that are allowed to reach groundwater. Some of these chemicals, particularly inorganic substances, may also be encountered as a consequence of natural contamination, but this may also be a by-product of industrial activity, such as mining, that changes drainage patterns. Many of these chemicals are used in small industrial units within human settlements, and, particularly where such units are found in groups of similar enterprises, they may be a significant source of pollution. Petroleum oils are widely used in human settlements, and improper handling or disposal can lead to significant pollution of surface water and groundwater. Where plastic pipes are used, the smaller aromatic molecules in petroleum oils can sometimes penetrate the pipes where they are surrounded by earth soaked in the oil, with subsequent pollution of the local water supply.

A number of chemicals can reach water as a consequence of disposal of general household chemicals; in particular, a number of heavy metals may be found in domestic wastewater. Where the wastewater receives treatment, these will usually partition out into the sludge. Some chemicals that are widely used both in industry and in materials used in a domestic setting are found widely in the environment, e.g., di(2-ethylhexyl)phthalate, and these may be found in water sources, although usually at low concentrations.

Where pit latrines and septic tanks are poorly sited, these can lead to contamination of drinking-water sources with nitrate (see section 8.7.3 on “Chemicals from Agricultural Activities”).

Identification of the potential for contamination by such chemicals requires that an assessment be made of activities in the catchment and of the risk that particular contaminants may reach water sources. The primary approach to addressing these contaminants is the prevention of contamination by encouraging good practices. However, if contamination has occurred, then it may be necessary to consider the introduction of treatment.

Some chemicals that reach drinking-water from industrial sources or human settlements have other primary sources and are therefore discussed in other sections of this chapter.

Chemicals listed in Table 8.6 have been excluded from guideline value derivation, as a review of the literature on occurrence and/or credibility of occurrence in drinking-water has shown evidence that the chemicals do not occur in drinking-water.

Table 8.16. Chemicals from industrial sources and human dwellings excluded from guideline value derivation

Chemical	Reason for exclusion
Beryllium	Unlikely to occur in drinking-water

Guideline values have not been established for the chemicals listed in Table 8.7 because:
evidence indicates that the chemical does not occur at concentrations at or near levels expected to cause a health concern, or
there is lack of evidence of health effects, or there are insufficient data to support the establishment of a guideline value.

Table 8.17. Chemicals from industrial sources and human dwellings for which guideline values have not been established

Chemical	Reason for exclusion
Dichlorobenzene, 1,3-	Toxicological data are insufficient to permit the recommendation of a health-based guideline value
Dichloroethane, 1,1-	Very limited database on toxicity and carcinogenicity
Di(2-ethylhexyl)adipate	Occurs at concentrations well below those at which toxic effects are observed
Monochlorobenzene	Occurs at concentrations well below those at which toxic effects are observed, and health-based value would far exceed lowest reported taste and odour threshold
Trichlorobenzenes (total)	Occurs at concentrations well below those at which toxic effects are observed, and health-based value would exceed lowest reported odour threshold
Trichloroethane, 1,1,1-	Occurs at concentrations well below those at which toxic effects are observed

Guideline values have been established for the chemicals listed in Table 8.8, which meet all of the criteria for inclusion.

Table 8.8. Guideline values for chemicals from industrial sources and human dwellings that are of health significance in drinking-water

Inorganics	Guideline value (mg/litre)	Remarks
Cadmium	0.003	
Cyanide	0.07	
Mercury	0.001	For total mercury (inorganic plus organic)

Organics	Guideline value[a] (µg/litre)	Remarks
Benzene	10[b]	
Carbon tetrachloride	4	
Di(2-ethylhexyl)phthalate	8	
Dichlorobenzene, 1,2-	1000	C[c]
Dichlorobenzene, 1,4-	300	C[c]
Dichloroethane, 1,2-	4[b]	
Dichloroethene, 1,1-	30	
Dichloroethene, 1,2-	50	
Dichloromethane	20	
Dioxane, 1,4-	50[b]	
Edetic acid (EDTA)	600	Applies to the free acid
Ethylbenzene	300	C[c]
Hexachlorobutadiene	0.6	
Nitrilotriacetic acid (NTA)	200	
Pentachlorophenol	9[b] (P)	
Styrene	20	C[c]
Tetrachloroethene	40	
Toluene	700	C[c]
Trichloroethene	70 (P)	
Xylenes	500	C[c]

[a] P = evidence of a potential hazard, but the available information on health effects is limited.

[b] For substances that are considered to be carcinogenic, the guideline value is the concentration in drinking-water associated with an upper bound excess lifetime cancer risk of 10^{-5} (one additional cancer per 100

000 of the population ingesting drinking-water containing the substance at the guideline value for 70 years). Concentrations associated with estimated upper bound excess lifetime cancer risks of 10^{-4} and 10^{-6} can be calculated by multiplying and dividing, respectively, the guideline value by 10.

In cases in which the concentration associated with an upper bound excess lifetime cancer risk of 10^{-5} is not feasible as a result of inadequate analytical or treatment technology, a provisional guideline value (designated A or T, respectively) is recommended at a practicable level.

It should be emphasized that the guideline values for carcinogenic substances have been computed from hypothetical mathematical models that cannot be verified experimentally and that the values should be interpreted differently from TDI-based values because of the lack of precision of the models. At best, these values must be regarded as rough estimates of cancer risk. However, the models used are conservative and probably err on the side of caution. Moderate short-term exposure to levels exceeding the guideline value for carcinogens does not significantly affect the risk.

[c] C = Concentrations of the substance at or below the health-based guideline value may affect the appearance, taste or odour of the water, leading to consumer complaints.

Inorganics

Cadmium

Cadmium metal is used in the steel industry and in plastics. Cadmium compounds are widely used in batteries. Cadmium is released to the environment in wastewater, and diffuse pollution is caused by contamination from fertilizers and local air pollution. Contamination in drinking-water may also be caused by impurities in the zinc of galvanized pipes and solders and some metal fittings. Food is the main source of daily exposure to cadmium. The daily oral intake is 10–35 µg. Smoking is a significant additional source of cadmium exposure.

Guideline value	0.003 mg/litre
Occurrence	Levels in drinking-water are usually less than 1 µg/litre
PTWI	7 µg/kg of body weight, on the basis that if levels of cadmium in the renal cortex are not to exceed 50 mg/kg, total intake of cadmium (assuming an absorption rate for dietary cadmium of 5% and a daily excretion rate of 0.005% of body burden) should not exceed 1 µg/kg of body weight per day
Limit of detection	0.01 µg/litre by inductively coupled plasma/mass spectrometry; 2 µg/litre by flame atomic absorption spectrometry
Treatment achievability	0.002 mg/litre should be achievable using coagulation or precipitation softening
Guideline derivation	
• *allocation to water*	10% of PTWI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Although new information indicates that a proportion of the general population may be at increased risk for tubular dysfunction when exposed at the current PTWI, the risk estimates that can be made at present are imprecise. It is recognized that the margin between the PTWI and the actual weekly intake of cadmium by the general population is small, less than 10-fold, and that this margin may be even smaller in smokers.

Toxicological Review

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 10–35 years. There is evidence that cadmium is carcinogenic by the inhalation route, and IARC has classified cadmium and cadmium compounds in Group 2A. However, there is no evidence of carcinogenicity by the oral route and no clear evidence for the genotoxicity of cadmium. The kidney is the main target organ for cadmium toxicity. The critical cadmium concentration in the renal cortex that would produce a 10% prevalence of low-molecular-weight proteinuria in the general population is about 200 mg/kg and would be reached after a daily dietary intake of about 175 µg per person for 50 years.

History of Guideline Development

The 1958 WHO International Standards for Drinking-water did not refer to cadmium. The 1963 International Standards recommended a maximum allowable concentration of 0.01 mg/litre, based on health concerns. This value was retained in the 1971 International Standards as a tentative upper concentration limit, based on the lowest concentration that could be conveniently measured. In the first edition of the Guidelines for Drinking-

water Quality, published in 1984, a guideline value of 0.005 mg/litre was recommended for cadmium in drinking-water. This value was lowered to 0.003 mg/litre in the 1993 Guidelines, based on the provisional tolerable weekly intake set by JECFA.

Primary Reference

JECFA (2000) Joint FAO/WHO Expert Committee on Food Additives. Fifty-fifth meeting. Summary and conclusions. Geneva, World Health Organization.

Health criteria and other supporting information

Cyanide

Cyanides can be found in some foods such as cassava, particularly in some developing countries, and they are occasionally found in drinking-water, primarily as a consequence of industrial contamination.

Guideline value	0.07 mg/litre
Occurrence	Occasionally found in drinking-water
TDI	12 µg/kg of body weight, based on a LOAEL of 1.2 mg/kg of body weight per day for effects on behavioural patterns and serum biochemistry in a 6-month study in pigs, using an uncertainty factor of 100 for inter- and intraspecies variation (no additional factor for LOAEL was considered necessary because of doubts over the biological significance of the observed changes)
Limit of detection	2 µg/litre by titrimetric and photometric techniques
Treatment achievability	Cyanide is not removed from water.
Guideline derivation	
• *allocation to water*	20% of TDI (because exposure to cyanide from other sources is normally small and because exposure from water is only intermittent)
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional considerations	The supporting document on cyanide will be revised in response to a CICAD that is in preparation. Guidance on short-term exposures to cyanide is also being prepared.

Toxicological Review

The acute toxicity of cyanides is high. Effects on the thyroid and particularly the nervous system were observed in some populations as a consequence of the long-term consumption of inadequately processed cassava containing high levels of cyanide.

History of Guideline Development

The 1958 WHO International Standards for Drinking-water recommended a maximum allowable concentration of 0.01 mg/litre for cyanide, based on health concerns. This value was raised to 0.2 mg/litre in the 1963 International Standards. The tentative upper concentration limit was lowered to 0.05 mg/litre in the 1971 International Standards upon consideration of the acceptable daily intake of hydrogen cyanide residues in some fumigated foods of 0.05 mg/kg of body weight and to ensure that the water source is not too highly contaminated by industrial effluents and that water treatment has been adequate. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, it was determined that a guideline value of 0.1 mg/litre would be a reasonable level for the protection of public health. A health-based guideline value of 0.07 mg/litre, which was considered to be protective for both acute and long-term exposure, was derived in the 1993 Guidelines.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.
Health criteria and other supporting information

Mercury

Mercury is used in the electrolytic production of chlorine, in electrical appliances, in dental amalgams and as a raw material for various mercury compounds. Methylation of inorganic mercury has been shown to occur in fresh water and in seawater, although almost all mercury in uncontaminated drinking-water is thought to be in the form of Hg^{2+}. Thus, it is unlikely that there is any direct risk of the intake of organic mercury compounds, especially of alkylmercurials, as a result of the ingestion of drinking-water. However, there is a real possibility that methylmercury will be converted into inorganic mercury. Food is the main source of mercury in non-occupationally exposed populations; the mean dietary intake of mercury in various countries ranges from 2 to 20 µg/day per person.

Guideline value	0.001 mg/litre for total mercury
Occurrence	Mercury is present in the inorganic form in surface water and groundwater at concentrations usually below 0.5 µg/litre, although local mineral deposits may produce higher levels in groundwater.
PTWI	5 µg/kg of body weight for total mercury, of which no more than 3.3 µg/kg of body weight should be present as methylmercury
Limit of detection	0.001 µg/litre by atomic fluorescence spectrometry; 0.05 µg/litre by cold vapour atomic absorption spectrometry; 0.6 µg/litre by inductively coupled plasma; 5 µg/litre by flame atomic absorption spectrometry
Treatment achievability	0.1 µg/litre should be achievable using coagulation

Guideline derivation

• *allocation to water*	10% of PTWI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

The toxic effects of inorganic mercury compounds are seen mainly in the kidney. Methylmercury affects mainly the central nervous system.

History of Guideline Development

The 1958 and 1963 WHO International Standards for Drinking-water did not mention mercury. Mercury was first mentioned in the 1971 International Standards, which gave the tentative upper concentration limit for mercury as 0.001 mg/litre (total mercury), based on health concerns. It was noted that this figure was related to levels found in natural water. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, the guideline value of 0.001 mg/litre, which applied to all chemical forms of mercury, was retained. The 1993 Guidelines also retained the guideline value for total mercury of 0.001 mg/litre, based on the provisional tolerable weekly intake for methylmercury established by JECFA. JECFA has since reaffirmed the provisional tolerable weekly intake.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Organics

The organic chemicals from industrial sources or human settlements are grouped as follows: Chlorinated alkanes, Chlorinated ethenes, Aromatic hydrocarbons, Chlorinated benzenes, and Miscellaneous organic constituents.

Carbon Tetrachloride

Carbon tetrachloride is used mainly in the production of chlorofluorocarbon refrigerants, foam-blowing agents and solvents. However, since the Montreal Protocol on Substances that Deplete the Ozone Layer (1987) and its amendments (1990 and 1992) have established a timetable for the phase-out of the production and consumption of carbon tetrachloride, manufacture and use have dropped and will continue to drop. Carbon tetrachloride is released mostly into the atmosphere but also into industrial wastewater. Although it readily migrates from surface water to the atmosphere, levels in anaerobic groundwater may remain elevated for months or even years. Carbon tetrachloride was used historically as a degreasing aagent and much pollution of groundwater by carbon tetrachloride relates to such past uses. Although available data on concentrations in food are limited, the intake from air is expected to be much greater than that from food or drinking-water.

Guideline value	4 µg/litre
Occurrence	Concentrations in drinking-water are generally less than 5 µg/litre.
TDI	1.4 µg/kg of body weight, based on hepatotoxic effects in a 12-week oral gavage study in rats, taking into account the less-than-lifetime study as well as the fact that this was a bolus study
Limit of detection	0.1–0.3 µg/litre by gas chromatography with electron capture detection or mass spectrometry
Treatment achievability	0.001 mg/litre should be achievable using air stripping
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value is lower than the range of values associated with lifetime upper bound excess cancer risks of 10^{-4}, 10^{-5} and 10^{-6} calculated by linear extrapolation.

Toxicological Review

The primary targets for carbon tetrachloride toxicity are liver and kidney, neurological damage being secondary to hepatotoxicity. In experiments with mice and rats, carbon tetrachloride proved to be capable of inducing hepatomas and hepatocellular carcinomas. The doses inducing hepatic tumours were higher than those inducing cell toxicity. It is likely that the carcinogenicity of carbon tetrachloride is secondary to its hepatotoxic effects. On the basis of available data, carbon tetrachloride can be considered as a non-genotoxic compound. Carbon tetrachloride is classified by IARC as being possibly carcinogenic to humans (Group 2B). There is sufficient evidence that carbon tetrachloride is carcinogenic in laboratory animals, but inadequate evidence in humans.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to carbon tetrachloride. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, a tentative guideline value of 0.003 mg/litre was recommended; the guideline was designated as tentative because reliable evidence on which to calculate a guideline value based on carcinogenicity was available in only one animal species, because of the good qualitative supporting data and because of its frequency of occurrence in water. The 1993 Guidelines established a health-based guideline value of 0.002 mg/litre for carbon tetrachloride.

Primary Reference

IPCS (1999) Carbon tetrachloride. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 208).

Health criteria and other supporting information

Dichloromethane

Dichloromethane, or methylene chloride, is widely used as a solvent for many purposes, including coffee decaffeination and paint stripping. Exposure from drinking-water is likely to be insignificant compared with that from other sources.

Guideline value	20 µg/litre
Occurrence	Dichloromethane has been found in surface water samples at concentrations ranging from 0.1 to 743 µg/litre. Levels are usually higher in groundwater because volatilization is restricted; concentrations as high as 3600 µg/litre have been reported. Mean concentrations in drinking-water were less than 1 µg/litre.
TDI	6 µg/kg of body weight, derived from a NOAEL of 6 mg/kg of body weight per day for hepatotoxic effects in a 2-year drinking-water study in rats, using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for concern about carcinogenic potential)
Limit of detection	0.3 µg/litre by purge-and-trap gas chromatography with mass spectrometric detection (note that dichloromethane vapour readily penetrates tubing during the procedure)
Treatment achievability	20 µg/litre should be achievable using air stripping
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The supporting document on dichloromethane will be revised once new data become available.

Toxicological Review

Dichloromethane is of low acute toxicity. An inhalation study in mice provided conclusive evidence of carcinogenicity, whereas a drinking-water study provided only suggestive evidence. IARC has placed dichloromethane in Group 2B; however, the balance of evidence suggests that it is not a genotoxic carcinogen and that genotoxic metabolites are not formed in relevant amounts in vivo.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to dichloromethane. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, no guideline value was recommended after a detailed evaluation of the compound. The 1993 Guidelines established a health-based guideline value of 0.02 mg/litre for dichloromethane, noting that widespread exposure from other sources is possible.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

1,1-Dichloroethane

1,1-Dichloroethane is used as a chemical intermediate and solvent. There are limited data showing that it can be present at concentrations of up to 10 µg/litre in drinking-water. However, because of the widespread use and disposal of this chemical, its occurrence in groundwater may increase.

1,1-Dichloroethane is rapidly metabolized by mammals to acetic acid and a variety of chlorinated compounds. It is of relatively low acute toxicity, and limited data are available on its toxicity from short- and long-term studies. There is limited in vitro evidence of genotoxicity. One carcinogenicity study by gavage in mice and rats provided no conclusive evidence of carcinogenicity, although there was some evidence of an increased incidence of haemangiosarcomas in treated animals.

In view of the very limited database on toxicity and carcinogenicity, it was concluded that no guideline value should be proposed.

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 1,1-dichloroethane. In view of the very limited database on toxicity and carcinogenicity, the 1993 Guidelines concluded that no guideline value for 1,1-dichloroethane should be proposed at this time.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

1,2-Dichloroethane

1,2-Dichloroethane is used mainly as an intermediate in the production of vinyl chloride and other chemicals and to a lesser extent as a solvent. It may enter surface waters via effluents from industries that manufacture or use the substance. It may also enter groundwater, where it may persist for long periods, following disposal in waste sites. It is found in urban air.

Guideline value	4 µg/litre
Occurrence	Has been found in drinking-water at levels of up to a few micrograms per litre
Basis of guideline derivation	The cancer potency, expressed as the dose associated with a 5% increase in tumour incidence, was calculated to be 6.2 to 34 mg/kg body weight per day. The values associated with a margin 50,000-fold less than this are 0.12 to 0.68 µg/kg body weight per day (WHO 1998). The guideline value associated with the lower of these figures, assuming a 60 kg adult drinking 2 l water is 4 µg/l (rounded value).
Limit of detection	0.06–2.8 µg/litre by gas chromatography/mass spectrometry; 0.03–0.2 µg/litre by gas chromatography with electrolytic conductivity detector; 5 µg/litre by gas chromatography with flame ionization detector; 0.03 µg/litre by gas chromatography with photoionization detection
Treatment achievability	0.0001 mg/litre should be achievable using GAC

Toxicological Review
IARC has classified 1,2-dichloroethane in Group 2B (possible human carcinogen). It has been shown to produce statistically significant increases in a number of tumour types in laboratory animals, including the relatively rare haemangiosarcoma, and the balance of evidence indicates that it is potentially genotoxic. Targets of 1,2-dichloroethane toxicity in orally exposed animals included the immune system, central nervous sytem, liver and kidney.

History of Guideline Development
The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to 1,2-dichloroethane. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, a health-based guideline value of 0.01 mg/litre was recommended for 1,2-dichloroethane, while noting that the mathematical model appropriate to chemical carcinogens that was used in its derivation involved considerable uncertainty. The 1993 Guidelines calculated a guideline value of 0.03 mg/litre for 1,2-dichloroethane on the basis of haemangiosarcomas observed in male rats, corresponding to an excess lifetime cancer risk of 10^{-5}.

Primary Reference
WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

WHO (1998) 1,2-Dichloroethane.Geneva, World Health Organization(Concise International Chemical Assessment Document)

Health criteria and other supporting information

1,1,1-Trichloroethane

1,1,1-Trichloroethane is widely used as a cleaning solvent for electrical equipment, as a solvent for adhesives, coatings and textile dyes, and as a coolant and lubricant. It is found mainly in the atmosphere, although it is mobile in soils and readily migrates to groundwaters. 1,1,1-Trichloroethane has been found in only a small proportion of surface waters and groundwaters, usually at concentrations of less than 20 µg/litre; higher concentrations (up to 150 µg/litre) have been observed in a few instances. There appears to be increasing exposure to 1,1,1-trichloroethane.

1,1,1-Trichloroethane is rapidly absorbed from the lungs and gastrointestinal tract, but only small amounts — about 6% in humans and 3% in experimental animals — are metabolized. Exposure to high concentrations can lead to hepatic steatosis (fatty liver) in both humans and laboratory animals. In a well conducted oral study in mice and rats, effects included reduced liver weight and changes in the kidney consistent with hyaline droplet neuropathy. IARC has placed 1,1,1-trichloroethane in Group 3. 1,1,1-Trichloroethane does not appear to be mutagenic.

A health-based value of 2 mg/litre can be calculated for 1,1,1-trichloroethane on the basis of a TDI of 0.6 mg/kg of body weight, based on changes in the kidney that were consistent with hyaline droplet nephropathy observed in a 13-week oral study in male rats, and taking into account the short duration of the study. However, because 1,1,1-trichloroethane occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to 1,1,1-trichloroethane. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, no guideline value was recommended after a detailed evaluation of the compound. The 1993 Guidelines proposed a provisional guideline value of 2 mg/litre for 1,1,1-trichloroethane. The value was provisional because it was based on an inhalation study rather than an oral study. It was strongly recommended that an adequate oral toxicity study be conducted to provide more acceptable data for the derivation of a guideline value.

Primary Reference

New background document

Health criteria and other supporting information

1,1-Dichloroethene

1,1-Dichloroethene, or vinylidene chloride, is used mainly as a monomer in the production of polyvinylidene chloride co-polymers and as an intermediate in the synthesis of other organic chemicals. It is an occasional contaminant of drinking-water, usually being found together with other chlorinated hydrocarbons. There are no data on levels in food, but levels in air are generally less than 40 ng/m^3 except at some manufacturing sites.

Guideline value	30 µg/litre
Occurrence	Detected in finished drinking-water taken from groundwater sources at median concentrations of 0.28–1.2 µg/litre and in public drinking-water supplies at concentrations ranging from 0.2 to 0.5 µg/litre
TDI	9 µg/kg of body weight, based on a LOAEL of 9 mg/kg of body weight per day in a 2-year drinking-water study in rats, using an uncertainty factor of 1000 (100 for intra- and interspecies variation and 10 for the use of a LOAEL instead of a NOAEL and the possibility of carcinogenicity)
Limit of detection	0.025 µg/litre by capillary gas chromatography with electron capture detection; 0.07 µg/litre by purge and trap packed column gas chromatography with electron capture or microcoulometric detector; 4.7 µg/litre by purge and trap packed column gas chromatography/mass spectrometry
Treatment achievability	0.01 mg/litre should be achievable using GAC or air stripping
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional considerations	The supporting document on 1,1-dichloroethene will be revised once a Concise International Chemical Assessment Document on the chemical becomes available.

Toxicological Review

1,1-Dichloroethene is a central nervous system depressant and may cause liver and kidney toxicity in occupationally exposed humans. It causes liver and kidney damage in laboratory animals. IARC has placed 1,1-dichloroethene in Group 3. It was found to be genotoxic in a number of test systems in vitro but was not active in the dominant lethal assay in vivo. It induced kidney tumours in mice in one inhalation study but was reported not to be carcinogenic in a number of other studies, including several in which it was given in drinking-water.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to 1,1-dichloroethene. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, a health-based guideline value of 0.0003 mg/litre was recommended for 1,1-dichloroethene, while noting that the mathematical model appropriate to chemical carcinogens that was used in its derivation involved considerable uncertainty. A health-based guideline value of 0.03 mg/litre for 1,1-dichloroethene was recommended in the 1993 Guidelines.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

1,2-Dichloroethene

1,2-Dichlorethene exists in a cis and a trans form. The cis form is more frequently found as a water contaminant. The presence of these two isomers, which are metabolites of other unsaturated halogenated hydrocarbons in wastewater and anaerobic groundwater, may indicate the simultaneous presence of more toxic organochlorine chemicals, such as vinyl chloride. Accordingly, their presence indicates that more intensive monitoring should be conducted. There are no data on exposure from food. Concentrations in air are low, with higher concentrations, in the microgram per cubic metre range, near production sites. The cis isomer was previously used as an anaesthetic.

Guideline value	50 µg/litre
Occurrence	Has been found in drinking-water supplies derived from groundwater at levels up to 120 µg/litre
TDI	17 µg/kg of body weight, based on a NOAEL of 17 mg/kg of body weight from a 90-day study in mice administered trans-1,2-dichloroethene in drinking-water, using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for the short duration of the study)
Limit of detection	0.17 µg/litre by gas chromatography with mass spectrometry
Treatment achievability	0.01 mg/litre should be achievable using GAC or air stripping
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Data on the trans isomer were used to calculate a joint guideline value for both isomers because toxicity for the trans isomer occurred at a lower dose than for the cis isomer and because data suggest that the mouse is a more sensitive species than the rat.

Toxicological Review

There is little information on the absorption, distribution and excretion of 1,2-dichloroethene. However, by analogy with 1,1-dichloroethene, it would be expected to be readily absorbed, distributed mainly to the liver, kidneys and lungs, and rapidly excreted. The cis isomer is more rapidly metabolized than the trans isomer in in vitro systems. Both isomers have been reported to cause increased serum alkaline phosphatase levels in rodents. In a 3-month study in mice given the trans isomer in drinking-water, there was a reported increase in serum alkaline phosphatase and reduced thymus and lung weights. Transient immunological effects were also reported, the toxicological significance of which is unclear. Trans-1,2-dichloroethene also caused reduced kidney weights in rats, but at higher doses. Only one rat toxicity study is available for the cis isomer, which produced toxic effects in rats similar in magnitude to those induced by the trans isomer in mice, but at higher doses. There are limited data to suggest that both isomers may possess some genotoxic activity. There is no information on carcinogenicity.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to 1,2-dichloroethene. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, no guideline value was recommended after a detailed evaluation of the compound. In the 1993 Guidelines, a joint guideline value of 0.05 mg/litre was calculated for both 1,2-dichloroethene isomers using toxicity data on the trans isomer.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Trichloroethene

Trichloroethene is used mainly in dry cleaning and metal degreasing operations. Its use in industrialized countries has declined sharply since 1970. It is released mainly to the atmosphere but may be introduced into surface water and groundwater in industrial effluents and poor disposal practices. It is expected that exposure to trichloroethene from air will be greater than that from food or drinking-water. Trichloroethene in anaerobic groundwater may degrade to more toxic compounds, including vinyl chloride.

Provisional guideline value	70 µg/litre
Occurrence	Found at a mean concentration of 2.1 µg/litre in a survey of drinking-water; also present in 24% of 158 non-random samples collected in a groundwater supply survey at a median level of 1 µg/litre and a maximum of 130 µg/litre
TDI	23.8 µg/kg of body weight (including allowance for 5 days/week dosing), based on a LOAEL of 100 mg/kg of body weight per day for minor effects on relative liver weight in a 6-week study in mice, using an uncertainty factor of 3000 (100 for intra- and interspecies variation, 10 for limited evidence of carcinogenicity and 3 in view of the short duration of the study and the use of a LOAEL rather than a NOAEL)
Limit of detection	0.037 µg/litre by capillary gas chromatography with electron capture detection; 0.12 µg/litre by purge and trap packed column gas chromatography with electron capture or microcoulometric detector; 0.2 µg/litre by purge and trap packed column gas chromatography/mass spectrometry
Treatment achievability	0.02 mg/litre should be achievable using air stripping
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional considerations	The guideline value is designated as provisional because of deficiencies in the toxicological database. The supporting document on trichloroethene will be revised, as IARC has re-evaluated it and changed its carcinogenicity classification.

Toxicological Review

The reactive epoxide trichloroethene oxide is an essential feature of the metabolic pathway. Trichloroethene has been classified by IARC in Group 3. It has been shown to induce lung and liver tumours in various strains of mice at toxic doses. However, there are no conclusive data that this chemical causes cancer in other species. Trichloroethene is a weakly active mutagen in bacteria and yeast.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to trichloroethene. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, a tentative guideline value of 0.03 mg/litre was recommended; the guideline was designated as tentative because, although carcinogenicity was observed in one species only, the compound occurs relatively frequently in drinking-water. The 1993 Guidelines established a provisional health-based guideline value of 0.07 mg/litre for trichloroethene. The value was provisional because an uncertainty factor of 3000 was used in its derivation.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Tetrachloroethene

Tetrachloroethene has been used primarily as a solvent in dry cleaning industries and to a lesser extent as a degreasing solvent. It is widespread in the environment and is found in trace amounts in water, aquatic organisms, air, foodstuffs and human tissue. The highest environmental levels of tetrachloroethene are associated with commercial dry cleaning and metal degreasing industries. Emissions can sometimes lead to high concentrations in groundwater. Tetrachloroethene in anaerobic groundwater may degrade to more toxic compounds, including vinyl chloride.

Guideline value	40 µg/litre
Occurrence	Concentrations in drinking-water are generally below 3 µg/litre, although much higher concentrations have been detected in well water (23 mg/litre) and in contaminated groundwater (1 mg/litre).
TDI	14 µg/kg of body weight, based on hepatotoxic effects observed in a 6-week gavage study in male mice and a 90-day drinking-water study in male and female rats, and taking into consideration carcinogenic potential (but not the short length of the study, in view of the database and considerations regarding the application of the dose via drinking-water in one of the two critical studies)
Limit of detection	0.2 µg/litre by gas chromatography with electron capture detection; 4.1 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	0.001 mg/litre should be achievable using air stripping

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

At high concentrations, tetrachloroethene causes central nervous system depression. Lower concentrations of tetrachloroethene have been reported to damage the liver and the kidneys. IARC has classified tetrachloroethene in Group 2A. It has been reported to produce liver tumours in male and female mice, with some evidence of mononuclear cell leukaemia in male and female rats and kidney tumours in male rats. The overall evidence from studies conducted to assess the genotoxicity of tetrachloroethene, including induction of single-strand DNA breaks, mutation in germ cells and chromosomal aberrations in vitro and in vivo, indicates that tetrachloroethene is not genotoxic.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to tetrachloroethene. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, a tentative guideline value of 0.01 mg/litre was recommended; the guideline was designated as tentative because, although the carcinogenicity data did not justify a full guideline value, the compound was considered to have important health implications when present in drinking-water. The 1993 Guidelines established a health-based guideline value of 0.04 mg/litre for tetrachloroethene.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Benzene

Benzene is used principally in the production of other organic chemicals. It is present in petrol, and vehicular emissions constitute the main source of benzene in the environment. Benzene may be introduced into water by industrial effluents, spills of petroleum and atmospheric pollution.

Guideline value	10 µg/litre
Occurrence	Concentrations in drinking-water are generally less than 5 µg/litre.
Basis of guideline derivation	Robust linear extrapolation model (because of statistical lack of fit of some of the data with the linearized multistage model) applied to leukaemia and lymphomas in female mice and oral cavity squamous cell carcinomas in male rats in a 2-year gavage study in rats and mice
Limit of detection	0.2 µg/litre by gas chromatography with photoionization detection and confirmation by mass spectrometry
Treatment achievability	0.01 mg/litre should be achievable using GAC or air stripping
Additional comments	Lower end of estimated range of concentrations in drinking-water corresponding to an upper bound excess lifetime cancer risk of 10^{-5} (10–80 µg/litre) corresponds to the estimate derived from data on leukaemia from epidemiological studies involving inhalation exposure, which formed the basis for the previous guideline value. The previous guideline value is therefore retained.

Toxicological Review

Acute exposure of humans to high concentrations of benzene primarily affects the central nervous system. At lower concentrations, benzene is toxic to the haematopoietic system, causing a continuum of haematological changes, including leukaemia. Because benzene is carcinogenic to humans, IARC has classified it in Group 1. Haematological abnormalities similar to those observed in humans have been observed in animal species exposed to benzene. In animal studies, benzene was shown to be carcinogenic following both inhalation and ingestion. It induced several types of tumours in both rats and mice in a 2-year carcinogenesis bioassay by gavage in corn oil. Benzene has not been found to be mutagenic in bacterial assays, but it has been shown to cause chromosomal aberrations in vivo in a number of species, including humans, and to be positive in the mouse micronucleus test.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to benzene. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, a health-based guideline value of 0.01 mg/litre was recommended for benzene based on human leukaemia data from inhalation exposure applied to a linear multistage extrapolation model. The 1993 Guidelines estimated the range of benzene concentrations in drinking-water corresponding to an excess lifetime cancer risk of 10^{-5} to be 0.01–0.08 mg/litre based on carcinogenicity in female mice and male rats. As the lower end of this estimate corresponds to the estimate derived from epidemiological data, which formed the basis for the previous guideline value of 0.01 mg/litre associated with a 10^{-5} excess lifetime cancer risk, the guideline value of 0.01 mg/litre was retained.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization. *Health criteria and other supporting information*

Toluene

Most toluene (in the form of benzene–toluene–xylene mixtures) is used in the blending of petrol. It is also used as a solvent and as a raw material in chemical production. The main exposure is via air. Exposure is increased by smoking and in traffic.

Guideline value	700 µg/litre
Occurrence	Concentrations of a few micrograms per litre have been found in surface water, groundwater and drinking-water; point emissions can lead to higher concentrations in groundwater (up to 1 mg/litre)
TDI	223 µg/kg of body weight, based on a LOAEL of 312 mg/kg of body weight per day for marginal hepatotoxic effects observed in a 13-week gavage study in mice, correcting for 5 days/week dosing and using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for the short duration of the study and use of a LOAEL instead of a NOAEL)
Limit of detection	0.13 µg/litre by gas chromatography with flame ionization detection; 6 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	0.001 mg/litre should be achievable using air stripping
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value exceeds the lowest reported odour threshold for toluene in water.

Toxicological Review

Toluene is absorbed completely from the gastrointestinal tract and rapidly distributed in the body, with a preference for adipose tissue. Toluene is rapidly metabolized and, following conjugation, excreted predominantly in urine. With occupational exposure to toluene by inhalation, impairment of the central nervous system and irritation of mucous membranes are observed. The acute oral toxicity is low. Toluene exerts embryotoxic and fetotoxic effects, but there is no clear evidence for teratogenic activity in laboratory animals and humans. In long-term inhalation studies in rats and mice, there is no evidence for carcinogenicity of toluene. Genotoxicity tests in vitro were negative, whereas in vivo assays showed conflicting results with respect to chromosomal aberrations.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to toluene. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, no guideline value was recommended after a detailed evaluation of the compound. The 1993 Guidelines established a health-based guideline value of 0.7 mg/litre for toluene, but noted that this value exceeds the lowest reported odour threshold for toluene in water (0.024 mg/litre).

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Xylenes

Xylenes are used in blending petrol, as a solvent and as a chemical intermediate. They are released to the environment largely via air. Exposure to xylenes is mainly from air, and exposure is increased by smoking.

Guideline value	500 µg/litre
Occurrence	Concentrations of up to 8 µg/litre have been reported in surface water, groundwater and drinking-water; levels of a few milligrams per litre were found in groundwater polluted by point emissions.
TDI	179 µg/kg of body weight, based on a NOAEL of 250 mg/kg of body weight per day for decreased body weight in a 103-week gavage study in rats, correcting for 5 days/week dosing and using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for the limited toxicological end-points)
Limit of detection	0.1 µg/litre by gas chromatography/mass spectrometry; 1 µg/litre by gas chromatography with flame ionization detector
Treatment achievability	0.005 mg/litre should be achievable using GAC or air stripping
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value exceeds the lowest reported odour threshold for xylenes in drinking-water.

Toxicological Review

Xylenes are rapidly absorbed by inhalation. Data on oral exposure are lacking. Xylenes are rapidly distributed in the body, predominantly in adipose tissue. They are almost completely metabolized and excreted in urine. The acute oral toxicity of xylenes is low. No convincing evidence for teratogenicity has been found. Long-term carcinogenicity studies have shown no evidence for carcinogenicity. In vitro as well as in vivo mutagenicity tests have proved negative.

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 xylenes. The 1993 Guidelines proposed a health-based guideline value of 0.5 mg/litre for xylenes, noting that this value exceeds the lowest reported odour threshold for xylenes in drinking-water (0.02 mg/litre).

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Ethylbenzene

The primary sources of ethylbenzene in the environment are the petroleum industry and the use of petroleum products. Because of its physical and chemical properties, more than 96% of ethylbenzene in the environment can be expected to be present in air. Values of up to 26 µg/m^3 in air have been reported. Ethylbenzene is found in trace amounts in surface water, groundwater, drinking-water and food.

Guideline value	300 µg/litre
Occurrence	Concentrations in drinking-water are generally below 1 µg/litre; levels up to 300 µg/litre have been reported in groundwater contaminated by point emissions.
TDI	97.1 µg/litre, based on a NOAEL of 136 mg/kg of body weight per day for hepatotoxicity and nephrotoxicity observed in a limited 6-month study in rats, correcting for 5 days/week dosing and using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for the limited database and short duration of the study)
Limit of detection	0.002–0.005 µg/litre by gas chromatography with photoionization detector; 0.03–0.06 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	0.001 mg/litre should be achievable using air stripping
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value exceeds the lowest reported odour threshold for ethylbenzene in drinking-water.

Toxicological Review

Ethylbenzene is readily absorbed by oral, inhalation or dermal routes. In humans, storage in fat has been reported. Ethylbenzene is almost completely converted to soluble metabolites, which are excreted rapidly in urine. The acute oral toxicity is low. No definite conclusions can be drawn from limited teratogenicity data. No data on reproduction, long-term toxicity or carcinogenicity are available. Ethylbenzene has shown no evidence of genotoxicity in in vitro or in in vivo systems.

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 ethylbenzene. The 1993 Guidelines proposed a health-based guideline value of 0.3 mg/litre for ethylbenzene, noting that this value exceeds the lowest reported odour threshold for ethylbenzene in drinking-water (0.002 mg/litre).

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Styrene

Styrene, which is used primarily for the production of plastics and resins, is found in trace amounts in surface water, drinking-water and food. In industrial areas, exposure levels from air can be a few hundred micrograms per day. Smoking may increase daily exposure by up to 10-fold.

Guideline value	20 µg/litre
Occurrence	Has been detected in drinking-water and surface water at concentrations below 1 µg/litre
TDI	7.7 µg/kg of body weight, based on a NOAEL of 7.7 mg/kg of body weight per day for decreased body weight observed in a 2-year drinking-water study in rats, and using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for the carcinogenicity and genotoxicity of the reactive intermediate styrene-7,8-oxide)
Limit of detection	0.3 µg/litre by gas chromatography with photoionization detection and confirmation by mass spectrometry
Treatment achievability	0.02 mg/litre may be achievable using GAC
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Styrene may affect the acceptability of drinking-water at the guideline value.

Toxicological Review

Following oral or inhalation exposure, styrene is rapidly absorbed and widely distributed in the body, with a preference for lipid depots. It is metabolized to the active intermediate styrene-7,8-oxide, which is conjugated with glutathione or further metabolized. Metabolites are rapidly and almost completely excreted in urine. Styrene has a low acute toxicity. Upon occupational exposure, irritation of mucous membranes, depression of the central nervous system and possibly hepatoxicity can occur. In short-term toxicity studies in rats, impairment of glutathione transferase activity and reduced glutathione concentrations were observed. In in vitro tests, styrene has been shown to be mutagenic in the presence of metabolic activation only. In in vitro as well as in in vivo studies, chromosomal aberrations have been observed, mostly at high doses of styrene. The reactive intermediate styrene-7,8-oxide is a direct-acting mutagen. In long-term studies, orally administered styrene increased the incidence of lung tumours in mice at high dose levels but had no carcinogenic effect in rats. Styrene-7,8-oxide was carcinogenic in rats after oral administration. IARC has classified styrene in Group 2B. The available data suggest that the carcinogenicity of styrene is due to overloading of the detoxification mechanism for styrene-7,8-oxide (e.g., glutathione depletion).

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 styrene. The 1993 Guidelines established a health-based guideline value of 0.02 mg/litre for styrene, noting that styrene may affect the acceptability of drinking-water at this concentration.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Monochlorobenzene

Releases of monochlorobenzene (MCB) to the environment are thought to be mainly due to volatilization losses associated with its use as a solvent in pesticide formulations, as a degreasing agent and from other industrial applications. MCB has been detected in surface water, groundwater and drinking-water; mean concentrations were less than 1 µg/litre in some potable water sources (maximum 5 µg/litre) in Canada. The major source of human exposure is probably air.

MCB is of low acute toxicity. Oral exposure to high doses of MCB affects mainly the liver, kidneys and haematopoietic system. There is limited evidence of carcinogenicity in male rats, with high doses increasing the occurrence of neoplastic nodules in the liver. The majority of evidence suggests that MCB is not mutagenic; although it binds to DNA in vivo, the level of binding is low.

A health-based value of 300 µg/litre can be calculated for MCB on the basis of a TDI of 85.7 µg/kg of body weight, based on neoplastic nodules identified in a 2-year rat study with dosing by gavage, and taking into consideration the limited evidence of carcinogenicity. However, because MCB occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value. It should also be noted that the health-based value far exceeds the lowest reported taste and odour threshold for MCB in water.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to monochlorobenzene. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, no guideline value for chlorobenzene was recommended after a detailed evaluation of the compound. Following consideration of the calculated toxicological limit for drinking-water of 0.005–0.05 mg/litre based on a tentative acceptable daily intake and the fact that the threshold odour concentration of monochlorobenzene in water is 0.03 mg/litre, no guideline value was recommended, and 0.003 mg/litre was recommended to avoid taste and odour problems in drinking-water. The 1993 Guidelines proposed a health-based guideline value of 0.3 mg/litre for monochlorobenzenes, noting that this value far exceeds the lowest reported taste and odour threshold for monochlorobenzene in water (0.01 mg/litre).

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Dichlorobenzenes (1,2-dichlorobenzene, 1,3-dichlorobenzene, 1,4-dichlorobenzene)

The dichlorobenzenes (DCBs) are widely used in industry and in domestic products such as odour-masking agents, chemical dyestuffs and pesticides. Sources of human exposure are predominantly air and food.

Guideline values	
1,2-dichlorobenzene	1000 µg/litre
1,4-dichlorobenzene	300 µg/litre
Occurrence	Have been found in raw water sources at levels as high as 10 µg/litre and in drinking-water at concentrations up to 3 µg/litre; much higher concentrations (up to 7 mg/litre) present in contaminated groundwater
TDI	
1,2-dichlorobenzene	429 µg/kg of body weight, based on a NOAEL of 60 mg/kg of body weight per day for tubular degeneration of the kidney identified in a 2-year mouse gavage study, correcting for 5 days/week dosing and using an uncertainty factor of 100 (for inter- and intraspecies variation)
1,4-dichlorobenzene	107 µg/kg of body weight, based on a LOAEL of 150 mg/kg of body weight per day for kidney effects identified in a 2-year rat study, correcting for 5 days/week dosing and using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for the use of a LOAEL instead of a NOAEL and the carcinogenicity end-point)
Limit of detection	0.01–0.25 µg/litre by gas–liquid chromatography with electron capture detection; 3.5 µg/litre by gas chromatography using a photoionization detector
Treatment achievability	0.01 mg/litre should be achievable using air stripping
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Guideline values for both 1,2- and 1,4-dichlorobenzene far exceed their lowest reported taste thresholds in water.

Toxicological Review

1,2-Dichlorobenzene

1,2-DCB is of low acute toxicity by the oral route of exposure. Oral exposure to high doses of 1,2-DCB affects mainly the liver and kidneys. The balance of evidence suggests that 1,2-DCB is not genotoxic, and there is no evidence for its carcinogenicity in rodents.

1,3-Dichlorobenzene

There are insufficient toxicological data on this compound to permit a guideline value to be proposed, but it should be noted that it is rarely found in drinking-water.

1,4-Dichlorobenzene

1,4-DCB is of low acute toxicity, but there is evidence that it increases the incidence of renal tumours in rats and of hepatocellular adenomas and carcinomas in mice after long-term exposure. IARC has placed 1,4-DCB in Group 2B. 1,4-DCB is not considered to be genotoxic, and the relevance for humans of the tumours observed in animals is doubtful.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to DCBs. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, no guideline value was recommended for 1,2- or 1,4-DCB after a detailed evaluation of the compounds. Toxicological limits for drinking-water of 0.005–0.05 mg/litre were derived based on an acceptable daily intake; given that the threshold odour concentrations are 0.003 mg/litre for 1,2-DCB and 0.001 mg/litre for 1,4-DCB, 10% of each of these values was recommended as a level unlikely to give rise to taste and odour problems in drinking-water supplies. The 1993 Guidelines calculated a health-based guideline value of 1 mg/litre for 1,2-DCB, which far exceeds the lowest reported taste threshold of 1,2-DCB in water (0.001 mg/litre). There were insufficient toxicological data on 1,3-DCB to permit a guideline value to be proposed, but the 1993 Guidelines noted that it is rarely found in drinking-water. A health-based guideline value of 0.3 mg/litre was proposed for 1,4-DCB, which far exceeds the lowest reported odour threshold of 1,4-DCB in water (0.0003 mg/litre).

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Trichlorobenzenes (total)

Releases of trichlorobenzenes (TCBs) into the environment occur through their manufacture and use as industrial chemicals, chemical intermediates and solvents. TCBs are found in drinking-water, but rarely at levels above 1 µg/litre. General population exposure will primarily result from air and food.

The TCBs are of moderate acute toxicity. After short-term oral exposure, all three isomers show similar toxic effects, predominantly on the liver. Long-term toxicity and carcinogenicity studies via the oral route have not been carried out, but the data available suggest that all three isomers are non-genotoxic.

A health-based value of 20 µg/litre can be calculated for total TCBs on the basis of a TDI of 7.7 µg/kg of body weight, based on liver toxicity identified in a 13-week rat study, taking into consideration the short duration of the study. However, because TCBs occur at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value. It should be noted that the health-based value exceeds the lowest reported odour threshold in water.

History of Guideline Development

The 1958, 1963 and 1971 WHO International Standards for Drinking-water did not refer to trichlorobenzenes. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, it was concluded that insufficient health data were available from which to derive a guideline value for 1,2,4-trichlorobenzene. The 1993 Guidelines proposed a health-based guideline value of 0.02 mg/litre for total trichlorobenzenes, because of the similarity in the toxicity of the three isomers, but noted that this value exceeds the lowest reported odour threshold in water (0.005 mg/litre for 1,2,4-trichlorobenzene).

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Di(2-ethylhexyl)adipate

Di(2-ethylhexyl)adipate (DEHA) is used mainly as a plasticizer for synthetic resins such as polyvinyl chloride (PVC). Reports of the presence of DEHA in surface water and drinking-water are scarce, but DEHA has occasionally been identified in drinking-water at levels of a few micrograms per litre. As a consequence of its use in PVC films, food is the most important source of human exposure (up to 20 mg/day).

DEHA is of low short-term toxicity; however, dietary levels above 6000 mg/kg of feed induce peroxisomal proliferation in the liver of rodents. This effect is often associated with the development of liver tumours. DEHA induced liver carcinomas in female mice at very high doses but not in male mice or rats. It is not genotoxic. IARC has placed DEHA in Group 3.

A health-based value of 80 µg/litre can be calculated for DEHA on the basis of a TDI of 280 µg/kg of body weight, based on fetotoxicity in rats, and allocating 1% of the TDI to drinking-water. However, because DEHA occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value.

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 di(2-ethylhexyl)adipate. The 1993 Guidelines proposed a health-based guideline value of 0.08 mg/litre for di(2-ethylhexyl)adipate in drinking-water.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Di(2-ethylhexyl)phthalate

Di(2-ethylhexyl)phthalate (DEHP) is used primarily as a plasticizer. Exposure among individuals may vary considerably because of the broad nature of products into which DEHP is incorporated. In general, food will be the main exposure route.

Guideline value	8 µg/litre
Occurrence	Found in surface water, groundwater and drinking-water in concentrations of a few micrograms per litre; in polluted surface water and groundwater, concentrations of hundreds of micrograms per litre have been reported
TDI	25 µg/kg of body weight, based on a NOAEL of 2.5 mg/kg of body weight per day for peroxisomal proliferation in the liver in rats, using an uncertainty factor of 100 for inter- and intraspecies variation
Limit of detection	0.1 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	No data available
Guideline derivation	
• *allocation to water*	1% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The reliability of some data on environmental water samples is questionable because of secondary contamination during sampling and working-up procedures. Concentrations that exceed the solubility more than 10-fold have been reported.

Toxicological Review

In rats, DEHP is readily absorbed from the gastrointestinal tract. In primates (including humans), absorption after ingestion is lower. Species differences are also observed in the metabolic profile. Most species excrete primarily the conjugated mono-ester in urine. Rats, however, predominantly excrete terminal oxidation products. DEHP is widely distributed in the body, with highest levels in liver and adipose tissue, without showing significant accumulation. The acute oral toxicity is low. The most striking effect in short-term toxicity studies is the proliferation of hepatic peroxisomes, indicated by increased peroxisomal enzyme activity and histopathological changes. The available information suggests that primates, including humans, are far less sensitive to this effect than rodents. In long-term oral carcinogenicity studies, hepatocellular carcinomas were found in rats and mice. IARC has concluded that DEHP is possibly carcinogenic to humans (Group 2B). In 1988, JECFA evaluated DEHP and recommended that human exposure to this compound in food be reduced to the lowest level attainable. The Committee considered that this might be achieved by using alternative plasticizers or alternatives to plastic material containing DEHP. In a variety of in vitro and in vivo studies, DEHP and its metabolites have shown no evidence of genotoxicity, with the exception of induction of aneuploidy and cell transformation.

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 di(2-ethylhexyl)phthalate. The 1993 Guidelines established a health-based guideline value of 0.008 mg/litre for di(2-ethylhexyl)phthalate in drinking-water.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Hexachlorobutadiene

Hexachlorobutadiene (HCBD) is used as a solvent in chlorine gas production, a pesticide, an intermediate in the manufacture of rubber compounds and a lubricant. Concentrations of up to 6 µg/litre have been reported in the effluents from chemical manufacturing plants. It is also found in air and food.

Guideline value	0.6 µg/litre
Occurrence	Has been detected in surface water at concentrations of a few micrograms per litre and in drinking-water at concentrations below 0.5 µg/litre
TDI	0.2 µg/kg of body weight, based on a NOAEL of 0.2 mg/kg of body weight per day for renal toxicity in a 2-year feeding study in rats, using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for limited evidence of carcinogenicity and genotoxicity of some metabolites)
Limit of detection	0.01 µg/litre by gas chromatography/mass spectrometry; 0.18 µg/litre by gas chromatography with electron capture detection
Treatment achievability	0.001 mg/litre should be achievable using GAC
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The practical quantification level for HCBD is of the order of 2 µg/litre, but concentrations in drinking-water can be controlled by specifying the HCBD content of products coming into contact with it.

Toxicological Review

HCBD is easily absorbed and metabolized via conjugation with glutathione. This conjugate can be further metabolized to a nephrotoxic derivative. Kidney tumours were observed in a long-term oral study in rats. HCBD has not been shown to be carcinogenic by other routes of exposure. IARC has placed HCBD in Group 3. Positive and negative results for HCBD have been obtained in bacterial assays for point mutation; however, several metabolites have given positive results.

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 hexachlorobutadiene. The 1993 Guidelines derived a health-based guideline value of 0.0006 mg/litre for hexachlorobutadiene, noting that although a practical quantification level for hexachlorobutadiene is of the order of 0.002 mg/litre, concentrations in drinking-water can be controlled by specifying the hexachlorobutadiene content of products coming into contact with it.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization. *Health criteria and other supporting information*

Edetic acid (EDTA)

Human exposure to EDTA arises directly from its use in food additives, medicines, and personal care and hygiene products. Exposure to EDTA from drinking-water is probably very small in comparison with that from other sources. Once EDTA is present in the aquatic environment, its speciation will depend on the water quality and the presence of trace metals with which it can combine. The removal of EDTA from communal wastewater by biodegradation in sewage purification plants is very limited.

Guideline value	600 µg/litre (for EDTA as the free acid)
Occurrence	Present in surface waters generally at concentrations below 70 µg/litre, although higher concentrations (900 µg/litre) have been measured; detected in drinking-water prepared from surface waters at concentrations of 10–30 µg/litre
ADI	1.9 mg/kg of body weight as the free acid (ADI of 2.5 mg/kg of body weight proposed by JECFA for calcium disodium edetate as a food additive)
Limit of detection	1 µg/litre by potentiometric stripping analyis
Treatment achievability	0.01 mg/litre using GAC plus ozonation
Guideline derivation	
• *allocation to water*	1% of ADI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Concern has been expressed over the ability of EDTA to complex, and therefore reduce the availability of, zinc. However, this is of significance only at elevated doses substantially in excess of those encountered in the environment.

Toxicological Review

Calcium disodium edetate is poorly absorbed from the gut. The long-term toxicity of EDTA is complicated by its ability to chelate essential and toxic metals. Those toxicological studies that are available indicate that the apparent toxicological effects of EDTA have in fact been due to zinc deficiency as a consequence of complexation. EDTA does not appear to be teratogenic or cacinogenic in animals. The vast clinical experience of the use of EDTA in the treatment of metal poisoning has demonstrated its safety in humans.

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 edetic acid. The 1993 Guidelines proposed a provisional health-based guideline value of 0.2 mg/litre for edetic acid, based on an ADI for calcium disodium edetate as a food additive proposed by JECFA in 1973 and assuming that a 10-kg child consumes 1 litre of water per day, in view of the possibility of zinc complexation. The value was considered provisional to reflect the fact that the JECFA ADI had not been considered since 1973. JECFA further evaluated the toxicological studies available on EDTA in 1993 and was unable to add any further important information regarding the toxicity of EDTA and its calcium and sodium salts to the 1973 evaluation. In the addendum to the second edition of the Guidelines, published in 1998, a guideline value of 0.6 mg/litre was derived for EDTA (free acid), using different assumptions from those used in the derivation of the provisional guideline value in the 1993 Guidelines. In particular, it was noted that the ability of EDTA to complex, and therefore reduce the availability of, zinc was of significance only at elevated doses substantially in excess of those encountered in the environment.

Primary Reference

WHO (1998) Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

Nitrilotriacetic acid (NTA)

Nitrilotriacetic acid (NTA) is used primarily in laundry detergents as a replacement for phosphates and in the treatment of boiler water to prevent accumulation of mineral scale.

Guideline value	200 µg/litre
Occurrence	Concentrations in drinking-water usually do not exceed a few micrograms per litre, although concentrations as high as 35 µg/litre have been measured.
TDI	10 µg/kg of body weight, based on nephritis and nephrosis in a 2-year study in rats, taking into consideration carcinogenic potential at high doses
Limit of detection	0.2 µg/litre using gas chromatography with a nitrogen-specific detector
Treatment achievability	No data available

Guideline derivation

• *allocation to water*	50% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

NTA is not metabolized in animals and is rapidly eliminated, although some may be briefly retained in bone. It is of low acute toxicity to animals, but it has been shown to produce kidney tumours in rodents following long-term exposure to high doses. IARC has placed NTA in Group 2B. It is not genotoxic, and the reported induction of tumours is believed to be due to cytotoxicity resulting from the chelation of divalent cations such as zinc and calcium in the urinary tract, leading to the development of hyperplasia and subsequently neoplasia.

History of Guideline Development

The 1958 and 1963 WHO International Standards for Drinking-water did not refer to nitrilotriacetic acid. The 1971 International Standards stated that nitrilotriacetate should be controlled in drinking-water, but that insufficient information was available to enable a tentative limit to be established. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, it was determined that no further action on nitrilotriacetic acid was required. A health-based guideline value of 0.2 mg/litre was established for nitrilotriacetic acid in the 1993 Guidelines.

Primary Reference

WHO (1996) Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.

Health criteria and other supporting information

1,4-Dioxane

1,4-Dioxane is used as a stabilizer in chlorinated solvents, as a solvent, for agricultural and biochemical intermediates and for adhesives, sealants, cosmetics, pharmaceuticals, rubber chemicals and surface coatings.

Guideline value	50 µg/litre (derived using TDI approach as well as linear multistage modelling)
Occurrence	Has been measured in surface water at concentrations up to 40 µg/litre and in groundwater at concentrations up to 80 µg/litre
TDI	16 µg/kg of body weight, based on a NOAEL of 16 mg/kg of body weight per day for hepatocellular tumours observed in a long-term drinking-water study in rats, using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for non-genotoxic carcinogenicity)
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Basis of guideline derivation based on carcinogenicity	Linear multistage model applied to data for hepatic tumours from drinking-water studies in rats, without body surface correction
Limit of detection	0.1 and 50 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	Not removed using conventional water treatment processes; effectively removed by biological activated carbon treatment
Additional comments	Similar guideline values were derived using the TDI approach (assuming 1,4-dioxane is not genotoxic in humans at low doses) and linear multistage modelling.

Toxicological Review

1,4-Dioxane caused hepatic and nasal cavity tumours in rodents in most long-term oral studies conducted. Tumours in peritoneum, skin and mammary gland were also observed in rats given a high dose. Lung tumours were specifically detected after intraperitoneal injection. Although cohort studies of workers did not reveal any elevation in the incidence of death by cancer, a significant increase in the incidence of liver cancer was found in a comparative mortality study. However, the evidence is inadequate for human carcinogenicity assessment because of small samples or lack of exposure data. IARC has classified 1,4-dioxane as Group 2B (possibly carcinogenic to humans).

History of Guideline Development

1,4-Dioxane was not referred to in the 1958, 1963 and 1971 WHO International Standards for Drinking-water, the first edition of the Guidelines for Drinking-water Quality, published in 1984, the second edition of the Guidelines, published in 1993, or the addendum to the second edition, published in 1998.

Primary Reference

New background document *Health criteria and other supporting information*

Pentachlorophenol (CAS No. 87-86-5)

Pentachlorophenol (PCP) and other chlorophenols are used primarily for protecting wood from fungal growth. Food is usually the major source of exposure to PCP unless there is a specific local chlorophenol contamination of drinking-water or exposure from log homes treated with PCP.

Provisional guideline value	9 µg/litre
Occurrence	Concentrations in water samples are usually below 10 µg/litre, although much higher concentrations in groundwater may be measured under certain conditions.
Basis of guideline derivation	Multistage modelling of tumour incidence in a US NTP bioassay without incorporation of a body surface area correction, recognizing that there are interspecies differences in metabolism between animals and humans, with an important metabolite in rat and a minor metabolite in humans
Limit of detection	0.005–0.01 µg/litre by gas chromatography with electron capture detection
Treatment achievability	0.4 µg/litre should be achievable using GAC
Additional comments	The concentration of PCP associated with an upper bound 10^{-5} excess lifetime cancer risk is similar to the guideline value established in the second edition, so that guideline value is retained. The guideline value for PCP is considered provisional because of the variations in metabolism between experimental animals and humans.

Toxicological Review

IARC classified PCP in Group 2B (the agent is possibly carcinogenic to humans) on the basis of inadequate evidence of carcinogenicity in humans but sufficient evidence in experimental animals. There is suggestive, although inconclusive, evidence of the carcinogenicity of PCP from epidemiological studies of populations exposed to mixtures that include PCP. Conclusive evidence of carcinogenicity has been obtained in one animal species (mice). Although there are notable variations in metabolism between experimental animals and humans, it was considered prudent to treat PCP as a potential carcinogen.

History of Guideline Development

The 1958 and 1963 WHO International Standards for Drinking-water did not refer to PCP, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the Guidelines for Drinking-water Quality, published in 1984, a health-based guideline value of 0.01 mg/litre was recommended for PCP. The 1993 Guidelines established a health-based guideline value of 0.009 mg/litre for PCP in drinking-water. This value was considered provisional because PCP was evaluated only at the final Task Group meeting on the basis of an Environmental Health Criteria monograph (No. 71). The concentration of PCP associated with a 10^{-5} excess lifetime cancer risk was found to be similar to the provisional guideline value established in 1993, and so that provisional guideline value was retained in the addendum to the Guidelines, published in 1998.

Primary Reference

WHO (1998) Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2. Health criteria and other supporting information. Geneva, World Health Organization.
Health criteria and other supporting information

8.7.3 Chemicals from Agricultural Activities

Chemicals are used in agriculture on crops and in animal husbandry. Nitrate may be present as a consequence of tillage when there is no growth to take up nitrate released from decomposing plants, from the application of excess inorganic or organic fertilizer, and in slurry from animal production. Most chemicals that may arise from agriculture are pesticides, although their presence will depend on many factors, and not all pesticides are used in all circumstances or climates. Contamination can result from application and subsequent movement following rainfall or from inappropriate disposal methods.

Some pesticides are also used in non-agricultural circumstances, such as the control of weeds on roads and railway lines. These pesticides are also included in this section.

Chemicals listed in Table 8.9 have been excluded from guideline value derivation, as a review of the literature on occurrence and/or credibility of occurrence in drinking-water has shown evidence that the chemicals do not occur in drinking-water.

Table 8.19. Chemicals from agricultural activities excluded from guideline value derivation

Chemical	Reason for exclusion
Amitraz	Degrades rapidly in the environment and is not expected to occur at measurable concentrations in drinking-water supplies
Chlorobenzilate	Unlikely to occur in drinking-water
Chlorothalonil	Unlikely to occur in drinking-water
Cypermethrin	Unlikely to occur in drinking-water
Diazinon	Unlikely to occur in drinking-water
Dinoseb	Unlikely to occur in drinking-water
Ethylene thiourea	Unlikely to occur in drinking-water
Fenamiphos	Unlikely to occur in drinking-water
Formothion	Unlikely to occur in drinking-water
Hexachlorocyclohexanes (mixed isomers)	Unlikely to occur in drinking-water
MCPB	Unlikely to occur in drinking-water
Methamidophos	Unlikely to occur in drinking-water
Methomyl	Unlikely to occur in drinking-water
Mirex	Unlikely to occur in drinking-water
Monocrotophos	Has been withdrawn from use in many countries and is unlikely to occur in drinking-water
Oxamyl	Unlikely to occur in drinking-water
Phorate	Unlikely to occur in drinking-water
Propoxur	Unlikely to occur in drinking-water
Pyridate	Not persistent and only rarely found in drinking-water
Quintozene	Unlikely to occur in drinking-water
Toxaphene	Unlikely to occur in drinking-water
Triazophos	Unlikely to occur in drinking-water
Tributyltin oxide	Unlikely to occur in drinking-water
Trichlorfon	Unlikely to occur in drinking-water

Guideline values have not been established for the chemicals listed in Table 8.10 because:
evidence indicates that the chemical does not occur at concentrations at or near levels expected to cause a health concern, or
there is lack of evidence of health effects, or there are insufficient data to support the establishment of a guideline value.

Table 8.20. Chemicals from agricultural activities for which guideline values have not been established

Chemical	Reason for exclusion
Ammonia	Occur at concentrations well below those at which toxic effects are observed
Bentazone	Occur at concentrations well below those at which toxic effects are observed
Dichloropropane, 1,3-	Data insufficient to permit recommendation of a health-based guideline value
Diquat	Rarely found in drinking-water, but may be used as an aquatic herbicide for the control of free-floating and submerged aquatic weeds in ponds, lakes and irrigation ditches
Endosulfan	Occur at concentrations well below those at which toxic effects are observed
Fenitrothion	Occur at concentrations well below those at which toxic effects are observed
Glyphosate and AMPA	Occur at concentrations well below those at which toxic effects are observed
Heptachlor and heptachlor epoxide	Occur at concentrations well below those at which toxic effects are observed
Malathion	Occurs at concentrations well below those at which toxic effects are observed
Parathion	Occurs at concentrations well below those at which toxic effects are observed
Parathion-methyl	Occurs at concentrations well below those at which toxic effects are observed
Permethrin	Occur at concentrations well below those at which toxic effects are observed
Phenylphenol, 2- and its sodium salt	Occurs at concentrations well below those at which toxic effects are observed
Propanil	Readily transformed into toxic metabolites

Guideline values have been established for the chemicals listed in Table 8.11, which meet all of the criteria for inclusion.

Table 8.21. Guideline values for chemicals from agricultural activities that are of health significance in drinking-water

Non-pesticides	Guideline value[a] (mg/litre)	Remarks
Nitrate (as NO_3^-)	50	Short-term exposure
Nitrite (as NO_2^-)	3	Short-term exposure
	0.2 (P)	Long-term exposure

Pesticides used in agriculture	Guideline value[a] (µg/litre)	Remarks
Alachlor	20[b]	
Aldicarb	10	Applies to aldicarb sulfoxide and aldicarb sulfone
Aldrin/dieldrin	0.03	For combined aldrin plus dieldrin
Atrazine	2	
Carbofuran	7	
Chlordane	0.2	
Chlorotoluron	30	
Cyanazine	0.6	
2,4-D (2,4-dichlorophenoxyacetic acid)	30	Applies to free acid
2,4-DB	90	
1,2-Dibromo-3-chloropropane	1[b]	
1,2-Dibromoethane	0.4–15[b] (P)	
1,2-Dichloropropane (1,2-DCP)	40 (P)	
1,3-Dichloropropene	20[b]	
Dichlorprop	100	
Dimethoate	6	
Endrin	0.6	
Fenoprop	9	
Hexachlorobenzene	1[b]	
Isoproturon	9	
Lindane	0.3	

Pesticides used in agriculture	Guideline value[a] (µg/litre)	Remarks
MCPA	2	
Mecoprop	10	
Methoxychlor	20	
Metolachlor	10	
Molinate	6	
Pendimethalin	20	
Pentachlorophenol	9[b] (P)	
Simazine	2	
2,4,5-T	9	
Terbuthylazine	7	
Trifluralin	20	

[a] P = evidence of a potential hazard, but the available information on health effects is limited.

[b] For substances that are considered to be carcinogenic, the guideline value is the concentration in drinking-water associated with an upper bound excess lifetime cancer risk of 10^{-5} (one additional cancer per 100 000 of the population ingesting drinking-water containing the substance at the guideline value for 70 years). Concentrations associated with estimated upper bound excess lifetime cancer risks of 10^{-4} and 10^{-6} can be calculated by multiplying and dividing, respectively, the guideline value by 10.

In cases in which the concentration associated with an upper bound excess lifetime cancer risk of 10^{-5} is not feasible as a result of inadequate analytical or treatment technology, a provisional guideline value (designated A or T, respectively) is recommended at a practicable level.

It should be emphasized that the guideline values for carcinogenic substances have been computed from hypothetical mathematical models that cannot be verified experimentally and that the values should be interpreted differently from TDI-based values because of the lack of precision of the models. At best, these values must be regarded as rough estimates of cancer risk. However, the models used are conservative and probably err on the side of caution. Moderate short-term exposure to levels exceeding the guideline value for carcinogens does not significantly affect the risk.

Non-Pesticides

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, which is formed from the reaction of chlorine with ammonia. 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.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Nitrate and nitrite

Nitrate and nitrite are naturally occurring ions that are part of the nitrogen cycle. Nitrate is used mainly in inorganic fertilizers, and sodium nitrite is used as a food preservative, especially in cured meats. The nitrate concentration in groundwater and surface water is normally low but can reach high levels as a result of leaching or runoff from agricultural land or contamination from human or animal wastes, as a consequence of the oxidation of ammonia and similar sources. Anaerobic conditions may result in the formation and persistence of nitrite. Nitrate/nitrite levels in groundwater are often higher that those in surface water supplies. Chloramination may give rise to the formation of nitrite within the distribution system if the formation of chloramine is not sufficiently controlled and there is free ammonia. The formation of nitrite is as a consequence of microbial activity and is likely to be intermittent. Nitrification in distribution systems can increase nitrite levels, usually by 0.2–1.5 mg/litre.

Guideline value for nitrate	50 mg/litre to protect against methaemoglobinaemia in bottle-fed infants (short-term exposure)
Guideline value / Provisional guideline value for nitrite	3 mg/litre for methaemoglobinaemia in infants (short-term exposure) 0.2 mg/litre (provisional) (long-term exposure)
Guideline value for combined nitrate plus nitrite	The sum of the ratios of the concentrations of each to its guideline value should not exceed 1.
Occurrence	In most countries, nitrate levels in drinking-water derived from surface water do not exceed 10 mg/litre, although nitrate levels in well-water often exceed 50 mg/litre; nitrite levels are normally lower, less than one milligrams per litre.
Basis of guideline derivation	nitrate (bottle-fed infants): in epidemiological studies, methaemoglobinaemia was not reported in infants in areas where drinking-water consistently contained less than 50 mg/litre nitrate nitrite (bottle-fed infants): nitrite is 10 times more potent than nitrate on a molar basis with respect to methaemoglobin formation nitrite (long-term exposure): based on allocation to drinking-water of 10% 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
Limit of detection	0.1 mg/litre (nitrate) and 0.05 mg/litre (nitrite) by liquid chromatography; 0.01–1 mg/litre (nitrate) by spectrometric techniques; 0.005–0.01 mg/litre (nitrite) by a molecular absorption spectrometric method; 22 µg/litre (nitrate) and 35 µg/litre (nitrite) by ion chromatography
Treatment achievability	nitrate: 5 mg/litre or lower should be achievable using biological denitrification (surface waters) or ion exchange (groundwaters) nitrite: 0.1 mg/litre should be achievable using chlorination (to form nitrate)
Additional comments	The guideline value for chronic effects of nitrite is 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 distribution as a consequence of chloramine use will be intermittent, and average exposures over time should not exceed the provisional guideline value.

Nitrite can occur in distribution at higher concentrations when chloramination is used, but the occurrence is almost invariably sporadic.
Methaemoglobinaemia is therefore the most important consideration, and the guideline derived for protection against methaemoglobinaemia would be the most appropriate under these circumstances, allowing for any nitrate that may also be present.
All water systems that practise chloramination should closely and regularly monitor their systems to verify disinfectant levels, microbiological quality and nitrite levels. If nitrification is detected (e.g., reduced disinfectant residuals and increased nitrite levels), steps should be taken to modify the treatment train or water chemistry in order to maintain a safe water quality. Efficient disinfection must never be compromised.

Toxicological Review

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 oxidize haemoglobin (Hb) to methaemoglobin (metHb), which is unable to transport oxygen around the body. The reduced oxygen transport becomes clinically manifest when metHb concentrations reach 10% 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%.

The Hb of young infants is more susceptible to metHb formation than that of older children and adults; this is believed to be the result of the large proportion of fetal Hb, which is more easily oxidized to metHb, still present in the blood of infants. In addition, there is a deficiency in infants of metHb reductase, the enzyme responsible for the reduction of metHb to Hb. The reduction of nitrate to nitrite by gastric bacteria is also higher in infants because of low gastric acidity. The level of nitrate in breast milk is relatively low; when bottle-fed, however, these young infants are at risk because of the potential for exposure to nitrate/nitrite in drinking-water and the relatively high intake of water in relation to body weight. The higher reduction of nitrate to nitrite in young infants is not very well quantified, but it appears that gastrointestinal infections exacerbate the conversion from nitrate to nitrite.

The weight of evidence is strongly against there being an association between nitrite and nitrate exposure in humans and the risk of cancer.

Studies with nitrite in laboratory rats have reported hypertrophy of the adrenal zona glomerulosa. The mechanism of induction of this effect and whether it occurs in other species is unclear. JECFA developed an ADI of 5 mg of potassium nitrite per kilogram of body weight based on the NOAEL in these studies.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* referred to nitrates, stating that the ingestion of water containing nitrates in excess of 50–100 mg/litre (as nitrate) may give rise to methaemoglobinaemia in infants under 1 year of age. In the 1963 International Standards, this value was lowered to 45 mg/litre (as nitrate), which was retained in the 1971 International Standards. The 1971 International Standards first mentioned concern over the possibility of nitrosamine formation *in vivo*; as nitrosamines are a possible hazard to human health, the 1971 Standards stated that it may eventually become necessary to reduce the level of nitrates in water if it is found that this source makes a significant contribution to the hazard to human health arising from nitrosamines. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 10 mg/litre for nitrate-nitrogen was recommended. It was also recommended that the guideline value for nitrite must be correspondingly lower than that for nitrate, and it was noted that the nitrite-nitrogen level should be considerably lower than 1 mg/litre where drinking-water is correctly treated. The 1993 Guidelines concluded that extensive epidemiological data support the current guideline value for nitrate-nitrogen of 10 mg/litre, but stated that this value should be expressed not on the basis of nitrate-nitrogen but on the basis of nitrate itself, which is the chemical entity of concern to health. The guideline value for nitrate is therefore 50 mg/litre. This guideline value

for methaemoglobinaemia in infants, an acute effect, was confirmed in the addendum to the Guidelines, published in 1998. It was also concluded in the 1993 Guidelines that a guideline value for nitrite should be proposed, although no suitable animal studies of methaemoglobinaemia were available. A provisional guideline value for nitrite of 3 mg/litre was therefore proposed by accepting a relative potency for nitrite and nitrate with respect to methaemoglobin formation of 10:1 (on a molar basis). In the addendum to the Guidelines, published in 1998, it was concluded that human data on nitrite reviewed by JECFA supported the current provisional guideline value of 3 mg/litre, based on induction of methaemoglobinaemia in infants. In addition, a guideline value of 0.2 mg/litre for nitrate ion associated with long-term exposure was derived in the addendum to the Guidelines, based on JECFA's ADI derived in 1995. However, because of the uncertainty surrounding the relevance of the observed adverse health effects for humans and the susceptibility of humans compared with animals, this guideline value was considered provisional. Because of the possibility of simultaneous occurrence of nitrite and nitrate in drinking-water, it was recommended in the 1993 and 1998 Guidelines that the sum of the ratios of the concentration of each to its guideline value should not exceed 1.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

Pesticides Used in Agriculture

Alachlor (CAS No. 15972-60-8)

Alachlor is a pre- and post-emergence herbicide used to control annual grasses and many broad-leaved weeds in maize and a number of other crops. It is lost from soil mainly through volatilization, photo-degradation and biodegradation. Many alachlor degradation products have been identified in soil.

Guideline value	20 µg/litre
Occurrence	Has been detected in groundwater and surface water; has also been detected in drinking-water at levels below 2 µg/litre
Basis of guideline derivation	Calculated by applying the linearized multistage model to data on the incidence of nasal tumours in rats
Limit of detection	0.1 µg/litre by gas–liquid chromatography with electrolytic conductivity detection in the nitrogen mode or by capillary column gas chromatography with a nitrogen–phosphorus detector
Treatment achievability	1 µg/litre should be achievable using GAC

Toxicological Review

On the basis of available experimental data, evidence for the genotoxicity of alachlor is considered to be equivocal. However, a metabolite of alachlor has been shown to be mutagenic. Available data from two studies in rats clearly indicate that alachlor is carcinogenic, causing benign and malignant tumours of the nasal turbinate, malignant stomach tumours and benign thyroid tumours.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to alachlor, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Alachlor was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines calculated a guideline value of 0.02 mg/litre for alachlor in drinking-water, corresponding to an excess lifetime cancer risk of 10^{-5}.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Aldicarb (CAS No. 116-06-3)

Aldicarb is a systemic pesticide used to control nematodes in soil and insects and mites on a variety of crops. It is very soluble in water and is highly mobile in soil. It degrades mainly by biodegradation and hydrolysis, persisting for weeks to months.

Guideline value	10 µg/litre, applies to aldicarb sulfoxide and aldicarb sulfone
Occurrence	Frequently found as a contaminant in groundwater, particularly when associated with sandy soil; concentrations in well-water as high as 500 µg/litre have been measured
ADI	3 µg/kg of body weight based on cholinesterase depression in a single oral dose study in human volunteers
Limit of detection	1 µg/litre by reverse-phase high-performance liquid chromatography with fluorescence detection
Treatment achievability	1 µg/litre should be achievable using GAC or ozonation
Guideline derivation	
• *allocation to water*	10% of ADI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value derived from the 1992 JMPR assessment was very similar to the previous guideline value, which was therefore retained.

Toxicological Review

Aldicarb is one of the most acutely toxic pesticides in use, although the only consistently observed toxic effect with both long-term and single-dose administration is acetylcholinesterase inhibition. It is metabolized to the sulfoxide and sulfone. The weight of evidence indicates that aldicarb is not genotoxic or carcinogenic. IARC has concluded that aldicarb is not classifiable as to its carcinogenicity (Group 3).

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to aldicarb, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Aldicarb was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but a health-based guideline value of 0.01 mg/litre was derived for aldicarb in the 1993 Guidelines.

Primary Reference

FAO/WHO (1993) *Pesticide residues in food — 1992*. Rome, Food and Agriculture Organization of the United Nations, 1993 (Joint FAO/WHO Meeting on Pesticide Residues: Report No. 116).

Health criteria and other supporting information

Aldrin (CAS No. 309-00-2) **and dieldrin** (CAS No. 60-57-1)

Aldrin and dieldrin are chlorinated pesticides that are used against soil-dwelling pests, for wood protection and, in the case of dieldrin, against insects of public health importance. The two compounds are closely related with respect to their toxicology and mode of action. Aldrin is rapidly converted to dieldrin under most environmental conditions and in the body. Dieldrin is a highly persistent organochlorine compound that has low mobility in soil and can be lost to the atmosphere. It is occasionally found in water. Dietary exposure to aldrin/dieldrin is very low and decreasing. Since the early 1970s, many countries have either severely restricted or banned the use of both compounds, particularly in agriculture.

Guideline value	0.03 µg/litre combined aldrin and dieldrin
Occurrence	Concentrations in drinking-water are normally less than 10 ng/litre; rarely present in groundwater
PTDI	0.1 µg/kg of body weight (combined total for aldrin and dieldrin), based on toxicity studies in dogs and rats and taking into consideration potential carcinogenicity in mice
Limit of detection	0.003 µg/litre for aldrin and 0.002 µg/litre for dieldrin by gas chromatography with electron capture detection
Treatment achievability	0.02 µg/litre should be achievable using coagulation, GAC or ozonation
Guideline derivation	
• *allocation to water*	1% of PTDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Aldrin and dieldrin are listed under the Stockholm Convention on Persistent Organic Pollutants. Hence, monitoring may occur in addition to that required by drinking-water guidelines.

Toxicological Review

Both compounds are highly toxic in experimental animals, and cases of poisoning in humans have occurred. Aldrin and dieldrin have more than one mechanism of toxicity. The target organs are the central nervous system and the liver. In long-term studies, dieldrin was shown to produce liver tumours in both sexes of two strains of mice. It did not produce an increase in tumours in rats and does not appear to be genotoxic. IARC has classified aldrin and dieldrin in Group 3. It is considered that all the available information on aldrin and dieldrin taken together, including studies on humans, supports the view that, for practical purposes, these chemicals make very little contribution, if any, to the incidence of cancer in humans.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to aldrin and dieldrin, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a health-based guideline value of 0.03 µg/litre was recommended for aldrin and dieldrin, based on the ADI recommended by JMPR in 1970 for aldrin and dieldrin residues separately or together and reaffirmed by toxicological data available in 1977. The 1993 Guidelines confirmed the health-based guideline value of 0.03 µg/litre for aldrin and dieldrin, based on the reaffirmation of the ADI recommended in 1977 by JMPR.

Primary Reference

FAO/WHO (1994) *Pesticide residues in food — 1994*. Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and WHO Toxicological and Environmental Core Assessment Groups (FAO Plant Production and Protection Paper 127, 1993).

Health criteria and other supporting information

Atrazine (CAS No. 1912-24-9)

Atrazine is a selective pre- and early post-emergence herbicide. It has been found in surface water and groundwater as a result of its mobility in soil. It is relatively stable in soil and aquatic environments, with a half-life measured in months, but is degraded by photolysis and microbial degradation in soil.

Guideline value	2 µg/litre
Occurrence	Found in groundwater and drinking-water at levels below 10 µg/litre
TDI	0.5 µg/kg of body weight based on a NOAEL of 0.5 mg/kg of body weight per day in a carcinogenicity study in the rat and taking into consideration potential neoplasia
Limit of detection	0.01 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	0.1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

The weight of evidence from a wide variety of genotoxicity assays indicates that atrazine is not genotoxic. There is evidence that atrazine can induce mammary tumours in rats. It is highly probable that the mechanism for this process is non-genotoxic. No significant increase in neoplasia has been observed in mice. IARC has concluded that there is inadequate evidence in humans and limited evidence in experimental animals for the carcinogenicity of atrazine (Group 2B).

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to atrazine, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Atrazine was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.002 mg/litre for atrazine in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Bentazone (CAS No. 25057-89-0)

Bentazone is a broad-spectrum herbicide used for a variety of crops. Photodegradation occurs in soil and water; however, bentazone is very mobile in soil and moderately persistent in the environment. Bentazone has been reported to occur in surface water, groundwater and drinking-water at concentrations of a few micrograms per litre or less. Although it has been found in groundwater and has a high affinity for the water compartment, it does not seem to accumulate in the environment. Exposure from food is unlikely to be high.

Long-term studies conducted in rats and mice have not indicated a carcinogenic potential, and a variety of *in vitro* and *in vivo* assays have indicated that bentazone is not genotoxic. A health-based value of 300 µg/litre can be calculated on the basis of an ADI of 0.1 mg/kg of body weight, based on haematological effects observed in a 2-year dietary study in rats. However, because bentazone occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to bentazone, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Bentazone was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.03 mg/litre for bentazone, based on an ADI established by JMPR in 1991. This guideline value was amended to 0.3 mg/litre in the addendum to the Guidelines, published in 1998, based on new information on the environmental behaviour of bentazone.

Primary Reference

FAO/WHO (1998) *Pesticide residues in food — 1998. Joint FAO/WHO Meeting on Pesticide Residues. Evaluations — 1998. Part II — Toxicology.* Geneva, World Health Organization (WHO/PCS/01.12).

Health criteria and other supporting information

Carbofuran (CAS No. 1563-66-2)

Carbofuran is used worldwide for many crops. Residues in treated crops are generally very low or not detectable. The physical and chemical properties of carbofuran and the few data on occurrence indicate that drinking-water from both groundwater and surface water sources is potentially the major route of exposure.

Guideline value	7 µg/litre
Occurrence	Has been detected in surface water, groundwater and drinking-water, generally at levels of a few micrograms per litre or lower; highest concentration (30 µg/litre) measured in groundwater
ADI	0.002 mg/kg of body weight based on acute (reversible) effects in dogs in a short-term (4-week) study conducted as an adjunct to a 13-week study in which inhibition of erythrocyte acetylcholinesterase activity was observed
Limit of detection	0.1 µg/litre by gas chromatography with a nitrogen–phosphorus detector; 0.9 µg/litre by reverse-phase high-performance liquid chromatography with a fluorescence detector
Treatment achievability	1 µg/litre should be achievable using GAC
Guideline derivation	
• *allocation to water*	10% of ADI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Use of a 4-week study was considered appropriate because the NOAEL is based on a reversible acute effect; the NOAEL will also be protective for chronic effects.

Toxicological Review

On the basis of available studies, carbofuran does not appear to be carcinogenic or genotoxic. The main systemic effect of carbofuran poisoning appears to be cholinesterase inhibition.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to carbofuran, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Carbofuran was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but a health-based guideline value of 0.005 mg/litre was established for carbofuran in the 1993 Guidelines, based on human data and supported by observations in laboratory animals. This value was amended to 0.007 mg/litre in the addendum to the Guidelines published in 1998, on the basis of the ADI established by JMPR in 1996.

Primary Reference

FAO/WHO (1997) *Pesticide residues in food — 1996. Joint FAO/WHO Meeting on Pesticide Residues. Evaluations 1996. Part II — Toxicological.* Geneva, World Health Organization, International Programme on Chemical Safety (WHO/PCS/97.1).

Health criteria and other supporting information

Chlordane (CAS No. 57-47-9)

Chlordane is a broad-spectrum insecticide that has been used since 1947. Its use has recently been increasingly restricted in many countries, and it is now used mainly to destroy termites by subsurface injection into soil. Chlordane may be a low-level source of contamination of groundwater when applied by subsurface injection. Technical chlordane is a mixture of compounds, with the *cis* and *trans* forms of chlordane predominating. It is very resistant to degradation, is highly immobile in soil and does not readily migrate to groundwater, where it has only rarely been found. It is readily lost to the atmosphere. Although levels of chlordane in food have been decreasing, it is highly persistent and has a high bioaccumulation potential.

Guideline value	0.2 µg/litre
Occurrence	Has been detected in both drinking-water and groundwater, usually at levels below 0.1 µg/litre
PTDI	0.5 µg/kg of body weight based on a NOAEL of 50 µg/kg of body weight per day derived from a long-term dietary study in rats
Limit of detection	0.014 µg/litre by gas chromatography with an electron capture detector
Treatment achievability	0.1 µg/litre should be achievable using GAC
Guideline derivation	
• *allocation to water*	1% of PTDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Chlordane is listed under the Stockholm Convention on Persistent Organic Pollutants. Hence, monitoring may occur in addition to that required by drinking-water guidelines.

Toxicological Review

In experimental animals, prolonged exposure in the diet causes liver damage. Chlordane produces liver tumours in mice, but the weight of evidence indicates that it is not genotoxic. Chlordane can interfere with cell communication *in vitro*, a characteristic of many tumour promoters. IARC re-evaluated chlordane in 1991 and concluded that there is inadequate evidence for its carcinogenicity in humans and sufficient evidence for its carcinogenicity in animals, classifying it in Group 2B.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to chlordane, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a health-based guideline value of 0.3 µg/litre was recommended for chlordane (total isomers), based on the ADI recommended by JMPR in 1977. The 1993 Guidelines established a health-based guideline value of 0.2 µg/litre for chlordane in drinking-water, based on an ADI established by JMPR in 1986.

Primary Reference

FAO/WHO (1994) *Pesticide residues in food — 1994.* Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and WHO Toxicological and Environmental Core Assessment Groups (FAO Plant Production and Protection Paper 127, 1993).

Health criteria and other supporting information

Chlorotoluron (CAS No. 15545-48-9)

Chlorotoluron is a pre- or early post-emergence herbicide that is slowly biodegradable and mobile in soil. There is only very limited exposure to this compound from food.

Guideline value	30 µg/litre
Occurrence	Chlorotoluron has been detected in drinking-water at concentrations of less than 1 µg/litre.
TDI	11.3 µg/kg of body weight, derived from a NOAEL of 11.3 mg/kg of body weight per day for systemic effects in a 2-year feeding study in mice using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for evidence of carcinogenicity)
Limit of detection	0.1 µg/litre by separation by reverse-phase high-performance liquid chromatography followed by ultraviolet and electrochemical detection
Treatment achievability	0.1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Chlorotoluron is of low toxicity in single, short-term and long-term exposures in animals, but it has been shown to cause an increase in adenomas and carcinomas of the kidneys of male mice given high doses for 2 years. Chlorotoluron and its metabolites have shown no evidence of genotoxicity.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to chlorotoluron, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Chlorotoluron was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.03 mg/litre for chlorotoluron in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Cyanazine (CAS No. 21725-46-2)

Cyanazine is a member of the triazine family of herbicides. It is used as a pre- and post-emergence herbicide for the control of annual grasses and broadleaf weeds. It can be degraded in soil and water by microorganisms and by hydrolysis.

Guideline value	0.6 µg/litre
Occurrence	Has been detected in surface water and groundwater, usually at concentrations of a few micrograms per litre, although levels as high as 1.3 and 3.5 mg/litre have been measured in surface water and groundwater, respectively
TDI	0.198 µg/kg of body weight based on a NOAEL of 0.198 mg/kg of body weight for hyperactivity in male rats in a 2-year toxicity/carcinogenicity study, with an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for limited evidence of carcinogenicity)
Limit of detection	0.01 µg/litre by gas chromatography with mass spectrometry
Treatment achievability	0.1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

On the basis of the available mutagenicity data on cyanazine, evidence for genotoxicity is equivocal. Cyanazine causes mammary gland tumours in Sprague-Dawley rats but not in mice. The mechanism of mammary gland tumour development in Sprague-Dawley rats is currently under investigation and may prove to be hormonal (cf. atrazine). Cyanazine is also teratogenic in Fischer 344 rats at dose levels of 25 mg/kg of body weight per day and higher.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to cyanazine, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, no guideline value for triazine herbicides, which include cyanazine, was recommended after a detailed evaluation of the compounds. Cyanazine was not evaluated in the second edition of the *Guidelines for Drinking-water Quality*, published in 1993. In the addendum to the second edition of these Guidelines, published in 1998, a health-based guideline value of 0.6 µg/litre was established for cyanazine in drinking-water.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

2,4-D (2,4-Dichlorophenoxyacetic acid) (CAS No. 94-75-7)

The term 2,4-D is used here to refer to the free acid, 2,4-dichlorophenoxyacetic acid. Commercial 2,4-D products are marketed as the free acid, alkali and amine salts, and ester formulations. 2,4-D itself is chemically stable, but its esters are rapidly hydrolysed to the free acid. 2,4-D is a systemic herbicide used for control of broad-leaved weeds, including aquatic weeds. Impurities may be present in the technical product as a result of the manufacturing process. 2,4-D is rapidly biodegraded in the environment. Residues of 2,4-D in food rarely exceed a few tens of micrograms per kilogram.

Guideline value	30 µg/litre
Occurrence	Levels in water are usually below 0.5 µg/litre, although concentrations as high as 30 µg/litre have been measured.
ADI	0.01 mg/kg of body weight for the sum of 2,4-D and its salts and esters, expressed as 2,4-D, on the basis of a NOAEL of 1 mg/kg of body weight per day in a 1-year study of toxicity in dogs and a 2-year study of toxicity and carcinogenicity in rats
Limit of detection	0.1 µg/litre by gas–liquid chromatography with electrolytic conductivity detection
Treatment achievability	1 µg/litre should be achievable using GAC
Guideline derivation	
• *allocation to water*	10% of ADI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value applies to 2,4-D, as salts and esters of 2,4-D are rapidly hydrolysed to the free acid in water

Toxicological Review

Epidemiological studies have suggested an association between exposure to chlorophenoxy herbicides, including 2,4-D, and two forms of cancer in humans: soft-tissue sarcomas and non-Hodgkin lymphoma. The results of these studies, however, are inconsistent; the associations found are weak, and conflicting conclusions have been reached by the investigators. Most of the studies did not provide information on exposure specifically to 2,4-D, and the risk was related to the general category of chlorophenoxy herbicides, a group that includes 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), which can be contaminated with dioxins. IARC has classified chlorophenoxy herbicides in Group 2B (the agent is possibly carcinogenic to humans) on the basis of limited evidence for carcinogenicity to humans and, for 2,4-D (and 2,4,5-T), inadequate evidence for carcinogenicity to animals.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to 2,4-D, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a health-based guideline value of 0.1 mg/litre was recommended for 2,4-D, based on the ADI recommended by WHO in 1976, but it was noted that some individuals may be able to detect 2,4-D by taste and odour at levels exceeding 0.05 mg/litre. The 1993 Guidelines established a health-based guideline value of 0.03 mg/litre for 2,4-D in drinking-water. This guideline value was retained in the addendum to these Guidelines, published in 1998, but was based on the more recent (1996) toxicological evaluation conducted by JMPR. This guideline value applies to 2,4-D, as salts and esters of 2,4-D are rapidly hydrolysed to the free acid in water.

Primary Reference

FAO/WHO (1997) *Pesticide residues in food — 1996. Joint FAO/WHO Meeting on Pesticide Residues. Evaluations 1996. Part II — Toxicological.* Geneva, World Health Organization, International Programme on Chemical Safety (WHO/PCS/97.1).

Health criteria and other supporting information

2,4-DB (CAS No. 94-82-6)

The half-lives for degradation of chlorophenoxy herbicides, including 2,4-DB, in the environment are in the order of several days. Chlorophenoxy herbicides are not often found in food.

Guideline value	90 µg/litre
Occurrence	Chlorophenoxy herbicides are not frequently found in drinking-water; when detected, their concentrations are usually no greater than a few micrograms per litre.
TDI	30 µg/kg of body weight, based on a NOAEL for effects on body and organ weights, blood chemistry and haematological parameters in a 2-year study in rats, with an uncertainty factor of 100 (for inter- and intraspecies variation)
Limit of detection	1 µg/litre to 1 mg/litre for various methods commonly used for the determination of chlorophenoxy herbicides in water, including solvent extraction, separation by gas chromatography, gas–liquid chromatography, thin-layer chromatography, or high-performance liquid chromatography, with electron capture or ultraviolet detection
Treatment achievability	0.1 µg/litre should be achievable using GAC
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional considerations	The NOAEL used in the guideline value derivation is similar to the NOAEL of 2.5 mg/kg of body weight per day obtained in a short-term study in beagle dogs and the NOAEL for hepatocytic hypertrophy of 5 mg/kg of body weight per day obtained in a 3-month study in rats.

Toxicological Review

Chlorophenoxy herbicides, as a group, have been classified in Group 2B by IARC. However, the available data from studies in exposed populations and animals do not permit assessment of the carcinogenic potential to humans of any specific chlorophenoxy herbicide. Therefore, drinking-water guidelines for these compounds are based on a threshold approach for other toxic effects.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to chlorophenoxy herbicides, including 2,4-DB, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. 2,4-DB was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.09 mg/litre for 2,4-DB.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

1,2-Dibromo-3-chloropropane (CAS No. 96-12-8)

1,2-Dibromo-3-chloropropane (DBCP) is a soil fumigant that is highly soluble in water. It has a taste and odour threshold in water of 10 µg/litre. DBCP was detected in vegetables grown in treated soils, and low levels have been detected in air.

Guideline value	1 µg/litre
Occurrence	Limited survey found levels of up to a few micrograms per litre in drinking-water
Basis of guideline derivation	Linearized multistage model was applied to the data on the incidence of stomach, kidney and liver tumours in the male rat in a 104-week dietary study
Limit of detection	0.02 µg/litre by gas chromatography with electron capture detection
Treatment achievability	1 µg/litre should be achievable using air stripping followed by GAC
Additional comments	The guideline value of 1 µg/litre should be protective for the reproductive toxicity of DBCP.

Toxicological Review

On the basis of animal data from different strains of rats and mice, DBCP was determined to be carcinogenic in both sexes by the oral, inhalation and dermal routes. DBCP was also determined to be a reproductive toxicant in humans and several species of laboratory animals. DBCP was found to be genotoxic in a majority of *in vitro* and *in vivo* assays. IARC has classified DBCP in Group 2B based upon sufficient evidence of carcinogenicity in animals. Recent epidemiological evidence suggests an increase in cancer mortality in individuals exposed to high levels of DBCP.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to DBCP, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. DBCP was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines calculated a guideline value of 0.001 mg/litre for DBCP in drinking-water, corresponding to an excess lifetime cancer risk of 10^{-5} and sufficiently protective for the reproductive toxicity of the pesticide. It was noted that for a contaminated water supply, extensive treatment would be required to reduce the level of DBCP to the guideline value.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

1,2-Dibromoethane (Ethylene dibromide) (CAS No. 106-93-4)

1,2-Dibromoethane is used as a lead scavenger in tetra-alkyl lead petrol and antiknock preparations and as a fumigant for soils, grains and fruits. However, with the phasing-out of leaded petrol and of the use of 1,2-dibromoethane in agricultural applications in many countries, use of this substance has declined significantly. In addition to its continued use as a petrol additive in some countries, 1,2-dibromoethane is currently used principally as a solvent and as an intermediate in the chemical industry.

Provisional guideline value	0.4–15 µg/litre (excess lifetime cancer risks for various tumour types in this range)
Occurrence	Detected in groundwater following its use as a soil fumigant at concentrations as high as 100 µg/litre
Basis of guideline derivation	Lifetime low-dose cancer risks calculated by linearized multistage modelling of the incidences of haemangiosarcomas and tumours in the stomach, liver, lung and adrenal cortex (adjusted for the observed high early mortality, where appropriate, and corrected for the expected rate of increase in tumour formation in rodents in a standard bioassay of 104 weeks) of rats and/or mice exposed to 1,2-dibromoethane by gavage
Limit of detection	0.01 µg/litre by microextraction gas chromatography/mass spectrometry; 0.03 µg/litre by purge and trap gas chromatography with halogen-specific detector; 0.8 µg/litre by purge and trap capillary column gas chromatography with photoionization and electrolytic conductivity detectors in series
Treatment achievability	0.1 µg/litre should be achievable using GAC
Additional comments	The guideline value is provisional due to serious limitations of critical studies.

Toxicological Review

1,2-Dibromoethane has induced an increased incidence of tumours at several sites in all carcinogenicity bioassays identified in which rats or mice were exposed to the compound by gavage, ingestion in drinking-water, dermal application and inhalation. However, many of these studies were characterized by high early mortality, limited histopathological examination, small group sizes or use of only one exposure level. The substance acted as an initiator of liver foci in an initiation/promotion assay but did not initiate skin tumour development. 1,2-Dibromoethane was consistently genotoxic in *in vitro* assays, although results of *in vivo* assays were mixed. Biotransformation to active metabolites, which have been demonstrated to bind to DNA, is probably involved in the induction of tumours. Available data do not support the existence of a non-genotoxic mechanism of tumour induction. The available data thus indicate that 1,2-dibromoethane is a genotoxic carcinogen in rodents. Data on the potential carcinogenicity in humans are inadequate; however, it is likely that 1,2-dibromoethane is metabolized similarly in rodent species and in humans (although there may be varying potential for the production of active metabolites in humans, owing to genetic polymorphism). IARC classified 1,2-dibromoethane in Group 2A (the agent is probably carcinogenic to humans).

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to 1,2-dibromoethane, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. 1,2-Dibromoethane was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines noted that 1,2-dibromoethane appears to be a genotoxic carcinogen. However, as the studies to date were inadequate for mathematical risk extrapolation, a guideline value for 1,2-dibromoethane was not

derived. The Guidelines recommended that 1,2-dibromoethane be re-evaluated as soon as new data became available. In the addendum to these Guidelines, published in 1998, the guideline value that corresponds to excess lifetime cancer risk for various tumour types of 10^{-5} was calculated to be in the range 0.0004–0.015 mg/litre. This guideline value was considered to be provisional because of the serious limitations of the critical studies.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

1,2-Dichloropropane (CAS No. 78-87-5)

1,2-Dichloropropane (1,2-DCP) is used as an insecticide fumigant on grain and soil and to control peach tree borers. It is also used as an intermediate in the production of perchloroethylene and other chlorinated products and as a solvent. 1,2-DCP is relatively resistant to hydrolysis, is poorly adsorbed onto soil and can migrate into groundwater.

Provisional guideline value	40 µg/litre
Occurrence	Detected in groundwater and drinking-water, usually at concentrations below 20 µg/litre, although levels as high as 440 µg/litre have been measured in well-water
TDI	14 µg/kg of body weight based on a LOAEL of 71.4 mg/kg of body weight per day for changes in haematological parameters in a 13-week study in male rats, with an uncertainty factor of 5000 (100 for inter- and intraspecies variation, 10 for use of a LOAEL and 5 to reflect limitations of the database, including the limited data on *in vivo* genotoxicity and use of a subchronic study)
Limit of detection	0.02 µg/litre by a purge-and-trap gas chromatographic method with an electrolytic conductivity detector or gas chromatography/mass spectrometry
Treatment achievability	1 µg/litre should be achievable using GAC
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value is provisional owing to limitations of the toxicological database.

Toxicological Review

1,2-DCP was evaluated by IARC in 1986 and 1987. The substance was classified in Group 3 (not classifiable as to its carcinogenicity to humans) on the basis of limited evidence for its carcinogenicity in experimental animals and the inability to evaluate its carcinogenicity in humans. Results from *in vitro* assays for mutagenicity were mixed. The *in vivo* studies, which were limited in number and design, were negative. In accordance with the IARC evaluation, the evidence from the long-term carcinogenicity studies in mice and rats was considered limited, and it was concluded that the use of a threshold approach for the toxicological evaluation of 1,2-DCP was appropriate.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to 1,2-DCP, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. 1,2-DCP was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines proposed a provisional health-based guideline value of 0.02 mg/litre for 1,2-DCP in drinking-water. The value was provisional because an uncertainty factor of 10 000 was used in its derivation. This guideline value was amended to 0.04 mg/litre in the addendum to these Guidelines, published in 1998, using a lower uncertainty factor. This guideline value was considered to be provisional owing to the magnitude of the uncertainty factor and the fact that the database had not changed since the previous guideline value had been derived.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

1,3-Dichloropropane (CAS No. 142-28-9)

1,3-Dichloropropane has several industrial uses and may be found as a contaminant of soil fumigants containing 1,3-dichloropropene. It is rarely found in water.

1,3-Dichloropropane is of low acute toxicity. There is some indication that it may be genotoxic in bacterial systems. No short-term, long-term, reproductive or developmental toxicity data pertinent to exposure via drinking-water could be located in the literature. The available data are considered insufficient to permit recommendation of a guideline value.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to 1,3-dichloropropane, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. 1,3-Dichloropropane was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines concluded that the available data were insufficient to permit recommendation of a guideline value for 1,3-dichloropropane in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

1,3-Dichloropropene (CAS Nos. 542-75-6 isomer mixture; 10061-01-5 *cis* isomer; 10061-02-6 *trans* isomer)

1,3-Dichloropropene is a soil fumigant, the commercial product being a mixture of *cis* and *trans* isomers. It is used to control a wide variety of soil pests, particularly nematodes in sandy soils. Notwithstanding its high vapour pressure, it is soluble in water at the gram per litre level and can be considered a potential water contaminant.

Guideline value	20 µg/litre
Occurrence	Has been found in surface water and groundwater at concentrations of a few micrograms per litre
Basis of guideline derivation	Calculated by applying the linearized multistage model to the observation of lung and bladder tumours in female mice in a 2-year gavage study
Limit of detection	0.34 and 0.20 µg/litre by purge-and-trap packed column gas chromatography using an electrolytic conductivity detector or microcoulometric detector for *cis*-1,3-dichloropropene and *trans*-1,3-dichloropropene, respectively
Treatment achievability	No information found on removal from water

Toxicological Review

1,3-Dichloropropene is a direct-acting mutagen that has been shown to produce forestomach tumours following long-term oral gavage exposure in rats and mice. Tumours have also been found in the bladder and lungs of female mice and the liver of male rats. Long-term inhalation studies in the rat have proved negative, whereas in inhalation studies in mice some benign lung tumours have been reported. IARC has classified 1,3-dichloropropene in Group 2B.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to 1,3-dichloropropene, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. 1,3-Dichloropropene was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines calculated a guideline value of 0.02 mg/litre for 1,3-dichloropropene in drinking-water, corresponding to an excess lifetime cancer risk of 10^{-5}.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Dichlorprop (2,4-DP) (CAS No. 120-36-5)

The half-lives for degradation of chlorophenoxy herbicides, including dichlorprop, in the environment are in the order of several days. Chlorophenoxy herbicides are not often found in food.

Guideline value	100 µg/litre
Occurrence	Chlorophenoxy herbicides are not frequently found in drinking-water; when detected, their concentrations are usually no greater than a few micrograms per litre.
TDI	36.4 µg/kg of body weight, based on a NOAEL for renal toxicity in a 2-year study in rats of 100 mg/kg of diet, equal to 3.64 mg/kg of body weight per day, applying an uncertainty factor of 100 (for intra- and interspecies variation)
Limit of detection	1 µg/litre to 1 mg/litre for various methods commonly used for the determination of chlorophenoxy herbicides in water, including solvent extraction, separation by gas chromatography, gas–liquid chromatography, thin-layer chromatography, or high-performance liquid chromatography, with electron capture or ultraviolet detection
Treatment achievability	No data available
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Chlorophenoxy herbicides, as a group, have been classified in Group 2B by IARC. However, the available data from studies in exposed populations and animals do not permit assessment of the carcinogenic potential to humans of any specific chlorophenoxy herbicide. Therefore, drinking-water guidelines for these compounds are based on a threshold approach for other toxic effects.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to chlorophenoxy herbicides, including dichlorprop, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Dichlorprop was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.1 mg/litre for dichlorprop.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Dimethoate (CAS No. 60-51-5)

Dimethoate is an organophosphorus insecticide used to control a broad range of insects in agriculture, as well as the housefly. It has a half-life of 18 h to 8 weeks and is not expected to persist in water, although it is relatively stable at pH 2–7. A total daily intake from food of 0.001 µg/kg of body weight has been estimated.

Guideline value	6 µg/litre
Occurrence	Detected at trace levels in a private well in Canada, but not detected in a Canadian survey of surface water or drinking-water supplies
ADI	0.002 mg/kg of body weight based on a NOAEL of 1.2 mg/kg of body weight per day for reproductive performance in a study of reproductive toxicity in rats, applying a safety factor of 500 to take into consideration concern regarding the possibility of adverse reproductive effects at the NOAEL
Limit of detection	0.05 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	1 µg/litre should be achievable using GAC and chlorination

Guideline derivation

• *allocation to water*	10% of ADI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Dimethoate is not carcinogenic to rodents. JMPR concluded that although *in vitro* studies indicate that dimethoate has mutagenic potential, this potential does not appear to be expressed *in vivo.* In a multigeneration study of reproductive toxicity in rats, the NOAEL appeared to be 1.2 mg/kg of body weight per day, but there was some indication that reproductive performance may have been affected at lower doses.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to dimethoate, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Dimethoate was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

FAO/WHO (1997) *Pesticide residues in food — 1996 evaluations. Part II — Toxicological*. Geneva, World Health Organization (WHO/PCS/97.1, 1997).

Health criteria and other supporting information

Diquat (CAS No. 2764-72-9)

Diquat is a non-selective contact herbicide and crop desiccant. Diquat may also be used (at or below 1 mg/litre) as an aquatic herbicide for the control of free-floating and submerged aquatic weeds in ponds, lakes and irrigation ditches. Because of its rapid degradation in water and strong adsorption onto sediments, diquat has rarely been found in drinking-water.

Diquat does not appear to be carcinogenic or genotoxic. The main toxicological finding in experimental animals is cataract formation. A health-based value of 6 µg/litre for diquat ion can be calculated on the basis of an ADI of 0.002 mg of diquat ion per kg of body weight, based on cataract formation at the next higher dose in a 2-year study in rats. However, because diquat has rarely been found in drinking-water, it is not considered necessary to derive a health-based guideline value. It should also be noted that the limit of detection of diquat in water is 0.001 mg/litre, and its practical quantification limit is about 0.01 mg/litre.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to diquat, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Diquat was not evaluated in the first two editions of the *Guidelines for Drinking-water Quality*, published in 1984 and 1993. In the addendum to the second edition of these Guidelines, published in 1998, a health-based value of 0.006 mg/litre was calculated for the diquat ion using the ADI established by JMPR in 1993. However, the limit of detection of diquat in water is 0.001 mg/litre, and its practical quantification limit is about 0.01 mg/litre. A provisional guideline value of 0.01 mg/litre was therefore established for diquat ion.

Primary Reference

FAO/WHO (1994) *Pesticide residues in food — 1993. Joint FAO/WHO Meeting on Pesticide Residues. Evaluations — 1993. Part II — Toxicology.* Geneva, World Health Organization (WHO/PCS/94.4).

Health criteria and other supporting information

Endosulfan (CAS No. 115-29-7)

Endosulfan is an insecticide used in countries throughout the world to control pests on fruit, vegetables and tea and on non-food crops such as tobacco and cotton. In addition to its agricultural use, it is used in the control of the tsetse fly, as a wood preservative and for the control of home garden pests. Endosulfan contamination does not appear to be widespread in the aquatic environment, but the chemical has been found in agricultural runoff and rivers in industrialized areas where it is manufactured or formulated, as well as in surface water and groundwater samples collected from hazardous waste sites in the USA. Surface water samples in the USA generally contain less than 1 µg/litre. The main source of exposure of the general population is food, but residues have generally been found to be well below the FAO/WHO maximum residue limits. Another important route of exposure to endosulfan for the general population is the use of tobacco products.

JMPR concluded that endosulfan is not genotoxic, and no carcinogenic effects were noted in long-term studies using mice and rats. The kidney is the target organ for toxicity. Several recent studies have shown that endosulfan, alone or in combination with other pesticides, may bind to estrogen receptors and perturb the endocrine system. A health-based value of 20 µg/litre can be calculated for endosulfan on the basis of an ADI of 0.006 mg/kg of body weight, based on results from a 2-year dietary study of toxicity in rats. However, because endosulfan occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to endosulfan, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Endosulfan was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

FAO/WHO (1999) *Pesticide residues in food — 1998 evaluations. Part II — Toxicological*. Geneva, World Health Organization (WHO/PCS/99.18).

Health criteria and other supporting information

Endrin *(CAS No. 72-20-8)*

Endrin is a broad-spectrum foliar insecticide that acts against a wide range of agricultural pests. It is also used as a rodenticide. Small amounts of endrin are present in food, but the total intake from food appears to be decreasing.

Guideline value	0.6 µg/litre
Occurrence	Traces of endrin have been found in the drinking-water supplies of several countries.
PTDI	0.0002 mg/kg of body weight, based on a NOAEL of 0.025 mg/kg of body weight per day in a 2-year study in dogs and applying a safety factor of 100
Limit of detection	0.002 µg/litre by gas chromatography with electron capture detection
Treatment achievability	0.2 µg/litre should be achievable using GAC
Guideline derivation	
• *allocation to water*	10% of PTDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Endrin is listed under the Stockholm Convention on Persistent Organic Pollutants. Hence, monitoring may occur in addition to that required by drinking-water guidelines.

Toxicological Review

Toxicological data are insufficient to indicate whether endrin is a carcinogenic hazard to humans. The primary site of action of endrin is the central nervous system.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to endrin, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Endrin was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

FAO/WHO (1994) *Pesticide residues in food — 1994.* Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and WHO Toxicological and Environmental Core Assessment Groups (FAO Plant Production and Protection Paper 127, 1993).

Health criteria and other supporting information

Fenitrothion *(CAS No. 122-14-5)*

Fenitrothion is mainly used in agriculture for controlling insects on rice, cereals, fruits, vegetables, stored grains and cotton and in forest areas. It is also used for the control of flies, mosquitos and cockroaches in public health programmes and/or indoor use. Fenitrothion is stable in water only in the absence of sunlight or microbial contamination. In soil, biodegradation is the primary route of degradation, although photolysis may also play a role. Fenitrothion residues detected in water were low (maximum 1.30 µg/litre) during the spruce budworm spray programme. Following the spraying of forests to control spruce budworm, water samples did not contain detectable amounts of fenitrothion; post-spray samples contained <0.01 µg/litre. Levels of fenitrothion residues in fruits, vegetables and cereal grains decline rapidly after treatment, with a half-life of 1–2 days. Intake of fenitrothion appears to be primarily (95%) from food.

On the basis of testing in an adequate range of studies *in vitro* and *in vivo*, JMPR concluded that fenitrothion is unlikely to be genotoxic. It also concluded that fenitrothion is unlikely to pose a carcinogenic risk to humans. In long-term studies of toxicity, inhibition of cholinesterase activity was the main toxicological finding in all species. A health-based value of 8 µg/litre can be calculated for fenitrothion on the basis of an ADI of 0.005 mg/kg of body weight, based on inhibition of brain and erythrocyte cholinesterase activity in a 2-year study of toxicity in rats, and allocating 5% of the ADI to drinking-water. However, because fenitrothion occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to fenitrothion, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Fenitrothion was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

FAO/WHO (2001) *Pesticide residues in food — 2000 evaluations. Part II — Toxicological*. Geneva, World Health Organization (WHO/PCS/01.3).

Health criteria and other supporting information

Fenoprop (2,4,5-TP; 2,4,5-trichlorophenoxy propionic acid) (CAS No. 93-72-1)

The half-lives for degradation of chlorophenoxy herbicides, including fenoprop, in the environment are in the order of several days. Chlorophenoxy herbicides are not often found in food.

Guideline value	9 µg/litre
Occurrence	Chlorophenoxy herbicides are not frequently found in drinking-water; when detected, their concentrations are usually no greater than a few micrograms per litre.
TDI	3 µg/kg of body weight, based on a NOAEL of 0.9 mg/kg of body weight for adverse effects on the liver in a study in which beagle dogs were administered fenoprop in the diet for 2 years, with an uncertainty factor of 300 (100 for inter- and intraspecies variation and 3 for limitations in the database)
Limit of detection	0.2 µg/litre by either packed or capillary column gas chromatography with electron capture detector
Treatment achievability	No data found; 0.001 mg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Chlorophenoxy herbicides, as a group, have been classified in Group 2B by IARC. However, the available data from studies in exposed populations and animals do not permit assessment of the carcinogenic potential to humans of any specific chlorophenoxy herbicide. Therefore, drinking-water guidelines for these compounds are based on a threshold approach for other toxic effects.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to chlorophenoxy herbicides, including fenoprop, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Fenoprop was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.009 mg/litre for fenoprop.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Glyphosate (CAS No. 1071-83-6) **and AMPA** (CAS No. 1066-51-9)

Glyphosate is a broad-spectrum herbicide used in both agriculture and forestry and for aquatic weed control. Microbial biodegradation of glyphosate occurs in soil, aquatic sediment and water, the major metabolite being aminomethylphosphonic acid (AMPA). Glyphosate is chemically stable in water and is not subject to photochemical degradation. The low mobility of glyphosate in soil indicates minimal potential for the contamination of groundwater. Glyphosate can, however, enter surface and subsurface waters after direct use near aquatic environments or by runoff or leaching from terrestrial applications.

Glyphosate and AMPA have similar toxicological profiles, and both are considered to exhibit low toxicity. A health-based value of 5 mg/litre can be calculated for glyphosate on the basis of an ADI of 1.75 mg/kg of body weight in a teratogenicity study in rabbits. Because of the low toxicity of glyphosate, this health-based value is orders of magnitude higher than the concentrations normally found in drinking-water. As AMPA is considered to be of no greater toxicological concern than its parent compound, under usual conditions, the presence of glyphosate and AMPA in drinking-water does not represent a hazard to human health, and the establishment of numerical health-based guideline values for glyphosate and AMPA is not deemed necessary.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to glyphosate, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Glyphosate was not evaluated in the first two editions of the *Guidelines for Drinking-water Quality*, published in 1984 and 1993. In the addendum to these Guidelines, published in 1998, a health-based value of 5 mg/litre was derived for glyphosate using the ADI derived in the Environmental Health Criteria monograph for glyphosate published in 1994. However, the health-based value is orders of magnitude higher than the concentrations normally found in drinking-water. Under usual conditions, therefore, the presence of glyphosate in drinking-water does not represent a hazard to human health, and it was not deemed necessary to establish a numerical guideline value for glyphosate. It was noted that most AMPA, the major metabolite of glyphosate, found in water comes from sources other than glyphosate degradation.

Primary References

WHO (1998) *Guidelines for drinking-water quality, 2nd ed. Addendum to Vol. 2. Health criteria and other supporting information.* Geneva, World Health Organization.

FAO/WHO (1998) *Pesticide residues in food — 1997. Joint FAO/WHO Meeting on Pesticide Residues. Evaluations — 1997. Part II — Toxicology*. Geneva, World Health Organization (FAO Plant Production and Protection Paper 78/2).

Health criteria and other supporting information

Heptachlor (CAS No. 76-44-8) **and heptachlor epoxide** (CAS No. 1024-57-3)

Heptachlor is a broad-spectrum insecticide, the use of which has been banned or restricted in many countries. At present, the major use of heptachlor is for termite control by subsurface injection into soil. Heptachlor is quite persistent in soil, where it is mainly transformed to its epoxide. Heptachlor epoxide is very resistant to further degradation. Heptachlor and heptachlor epoxide bind to soil particles and migrate very slowly. Heptachlor and heptachlor epoxide have been found in drinking-water at levels of nanograms per litre. Diet is considered to represent the major source of exposure to heptachlor, although intake is decreasing.

Prolonged exposure to heptachlor has been associated with damage to the liver and central nervous system toxicity. In 1991, IARC reviewed the data on heptachlor and concluded that the evidence for carcinogenicity was sufficient in animals and inadequate in humans, classifying it in Group 2B. A health-based value of 0.03 µg/litre can be calculated for heptachlor and heptachlor epoxide on the basis of a PTDI of 0.1 µg/kg of body weight, based on a NOAEL for heptachlor of 0.025 mg/kg of body weight per day from two studies in the dog, taking into consideration inadequacies of the database and allocating 1% of the PTDI to drinking-water. However, because heptachlor and heptachlor epoxide occur at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value. It should also be noted that concentrations below 0.1 µg/litre are generally not achievable using conventional treatment technology.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to heptachlor and heptachlor epoxide, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a health-based guideline value of 0.1 µg/litre was recommended for heptachlor and heptachlor epoxide, based on the ADI recommended by JMPR. It was noted that this guideline value was less than the value that would have been calculated by applying the multistage model at a projected incremental cancer risk of 1 per 100 000 per lifetime. The 1993 Guidelines established a health-based guideline value of 0.03 µg/litre for heptachlor, based on an ADI established by JMPR in 1991 and taking into consideration the fact that the main source of exposure seems to be food.

Primary Reference

FAO/WHO (1994) *Pesticide residues in food — 1994.* Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and WHO Toxicological and Environmental Core Assessment Groups (FAO Plant Production and Protection Paper 127, 1993).

Health criteria and other supporting information

Hexachlorobenzene (CAS No. 118-74-1)

The major agricultural application for hexachlorobenzene (HCB) was as a seed dressing for crops to prevent the growth of fungi, but its use is now uncommon. At present, it appears mainly as a by-product of several chemical processes or an impurity in some pesticides. HCB is distributed throughout the environment because it is mobile and resistant to degradation. It bioaccumulates in organisms because of its physicochemical properties and its slow elimination. HCB is commonly detected at low levels in food, and it is generally present at low concentrations in ambient air.

Guideline value	1 µg/litre
Occurrence	Has been detected infrequently, and at very low concentrations, in drinking-water supplies
Basis of guideline derivation	Applying the linearized multistage low-dose extrapolation model to liver tumours observed in female rats in a 2-year dietary study (IPCS 1997)
Limit of detection	5 ng/litre by gas chromatography with electron capture detection
Treatment achievability	1 µg/litre should be achievable using GAC
Additional comments	Hexachlorobenzene is listed under the Stockholm Convention on Persistent Organic Pollutants. Hence, monitoring may occur in addition to that required by drinking-water guidelines.

Toxicological Review

Hexachlorobenzene is highly toxic on repeat exposure and has been shown to be a developmental toxin in rats and mice, but not rabbits. IARC has evaluated the evidence for the carcinogenicity of HCB in animals and humans and assigned it to Group 2B. HCB has been shown to induce tumours in three animal species and at a variety of sites. The weight of evidence indicates that it is not genotoxic.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to HCB, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a health-based guideline value of 0.01 µg/litre was recommended for HCB, derived from the linear multistage extrapolation model for a cancer risk of less than 1 in 100 000 for a lifetime of exposure; it was noted that the mathematical model used involved considerable uncertainty. The 1993 Guidelines calculated a guideline value of 1 µg/litre for HCB in drinking-water, corresponding to an excess lifetime cancer risk of 10^{-5}.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Isoproturon (CAS No. 34123-59-6)

Isoproturon is a selective, systemic herbicide used in the control of annual grasses and broad-leaved weeds in cereals. It can be photodegraded, hydrolysed and biodegraded and persists for periods ranging from days to weeks. It is mobile in soil. There is evidence that exposure to this compound through food is low.

Guideline value	9 µg/litre
Occurrence	Has been detected in surface water and groundwater, usually at concentrations below 0.1 µg/litre; levels above 0.1 µg/litre have occasionally been detected in drinking-water
TDI	3 µg/kg of body weight based on a NOAEL of approximately 3 mg/kg of body weight in a 90-day study in dogs and a 2-year feeding study in rats, with an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for evidence of non-genotoxic carcinogenicity in rats)
Limit of detection	10–100 ng/litre by reverse-phase high-performance liquid chromatography followed by ultraviolet or electrochemical detection
Treatment achievability	0.1 µg/litre should be achievable using ozonation

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Isoproturon is of low acute toxicity and low to moderate toxicity following short- and long-term exposures. It does not possess significant genotoxic activity, but it causes marked enzyme induction and liver enlargement. Isoproturon caused an increase in hepatocellular tumours in male and female rats, but this was apparent only at doses that also caused liver toxicity. Isoproturon appears to be a tumour promoter rather than a complete carcinogen.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to isoproturon, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Isoproturon was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines calculated a health-based guideline value of 0.009 mg/litre for isoproturon in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Lindane (CAS No. 58-89-9)

Lindane (γ-hexachlorocyclohexane, γ-HCH) is used as an insecticide on fruit and vegetable crops, seed treatment and in forestry. It is also used as a therapeutic pesticide in humans and animals. Several countries have restricted the use of lindane. Lindane can be degraded in soil and rarely leaches to groundwater. In surface waters, it is removed by evaporation. Exposure of humans occurs mainly via food, but this is decreasing. There may also be exposure from its use in public health and as a wood preservative.

Guideline value	0.3 µg/litre
Occurrence	Has been detected in both surface water and groundwater, usually at concentrations below 0.1 µg/litre, although concentrations as high as 12 µg/litre have been reported in wastewater-contaminated rivers
Temporary ADI	0.001 mg/kg of body weight on the basis of a NOAEL of 0.5 mg/kg of body weight per day in a 2-year toxicity/carcinogenicity study in rats
Limit of detection	0.01 µg/litre using gas chromatography
Treatment achievability	0.1 µg/litre should be achievable using GAC
Guideline derivation	
• *allocation to water*	1% of temporary ADI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Pending clarification of the immunotoxicity of lindane that meets FAO specifications, this ADI provides a 10-fold margin of safety over the LOAEL of 0.012 mg/kg of body weight per day in a study of immunotoxicity in mice.

Toxicological Review

Lindane causes liver tumours in mice given very high doses, but there is evidence that this is a result of tumour promotion. In 1987, IARC classified lindane in Group 2B. JMPR has concluded that there was no evidence of genotoxicity.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to lindane, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a health-based guideline value of 3 µg/litre was recommended for lindane, based on the ADI recommended by JMPR. The 1993 Guidelines established a health-based guideline value of 2 µg/litre for lindane in drinking-water, on the basis of a study used to establish an ADI by JMPR in 1989 but using a compound intake estimate considered to be more appropriate in light of additional data and recognizing that there may be substantial exposure to lindane from its use in public health and as a wood preservative.

Primary Reference

FAO/WHO (1998) *Pesticide residues in food — 1997. Joint FAO/WHO Meeting on Pesticide Residues. Evaluations — 1997. Part II — Toxicology*. Geneva, World Health Organization (WHO/PCS/98.6).
Health criteria and other supporting information

Malathion (CAS No. 121-75-5)

Malathion is commonly used to control mosquitos and a variety of insects that attack fruits, vegetables, landscaping plants and shrubs. It can also be found in other pesticide products used indoors, on pets to control ticks and insects, and to control human head and body lice. Under least favourable conditions (i.e., low pH and little organic content), malathion may persist in water with a half-life of months or even years. However, under most conditions, the half-life appears to be roughly 7–14 days. Malathion has been detected in surface water and drinking-water at concentrations below 2 µg/litre.

A health-based value of 0.9 mg/litre can be calculated for malathion based on an allocation of 10% of the JMPR ADI — based on a NOAEL of 29 mg/kg of body weight per day in a 2-year study of toxicity and carcinogenicity in rats, using an uncertainty factor of 100 — to drinking-water. However, the chemical occurs at concentrations much lower than this health-based value. Under usual conditions, therefore, the presence of malathion in drinking-water is unlikely to represent a hazard to human health. For this reason, it is considered unnecessary to derive a guideline value for malathion in drinking-water.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to malathion, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Malathion was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

FAO/WHO (1998) *Pesticide residues in food — 1997 evaluations. Part II — Toxicological and environmental*. Geneva, World Health Organization (WHO/PCS/98.6).

Health criteria and other supporting information

MCPA [4-(2-methyl-4-chlorophenoxy)acetic acid] (CAS No. 94-74-6)

MCPA is a chlorophenoxy post-emergence herbicide that is very soluble, is highly mobile and can leach from the soil. It is metabolized by bacteria and can be photochemically degraded. MCPA has only limited persistence in water.

Guideline value	2 µg/litre
Occurrence	Not been frequently detected in drinking-water; has been measured in surface water and groundwater at concentrations below 0.54 and 5.5 µg/litre, respectively
TDI	0.5 µg/kg of body weight, based on the NOAEL of 0.15 mg/kg of body weight for renal and liver toxicity observed at higher dose levels in a 1-year feeding study in dogs, with an uncertainty factor of 300 (100 for inter- and intraspecies variation and 3 for inadequacies in the database)
Limit of detection	0.01 µg/litre by gas chromatography/mass spectrometry and by gas chromatography with electron capture detector
Treatment achievability	0.1 µg/litre should be achievable using GAC or ozonation

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

There are only limited and inconclusive data on the genotoxicity of MCPA. IARC evaluated MCPA in 1983 and concluded that the available data on humans and experimental animals were inadequate for an evaluation of carcinogenicity. Further evaluations by IARC on chlorophenoxy herbicides in 1986 and 1987 concluded that evidence for their carcinogenicity was limited in humans and inadequate in animals (Group 2B). Recent carcinogenicity studies on rats and mice did not indicate that MCPA was carcinogenic. No adequate epidemiological data on exposure to MCPA alone are available.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to MCPA, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. MCPA was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.002 mg/litre for MCPA in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Mecoprop (MCPP; [2(2-methyl-chlorophenoxy) propionic acid]) (CAS No. 93-65-2; 7085-19-0 racemic mixture)

The half-lives for degradation of chlorophenoxy herbicides, including mecoprop, in the environment are in the order of several days. Chlorophenoxy herbicides are not often found in food.

Guideline value	10 µg/litre
Occurrence	Chlorophenoxy herbicides are not frequently found in drinking-water; when detected, their concentrations are usually no greater than a few micrograms per litre.
TDI	3.33 µg/kg of body weight, based on a NOAEL of 1 mg/kg of body weight for effects on kidney weight in 1- and 2-year studies in rats, with an uncertainty factor of 300 (100 for inter- and intraspecies variation and 3 for limitations in the database)
Limit of detection	0.01 µg/litre by gas chromatography/mass spectrometry; 0.01–0.02 µg/litre by gas chromatography with electron capture detector
Treatment achievability	0.1 µg/litre should be achievable using GAC or ozonation

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Chlorophenoxy herbicides, as a group, have been classified in Group 2B by IARC. However, the available data from studies in exposed populations and animals do not permit assessment of the carcinogenic potential to humans of any specific chlorophenoxy herbicide. Therefore, drinking-water guidelines for these compounds are based on a threshold approach for other toxic effects.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to chlorophenoxy herbicides, including mecoprop, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Mecoprop was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.01 mg/litre for mecoprop.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Methoxychlor (CAS No. 72-43-5)

Methoxychlor is an insecticide used on vegetables, fruit, trees, fodder and farm animals. It is poorly soluble in water and highly immobile in most agricultural soils. Under normal conditions of use, methoxychlor does not seem to be of environmental concern. Daily intake from food and air is expected to be below 1 µg per person. Environmental metabolites are formed preferentially under anaerobic rather than aerobic conditions and include mainly the dechlorinated and demethylated products. There is some potential for the accumulation of the parent compound and its metabolites in surface water sediments.

Guideline value	20 µg/litre
Occurrence	Detected occasionally in drinking-water, at concentrations as high as 300 µg/litre in rural areas
TDI	5 µg/kg of body weight, based on a systemic NOAEL of 5 mg/kg of body weight in a teratology study in rabbits, with an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 reflecting concern for threshold carcinogenicity and the limited database)
Limit of detection	0.001–0.01 µg/litre by gas chromatography
Treatment achievability	0.1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

The genotoxic potential of methoxychlor appears to be negligible. In 1979, IARC assigned methoxychlor to Group 3. Subsequent data suggest a carcinogenic potential of methoxychlor for liver and testes in mice. This may be due to the hormonal activity of proestrogenic mammalian metabolites of methoxychlor and may therefore have a threshold. The study, however, was inadequate because only one dose was used and because this dose may have been above the maximum tolerated dose. The database for studies on long-term, short-term and reproductive toxicity is inadequate. A teratology study in rabbits reported a systemic NOAEL of 5 mg/kg of body weight per day, which is lower than the LOAELs and NOAELs from other studies. This NOAEL was therefore selected for use in the derivation of a TDI.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to methoxychlor, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a health-based guideline value of 0.03 mg/litre was recommended for methoxychlor, based on the ADI recommended by JMPR in 1965 and reaffirmed in 1977. The 1993 Guidelines established a health-based guideline value of 0.02 mg/litre for methoxychlor in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.
Health criteria and other supporting information

Metolachlor (CAS No. 51218-45-2)

Metolachlor is a selective pre-emergence herbicide used on a number of crops. It can be lost from the soil through biodegradation, photodegradation and volatilization. It is fairly mobile and under certain conditions can contaminate groundwater, but it is mostly found in surface water.

Guideline value	10 µg/litre
Occurrence	Detected in surface water and groundwater at concentrations that can exceed 10 µg/litre
TDI	3.5 µg/kg of body weight, based on a NOAEL of 3.5 mg/kg of body weight for an apparent decrease in kidney weight at the two highest dose levels in a 1-year dog study, with an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 reflecting some concern regarding carcinogenicity)
Limit of detection	0.75–0.01 µg/litre by gas chromatography with nitrogen–phosphorus detection
Treatment achievability	0.1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

There is no evidence from available studies that metolachlor is carcinogenic in mice. In rats, an increase in liver tumours in females as well as a few nasal tumours in males have been observed. Metolachlor is not genotoxic.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to metolachlor, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Metolachlor was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.01 mg/litre for metolachlor in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Molinate (CAS No. 2212-67-1)

Molinate is a herbicide used to control broad-leaved and grassy weeds in rice. The available data suggest that groundwater pollution by molinate is restricted to some rice-growing regions. Data on the occurrence of molinate in the environment are limited. Molinate is of low persistence in water and soil, with a half-life of about 5 days.

Guideline value	6 µg/litre
Occurrence	Concentrations in water rarely exceed 1 µg/litre
TDI	2 µg/kg of body weight, based on a NOAEL for reproductive toxicity in the rat of 0.2 mg/kg of body weight, with an uncertainty factor of 100 (for inter- and intraspecies variation)
Limit of detection	0.01 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	0.001 mg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

On the basis of the limited information available, molinate does not seem to be carcinogenic or mutagenic in animals. Evidence suggests that impairment of the reproductive performance of the male rat represents the most sensitive indicator of molinate exposure. However, epidemiological data based on the examination of workers involved in molinate production do not indicate any effect on human fertility.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to molinate, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Molinate was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.006 mg/litre for molinate in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Parathion (CAS No. 56-38-2)

Parathion is a non-systemic insecticide that is used in many countries throughout the world. It is used as a fumigant and acaricide and as a pre-harvest soil and foliage treatment on a wide variety of crops, both outdoors and in greenhouses. Parathion released to the environment will adsorb strongly to the top layer of soil and is not likely to leach significantly. Parathion disappears from surface waters in about a week. The general population is not usually exposed to parathion from air or water. Parathion residues in food are the main source of exposure.

A health-based value of 10 µg/litre can be calculated for parathion on the basis of an ADI of 0.004 mg/kg of body weight based on a NOAEL of 0.4 mg/kg body weight per day in a 2-year study in rats for retinal atrophy and inhibition of brain acetylcholinesterase at the higher dose. However, this value is much higher than parathion concentrations likely to be found in drinking-water. Under usual conditions, therefore, the presence of parathion in drinking-water is unlikely to represent a hazard to human health. For this reason, the establishment of a numerical guideline value for parathion is not deemed necessary.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to parathion, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Parathion was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

FAO/WHO (1996) *Pesticide residues in food — 1995 evaluations. Part II — Toxicological and environmental*. Geneva, World Health Organization (WHO/PCS/96.48).

Health criteria and other supporting information

Parathion-methyl (CAS No. 298-00-0)

Parathion-methyl is a non-systemic insecticide and acaricide that is produced throughout the world and has been registered for use on many crops, in particular cotton. It partitions mainly to air and soil in the environment. There is virtually no movement through soil, and neither the parent compound nor its breakdown products will reach groundwater. By far the most important route for the environmental degradation of parathion-methyl is microbial degradation. Half-lives of parathion-methyl in water are in the order of weeks to months. Concentrations of parathion-methyl in natural waters of agricultural areas in the USA ranged up to 0.46 µg/litre, with highest levels in summer. The general population can come into contact with parathion-methyl via air, water or food.

A health-based value of 9 µg/litre can be calculated for parathion-methyl on the basis of an ADI of 0.003 mg/kg of body weight, based on a NOAEL of 0.25 mg/kg of body weight per day in a 2-year study in rats for retinal degeneration, sciatic nerve demyelination, reduced body weight, anaemia and decreased brain acetylcholinesterase activity. However, this value is much higher than parathion-methyl concentrations likely to be found in drinking-water. Under usual conditions, therefore, the presence of parathion-methyl in drinking-water is unlikely to represent a hazard to human health. For this reason, the establishment of a numerical guideline value for parathion-methyl is not deemed necessary.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to parathion-methyl, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Parathion-methyl was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

***Primary Reference*s**

FAO/WHO (1996) *Pesticide residues in food — 1995 evaluations. Part II — Toxicological and environmental.* Geneva, World Health Organization (WHO/PCS/96.48).

IPCS (1992) *Methyl parathion*. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 145).

Health criteria and other supporting information

Pendimethalin (CAS No. 40487-42-1)

Pendimethalin is a pre-emergence herbicide that is fairly immobile and persistent in soil. It is used in large amounts in Japan (5000 tonnes per year). It is lost through photodegradation, biodegradation and volatilization. The leaching potential of pendimethalin appears to be very low, but little is known about its more polar degradation products.

Guideline value	20 µg/litre
Occurrence	Rarely been found in drinking-water in the limited studies available (detection limit 0.01 µg/litre)
TDI	5 µg/kg of body weight, based on evidence of slight liver toxicity even at the lowest dose tested (5 mg/kg of body weight) in a long-term rat feeding study, with an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for a combination of the use of a LOAEL instead of a NOAEL and limitations of the database)
Limit of detection	0.01 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

On the basis of available data, pendimethalin does not appear to have significant mutagenic activity. Long-term studies in mice and rats have not provided evidence of carcinogenicity; however, these studies have some important limitations.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to pendimethalin, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Pendimethalin was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.02 mg/litre for pendimethalin in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Permethrin (CAS No. 52645-53-1)

Permethrin is a contact insecticide effective against a broad range of pests in agriculture, forestry and public health. It is also used to control aquatic invertebrates in water mains. Permethrin is photodegraded both in water and on soil surfaces. Concentrations as high as 0.8 mg/litre have been recorded in surface water; levels in drinking-water have not been reported. In soil, permethrin is rapidly degraded by hydrolysis and microbial action under aerobic conditions. Exposure of the general population to permethrin is mainly via the diet.

Technical-grade permethrin is of low acute toxicity. The *cis* isomer is considerably more toxic than the *trans* isomer. IARC has classified permethrin in Group 3, as there are no human data and only limited data from animal studies. Permethrin is not genotoxic. A health-based value of 20 µg/litre can be calculated for permethrin on the basis of an ADI of 0.05 mg/kg of body weight for 2:3 and 1:3 *cis*:*trans*-permethrin, based on clinical signs and changes in body and organ weights and blood chemistry in a dietary rat study, and allocating 1% of the ADI to drinking-water. However, because permethrin occurs at concentrations well below those at which toxic effects are observed, it is not considered necessary to derive a health-based guideline value.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to permethrin, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Permethrin was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.02 mg/litre for permethrin in drinking-water, based on an ADI established by JMPR in 1987 for 2:3 and 1:3 *cis*:*trans*-permethrin and recognizing the significant exposure to permethrin from the environment. It was noted that if permethrin is to be used as a larvicide for the control of mosquitos and other insects of health significance in drinking-water sources, the share of the ADI allocated to drinking-water may be increased.

Primary Reference

FAO/WHO (2000) *Pesticide residues in food — 1999. Joint FAO/WHO Meeting on Pesticide Residues. Evaluations — 1999. Part II — Toxicology*. Geneva, World Health Organization (WHO/PCS/00.4).

Health criteria and other supporting information

2-Phenylphenol (CAS No. 90-43-7) **and its sodium salt**

2-Phenylphenol is used as a disinfectant, bactericide and virucide. In agriculture, it is used in disinfecting fruits, vegetables and eggs. It is also used as a general surface disinfectant in hospitals, nursing homes, veterinary hospitals, poultry farms, dairy farms, commercial laundries, barbershops and food processing plants. 2-Phenylphenol is readily degraded in surface waters, with a half-life of about 1 week in river water.

2-Phenylphenol has been determined to be of low toxicity. A health-based value of 1 mg/litre can be calculated for 2-phenylphenol on the basis of an ADI of 0.4 mg/kg of body weight, based on a NOAEL of 39 mg/kg of body weight per day in a 2-year study of toxicity (for decreased body weight gain and hyperplasia of the urinary bladder) and carcinogenicity of the urinary bladder in male rats and using an uncertainty factor of 100. Because of its low toxicity, however, the health-based value derived for 2-phenylphenol is much higher than 2-phenylphenol concentrations likely to be found in drinking-water. Under usual conditions, therefore, the presence of 2-phenylphenol in drinking-water is unlikely to represent a hazard to human health. For this reason, the establishment of a numerical guideline value for 2-phenylphenol is not deemed necessary.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to 2-phenylphenol, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. 2-Phenylphenol was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

FAO/WHO (2000) *Pesticide residues in food — 1999 evaluations. Part II — Toxicological*. Geneva, World Health Organization (WHO/PCS/00.4).

Health criteria and other supporting information

Propanil (CAS No. 709-98-8)

Propanil is a contact post-emergence herbicide used to control broad-leaved and grassy weeds, mainly in rice. It is a mobile compound with affinity for the water compartment. Propanil is not, however, persistent, being easily transformed under natural conditions to several metabolites. Two of these metabolites, 3,4-dichloroaniline and 3,3',4,4'-tetrachloroazobenzene (TCAB), are more toxic and more persistent than the parent compound. Although used in a number of countries, propanil has only occasionally been detected in groundwater.

Although a health-based value for propanil can be derived, this has not been done, because it is readily transformed into metabolites that are more toxic; therefore, a guideline value for the parent compound is considered inappropriate, and there are inadequate data on the metabolites to derive a guideline value for them.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to propanil, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Propanil was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.02 mg/litre for propanil in drinking-water, noting that in applying this guideline, authorities should consider the possible presence of more toxic metabolites in water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Simazine (CAS No. 122-34-9)

Simazine is a pre-emergence herbicide used on a number of crops as well as in non-crop areas. It is fairly resistant to physical and chemical dissipation processes in the soil. It is persistent and mobile in the environment.

Guideline value	2 µg/litre
Occurrence	Frequently detected in groundwater and surface water at concentrations of up to a few micrograms per litre
TDI	0.52 µg/kg of body weight, based on a NOAEL of 0.52 mg/kg of body weight from a long-term study in the rat and an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for possible non-genotoxic carcinogenicity)
Limit of detection	0.01 µg/litre by gas chromatography/mass spectrometry; 0.1–0.2 µg/litre by gas chromatography with flame thermionic detection
Treatment achievability	0.1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Simazine does not appear to be genotoxic in mammalian systems. Recent studies have shown an increase in mammary tumours in the female rat but no effects in the mouse. IARC has classified simazine in Group 3.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to simazine, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Simazine was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.002 mg/litre for simazine in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

2,4,5-T (2,4,5-Trichlorophenoxyacetic acid) (CAS No. 93-76-5)

The half-lives for degradation of chlorophenoxy herbicides, including 2,4,5-T, in the environment are in the order of several days. Chlorophenoxy herbicides are not often found in food.

Guideline value	9 µg/litre
Occurrence	Chlorophenoxy herbicides are not frequently found in drinking-water; when detected, their concentrations are usually no greater than a few micrograms per litre.
TDI	3 µg/kg of body weight, based on a NOAEL of 3 mg/kg of body weight for reduced body weight gain, increased liver and kidney weights, and renal toxicity in a 2-year study in rats, with an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 to take into consideration the suggested association between 2,4,5-T and soft tissue sarcoma and non-Hodgkin lymphoma in epidemiological studies)
Limit of detection	0.02 µg/litre by gas chromatography with an electron capture detector
Treatment achievability	1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Chlorophenoxy herbicides, as a group, have been classified in Group 2B by IARC. However, the available data from studies in exposed populations and animals do not permit assessment of the carcinogenic potential to humans of any specific chlorophenoxy herbicide. Therefore, drinking-water guidelines for these compounds are based on a threshold approach for other toxic effects.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to chlorophenoxy herbicides, including 2,4,5-T, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. 2,4,5-T was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.009 mg/litre for 2,4,5-T.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Terbuthylazine (CAS No. 5915-41-3)

Terbuthylazine (TBA), a herbicide that belongs to the chlorotriazine family, is used in both pre- and post-emergence treatment of a variety of agricultural crops and in forestry. Degradation of TBA in natural water depends on the presence of sediments and biological activity.

Guideline value	7 µg/litre
Occurrence	Concentrations found in water seldom exceed 0.2 µg/litre, although higher concentrations have been observed.
TDI	2.2 µg/kg of body weight, based on a NOAEL of 0.22 mg/kg of body weight for decreased body weight gain at the next higher dose in a 2-year toxicity/carcinogenicity study in rats, with an uncertainty factor of 100 (for inter- and intraspecies variation)
Limit of detection	0.1 µg/litre by high-performance liquid chromatography with ultraviolet detection
Treatment achievability	0.1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

There is no evidence that TBA is carcinogenic or mutagenic.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to TBA, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, no guideline value for triazine herbicides, which include TBA, was recommended after a detailed evaluation of the compounds. TBA was not evaluated in the second edition of the *Guidelines for Drinking-water Quality*, published in 1993. In the addendum to the second edition of the Guidelines, published in 1998, a health-based guideline value of 0.007 mg/litre was derived for TBA in drinking-water.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

Trifluralin (CAS No. 1582-09-8)

Trifluralin is a pre-emergence herbicide used in a number of crops. It has low water solubility and a high affinity for soil. However, biodegradation and photodegradation processes may give rise to polar metabolites that may contaminate drinking-water sources. Although this compound is used in many countries, relatively few data are available concerning contamination of drinking-water.

Guideline value	20 µg/litre
Occurrence	Not detected in the small number of drinking-water samples analysed; has been detected in surface water at concentrations above 0.5 µg/litre and rarely in groundwater
TDI	7.5 µg/kg of body weight, based on a NOAEL of 0.75 mg/kg of body weight for mild hepatic effects in a 1-year feeding study in dogs, with an uncertainty factor of 100 (for inter- and intraspecies variation)
Limit of detection	0.05 µg/litre by gas chromatography with nitrogen–phosphorus detection
Treatment achievability	1 µg/litre should be achievable using GAC
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Authorities should note that some impure technical grades of trifluralin could contain potent carcinogenic compounds and therefore should not be used.

Toxicological Review

Trifluralin of high purity does not possess mutagenic properties. Technical trifluralin of low purity may contain nitroso contaminants and has been found to be mutagenic. No evidence of carcinogenicity was demonstrated in a number of long-term toxicity/carcinogenicity studies with pure (99%) test material. IARC recently evaluated technical-grade trifluralin and assigned it to Group 3.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to trifluralin, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Trifluralin was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines established a health-based guideline value of 0.02 mg/litre for trifluralin in drinking-water, noting that authorities should be aware that some impure technical grades of trifluralin could contain potent carcinogenic compounds and therefore should not be used.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.
Health criteria and other supporting information

8.7.4 Chemicals Used in Water Treatment or Materials in Contact with Drinking-water

Chemicals are used in water treatment and may give rise to residuals in the final water. In some cases, such as chloramine and chlorine, this is intentional, and their presence confers a

direct benefit to the consumer. Some arise as unwanted by-products of the disinfection process (see Table 8.12) and some as residuals from other parts of the treatment process, such as coagulation. Some may arise as contaminants in treatment chemicals, and others may arise as contaminants in, or as corrosion products from, materials used as pipes or in other parts of the water system. Some chemicals used in water treatment (e.g., fluoride) or in materials in contact with drinking-water (e.g., styrene) have other primary sources and are therefore discussed in detail in other sections of this chapter.

The approach to monitoring may be through control of the material or chemical, and this is covered in more detail in section 4.2. In particular, it is important to optimize treatment processes and to ensure that such processes remain optimized in order to control residuals of chemicals used in treatment or the formation of disinfection by-products (see treatment monograph).

Table 8.22. Disinfection by-products present in disinfected waters (from IPCS 2000)

Disinfectant	Significant organohalogen products	Significant inorganic products	Significant non-halogenated products
Chlorine/ hypochlorous acid	trihalomethanes, haloacetic acids, haloacetonitriles, chloral hydrate, chloropicrin, chlorophenols, *N*-chloramines, halofuranones, bromohydrins	chlorate (mostly from hypochlorite use)	aldehydes, cyanoalkanoic acids, alkanoic acids, benzene, carboxylic acids
Chlorine dioxide		chlorite, chlorate	unknown
Chloramine	haloacetonitriles, cyanogen chloride, organic chloramines, chloramino acids, chloral hydrate, haloketones	nitrate, nitrite, chlorate, hydrazine	aldehydes, ketones
Ozone	bromoform, monobromoacetic acid, dibromoacetic acid, dibromoacetone, cyanogen bromide	chlorate, iodate, bromate, hydrogen peroxide, hypobromous acid, epoxides, ozonates	aldehydes, ketoacids, ketones, carboxylic acids

Guideline values have not been established for the chemicals listed in Table 8.13 because: evidence indicates that the chemical does not occur at concentrations at or near levels expected to cause a health concern, or there is lack of evidence of health effects, or there are insufficient data to support the establishment of a guideline value.

Table 8.23. Chemicals used in water treatment or materials in contact with drinking-water for which guideline values have not been established

Chemical	Reason for exclusion
Disinfectants	
Chlorine dioxide	Guideline value not established because of the rapid breakdown of chlorine dioxide and because the chlorite provisional guideline value is adequately protective for potential toxicity from chlorine dioxide
Dichloramine	Available data considered inadequate to permit recommendation of health-based guideline value
Iodine	Available data considered inadequate to permit recommendation of health-based guideline value, and lifetime exposure to iodine through water disinfection is unlikely
Silver	Available data considered inadequate to permit recommendation of health-based guideline value

Chemical	Reason for exclusion
Trichloramine	Available data considered inadequate to permit recommendation of health-based guideline value
Disinfection by-products	
Bromochloroacetate	Available data considered inadequate to permit recommendation of health-based guideline value
Bromochloroacetonitrile	Available data considered inadequate to permit recommendation of health-based guideline value
Chloroacetones	Available data considered inadequate to permit recommendation of health-based guideline values for any of the chloroacetones
Chlorophenol, 2-	Available data considered inadequate to permit recommendation of health-based guideline value
Chloropicrin	Available data considered inadequate to permit the recommendation of a health-based guideline value
Dibromoacetate	Available data considered inadequate to permit recommendation of health-based guideline value
Dichlorophenol, 2,4-	Available data considered inadequate to permit recommendation of health-based guideline value
Monobromoacetate	Available data considered inadequate to permit recommendation of health-based guideline value
MX	Occurs at concentrations well below those at which toxic effects are observed, and difficult to measure at low concentrations
Trichloroacetonitrile	Available data considered inadequate to permit recommendation of health-based guideline value
Contaminants from treatment chemicals	
Aluminium	Owing to limitations in the animal data as a model for humans and the uncertainty surrounding the human data, a health-based guideline value cannot be derived.
Iron	Not of health concern at concentrations normally observed in drinking-water, and taste and appearance of water are affected below the health-based value
Contaminants from pipes and fittings	
Asbestos	No consistent evidence that ingested asbestos is hazardous to health
Dialkyltins	Available data considered inadequate to permit recommendation of health-based guideline values for any of the dialkyltins
Fluoranthene	Occurs at concentrations well below those at which toxic effects are observed
Inorganic tin	Occurs at concentrations well below those at which toxic effects are observed
Zinc	Not of health concern at concentrations normally observed in drinking-water, but may affect the acceptability of water

Guideline values have been established for the chemicals listed in Table 8.14, which meet all of the criteria for inclusion.

Table 8.24. Guideline values for chemicals used in water treatment or materials in contact with drinking-water that are of health significance in drinking-water

Disinfectants	Guideline value (mg/litre)	Remarks
Chlorine	5	C.[c] For effective disinfection, there should be a residual concentration of free chlorine of ≥0.5 mg/litre after at least 30 min contact time at pH <8.0.
Monochloramine	3	

Disinfection by-products	Guideline value[a] (µg/litre)	Remarks
Bromate	10[b] (T)	
Bromodichloromethane	60[b]	

Disinfection by-products	Guideline value[a] (µg/litre)	Remarks
Bromoform	100	
Chloral hydrate (trichloroacetaldehyde)	10	
Chlorate	700 (D)	
Chlorite	700 (D)	
Chloroform	200	
Cyanogen chloride	70	For cyanide as total cyanogenic compounds
Dibromoacetonitrile	70	
Dibromochloromethane	100	
Dichloroacetate	40	
Dichloroacetonitrile	20 (P)	
Formaldehyde	900	
Monochloroacetate	20	
Trichloroacetate	200	
Trichlorophenol, 2,4,6-	200[b]	C[c]
Trihalomethanes		The sum of the ratio of the concentration of each to its respective guideline value should not exceed 1

Contaminants from treatment chemicals	**Guideline value (µg/litre)**	**Remarks**
Acrylamide	0.5[b]	
Epichlorohydrin	0.4 (P)	

Contaminants from pipes and fittings	**Guideline value (µg/litre)**	**Remarks**
Antimony	18	
Benzo[*a*]pyrene	0.7	
Copper	2000 (P)	Staining of laundry and sanitary ware may occur below guideline value
Lead	10	
Nickel	20 (P)	
Vinyl chloride	0.3[b]	

[a] Abbreviations used for provisional guideline values are as follows: P = evidence of a potential hazard, but the available information on health effects is limited; A = calculated guideline value is below the practical quantification level; T = calculated guideline value is below the level that can be achieved through practical treatment methods or source control; D = disinfection is likely to result in the guideline value being exceeded.

[b] For substances that are considered to be carcinogenic, the guideline value is the concentration in drinking-water associated with an upper bound excess lifetime cancer risk of 10^{-5} (one additional cancer per 100 000 of the population ingesting drinking-water containing the substance at the guideline value for 70 years). Concentrations associated with estimated upper bound excess lifetime cancer risks of 10^{-4} and 10^{-6} can be calculated by multiplying and dividing, respectively, the guideline value by 10.
In cases in which the concentration associated with an upper bound excess lifetime cancer risk of 10^{-5} is not feasible as a result of inadequate analytical or treatment technology, a provisional guideline value is recommended at a practicable level.
It should be emphasized that the guideline values for carcinogenic substances have been computed from hypothetical mathematical models that cannot be verified experimentally and that the values should be interpreted differently from TDI-based values because of the lack of precision of the models. At best, these values must be regarded as rough estimates of cancer risk. However, the models used are conservative and probably err on the side of caution. Moderate short-term exposure to levels exceeding the guideline value for carcinogens does not significantly affect the risk.

[c] C — Concentrations of the substance at or below the health-based guideline value may affect the appearance, taste or odour of the water, causing consumer complaints.

Disinfectants

Chloramines (monochloramine, dichloramine, trichloramine)

Mono-, di- and trichloramines are considered by-products of drinking-water chlorination, being formed when ammonia is added to chlorinated water. Monochloramine may also be added to maintain residual disinfection activity in potable water distribution systems. The use of chloramines for disinfection instead of chlorine reduces the formation of trihalomethanes in drinking-water supplies. However, formation of other by-products, such as haloketones, chloropicrin, cyanogen chloride, haloacetic acids, haloacetonitriles, aldehydes and chlorophenols, has been reported. Monochloramine is recognized as a less effective disinfectant than chlorine.

Guideline value for monochloramine	3 mg/litre
Occurrence	Typical chloramine concentrations of 0.5–2 mg/litre are found in drinking-water supplies where chloramine is used as a primary disinfectant or to provide a chlorine residual in the distribution system.
TDI	94 µg/kg of body weight, based on a NOAEL of 9.4 mg/kg of body weight per day, the highest dose administered to male rats in a 2-year NTP drinking-water study (although mean body weights of rats given the highest dose were lower than those of their respective control groups, it is probable that the lower body weights were caused by the unpalatability of the drinking-water)
Limit of detection	10 µg/litre by colorimetric methods
Treatment achievability	It is possible to reduce the concentration of chloramine effectively to zero (<0.1 mg/litre) by reduction; however, it is normal practice to supply water with a chloramine residual of a few tenths of a milligram per litre to act as a preservative during distribution.
Guideline derivation	
• *allocation to water*	100% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	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. Most individuals are able to taste chloramines at concentrations below 5 mg/litre, and some at levels as low as 0.3 mg/litre. The odour thresholds of dichloramine and trichloramine are much lower than that for monochloramine.

Toxicological Review

Although monochloramine has been shown to be mutagenic in some *in vitro* studies, it has not been found to be genotoxic *in vivo*. IARC has classified chloramine in Group 3 and the US EPA has classified monochloramine in group D (not classifiable as to human carcinogenicity, as there is inadequate human and animal evidence). In the NTP bioassay in two species, the incidence of mononuclear cell leukaemias in female F344/N rats was increased, but no other increases in tumour incidence were observed. IPCS (2000) did not consider that the increase in mononuclear cell leukaemia was treatment-related.

Available data are insufficient for the establishment of guideline values for dichloramine and trichloramine.

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 chloramines. The 1993 Guidelines established a health-based guideline value of 3 mg/litre for monochloramine in drinking-water. Available data were insufficient for the establishment of guideline values for dichloramine and trichloramine. It was noted that the odour thresholds for dichloramine and trichloramine are much lower than that for monochloramine.

Primary Reference

IPCS (2000) *Disinfectants and disinfectant by-products*. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 216).

Health criteria and other supporting information

Chlorine

Chlorine is produced in large amounts and widely used both industrially and domestically as a disinfectant and bleach. In particular, it is widely used in the disinfection of swimming pools and is the most commonly used disinfectant and oxidant in drinking-water treatment. In water, chlorine reacts to form hypochlorous acid and hypochlorites.

Guideline value	5 mg/litre
Occurrence	Present in most disinfected drinking-water at concentrations of 0.2–1 mg/litre
TDI	150 µg/kg of body weight, derived from a NOAEL for the absence of toxicity in rodents ingesting chlorine in drinking-water for 2 years
Limit of detection	0.01 µg/litre following pre-column derivatization to 4-bromoacetanilide by high-performance liquid chromatography; 10 µg/litre as free chlorine by colorimetry; 0.2 mg/litre by ion chromatography
Treatment achievability	It is possible to reduce the concentration of chlorine effectively to zero (<0.1 mg/litre) by reduction. However, it is normal practice to supply water with a chlorine residual of a few tenths of a milligram per litre to act as a preservative during distribution.
Guideline derivation	
• *allocation to water*	100% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value is conservative, as no adverse effect level was identified in the critical study. Most individuals are able to taste chlorine at the guideline value.

Toxicological Review

In humans and animals exposed to chlorine in drinking-water, no specific adverse treatment-related effects have been observed. IARC has classified hypochlorite in Group 3.

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 chlorine. The 1993 Guidelines established a guideline value of 5 mg/litre for free chlorine in drinking-water, but noted that this value is conservative, as no adverse effect level was identified in the study used. It was also noted that most individuals are able to taste chlorine at the guideline value.

Primary Reference

IPCS (2000) *Disinfectants and disinfectant by-products*. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 216).

Health criteria and other supporting information

Chlorine dioxide (includes Chlorate, Chlorite)

Chlorine dioxide is used for disinfection and odour/taste control of water and as a bleaching agent for cellulose, paper pulp, flour and oils. Sodium chlorite and sodium chlorate are both used in the production of chlorine dioxide as well as for other commercial purposes. Chlorine dioxide rapidly decomposes into chlorite, chlorate and chloride ions in treated water, chlorite being the predominant species; this reaction is favoured by alkaline conditions. The major route of environmental exposure to chlorine dioxide, sodium chlorite and sodium chlorate is through drinking-water.

Provisional guideline values

Chlorite	0.7 mg/litre
Chlorate	0.7 mg/litre

Occurrence	Levels of chlorite in water reported in one study ranged from 3.2 to 7.0 mg/litre.

TDIs

Chlorite	30 µg/kg of body weight based on a NOAEL of 2.9 mg/kg of body weight per day identified in a two-generation study in rats, based on lower startle amplitude, decreased absolute brain weight in the F_1 and F_2 generations and altered liver weights in two generations, using an uncertainty factor of 100 (10 each for inter- and intraspecies variation)
Chlorate	30 µg/kg of body weight based on a NOAEL of 30 mg/kg of body weight per day in a recent well conducted 90-day study in rats, based on thyroid gland colloid depletion at the next higher dose, and using an uncertainty factor of 1000 (10 each for inter- and intraspecies variation and 10 for the short duration of the study)
Limit of detection	5 µg/litre by ion chromatography with suppressed conductivity detection for chlorate
Treatment achievability	It is possible to reduce the concentration of chlorine dioxide effectively to zero (<0.1 mg/litre) by reduction; however, it is normal practice to supply water with a chlorine dioxide residual of a few tenths of a milligram per litre to act as a preservative during distribution. Chlorate concentrations arising from the use of sodium hypochlorite are generally around 0.1 mg/litre, although concentrations above 1 mg/litre have been reported. With chlorine dioxide disinfection, the concentration of chlorate depends heavily on process conditions (in both the chlorine dioxide generator and the water treatment plant) and applied dose of chlorine dioxide. As there is no viable option for reducing chlorate concentrations, control of chlorate concentration must rely on preventing its addition (from sodium hypochlorite) or formation (from chlorine dioxide). Chlorite ion is an inevitable by-product arising from the use of chlorine dioxide. When chlorine dioxide is used as the final disinfectant at typical doses, the resulting chlorite concentration should be <0.2 mg/litre. If chlorine dioxide is used as a pre-oxidant, the resulting chlorite concentration may need to be reduced using ferrous iron or activated carbon.

Guideline derivation

• *allocation to water*	80% of TDI
• *weight*	60-kg adult 2 litres/day

- *consumption*

Additional comments The guideline values for chlorite and chlorate are designated as provisional because use of chlorine dioxide as a disinfectant may result in the chlorite and chlorate guideline values being exceeded, and difficulties in meeting the guideline value must never be a reason for compromising adequate disinfection.

Toxicological Review

Chlorine dioxide - Chlorine dioxide has been shown to impair neurobehavioural and neurological development in rats exposed perinatally. Significant depression of thyroid hormones has also been observed in rats and monkeys exposed to it in drinking-water studies. A guideline value has not been established for chlorine dioxide because of its rapid hydrolysis to chlorite and because the chlorite provisional guideline value is adequately protective for potential toxicity from chlorine dioxide. The taste and odour threshold for this compound is 0.4 mg/litre.

Chlorite - IARC has concluded that chlorite is not classifiable as to its carcinogenicity to humans. The primary and most consistent finding arising from exposure to chlorite is oxidative stress resulting in changes in the red blood cells. This end-point is seen in laboratory animals and, by analogy with chlorate, in humans exposed to high doses in poisoning incidents.

Chlorate - Like chlorite, the primary concern with chlorate is oxidative damage to red blood cells. The database for chlorate is less extensive than that for chlorite. A long-term study is in progress, which should provide more information on chronic exposure to chlorate.

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 chlorine dioxide, chlorate or chlorite. The 1993 Guidelines established a provisional health-based guideline value of 0.2 mg/litre for chlorite in drinking-water. The guideline value 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 disinfection. The 1993 Guidelines did not establish a health-based guideline value for chlorine dioxide in drinking-water because of its rapid breakdown and because the provisional guideline value for chlorite is adequately protective for potential toxicity from chlorine dioxide. The 1993 Guidelines concluded that available data on the effects of chlorate in humans and experimental animals are insufficient to permit development of a guideline value and recommended that further research was needed to characterize the non-lethal effects of chlorate. It was noted that the taste and odour threshold for chlorine dioxide is 0.4 mg/litre.

Primary Reference

IPCS (2000) *Disinfectants and disinfectant by-products.* Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 216).

Health criteria and other supporting information

Iodine

Iodine occurs naturally in water in the form of iodide. Traces of iodine are produced by oxidation of iodide during water treatment. Iodine is occasionally used for water disinfection in the field or in emergency situations.

Iodine is an essential element for the synthesis of thyroid hormones. Estimates of the dietary requirement for adult humans range from 80 to 150 µg/day; in many parts of the world, there are dietary deficiencies in iodine. In 1988, JECFA set a PMTDI for iodine of 1 mg/day (17 µg/kg of body weight per day) from all sources, based primarily 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 the 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 concentrations such as might occur from water disinfection is unlikely. For these reasons, a guideline value for iodine has not been established at this time.

However, as there are concerns over the toxicological aspects of using iodine as a disinfectant in emergency situations and for travellers, iodine has been referred to JECFA for detailed evaluation.

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 iodine. The 1993 Guidelines did not establish a guideline value for iodine because available data 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; also, because iodine is not recommended for long-term disinfection, lifetime exposure to iodine concentrations such as might occur from water disinfection is unlikely.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Silver

Silver occurs naturally mainly in the form of its very insoluble and immobile oxides, sulfides and some salts. It has occasionally been found in groundwater, surface water and drinking-water at concentrations above 5 µg/litre. Silver ions are bacteriastatic and silver is used both as an emergency drinking-water disinfectant and impregnated in some filters and materials used at the point of use to prevent microbial regrowth. Silver ions are also used with copper ions in some water systems in building to control *Legionella*. Levels in drinking-water treated with silver for disinfection may be above 50 µg/litre. Recent estimates of daily intake are about 7 µg per person.

Only a small percentage of silver is absorbed. Retention rates in humans and laboratory animals range between 0 and 10%.

The only obvious sign of silver overload is argyria, a condition in which skin and hair are heavily discoloured by silver in the tissues. An oral NOAEL for argyria in humans for a total lifetime intake of 10 g of silver was estimated on the basis of human case reports and long-term animal experiments.

The low levels of silver in drinking-water, generally below 5 µg/litre, 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 bacteriological quality of drinking-water. Higher levels of silver, up to 0.1 mg/litre (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.

There are no adequate data with which to derive a health-based guideline value for silver in drinking-water.

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to silver. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was not considered necessary to establish a guideline value for silver in drinking-water. No health-based guideline value for silver was proposed in the 1993 Guidelines. Where silver salts are used to maintain the bacteriological quality of drinking-water, levels of silver up to 0.1 mg/litre can be tolerated without risk to health.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Disinfection by-products

Bromate

Sodium and potassium bromate are powerful oxidizers used mainly in permanent wave neutralizing solutions and the dyeing of textiles using sulfur dyes. Potassium bromate is also used as an oxidizer to mature flour during milling, in treating barley in beer making and in fish-paste products, although JECFA has concluded that the use of potassium bromate in food processing is not appropriate. Bromate is not normally found in water, but may be formed during ozonation when the bromide ion is present in water. Under certain conditions, bromate may also be formed in concentrated hypochlorite solutions used to disinfect drinking-water. In chlorine dioxide-treated waters, bromide, in the presence of sunlight, can be oxidized to bromate over a wide range of pH values.

Provisional guideline value	10 µg/litre
Occurrence	Has been reported in drinking-water with a variety of source water characteristics after ozonation at concentrations ranging from <2 to 293 µg/litre, depending on bromide ion concentration, ozone dosage, pH, alkalinity and dissolved organic carbon
Guideline derivation	0.19 mg/kg of body weight per day based on low-dose liner extrapolation, a one-stage Weibull time-to-tumor model applied to the incidence of mesotheliomas, renal tubule tumors and thyroid follicular tumors in male rats given potassium bomate in drinking water, using the 12-, 26-, 52- and 77- week interim kill data, leads to a health based value of 2 µg/L 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 2 - 6 µg/l. The provisional guideline value is 10 µg/L based on practical achievability.
Limit of detection	1.5 µg/litre by ion chromatography with suppressed conductivity detection; 0.2 µg/litre by ion chromatography with ultraviolet/visible absorbance detection; 0.3 µg/litre by ion chromatography with detection by inductively coupled plasma/mass spectrometry
Treatment achievability	Bromate is difficult to remove once formed. By appropriate control of disinfection conditions, it is possible to achieve bromate concentrations below 0.010 mg/litre.

Toxicological Review

IARC has concluded that although there is inadequate evidence of carcinogenicity in humans, there is sufficient evidence for the carcinogenicity of potassium bromate in experimental animals and has classified it in Group 2B (possibly carcinogenic to humans). Bromate is mutagenic both *in vitro* and *in vivo*. At this time, there is not sufficient evidence to conclude the mode of carcinogenic action for potassium bromate. Although there is limited evidence to suggest that the DNA reactivity in kidney tumours may have a non-linear dose–response relationship, there is no evidence to suggest that this same dose–response relationship operates in the development of mesotheliomas or thyroid tumours. Oxidative stress may play a role in the formation of kidney tumours, but the evidence is insufficient to establish lipid peroxidation and free radical production as key events responsible for induction of kidney tumours. Also, there are no data currently available to suggest that any single mechanism, including oxidative stress, is responsible for the production of thyroid and peritoneal tumours by bromate.

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 bromate. The 1993 Guidelines calculated the concentration of bromate in drinking-water associated with an excess lifetime cancer risk of 10^{-5} to be 0.003 mg/litre. However, because of limitations in available analytical and treatment methods, a provisional guideline value of 0.025 mg/litre was recommended.

Primary Reference

New background document

Health criteria and other supporting information

Chlorophenols (2-chlorophenol, 2,4-dichlorophenol, 2,4,6-trichlorophenol)

Chlorophenols are present in drinking-water as a result of the chlorination of phenols, as by-products of the reaction of hypochlorite with phenolic acids, as biocides or as degradation products of phenoxy herbicides. Those most likely to occur in drinking-water as by-products of chlorination are 2-chlorophenol, 2,4-dichlorophenol and 2,4,6-trichlorophenol. The taste thresholds for chlorophenols in drinking-water are low.

Guideline value for 2,4,6-trichlorophenol	200 µg/litre
Occurrence	Concentrations of chlorophenols in drinking-water are usually less than 1 µg/litre.
Basis of guideline derivation	Applying the linearized multistage model to leukaemias in male rats observed in a 2-year feeding study (hepatic tumours found in this study were not used for risk estimation because of the possible role of contaminants in their induction)
Limit of detection	0.5–5 µg/litre by formation of pentafluorobenzyl ether derivatives; 1–10 µg/litre (monochlorophenols), 0.5 µg/litre (dichlorophenols) and 0.01 µg/litre (trichlorophenols) using gas chromatography with electron capture detector
Treatment achievability	2,4,6-Trichlorophenol concentrations are generally less than 0.001 mg/litre. If necessary, 2,4,6-trichlorophenol concentrations can be reduced using GAC.
Additional comments	The guideline value for 2,4,6-trichlorophenol exceeds its lowest reported taste threshold.

Toxicological Review

2-Chlorophenol - Data on the toxicity of 2-chlorophenol are limited. Therefore, no health-based guideline value has been derived.

2,4-Dichlorophenol - Data on the toxicity of 2,4-dichlorophenol are limited. Therefore, no health-based guideline value has been derived.

2,4,6-Trichlorophenol - 2,4,6-Trichlorophenol has been reported to induce lymphomas and leukaemias in male rats and hepatic tumours in male and female mice. The compound has not been shown to be mutagenic in the Ames test but has shown weak mutagenic activity in other *in vitro* and *in vivo* studies. IARC has classified 2,4,6-trichlorophenol in Group 2B.

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to chlorophenols. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, no guideline values for 2-chlorophenol, 4-chlorophenol, 2,4-dichlorophenol, 2,6-dichlorophenol or 2,4,5-trichlorophenol were recommended after a detailed evaluation of the compounds, although it was suggested that individual chlorophenols should not be present in drinking-water at a level above 0.0001 mg/litre for organoleptic reasons (and the total phenol content of water to be chlorinated should be kept below 0.001 mg/litre). In the same edition, a health-based guideline value of 0.01 mg/litre was recommended for 2,4,6-trichlorophenol, while noting that the linear multi-stage extrapolation model appropriate for chemical carcinogens that was used in its derivation involved considerable uncertainty. It was also noted that 2,4,6-trichlorophenol may be detected by its taste and odour at a concentration of 0.0001 mg/litre. No health-based guidelines for 2-chlorophenol or 2,4-dichlorophenol were derived in the 1993 Guidelines, as data on their toxicity were limited. A guideline value of 0.2 mg/litre,

associated with a 10^{-5} excess lifetime cancer risk, was calculated for 2,4,6-trichlorophenol. This concentration exceeds the lowest reported taste threshold for the chemical (0.002 mg/litre).

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Formaldehyde

Formaldehyde occurs in industrial effluents and is emitted into air from plastic materials and resin glues. Formaldehyde in drinking-water results primarily from the oxidation of natural organic matter during ozonation and chlorination. It is also found in drinking-water as a result of release from polyacetal plastic fittings.

Guideline value	900 µg/litre
Occurrence	Concentrations of up to 30 µg/litre have been found in ozonated drinking-water.
TDI	150 µg/kg of body weight, derived from a NOAEL (for a variety of effects, including increased relative kidney weights in females and an increased incidence of renal papillary necrosis in both sexes) of 15 mg/kg of body weight per day in a 2-year study in rats, incorporating an uncertainty factor of 100 (for intro- and interspecies variation); no account was taken of potential carcinogenicity from the inhalation of formaldehyde from various indoor water uses, such as showering
Limit of detection	6.2 µg/litre by high-performance liquid chromatography following derivatization with 2,4-dinitrophenylhydrazine and liquid–solid extraction
Treatment achievability	<0.03 mg/litre by process control/modification
Guideline derivation	
• *allocation to water*	20% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

Rats and mice exposed to formaldehyde by inhalation exhibited an increased incidence of carcinomas of the nasal cavity at doses that caused irritation of the nasal epithelium. Ingestion of formaldehyde in drinking-water for 2 years caused stomach irritation in rats. Papillomas of the stomach associated with severe tissue irritation were observed in one study. IARC has classified formaldehyde in Group 2A. The weight of evidence indicates that formaldehyde is not carcinogenic by the oral route.

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 formaldehyde. The 1993 Guidelines established a health-based guideline value of 0.9 mg/litre for formaldehyde in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

MX

MX, which is the common name for 3-chloro-4-dichloromethyl-5-hydroxy-2(5H)-furanone, is formed by the reaction of chlorine with complex organic matter in drinking-water. It has been identified in chlorinated humic acid solutions and drinking-water in Finland, the United Kingdom and the USA and was found to be present in 37 water sources at levels of 2–67 ng/litre. Five drinking-water samples from different Japanese cities contained MX at concentrations ranging from <3 to 9 ng/litre.

MX is a potent mutagen in bacteria and in cells *in vitro* and has undergone a lifetime study in rats in which some tumorigenic responses were observed. These data indicate that MX induced thyroid and bile duct tumours. IARC has classified MX in Group 2B on the basis of its strong mutagenicity.

A health-based value of 1.8 µg/litre can be calculated for MX on the basis of the increase in cholangiomas and cholangiocarcinomas in female rats using the linearized multistage model (without a body surface area correction). However, this is significantly above the concentrations that would be found in drinking-water, and, in view of the analytical difficulties in measuring this compound at such low concentrations, it is considered unnecessary to propose a formal guideline value.

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 MX, or 3-chloro-4-dichloromethyl-5-hydroxy-2(5H)-furanone. The 1993 Guidelines concluded that available data were inadequate to permit a guideline value for MX to be established.

Primary Reference

IPCS (2000) *Disinfectants and disinfectant by-products*. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 216).
New background document

Health criteria and other supporting information

Trihalomethanes (bromoform, bromodichloromethane, dibromochloromethane, chloroform)

Trihalomethanes (THMs) are generated principally as by-products of the chlorination of drinking-water, being formed from naturally occurring organic compounds. Hypochlorous acid oxidizes bromide ion to form hypobromous acid, which reacts with endogenous organic materials (e.g., humic or fulvic acids) to form brominated trihalomethanes. The amount of each trihalomethane formed depends on the temperature, pH, and chlorine and bromide ion concentrations. It is assumed that most THMs present in water are ultimately transferred to air as a result of their volatility. For chloroform, for example, individuals may be exposed during showering to elevated concentrations from chlorinated tap water. Based on estimates of mean exposure from various media, the general population is exposed to chloroform principally in food, drinking-water and indoor air, in approximately equivalent amounts.

Guideline values

Chloroform	200 µg/litre
Bromoform	100 µg/litre
Dibromochloromethane	100 µg/litre
Bromodichloromethane	60 µg/litre

Occurrence Trihalomethanes are rarely found in raw water but are often present in finished water; concentrations are generally below 100 µg/litre

TDIs

Chloroform 13 µg/kg of body weight, based on slight hepatotoxicity (increases in hepatic serum enzymes and fatty cysts) observed in beagle dogs ingesting 15 mg chloroform/kg of body weight per day in toothpaste for 7.5 years, incorporating an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for use of a LOAEL rather than a NOAEL and a subchronic study) and correcting for 6 days/week dosing

Bromoform 17.9 µg/kg of body weight, based on the absence of histopathological lesions in the liver in a well conducted and well documented 90-day study in rats, using an uncertainty factor of 1000 (100 for intra- and interspecies variation and 10 for possible carcinogenicity and short duration of exposure)

Dibromochloromethane 21.4 µg/kg of body weight, based on absence of histopathological effects in the liver in a well conducted and well documented 90-day study in rats, using an uncertainty factor of 1000 (100 for intra- and interspecies variation and 10 for the short duration of the study); an additional uncertainty factor for potential carcinogenicity was not applied because of the questions regarding mouse liver tumours from corn oil vehicles and inconclusive evidence of genotoxicity

Basis of guideline derivation for bromodichloromethane Application of the linearized multi-stage model for the observed increases in incidence of kidney tumours in male mice observed in an NTP bioassay, as these tumours yield the most protective value (guideline value is supported by a recently published feeding study in rats that was not available for full evaluation)

Limit of detection 0.1 µg/litre by gas chromatography with electron capture detection; 2.2 µg/litre by gas chromatography/mass spectrometry

Treatment achievability Concentrations of chloroform, bromoform, bromodichloromethane and dibromochloromethane in drinking-water are generally <0.05 mg/litre. Concentrations can be reduced by changes to disinfection practice

(reducing organic THM precursors) or using air stripping.

Guideline derivation

- *allocation to water* — 20% of TDI for bromoform and dibromochloromethane
  50% of TDI for chloroform (based on estimates indicating that the general population is exposed to chloroform principally in food, drinking-water, and indoor air in approximately equivalent amounts and that most of the chloroform in indoor air is present as a result of volatilization from drinking-water)
- *weight* — 60-kg adult
- *consumption* — 2 litres/day

Additional comments

For authorities wishing to establish a total trihalomethane standard to account for additive toxicity, the following fractionation approach could be taken:

$$\frac{C_{bromoform}}{GV_{bromoform}} + \frac{C_{DBCM}}{GV_{DBCM}} + \frac{C_{BDCM}}{GV_{BDCM}} + \frac{C_{chloroform}}{GV_{chloroform}} \leq 1$$

Where C = concentration and GV = guideline value.

It is emphasised that disinfection efficiency should never be compromised by attempting to achieve guideline values for THMs.

Toxicological Review

The weight of evidence for genotoxicity of chloroform is considered negative. The weight of evidence for liver tumours in mice is consistent with a threshold mechanism of induction. Although it is plausible that kidney tumours in rats may similarly be associated with a threshold mechanism, there are some limitations of the database in this regard. The most universally observed toxic effect of chloroform is damage to the centrilobular region of the liver. The severity of these effects per unit dose administered depends on the species, vehicle and method by which the chloroform is administered.

In an NTP bioassay, bromoform induced a small increase in relatively rare tumours of the large intestine in rats of both sexes but did not induce tumours in mice. Data from a variety of assays on the genotoxicity of bromoform are equivocal. IARC has classified bromoform in Group 3 (not classifiable as to its carcinogenicity to humans).
In an NTP bioassay, DBCM induced hepatic tumours in female and possibly in male mice but not in rats. The genotoxicity of DBCM has been studied in a number of assays, but the available data are considered inconclusive. IARC has classified DBCM in Group 3 (not classifiable as to its carcinogenicity to humans).

IARC has classified BDCM in Group 2B (possibly carcinogenic to humans). BDCM gave both positive and negative results in a variety of *in vitro* and *in vivo* genotoxicity assays. In an NTP bioassay, BDCM induced renal adenomas and adenocarcinomas in both sexes of rats and male mice, rare tumours of the large intestine (adenomatous polyps and adenocarcinomas) in both sexes of rats, and hepatocellular adenomas and adenocarcinomas in female mice.

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to THMs. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, no guideline values for THMs other than chloroform were recommended after a detailed evaluation of the compounds. A health-based guideline value of 0.03 mg/litre was established for chloroform only, as few data existed for the remaining THMs and, for most water supplies, chloroform was the most commonly encountered member of the group. It was noted that the guideline value for chloroform was obtained using a linear multistage extrapolation of data obtained from male rats, a mathematical model appropriate to chemical carcinogens that involves considerable uncertainty. It was

also mentioned that although the available toxicological data were useful in establishing a guideline value for chloroform only, the concentrations of the other THMs should also be minimized. Limits ranging from 0.025 to 0.25 mg/litre, which represent a balance between the levels that can be achieved given certain circumstances and those that are desirable, have been set in several countries for the sum of bromoform, dibromochloromethane, bromodichloromethane and chloroform. In the 1993 Guidelines, no guideline value was set for total THMs, but guideline values were established separately for all four THMs. Authorities wishing to establish a total trihalomethane standard to account for additive toxicity could use a fractionation approach in which the sum of the ratios of each of the four THMs to their respective guideline values is less than 1. The 1993 Guidelines established health-based guideline values of 0.1 mg/litre for both bromoform and dibromochloromethane. Guideline values of 0.06 mg/litre for bromodichloromethane and 0.2 mg/litre for chloroform, associated with an excess lifetime cancer risk of 10^{-5}, were also recommended. The guideline value of 0.2 mg/litre for chloroform was retained in the addendum to the second edition of the Guidelines, published in 1998, but was developed on the basis of a TDI for threshold effects.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

Brominated acetates

Brominated acetic acids are formed during disinfection of water that contains bromide ions and organic matter. Bromide ions occur naturally in surface water and groundwater and exhibit seasonal fluctuations in levels. Bromide ion levels can increase due to saltwater intrusion resulting from drought conditions or due to pollution. Brominated acetates are generally present in surface water and groundwater distribution systems at concentrations below 50 µg/litre.

The database for dibromoacetate is considered inadequate for the development of a TDI, because there are no systemic toxicity studies of subchronic duration or longer. The database also lacks suitable toxicokinetic studies, a carcinogenicity study and a developmental study in a second species. Available mutagenicity data suggest that dibromoacetate is genotoxic. Currently, the US NTP is conducting a 2-year cancer bioassay via the oral route. In the current assessment, the database is considered to be inadequate for derivation of a guideline value for dibromoacetate.

Data are also limited on the oral toxicity of monobromoacetate and bromochloroacetate. Limited mutagenicity and genotoxicity data give mixed results for monobromoacetate and generally positive results for bromochloroacetate. Data gaps include subchronic or chronic toxicity studies, multigeneration reproductive toxicity studies, standard developmental toxicity studies and carcinogenicity studies. The available data are considered inadequate to establish guideline values for these chemicals.

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to brominated acetates. Brominated acetates were not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

New background document

Health criteria and other supporting information

Chlorinated acetates (monochloroacetate, dichloroacetate, trichloroacetate)

Chlorinated acetic acids are formed from organic material during water chlorination. Trichloroacetic acid may also be present from raw water as a consequence of its use as a herbicide.

Guideline values	
Monochloroacetate	20 µg/litre
Dichloroacetate	40 µg/litre
Trichloroacetate	200 µg/litre
Occurrence	Monochloroacetate, dichloroacetate and trichloroacetate have all been found in surface water and groundwater distribution systems, generally at concentrations below 100 µg/litre.
TDIs	
Monochloroacetate	3.5 µg/kg of body weight, based on a LOAEL of 3.5 mg/kg of body weight per day from a study in which increased absolute and relative spleen weights were observed in rats exposed to monochloroacetate in drinking-water for 2 years, and using an uncertainty factor of 1000 to take into account inter- and intraspecies variation, use of a minimal LOAEL instead of a NOAEL and database deficiencies
Trichloroacetate	32.5 µg/kg of body weight, based on a NOAEL of 32.5 mg/kg of body weight per day from a study in which decreased body weight, increased liver serum enzyme activity and liver histopathology were seen in rats exposed to trichloroacetate in drinking-water for 2 years, incorporating an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for database deficiencies)
Basis of guideline derivation for dichloroacetate	Tumour prevalence data in male mice: extrapolation from the point of departure (lower-bound confidence limit on the benchmark dose of 2.1 mg/kg of body weight per day, derived from the fit of the multistage model) to low dose performed by assuming linear dose–response curve between point of departure and origin; slope factor, which does not include body weight correction, calculated to be 0.007 (mg/kg of body weight per day)$^{-1}$
Limit of detection	2 µg/litre for monochloroacetate and 1 µg/litre for dichloro- and trichloroacetate by gas chromatography with electron capture detector; 5 µg/litre for monochloroacetate, 2 µg/litre for dichloroacetate and 1 µg/litre for trichloroacetate by gas chromatography/mass spectrometry

Treatment achievability Dichloroacetic acid and trichloroacetic acid concentrations in drinking-water are generally below 0.05 and 0.1 mg/litre, respectively. Concentrations may be reduced by installing or optimizing coagulation to remove precursors and/or by controlling the pH during chlorination.

Guideline derivation

- *allocation to water* 20% of TDI
- *weight* 60-kg adult
- *consumption* 2 litres/day

Additional comments A similar TDI for trichloroacetate was established by IPCS (2000) based on a NOAEL for hepatic toxicity in a long-term study in mice.

Toxicological Review

Monochloroacetate - No evidence of carcinogenicity of monochloroacetate was found in 2-year gavage bioassays with rats and mice. Monochloroacetate has given mixed results in a limited number of mutagenicity assays and has been negative for clastogenicity in genotoxicity studies. IARC has not classified the carcinogenicity of monochloroacetic acid.

Dichloroacetate - Dichloroacetic acid is considered to be carcinogenic, based primarily on findings of liver tumours in rats and mice. IARC has classified dichloroacetate in Group 2B. Available data are not sufficient to establish a cancer mode of action with reasonable certainty, especially at the very low exposure levels expected to apply to humans ingesting chlorinated drinking-water.

Trichloroacetate - Trichloroacetic acid has been shown to induce tumours in the liver of mice. It has given mixed results in *in vitro* assays for mutations and chromosomal aberrations and has been reported to cause chromosomal aberrations in *in vivo* studies. IARC has classified trichloroacetic acid in Group 3, not classifiable as to its carcinogenicity to humans.

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 halogenated acetic acids. The 1993 Guidelines did not establish a guideline value for monochloroacetic acid, as available toxicity data were considered insufficient. A provisional guideline value of 0.05 mg/litre was derived for dichloroacetic acid; the guideline value was designated as provisional because the data were insufficient to ensure that the value was technically achievable. A provisional guideline value of 0.1 mg/litre was derived for trichloroacetic acid, with the provisional designation because of the limitations of the available toxicological database and because there were inadequate data to judge whether the guideline value was technically achievable. It was emphasized that difficulties in meeting the guideline value must never be a reason for compromising adequate disinfection.

Primary Reference

New background document

Health criteria and other supporting information

Chloral hydrate (trichloroacetaldehyde)

Chloral hydrate is formed as a by-product of chlorination when chlorine reacts with humic acids. It has been widely used as a sedative or hypnotic drug in humans at oral doses of up to 14 mg/kg of body weight.

Guideline value	10 µg/litre
Occurrence	Found in drinking-water at concentrations of up to 100 µg/litre
TDI	1.89 µg/kg of body weight, based on a NOAEL of 1.89 mg/kg of body weight per day for mild vacuolation of the myelin sheath of the optic nerve in male rats treated with chloral hydrate in their drinking-water for 13 weeks, using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and 10 for the short duration of the study)
Quantification limit	1 µg/litre by gas chromatography with electron capture detection; 3 µg/litre by gas chromatography/mass spectrometry
Treatment achievability	Chloral hydrate concentrations in drinking-water are generally below 0.05 mg/litre. Chloral hydrate concentrations may be reduced by changes to disinfection practice or by GAC treatment.

Guideline derivation

• *allocation to water*	20% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

The results of bacterial mutagenicity assays using chloral hydrate are inconsistent; in general, the results indicate that high doses are required to induce mutagenic events. IARC has classified chloral hydrate as not classifiable as to its carcinogenicity in humans. Evidence of carcinogenicity comes from three separate 2-year bioassays with mice, which showed an increase in hepatocellular adenoma. However, two chloral hydrate metabolites, trichloroacetic acid and dichloroacetic acid, have been shown to cause hepatocellular tumours in rodents. The hepatocellular tumours are supported by subchronic and chronic toxicity studies indicating that the liver is the main target organ, in which an increase in many liver enzymes was observed. There is considerable evidence that the toxic effects are caused by a genotoxic mechanism; however, the data suggest that these effects require concentrations that are unlikely to occur under physiological conditions at the exposures typically encountered in the environment.

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 chloral hydrate. The 1993 Guidelines established a provisional health-based guideline value of 0.01 mg/litre for chloral hydrate in drinking-water. The guideline value was designated as provisional because of the limitations of the available database, necessitating the use of an uncertainty factor of 10 000.

Primary References

IPCS (2000) *Disinfectants and disinfectant by-products.* Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 216).

New background document

Health criteria and other supporting information

Chloroacetones

1,1-Dichloroacetone is formed from the reaction between chlorine and organic precursors and has been detected in chlorinated drinking-water.

The toxicological data on 1,1-dichloroacetone are very limited, although studies with single doses indicate that it affects the liver.

There are insufficient data at present to permit the proposal of guideline values for 1,1-dichloroacetone or any of the other chloroacetones.

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 chloroacetones. The 1993 Guidelines concluded that there were insufficient data available to permit the proposal of guideline values for any of the chloroacetones.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Halogenated acetonitriles (dichloroacetonitrile, dibromoacetonitrile, bromochloroacetonitrile, trichloroacetonitrile)

Halogenated acetonitriles are produced during water chlorination or chloramination from naturally occurring substances, including algae, fulvic acid and proteinaceous material. In general, increasing temperature and/or decreasing pH have been associated with increasing concentrations of halogenated acetonitriles. Ambient bromide levels appear to influence, to some degree, the speciation of halogenated acetonitrile compounds. Dichloroacetonitrile is by far the most predominant halogenated acetonitrile species detected in drinking-water.

Provisional guideline value dichloroacetonitrile	20 µg/litre
Guideline value for dibromoacetonitrile	70 µg/litre
Occurrence	Halogenated acetonitriles have been found in surface water and groundwater distribution systems at concentrations generally below 10 µg/litre and usually below 1 µg/litre.
TDIs	
Dichloroacetonitrile	2.7 µg/kg of body weight based on a LOAEL of 8 mg/kg of body weight per day for increased relative liver weight in male and female rats in a 90-day study, using an uncertainty factor of 3000 (taking into consideration intra- and interspecies variation, the short duration of the study, the use of a minimal LOAEL, and database deficiencies)
Dibromoacetonitrile	11 µg/kg of body weight, based on a NOAEL of 11.3 mg/kg of body weight per day for decreased body weight in male F344 rats in a 90-day drinking-water study and an uncertainty factor of 1000 (accounting for inter- and intraspecies variation, subchronic to chronic extrapolation and database insufficiencies)
Limit of detection	0.03 µg/litre by gas chromatography with an electron capture detector
Treatment achievability	Concentrations of individual halogenated acetonitriles can exceed 0.01 mg/litre, although levels of 0.002 mg/litre or less are more usual. Trichloroacetonitrile concentrations are likely to be much less than 0.001 mg/litre. If necessary, HAN concentrations can be reduced using GAC.
Guideline derivation	
• *allocation to water*	20% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value for dichloroacetonitrile is provisional due to limitations of the toxicological database.

Toxicological Review

IARC has concluded that dichloro-, dibromo-, bromochloro- and trichloroacetonitrile are not classifiable as to their carcinogenicity in humans. Dichloroacetonitrile and bromochloroacetonitrile have been shown to be mutagenic in bacterial assays, whereas results for dibromoacetonitrile and trichloroacetonitrile were negative. All four of these halogenated acetonitriles induced sister chromatid exchange and DNA strand breaks and adducts in mammalian cells *in vitro* but were negative in the mouse micronucleus test.

The majority of reproductive and developmental toxicity studies of the halogenated acetonitriles were conducted using tricaprylin as a vehicle for gavage administration of the compound under study. As tricaprylin was subsequently demonstrated to be a developmental toxicant that potentiated the effects of trichloroacetonitrile and, presumably, other halogenated acetonitriles, results reported for developmental studies using tricaprylin as the gavage vehicle are likely to overestimate the developmental toxicity of these halogenated acetonitriles.

Dichloroacetonitrile - Dichloroacetonitrile induced decreases in body weight and increases in relative liver weight in short-term studies. Although developmental toxicity has been demonstrated, the studies used tricaprylin as the vehicle for gavage administration.

Dibromoacetonitrile - Dibromoacetonitrile is currently under test for chronic toxicity in mice and rats. None of the available reproductive or developmental studies were adequate to use in the quantitative dose–response assessment. The data gap may be particularly relevant since cyanide, a metabolite of dibromoacetonitrile, induces male reproductive system toxicity, and due to uncertainty regarding the significance of the testes effects observed in the 14-day NTP rat study for dibromoacetonitrile.

Bromochloroacetonitrile - Available data are insufficient to serve as a basis for derivation of a guideline value for bromochloroacetonitrile.

Trichloroacetonitrile - Available data are also insufficient to serve as a basis for derivation of a guideline value for trichloroacetonitrile. The previous provisional guideline value of 1 µg/litre was based on a developmental toxicity study in which trichloroacetonitrile was administered by gavage in tricaprylin vehicle, and a recent re-evaluation judged this study to be unreliable in light of the finding in a more recent study that tricaprylin potentiates the developmental and teratogenic effects of halogenated acetonitriles and alters the spectrum of malformations in the fetuses of treated dams.

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 halogenated acetonitriles. The 1993 Guidelines established provisional health-based guideline values of 0.09 mg/litre for dichloroacetonitrile, 0.1 mg/litre for dibromoacetonitrile and 0.001 mg/litre for trichloroacetonitrile. The guideline values were designated as provisional because of the limitations of the databases (i.e., lack of long-term toxicity and carcinogenicity bioassays). Available data were insufficient to serve as a basis for derivation of a guideline value for bromochloroacetonitrile.

Primary References

New background document

IPCS (2000) *Disinfectants and disinfectant by-products.* Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 216).

Health criteria and other supporting information

Cyanogen Chloride

Cyanogen chloride is a by-product of chloramination. It is a reaction product of organic precursors with hypochlorous acid in the presence of ammonium ion. Concentrations detected in drinking-water treated with chlorine and chloramine were 0.4 and 1.6 µg/litre, respectively.

Cyanogen chloride is rapidly metabolized to cyanide in the body. There are few data on the oral toxicity of cyanogen chloride, and the guideline value is based, therefore, on cyanide. The guideline value is 70 µg/litre for cyanide as total cyanogenic compounds (see Cyanide in section 8.7.2, Chemicals from Industrial Sources and Human Dwellings).

The supporting document for cyanogen chloride will be revised once the CICAD on cyanide that is currently in preparation is finalized.

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 cyanogen chloride. The 1993 Guidelines derived a health-based guideline value for cyanogen chloride based on cyanide, as cyanogen chloride is rapidly metabolized to cyanide in the body and as there are few data on the oral toxicity of cyanogen chloride. The guideline value is 0.07 mg/litre for cyanide as total cyanogenic compounds (see cyanide).

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Chloropicrin

Chloropicrin, or trichloronitromethane, is formed by the reaction of chlorine with humic and amino acids and with nitrophenols. Its formation is increased in the presence of nitrates. Limited data from the USA indicate that concentrations in drinking-water are usually less than 5 µg/litre.

Decreased survival and body weights have been reported following long-term oral exposure in laboratory animals. Chloropicrin has been shown to be mutagenic in bacterial tests and in *in vitro* assays in lymphocytes. Because of the high mortality in a carcinogenesis bioassay and the limited number of end-points examined in the 78-week toxicity study, the available data were considered inadequate to permit the establishment of a guideline value for chloropicrin.

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 chloropicrin. The 1993 Guidelines considered the available data to be inadequate to permit the establishment of a guideline value for chloropicrin in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Contaminants from treatment chemicals

Aluminium

Aluminium is the most abundant metallic element and constitutes about 8% of the Earth's crust. Aluminium salts are widely used in water treatment as coagulants to reduce organic matter, colour, turbidity and microorganism levels. Such use may lead to increased concentrations of aluminium in finished water. Where residual concentrations are high, undesirable colour and turbidity may ensue. Concentrations of aluminium at which such problems may occur are highly dependent on a number of water quality parameters and operational factors at the water treatment plant. Aluminium intake from foods, particularly those containing aluminium compounds used as food additives, represents the major route of aluminium exposure for the general public. The contribution of drinking-water to the total oral exposure to aluminium is usually less than 5% of the total intake.

In humans, aluminium and its compounds appear to be poorly absorbed, although the available data are limited to studies on healthy males. The degree of aluminium absorption depends on a number of factors, such as the aluminium salt administered, pH (for aluminium speciation and solubility), bioavailability and dietary factors. The use of currently available animal studies to develop a guideline value for aluminium is not appropriate because of these specific toxicokinetic/dynamic considerations.

There is little indication that orally ingested aluminium is acutely toxic to humans despite the widespread occurrence of the element in foods, drinking-water and many antacid preparations. It has been hypothesized that aluminium exposure is a risk factor for the development or acceleration of onset of Alzheimer disease (AD) in humans. The 1997 WHO Environmental Health Criteria document for aluminium concludes that:

On the whole, the positive relationship between aluminium in drinking-water and AD, which was demonstrated in several epidemiological studies, cannot be totally dismissed. However, strong reservations about inferring a causal relationship are warranted in view of the failure of these studies to account for demonstrated confounding factors and for total aluminium intake from all sources.

Taken together, the relative risks for AD from exposure to aluminium in drinking-water above 100 µg/litre, as determined in these studies, are low (less than 2.0). But, because the risk estimates are imprecise for a variety of methodological reasons, a population-attributable risk cannot be calculated with precision. Such imprecise predictions may, however, be useful in making decisions about the need to control exposures to aluminium in the general population.

Owing to the limitations of the animal data as a model for humans and the uncertainty surrounding the human data, a health-based guideline value for aluminium cannot be derived at this time.

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 approach is proposed, 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/litre 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/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 Q*uality, 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 proposed 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.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

Iron

Iron is one of the most abundant metals in the Earth's crust. It is found in natural fresh waters at levels ranging from 0.5 to 50 mg/litre. Iron may also be present in drinking-water as a result of the use of iron coagulants or the corrosion of steel and cast iron pipes during water distribution.

Iron is an essential element in human nutrition. Estimates of the minimum daily requirement for iron depend on age, sex, physiological status and iron bioavailability and range from about 10 to 50 mg/day.

As a precaution against storage in the body of excessive iron, in 1983 JECFA established a provisional maximum tolerable daily intake (PMTDI) of 0.8 mg/kg of body weight, which applies to iron from all sources except for iron oxides used as colouring agents and iron supplements taken during pregnancy and lactation or for specific clinical requirements. An allocation of 10% of this PMTDI to drinking-water gives a value of about 2 mg/litre, which does not present a hazard to health. The taste and appearance of drinking-water will usually be affected below this level (see chapter 10).

No health-based guideline value for iron in drinking-water is proposed.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* suggested that concentrations of iron greater than 1.0 mg/litre would markedly impair the potability of the water. The 1963 and 1971 International Standards retained this value as a maximum allowable or permissible concentration. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 0.3 mg/litre was established, as a compromise between iron's use in water treatment and aesthetic considerations. No health-based guideline value for iron in drinking-water was proposed in the 1993 Guidelines, but it was mentioned that a value of about 2 mg/litre can be derived from the PMTDI established in 1983 by JECFA as a precaution against storage in the body of excessive iron. Iron stains laundry and plumbing fixtures at levels above 0.3 mg/litre; there is usually no noticeable taste at iron concentrations below 0.3 mg/litre, and concentrations of 1–3 mg/litre can be acceptable for people drinking anaerobic well-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

Acrylamide

Residual acrylamide monomer occurs in polyacrylamide coagulants used in the treatment of drinking-water. In general, the maximum authorized dose of polymer is 1 mg/litre. At a monomer content of 0.05%, this corresponds to a maximum theoretical concentration of 0.5 µg/litre of the monomer in water. Practical concentrations may be lower by a factor of 2–3. This applies to the anionic and non-ionic polyacrylamides, but residual levels from cationic polyacrylamides may be higher. Polyacrylamides are also used as grouting agents in the construction of drinking-water reservoirs and wells. Additional human exposure might result from food, owing to the use of polyacrylamide in food processing and the potential formation of acrylamide in foods cooked at high temperatures.

Guideline value	0.5 µg/litre
Occurrence	Concentrations of a few micrograms per litre have been detected in tap water.
Basis of guideline derivation	Combined mammary, thyroid and uterine tumours observed in female rats in a drinking-water study, and using the linearized multistage model
Limit of detection	0.032 µg/litre by gas chromatography; 0.2 µg/litre by high-performance liquid chromatography; 10 µg/litre by high-performance liquid chromatography with ultraviolet detection
Treatment achievability	Conventional treatment processes do not remove acrylamide. Acrylamide concentrations in drinking-water are controlled by limiting either or both the acrylamide content of polyacrylamide flocculants and the dose used.
Additional comments	Although the practical quantification level for acrylamide in most laboratories is above the guideline value (generally in the order of 1 µg/litre), concentrations in drinking-water can be controlled by product and dose specification.

Toxicological Review

Following ingestion, acrylamide is readily absorbed from the gastrointestinal tract and widely distributed in body fluids. Acrylamide can cross the placenta. It is neurotoxic, affects germ cells and impairs reproductive function. In mutagenicity assays, acrylamide was negative in the Ames test but induced gene mutations in mammalian cells and chromosomal aberrations *in vitro* and *in vivo*. In a long-term carcinogenicity study in rats exposed via drinking-water, acrylamide induced scrotal, thyroid and adrenal tumours in males and mammary, thyroid and uterine tumours in females. IARC has placed acrylamide in Group 2A.

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 acrylamide. The 1993 Guidelines established a guideline value of 0.0005 mg/litre associated with an excess lifetime cancer risk of 10^{-5}, noting that although the practical quantification level for acrylamide is generally in the order of 0.001 mg/litre, concentrations in drinking-water can be controlled by product and dose specification.

Primary Reference

New background document *Health criteria and other supporting information*

Epichlorohydrin

Epichlorohydrin is used for the manufacture of glycerol, unmodified epoxy resins and water treatment resins. No quantitative data are available on its occurrence in food or drinking-water. Epichlorohydrin is hydrolysed in aqueous media.

Provisional guideline value	0.4 µg/litre
Occurrence	No quantitative data available
TDI	0.14 µg/kg of body weight, on the basis of a LOAEL of 2 mg/kg of body weight per day for forestomach hyperplasia observed in a 2-year gavage study in rats, correcting for 5 days/week dosing and using an uncertainty factor of 10 000 to take into consideration inter- and intraspecies variation (100), the use of a LOAEL instead of a NOAEL (10) and carcinogenicity (10)
Limit of detection	0.01 µg/litre by gas chromatography with electron capture detection; 0.1 and 0.5 µg/litre by gas chromatography/mass spectrometry; 0.01 mg/litre by gas chromatography with flame ionization
Treatment achievability	Conventional treatment processes do not remove epichlorohydrin. Epichlorohydrin concentrations in drinking-water are controlled by limiting either or both the epichlorohydrin content of polyamine flocculants and the dose used.
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	Although epichlorohydrin is a genotoxic carcinogen, the use of the linearized multistage model for estimating cancer risk was considered inappropriate because tumours are seen only at the site of administration, where epichlorohydrin is highly irritating. The guideline value is considered to be provisional because of the large uncertainty associated with its calculation.

Toxicological Review

Epichlorohydrin is rapidly and extensively absorbed following oral, inhalation or dermal exposure. It binds easily to cellular components. Major toxic effects are local irritation and damage to the central nervous system. It induces squamous cell carcinomas in the nasal cavity by inhalation and forestomach tumours by the oral route. It has been shown to be genotoxic *in vitro* and *in vivo*. IARC has placed epichlorohydrin in Group 2A (probably carcinogenic to humans).

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 epichlorohydrin. The 1993 Guidelines proposed a provisional health-based guideline value of 0.0004 mg/litre for epichlorohydrin. The value was provisional because it was derived using an uncertainty factor of 10 000. It was noted that a practical quantification level for epichlorohydrin is of the order of 0.03 mg/litre, but concentrations in drinking-water can be controlled by specifying the epichlorohydrin content of products coming into contact with it.

Primary Reference

New background document
Health criteria and other supporting information

Contaminants from pipes and fittings

Antimony

Elemental antimony forms very hard alloys with copper, lead and tin. Antimony compounds have various therapeutic uses. Antimony is 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. It may reach drinking-water as a consequence of its presence as a contaminant in metal fittings. 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	18 µg/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 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 electrothermal atomic absorption spectrometry; 0.1–1 µg/litre by inductively coupled plasma/mass spectrometry; 0.8 µg/litre by graphite furnace atomic absorption spectrophotometry; 5 µg/litre by hydride generation atomic absorption spectrometry
Treatment achievability	Conventional treatment processes do not remove antimony. However, antimony is not normally a raw water contaminant.

Guideline derivation

• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Additional comments It is noted that the guideline value may be highly conservative.

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 was 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 health concerns.

Primary Reference

New background document

Health criteria and other supporting information

Asbestos

Asbestos is introduced into water by the dissolution of asbestos-containing minerals and ores as well as from industrial effluents, atmospheric pollution and asbestos-cement pipes in the distribution system. Exfoliation of asbestos fibres from asbestos-cement pipes is related to the aggressiveness of the water supply. Limited data indicate that exposure to airborne asbestos released from tap water during showers or humidification is negligible.

Asbestos is a known human carcinogen by the inhalation route. Although well studied, there has been 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 animal species, asbestos has not consistently increased the incidence of tumours of the gastrointestinal tract. 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.

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to asbestos. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was noted that available data were insufficient to determine whether a guideline value was needed for asbestos. The 1993 Guidelines concluded that there was no consistent evidence that ingested asbestos was hazardous to health and that there was therefore no need to establish a health-based guideline value for asbestos in drinking-water.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Copper

Copper is both an essential nutrient and a drinking-water contaminant. It has many commercial uses. It is used to make pipes, valves and fittings and is present in alloys and coatings. Copper sulfate pentahydrate is sometimes added to surface water for the control of algae. Copper concentrations in drinking-water vary widely, with the primary source most often being the corrosion of interior copper plumbing. Levels in running or fully flushed water tend to be low, whereas those in standing or partially flushed water samples are more variable and can be substantially higher (frequently >1 mg/litre). Copper concentrations in treated water often increase during distribution, especially in systems with an acid pH or high-carbonate waters with an alkaline pH. Food and water are the primary sources of copper exposure in developed countries. Consumption of standing or partially flushed water from a distribution system that includes copper pipes or fittings can considerably increase total daily copper exposure, especially for infants fed formula reconstituted with tap water.

Provisional guideline value	2000 µg/litre
Occurrence	Concentrations in drinking-water range from 0.005 to >30 mg/litre
Basis of guideline derivation	To be protective against adverse effects of copper and provide an adequate margin of safety in populations with normal copper homeostasis
Limit of detection	0.02–0.1 µg/litre by inductively coupled plasma/mass spectrometry; 0.3 µg/litre by inductively coupled plasma/optical emission spectroscopy; 0.5 µg/litre by flame atomic absorption spectrometry
Treatment achievability	Copper is not removed by conventional treatment processes. However, copper is not normally a raw water contaminant.
Additional comments	For adults with normal copper homeostasis, the guideline value should permit consumption of 2 or 3 litres of water per day, use of a nutritional supplement and copper from foods without exceeding the recommended dietary upper limit of 10 mg/day or eliciting an adverse gastrointestinal response. Owing to limitations of the available data for sensitive populations, it is not possible to establish a clear effect level with any precision. Thus, it is recommended that the guideline value for copper remain provisional. Staining of laundry and sanitary ware occurs at copper concentrations above 1 mg/litre. At levels above 2.5 mg/litre, copper imparts an undesirable bitter taste to water; at higher levels, the colour of water is also impacted.

Toxicological Review

IPCS 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 (more than 2 or 3) but not many milligrams per day in adults. This evaluation was based solely on studies of gastrointestinal effects of copper-contaminated drinking-water. However, the data on the gastrointestinal effects of copper must be used with caution, since the effects observed are influenced by temporal aspects of exposure and the concentration of ingested copper to a greater extent than the total mass or dose ingested in a 24-h period. Recent studies have delineated the threshold for the effects of copper in drinking-water on the gastrointestinal tract, but there is still uncertainty regarding the long-term effects of copper on sensitive populations, such as carriers of the gene for Wilson's disease and other metabolic disorders of copper homeostasis.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* suggested that concentrations of copper greater than 1.5 mg/litre would markedly impair the potability of the water. The 1963 and 1971 International Standards

retained this value as a maximum allowable or permissible concentration. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 1.0 mg/litre was established for copper, based on its laundry and other staining properties. The 1993 Guidelines derived a provisional health-based guideline value of 2 mg/litre for copper from the provisional maximum tolerable daily intake proposed by JECFA, based on a rather old study in dogs that did not take into account differences in copper metabolism between infants and adults. The guideline value was considered provisional because of the uncertainties regarding copper toxicity in humans. This guideline value was retained in the addendum to the Guidelines published in 1998 and remained provisional as a result of uncertainties in the dose–response relationship between copper in drinking-water and acute gastrointestinal effects in humans. It was stressed that the outcome of epidemiological studies in progress in Chile, Sweden and the USA may permit more accurate quantification of effect levels for copper-induced toxicity in humans, including sensitive subpopulations. Copper can also give rise to taste problems at concentrations above 5 mg/litre and can stain laundry and sanitary ware at concentrations above 1 mg/litre.

Primary Reference

IPCS (1998) *Copper*. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 200).

Health criteria and other supporting information

Lead

Lead is used principally in the production of lead-acid batteries, solder and alloys. The organolead compounds tetraethyl and tetramethyl lead have also been used extensively as antiknock and lubricating agents in petrol, although their use for these purposes in many countries is being phased out. 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, although total intake is decreasing. Lead is present in tap water to some extent as a result of its dissolution from natural sources, but primarily from household plumbing systems containing lead in pipes, solder, fittings or the service connections to homes. The amount of lead dissolved from the plumbing system depends on several factors, including pH, temperature, water hardness and standing time of the water, with soft, acidic water being the most plumbosolvent.

Guideline value	10 µg/litre
Occurrence	Concentrations in drinking-water are generally below 5 µg/litre, although much higher concentrations (above 100 µg/litre) have been measured.
PTWI	25 µg/kg of body weight (equivalent to 3.5 µg/kg of 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
Limit of detection	1 µg/litre by atomic absorption spectrometry
Treatment achievability	Not a raw water contaminant; treatment not applicable
Guideline derivation	
• *allocation to water*	50% of PTWI
• *weight*	5-kg infant
• *consumption*	0.75 litre/day
Additional comments	As infants are considered to be the most sensitive subgroup of the population, this guideline value will also be protective for other age groups. Lead is exceptional in that most lead in drinking-water arises from plumbing in buildings and the remedy consists principally of removing plumbing and fittings containing lead. This requires much time and money, and it is recognized 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.

Toxicological Review

Placental transfer of lead occurs in humans as early as the 12th week of gestation and continues throughout development. Young children absorb 4–5 times as much lead as adults, and the biological half-life may be considerably longer in children than in adults. Lead is a general toxicant that accumulates in the skeleton. Infants, children up to 6 years of age and pregnant women are most susceptible to its adverse health effects. Inhibition of the activity of d-aminolaevulinic dehydratase (porphobilinogen synthase; one of the major enzymes involved in the biosynthesis of haem) in children has been observed at blood lead levels as low as 5 µg/dl, although adverse effects are not associated with its inhibition at this level. Lead also interferes with calcium metabolism, both directly and by interfering with vitamin D metabolism. These effects have been observed in children at blood lead levels ranging from 12 to 120 µg/dl, with no evidence of a threshold. Lead is toxic to both the central and peripheral nervous systems, inducing subencephalopathic neurological and behavioural effects.

There is electrophysiological evidence of effects on the nervous system in children with blood levels well below 30 µg/dl. The balance of evidence from cross-sectional epidemiological studies indicates that there are statistically significant associations between blood lead levels of 30 µg/dl and more and intelligence quotient deficits of about four points in children. Results from prospective (longitudinal) epidemiological studies suggest that prenatal exposure to lead may have early effects on mental development that do not persist to the age of 4 years. Research on primates has supported the results of the epidemiological studies, in that significant behavioural and cognitive effects have been observed following postnatal exposure resulting in blood lead levels ranging from 11 to 33 µg/dl. Renal tumours have been induced in experimental animals exposed to high concentrations of lead compounds in the diet, and IARC has classified lead and inorganic lead compounds in Group 2B (possible human carcinogen). 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 guideline value derived on this basis would also be protective for carcinogenic effects.

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* recommended a maximum allowable concentration of 0.1 mg/litre for lead, based on health concerns. This value was lowered to 0.05 mg/litre in the 1963 International Standards. The tentative upper concentration limit was increased to 0.1 mg/litre in the 1971 International Standards, because this level was accepted in many countries and the water had been consumed for many years without apparent ill effects, and it was difficult to reach a lower level in countries where lead pipes were used. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a health-based guideline value of 0.05 mg/litre was recommended. The 1993 Guidelines proposed a health-based guideline value of 0.01 mg/litre, using the PTWI established by JECFA 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. As infants are considered to be the most sensitive subgroup of the population, this guideline value would also be protective for other age groups. The Guidelines also recognized that lead is exceptional, in that most lead in drinking-water arises from plumbing, and the remedy consists principally of removing plumbing and fittings containing lead. As this requires much time and money, it is recognized 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. JECFA has reassessed lead and confirmed the previously derived PTWI.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Nickel

Nickel is used mainly in the production of stainless steel and nickel alloys. 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. However, where there is heavy pollution or use of certain types of kettles, of non-resistant material for lining wells or of water that has stood for an extended time in contact with chromium or nickel plated fittings, particularly taps, the nickel contribution from water may be significant.

Provisional guideline value	20 µg/litre
Occurrence	The concentration of nickel in drinking-water is normally less than 0.02 mg/litre, although nickel released from taps and fittings may contribute up to 1 mg/litre. In special cases of release from natural or industrial nickel deposits in the ground, the nickel concentrations in drinking-water may be even higher.
TDI	5 µg/kg of body weight, derived from a NOAEL of 5 mg/kg of body weight per day from a dietary study in rats in which altered organ-to-body weight ratios were observed, using an uncertainty factor of 1000 (100 for inter- and intraspecies variation and an extra factor of 10 to compensate for the lack of adequate studies on long-term exposure and reproductive effects, a lack of data on carcinogenicity by the oral route and a much higher intestinal absorption when taken on an empty stomach in drinking-water than when taken together with food)
Limit of detection	0.1 µg/litre by inductively coupled plasma–mass spectrometry; 1 µg/litre by electrothermal atomic absorption spectrometry or inductively coupled plasma/optical emission spectroscopy; 15 µg/litre by inductively coupled plasma; 20 µg/litre by flame atomic absorption spectrometry
Treatment achievability	20 µg/litre should be achievable by conventional treatment, e.g., coagulation. However, nickel is not usually a raw water contaminant.
Guideline derivation	
• *allocation to water*	10% of TDI
• *weight*	60-kg adult
• *consumption*	2 litres/day
Additional comments	The guideline value is considered provisional owing to uncertainties about the effect level for perinatal mortality in studies in rats. The supporting document on nickel will be revised once the results of a new reproductive study have been reviewed.

Toxicological Review

IARC concluded that inhaled nickel compounds are carcinogenic to humans (Group 1) and metallic nickel is possibly carcinogenic (Group 2B). However, there is a lack of evidence of a carcinogenic risk from oral exposure to nickel. Dose-related increases in perinatal mortality were observed in a carefully conducted two-generation study in rats, but variations in response between successive litters make it difficult to draw firm conclusions from this study.

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to nickel. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was concluded that the toxicological data available indicate that a guideline value for nickel in drinking-water was not required. A health-based

guideline value of 0.02 mg/litre was derived in the 1993 Guidelines, which should provide sufficient protection for individuals who are sensitive to nickel. This guideline value was maintained in the addendum to the Guidelines published in 1998 because, on the basis of the available data, it was considered to provide sufficient protection for individuals who are sensitive to nickel. However, the guideline value was designated as provisional owing to uncertainties about the effect level for perinatal mortality.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

Inorganic Tin

Tin is used principally in the production of coatings used in the food industry. Food, particularly canned food, therefore represents the major route of human exposure to tin. For the general population, drinking-water is not a significant source of tin, and levels in drinking-water greater than 1–2 µg/litre are exceptional. However, there is increasing use of tin as a lead corrosion inhibitor.

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 stannous 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 of body weight per day.

The main adverse effect on humans of excessive levels of tin in foods (above 150 mg/kg), such as canned fruit, has been acute gastric irritation. There is no evidence of adverse effects in humans associated with chronic exposure to tin.

JECFA considered inorganic tin in 1988 at the 33rd meeting, at which a PTWI of 14 mg/kg of body weight was established. It was concluded that, because of the low toxicity of inorganic tin, a tentative guideline value could be derived that is 3 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.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to inorganic tin. The 1971 International Standards stated that tin should be controlled in drinking-water, but that insufficient information was available to enable a tentative limit to be established. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, it was concluded that no action was required for tin. The establishment of a numerical guideline value for inorganic tin was not deemed necessary in the 1993 Guidelines, as, because of the low toxicity of inorganic tin, a tentative guideline value could be derived 3 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.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

Dialkyltins

The group of chemicals known as the organotins is composed of a large number of compounds with differing properties and applications. The most widely used of the organotins are the disubstituted compounds, which are employed as stabilizers in plastics, including polyvinyl chloride (PVC) water pipes, and the trisubstituted compounds, which are widely used as biocides.

The disubstituted compounds that may leach from PVC water pipes for a short time after installation are primarily immunotoxins, although they appear to be of low general toxicity. The data available are insufficient to permit the proposal of guideline values for individual dialkyltins.

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 dialkyltins. The 1993 Guidelines concluded that the data available were insufficient to permit the proposal of guideline values for individual dialkyltins.

Primary Reference

New background document

Health criteria and other supporting information

Polynuclear Aromatic Hydrocarbons

Polynuclear aromatic hydrocarbons (PAHs) form a class of diverse organic compounds each containing two or more fused aromatic rings of carbon and hydrogen atoms. Most PAHs enter the environment via the atmosphere from a variety of combustion processes and pyrolysis sources. Owing to their low solubility and high affinity for particulate matter, they are not usually found in water in notable concentrations. The main source of PAH contamination in drinking-water is usually the coal-tar coating of drinking-water distribution pipes, used to protect the pipes from corrosion. Fluoranthene is the most commonly detected PAH in drinking-water and is associated primarily with coal-tar linings of cast iron or ductile iron distribution pipes. PAHs have been detected in a variety of foods as a result of the deposition of airborne PAHs and in fish from contaminated waters. PAHs are also formed during some methods of food preparation, such as char-broiling, grilling, roasting, frying or baking. For the general population, the major routes of exposure to PAHs are from food and ambient and indoor air. The use of open fires for heating and cooking may increase PAH exposure, especially in developing countries. Where there are elevated levels of contamination by coal-tar coatings of water pipes, PAH intake from drinking-water could be equal to or even exceed that from food.

Guideline value for benzo[a]pyrene (BaP)	0.7 µg/litre
Occurrence	PAH levels in uncontaminated groundwater usually in range 0–5 ng/litre; concentrations in contaminated groundwater may exceed 10 µg/litre; typical concentration range for sum of selected PAHs in drinking-water is from about 1 ng/litre to 11 µg/litre
Basis of guideline derivation	Based on an oral carcinogenicity study in mice and calculated using a two-stage birth–death mutation model, which incorporates variable dosing patterns and time of killing; quantification of dose–response for tumours, on the basis of new studies in which the carcinogenicity of BaP was examined following oral administration in mice, but for which the number of dose groups was smaller, confirms this value
Limit of detection	0.01 µg/litre by gas chromatography/mass spectrometry and reverse-phase high-performance liquid chromatography with a fluorescence detector
Treatment achievability	0.05 µg/litre should be achievable using coagulation
Additional comments	The presence of significant concentrations of BaP in drinking-water in the absence of very high concentrations of fluoranthene indicates the presence of coal-tar particles, which may arise from seriously deteriorating coal-tar pipe linings. It is recommended that the use of coal-tar-based and similar materials for pipe linings and coatings on storage tanks be discontinued.

Toxicological Review

Evidence that mixtures of PAHs are carcinogenic to humans comes primarily from occupational studies of workers following inhalation and dermal exposure. No data are available for humans for the oral route of exposure. There are few data on the oral toxicity of PAHs other than BaP, particularly in drinking-water. Relative potencies of carcinogenic PAHs have been determined by comparison of data from dermal and other studies. The order of potencies is consistent, and this scheme therefore provides a useful indicator of PAH potency relative to BaP.

A health-based value of 4 µg/litre can be calculated for fluoranthene on the basis of a NOAEL of 125 mg/kg of body weight per day for increased serum glutamate–pyruvate transaminase levels, kidney and

liver pathology, and clinical and haematological changes in a 13-week oral gavage study in mice, using an uncertainty factor of 10 000 (100 for inter- and intraspecies variation, 10 for the use of a subchronic study and inadequate database, and 10 because of clear evidence of co-carcinogenicity with BaP in mouse skin painting studies). However, this health-based value is significantly above the concentrations normally found in drinking-water. Under usual conditions, therefore, the presence of FA in drinking-water does not represent a hazard to human health. For this reason, the establishment of a numerical guideline value for FA is not deemed necessary.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to polynuclear aromatic hydrocarbons (PAHs). The 1971 International Standards stated that some PAHs are known to be carcinogenic and that the concentrations of six representative PAH compounds (fluoranthene, 3,4-benzfluoranthene, 11,12-benzfluoranthene, 3,4-benzpyrene, 1,12-benzpyrene and indeno [1,2,3-cd] pyrene) should therefore not, in general, exceed 0.0002 mg/litre. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, the only PAH for which there was sufficient substantiated toxicological evidence to set a guideline value was BaP. A health-based guideline value of 0.000 01 mg/litre was recommended for BaP, while noting that the mathematical model appropriate to chemical carcinogens that was used in its derivation involved considerable uncertainty. It was also recommended that the control of PAHs in drinking-water should be based on the concept that the levels found in unpolluted groundwater should not be exceeded. The 1993 Guidelines concluded that there were insufficient data available to derive drinking-water guidelines for PAHs other than BaP. The guideline value for BaP, corresponding to an excess lifetime cancer risk of 10^{-5}, was calculated to be 0.0007 mg/litre. This guideline value was retained in the addendum to the second edition of the Guidelines, published in 1998, as it was confirmed by new studies on the carcinogenicity of the compound. It was also recommended that the use of coal-tar-based and similar materials for pipe linings and coatings on storage tanks be discontinued. Although a health-based value for fluoranthene was calculated in the addendum, it was significantly above the concentrations found in drinking-water, and it was concluded that, under usual conditions, the presence of fluoranthene in drinking-water does not represent a hazard to human health; thus, the establishment of a numerical guideline value for fluoranthene was not deemed necessary. As there are few data on the oral toxicity of other PAHs, particularly in drinking-water, relative potencies of carcinogenic PAHs were determined by comparison of data from dermal and other studies, which provides a useful indicator of PAH potency relative to BaP.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information

Vinyl chloride

Vinyl chloride is used primarily for the production of polyvinyl chloride (PVC). Owing to its high volatility, vinyl chloride has rarely been detected in surface waters, except in contaminated areas. Unplasticized PVC is increasingly being used in some countries for water mains supplies. Migration of vinyl chloride monomer from unplasticized PVC is a possible source of vinyl chloride in drinking-water. It appears that inhalation is the most important route of vinyl chloride intake, although drinking-water may contribute a significant portion of daily intake where PVC piping with a high residual content of vinyl chloride monomer is used in the distribution network. There is some evidence that vinyl chloride can be formed from the break-down of other chlorinated hydrocarbon solvents polluting groundwater.

Guideline value	0.3 μg/litre
Occurrence	Rarely detected in surface waters, the concentrations measured generally not exceeding 10 μg/litre; much higher concentrations found in groundwater and well water in contaminated areas; concentrations up to 10 μg/litre detected in drinking-water
Basis for guideline derivation	Application of a linear extrapolation by drawing a straight line between the dose, determined using a pharmocokinetic model, resulting in tumours in 10 per cent of animals in rat bioassays involving oral exposure and zero dose, determining the value associated with the upper bound risk of 10^{-5} and assuming a doubling of the risk for exposure from birth.
Limit of detection	0.01 μg/litre by gas chromatography with electron capture detection or flame ionization detection with mass spectrometry for confirmation
Treatment achievability	0.001 mg/litre should be achievable using air stripping
Additional comments	Vinyl chloride is primarily of concern as a potential contaminant from some grades of PVC pipe and is best controlled by specification of material quality.

Toxicological Review

There is sufficient evidence of the carcinogenicity of vinyl chloride in humans from industrial populations exposed to high concentrations via the inhalation route, and IARC has classified vinyl chloride in Group 1. Studies of workers employed in the vinyl chloride industry have shown a marked exposure response for all liver cancers, angiosarcomas, and hepatocellular carcinoma but no strong relationship between cumulative vinyl chloride exposure and other cancers. Animal data show vinyl chloride to be a multisite carcinogen. When administered orally or by inhalation to mice, rats and hamsters, it produced tumours in the mammary gland, lungs, Zymbal gland and skin, as well as angiosarcomas of the liver and other sites. Evidence indicates that vinyl chloride metabolites are genotoxic, interacting directly with DNA. DNA adducts by vinyl chloride have also been identified. Occupational exposure has resulted in chromosomal aberrations, micronuclei and sister chromatid exchanges; response levels were correlated with exposure levels.

History of Guideline Development

The 1958, 1963 and 1971 WHO *International Standards for Drinking-water* did not refer to vinyl chloride. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, no guideline value was recommended, because the occurrence of vinyl chloride in water seemed to be associated primarily with the use of poorly polymerized poly(vinyl chloride) water pipes, a problem that was more appropriately controlled by product specification. The 1993 Guidelines calculated a guideline value of 0.005 mg/litre for vinyl chloride based on an excess lifetime cancer risk of 10^{-5}.

Primary Reference

IPCS (1999) *Vinyl chloride*. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 215).

Health criteria and other supporting information

Zinc

Zinc is an essential trace element found in virtually all food and potable water in the form of salts or organic complexes. The diet is normally the principal source of zinc. Although levels of zinc in surface water and groundwater normally do not exceed 0.01 and 0.05 mg/litre, respectively, concentrations in tap water can be much higher as a result of dissolution of zinc from pipes.

In 1982, JECFA proposed a provisional maximum tolerable daily intake for zinc of 1 mg/kg of body weight. The daily requirement for adult men is 15–20 mg/day. It was considered that, taking into account recent studies on humans, the derivation of a health-based guideline value is not required at this time. However, drinking-water containing zinc at levels above 3 mg/litre may not be acceptable to consumers (see chapter 10).

History of Guideline Development

The 1958 WHO *International Standards for Drinking-water* suggested that concentrations of zinc greater than 15 mg/litre would markedly impair the potability of the water. The 1963 and 1971 International Standards retained this value as a maximum allowable or permissible concentration. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a guideline value of 5.0 mg/litre was established for zinc, based on taste considerations. The 1993 Guidelines concluded that, taking into account recent studies on humans, the derivation of a health-based guideline value was not required at this time. However, drinking-water containing zinc at levels above 3 mg/litre may not be acceptable to consumers.

Primary Reference

WHO (1996) *Guidelines for drinking-water quality*, 2nd ed. Vol. 2. *Health criteria and other supporting information.* Geneva, World Health Organization.

Health criteria and other supporting information

8.7.5 Pesticides Used in Water for Public Health Purposes

Some pesticides are used to control the aquatic larval stages of insects of public health significance. In considering those pesticides that may enter water used for drinking-water purposes, every effort should be made not to develop guidelines that are unnecessarily stringent as to impede their use. This approach enables a suitable balance to be achieved between the protection of drinking-water quality and the control of insects of public health significance. However, it is stressed that every effort should be made to keep the concentration of any larvicide as low as possible.

Guideline values that have been derived for these larvicides are provided in Table 8.15.

Table 8.25. Guideline values for larvicides used for public health purposes that are of health significance in drinking-water

Larvicides used for public health purposes	Guideline value (µg/litre)
Chlorpyrifos	30
DDT and metabolites	1
Pyriproxyfen	300

Chlorpyrifos (CAS No. 2921-88-2)

Chlorpyrifos is a broad-spectrum organophosphorus insecticide used for the control of mosquitos, flies, various crop pests in soil and on foliage, household pests and aquatic larvae. It is a WHOPES-recommended insecticide for the control of mosquito larvae, but not for use on drinking-water sources. Chlorpyrifos is strongly absorbed by soil and does not readily leach from it, degrading slowly by microbial action. It has a low solubility in water and great tendency to partition from aqueous into organic phases in the environment. It has been detected in surface water and groundwater in the USA.

Guideline value	30 µg/litre
ADI	0.01 mg/kg of body weight based on inhibition of brain acetylcholinesterase activity in studies in mice, rats and dogs and on inhibition of erythrocyte acetylcholinesterase activity in a study of human subjects
Limit of detection	1 µg/litre by gas chromatography using an electron capture detector or flame photometric detection
Treatment achievability	No data available; should be amenable to treatment by coagulation (10–20% removal), activated carbon adsorption and ozonation
Guideline derivation	
• *allocation to water*	10% of ADI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

JMPR concluded that chlorpyrifos is unlikely to pose a carcinogenic risk to humans. Chlorpyrifos was not genotoxic in an adequate range of studies *in vitro* and *in vivo*. In long-term studies, inhibition of cholinesterase activity was the main toxicological finding in all species.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to chlorpyrifos, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Chlorpyrifos was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

FAO/WHO (2000) *Pesticide residues in food — 1999 evaluations. Part II — Toxicological*. Geneva, World Health Organization (WHO/PCS/00.4).

Health criteria and other supporting information

DDT (CAS No. 107917-42-0) **and metabolites**

The structure of DDT permits several different isomeric forms, and commercial products consist predominantly of *p*,*p*'-DDT. Its use has been restricted or banned in several countries, although DDT is still used in some countries for the control of vectors that transmit yellow fever, sleeping sickness, typhus, malaria and other insect-transmitted diseases. DDT and its metabolites are persistent in the environment and resistant to complete degradation by microorganisms. Food is the major source of intake of DDT and related compounds for the general population.

Guideline value	1 µg/litre
PTDI	0.01 mg/kg of body weight based on developmental toxicity in rats
Limit of detection	0.011 µg/litre by gas chromatography using an electron capture detector
Treatment achievability	0.1 µg/litre should be achievable using coagulation or GAC
Guideline derivation	
• *allocation to water*	1% of PTDI
• *weight*	10-kg child
• *consumption*	1 litre/day
Additional comments	DDT is listed under the Stockholm Convention on Persistent Organic Pollutants. Hence, monitoring may occur in addition to that required by drinking-water guidelines.

Toxicological Review

IARC has concluded that there is insufficient evidence in humans and sufficient evidence in experimental animals for the carcinogenicity of DDT (Group 2B) based upon liver tumours observed in rats and mice. In most studies, DDT did not induce genotoxic effects in rodent or human cell systems, nor was it mutagenic to fungi or bacteria. The ATSDR concluded that the DDT complex could impair reproduction and/or development in several species.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to DDT, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. In the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, a health-based guideline value of 0.001 mg/litre was recommended for DDT (total isomers), based on the ADI recommended by JMPR in 1969. The 1993 Guidelines established a health-based guideline value of 0.002 mg/litre for DDT and its metabolites in drinking-water, derived from the ADI recommended by JMPR in 1984 and taking into consideration the fact that infants and children may be exposed to greater amounts of chemicals in relation to their body weight, concern over the bioaccumulation of DDT and the significant exposure to DDT by routes other than water. It was noted that the guideline value exceeds the water solubility of DDT of 0.001 mg/litre, but that some DDT may be adsorbed onto the small amount of particulate matter present in drinking-water, so the guideline value could be reached under certain circumstances. It was also emphasized that the benefits of DDT use in malaria and other vector control programmes far outweigh any health risk from the presence of DDT in drinking-water.

Primary Reference

FAO/WHO (2001) *Pesticide residues in food — 2000. Joint FAO/WHO Meeting on Pesticide Residues. Evaluations — 2000. Part II — Toxicology.* Geneva, World Health Organization (WHO/PCS/01.3).
Health criteria and other supporting information

Pyriproxyfen (CAS No. 95737-68-1)

Pyriproxyfen is a broad-spectrum insect growth regulator with insecticidal activity against public health insect pests. It is a WHOPES-recommended insecticide for the control of mosquito larvae. In agriculture and horticulture, pyriproxyfen has registered uses for the control of scale, whitefly, bollworm, jassids, aphids and cutworms. Pyriproxyfen degrades rapidly in soil under aerobic conditions, with a half-life of 6.4–36 days. It disappeared from aerobic lake water–sediment systems with half-lives of 16 and 21 days. Pyriproxyfen appeared to be degraded much more slowly in anaerobic lake water–sediment systems. As pyriproxyfen is a new pesticide, few environmental data have been collected. No detectable concentrations of pyriproxyfen were found in surface water in the USA. Intake of pyriproxyfen from all sources is generally low and below the ADI.

Guideline value	300 µg/litre
ADI	0.1 mg/kg of body weight based on increased relative liver weight and increased total plasma cholesterol concentration in male dogs in 1-year toxicity studies
Limit of detection	No information found
Treatment achievability	No data available; 1 µg/litre should be achievable using GAC

Guideline derivation

• *allocation to water*	10% of ADI
• *weight*	60-kg adult
• *consumption*	2 litres/day

Toxicological Review

JMPR concluded that pyriproxyfen was not carcinogenic or genotoxic. In short- and long-term studies of the effects of pyriproxyfen in mice, rats and dogs, the liver (increases in liver weight and changes in plasma lipid concentrations, particularly cholesterol) was the main toxicological target.

History of Guideline Development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to pyriproxyfen, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. Pyriproxyfen was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, in the second edition, published in 1993, or in the addendum to the second edition, published in 1998.

Primary Reference

FAO/WHO (2000) *Pesticide residues in food — 1999 evaluations. Part II — Toxicological*. Geneva, World Health Organization (WHO/PCS/00.4).

Health criteria and other supporting information

8.7.6 Cyanobacterial Toxins

Cyanobacteria occur widely in lakes, reservoirs, ponds and slow flowing rivers. Many species are known to produce toxins, a number of which are of concern for health. There is a number of cyanotoxins, which vary in structure and may be found within cells or released into water. There is wide variation in the toxicity of recognised toxins (including amongst different varieties of a single toxin, e.g., Microcystins) and it is likely that further toxins remain unrecognized.

The health hazard is primarily associated with overgrowth, (bloom) events. Such blooms may develop rapidly and they may be of short duration. In most circumstances, but not all, they are seasonal.

Analysis of these substances is also difficult although rapid methods are becoming available for a small number, e.g. microcystins, in addition analytical standards are frequently not available. The preferred approach is therefore, monitoring of source water for evidence of blooms, or bloom forming potential, and increased vigilance where such events occur.

A variety of actions are available to decrease the probability of bloom occurrence and some effective treatments are available for removal of cyanobacteria or cyanotoxins. For these reasons, chemical monitoring of cyanotoxins is not the preferred focus of routine monitoring and is primarily used in response to bloom events. Whilst guideline values are derived where sufficient data exist, they are intended to inform the interpretation of data from the above monitoring and not to indicate that there is a requirement for routine monitoring by chemical analysis.

A guideline value has been established for microcystin-LR, which meets all of the criteria for inclusion.

	Guideline value[a] (µg/litre)	**Remarks**
microcystin-LR	1 (P)	For total microcystin-LR (free plus cell-bound)

[a] P = provisional guideline value, as there is evidence of a potential hazard, but the available information on health effects is limited.

Microcystin-LR

Cyanobacteria, or blue-green algae, occur widely in lakes, reservoirs, ponds and slow-flowing rivers. Many species of cyanobacteria produce cytotoxins, which vary in structure and may be found within cells or released into water. The health hazard is primarily associated with overgrowth (bloom) events. Such blooms may develop rapidly, and they may be of short duration. In most circumstances, but not all, they are seasonal. The major route of human exposure to cyanobacterial toxins is the consumption of drinking-water.

Provisional guideline value 1 µg/litre (for total microcystin-LR, free plus cell-bound)

TDI 0.04 µg/kg of body weight, based on liver pathology observed in a 13-week study in mice and taking into consideration limitations in the database, in particular lack of data on chronic toxicity and carcinogenicity

Analysis Analysis of cyanobacterial toxins is difficult, although rapid methods are

becoming available for a small number, e.g., microcystins. In addition, analytical standards are frequently not available.

Monitoring and treatment

The preferred approach is monitoring of source water for evidence of blooms, or bloom-forming potential, and increased vigilance where such events occur. A variety of actions are available to decrease the probability of bloom occurrence, and some effective treatments are available for the removal of cyanobacteria or cyanotoxins. For these reasons, monitoring of cyanotoxins is not the preferred focus of routine monitoring and is primarily used in response to bloom events.

Guideline derivation

- *allocation to water* — 80% of TDI
- *weight* — 60-kg adult
- *consumption* — 2 litres/day

Additional comments

The guideline value is provisional, as it covers only microcystin-LR, the database is limited and new data for the toxicity of cyanobacterial toxins are being generated. While guideline values are derived where sufficient data exist, they are intended to inform the interpretation of monitoring data and not to indicate that there is a requirement for routine monitoring by chemical analysis. Further information may be found in *Toxic cyanobacteria in water: A guide to their public health consequences, monitoring and management*, by Chorus and Bartram (1999).

Toxicological Review

Many species of cyanobacteria produce toxins, a number of which are of concern for health. The toxins are classified, according to their mode of action, as hepatotoxins (e.g., microcystins), neurotoxins (e.g., anatoxins) and skin irritants. The hepatotoxins are produced by various species within the genera *Microcystis*, *Anabaena*, *Oscillatoria*, *Nodularia*, *Nostoc*, *Cylindrospermopsis* and *Umezakia.* Most hepatotoxins (all cyclic heptapeptides) are microcystins. There is wide variation in the toxicity of recognized toxins (including among different varieties of a single toxin, e.g., microcystins), and it is likely that further toxins remain unrecognized.

History of Guideline Development

Cyanobacterial toxins were not evaluated in the 1958, 1963 and 1971 WHO *International Standards for Drinking-water* or in the first two editions of the *Guidelines for Drinking-water Quality*, published in 1984 and 1993. In the addendum to the second edition of the Guidelines, published in 1998, it was concluded that there were insufficient data to allow a guideline value to be derived for any cyanobacterial toxins other than microcystin-LR. A health-based guideline value for total microcystin-LR (free plus cell-bound) of 0.001 mg/litre was derived, assuming significant exposure from drinking-water. The guideline value was designated as provisional, as it covers only microcystin-LR, the database is limited and new data for the toxicity of cyanobacterial toxins are being generated.

Primary Reference

WHO (1998) *Guidelines for drinking-water quality*, 2nd ed. Addendum to Vol. 2. *Health criteria and other supporting information*. Geneva, World Health Organization.

Health criteria and other supporting information