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Asbestos in drinking-water

Background document for development of WHO *Guidelines for drinking-water quality*

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Preface

Access to safe drinking-water is essential to health, a basic human right and a component of effective policy for health protection. A major World Health Organization (WHO) function to support access to safe drinking-water is the responsibility "to propose ... regulations, and to make recommendations with respect to international health matters ...", including those related to the safety and management of drinking-water.

The first WHO document dealing specifically with public drinking-water quality was published in 1958 as *International standards for drinking-water*. It was revised in 1963 and 1971 under the same title. In 1984–1985, the first edition of the WHO *Guidelines for drinking-water quality* (GDWQ) was published in three volumes: Volume 1, Recommendations; Volume 2, Health criteria and other supporting information; and Volume 3, Surveillance and control of community supplies. Second editions of these volumes were published in 1993, 1996 and 1997, respectively. Addenda to Volumes 1 and 2 of the second edition were published in 1998, addressing selected chemicals. An addendum on microbiological aspects, reviewing selected microorganisms, was published in 2002. The third edition of the GDWQ was published in 2004, the first addendum to the third edition was published in 2006, and the second addendum to the third edition was published in 2008. The fourth edition was published in 2011, and the first addendum to the fourth edition was published in 2017.

The GDWQ are subject to a rolling revision process. Through this process, microbial, chemical and radiological aspects of drinking-water are subject to periodic review, and documentation relating to aspects of protection and control of drinking-water quality is accordingly prepared and updated.

Since the first edition of the GDWQ, WHO has published information on health criteria and other information to support the GDWQ, describing the approaches used in deriving guideline values, and presenting critical reviews and evaluations of the effects on human health of the substances or contaminants of potential health concern in drinking-water. In the first and second editions, these constituted Volume 2 of the GDWQ. Since publication of the third edition, they comprise a series of free-standing monographs, including this one.

For each chemical contaminant or substance considered, a background document evaluating the risks to human health from exposure to that chemical in drinking-water was prepared. The draft health criteria document was submitted to a number of scientific institutions and selected experts for peer review. The draft document was also released to the public domain for comment. Comments were carefully considered and addressed, as appropriate, taking into consideration the processes outlined in *Policies and procedures used in updating the WHO guidelines for drinking-water quality* and the WHO *Handbook for guideline development*. The revised draft was submitted for final evaluation at expert consultations.

During preparation of background documents and at expert consultations, careful consideration was given to information available in previous risk assessments carried out by the International Programme on Chemical Safety, in its Environmental Health Criteria monographs and Concise International Chemical Assessment Documents; the International Agency for Research on Cancer; the Joint Food and Agriculture Organization of the United Nations (FAO)/WHO Meeting on Pesticide Residues; and the Joint FAO/WHO Expert Committee on Food Additives (which evaluates contaminants such as lead, cadmium, nitrate and nitrite, in addition to food additives).

Further up-to-date information on the GDWQ and the process of their development is available on the WHO website and in the current edition of the GDWQ.

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The coordinator was Ms J De France, WHO. Strategic direction was provided by Mr B Gordon, WHO. Dr K Petersen and Dr S Madsen, WHO, provided liaisons with the Joint FAO/WHO Expert Committee on Food Additives and the Joint FAO/WHO Meeting on Pesticide Residues, respectively. Dr R Brown and Ms C Vickers, WHO, provided liaisons with the International Programme on Chemical Safety. Dr M Perez contributed on behalf of the WHO Radiation Programme. Dr A Faragher, Biotext, Australia, was responsible for the scientific editing of the document.

Many individuals from various countries contributed to the development of the GDWQ. The efforts of all who contributed to the preparation of this document are greatly appreciated.

Acronyms and abbreviations

A/C	asbestos–cement
ATSDR	Agency for Toxic Substances and Disease Registry (USA)
bw	body weight
CI	confidence interval
F-yr/mL	total number of fibres in one year per mL of air
GI	gastrointestinal
IARC	International Agency for Research on Cancer
MFL	million fibres per litre
SIR	standardized incidence ratio
USA	United States of America
WHO	World Health Organization

Contents

Executive summary

Asbestos minerals are naturally occurring and widespread in the environment, with the erosion of asbestiform rocks leading to naturally occurring asbestos fibres in water sources used for drinking-water. Human activities may also lead to contamination of surface waters. Asbestos was historically used in a large number of construction materials, including asbestos–cement (A/C) pipes carrying drinking-water. Although these would be replaced at the end of their operational life, many hundreds (and possibly thousands) of kilometres of A/C pipes are likely to remain in use globally as a result of the longevity of asbestos products and the risk management advice to keep products in place, where possible, to reduce health risks associated with removal. A/C pipes in contact with drinking-water are considered the main exposure source of asbestos in drinking-water. The ubiquitous nature of asbestos in the environment means that a background level of human exposure will occur via water, food and air.

The toxicological profile of asbestos fibres depends heavily on the route of exposure; inhalation exposure is associated with the most concern. Asbestos fibres are known human carcinogens by the inhalation route. Although, the data on adverse effects following ingestion are less clear, the overall weight of evidence from available epidemiology and animal studies does not suggest an increased risk of cancer following ingestion of asbestos in drinking-water. Epidemiological studies have evaluated the correlation between asbestos exposure via drinking-water and incidence of cancers of the stomach and gastrointestinal tract. The results are inconclusive, with some studies suggesting a weak positive correlation and others finding no evidence of a correlation. In addition, chronic feeding studies in rats and hamsters have similarly failed to produce any evidence of cancer effects or systemic toxicity. Thus, no causal association between asbestos exposure via drinking-water and cancer development has been reported for any asbestos fibre type.

Based on the above, it is not considered appropriate or necessary to establish a guideline value for asbestos fibres in drinking-water. Furthermore, epidemiological studies have a number of limitations that would preclude their use for deriving a guideline value. However, in view of the uncertainties and limitations of the data, it is appropriate to minimise the concentrations of asbestos fibres in drinking-water as far as practical. Where A/C materials are used, such as in pipes and storage containers, degradation and release of fibres into drinking-water should be minimized by controlling water corrosivity or coating A/C pipes with suitable structural linings. As these materials fail or deteriorate significantly, the A/C materials should be replaced with non-asbestos-containing materials. No new sources of asbestos fibres in drinking-water should be introduced. Where replacement or repair of parts is required, it is essential to prevent worker exposure to asbestos dust.

In addition, in view of the limited data available on occurrence of asbestos in drinking-water, investigative monitoring should be considered, to provide additional information on the contribution of older A/C pipes to numbers, types, size and shape of fibres in drinking-water.

1 General description

1.1 Identity

Asbestos is a general term for a group of naturally occurring fibrous silicate minerals containing iron, magnesium, calcium, sodium, titanium, manganese, and combinations thereof. These minerals are divided into two groups: serpentine (chrysotile, characterized by generally curly fibres – also called "white" asbestos) and amphibole (amosite – also called "brown" asbestos, crocidolite – also called "blue" asbestos, anthophyllite, actinolite and tremolite, characterized by generally straight fibres) (Klein & Hurlbut, 1993; IARC, 2012; WHO, 2014).

1.2 Physicochemical properties

Asbestos minerals are polyfilamentous bundles comprising long, flexible fibres of small diameter (≤3 µm), which are easily separated (IARC, 2012). These fibres are regarded as chemically inert because they do not evaporate, burn, dissolve or react with most chemicals. Chrysotile is easily degraded by strong acids, whereas amphiboles are more resistant. The various forms of asbestos are generally resistant to alkalis. The chemical nature and crystalline structure of asbestos impart several characteristics, including high tensile strength, durability, flexibility, and resistance to heat and chemicals (ATSDR, 2001; IARC, 2012).

1.3 Organoleptic properties

Asbestos fibres would not be expected to affect the taste or odour of water since they are tasteless and odourless (ATSDR, 2001). They would not affect the appearance of water at levels that have been detected in drinking-water.

1.4 Major uses and sources

Asbestos minerals are naturally occurring and thus widespread in the environment, predominantly in metamorphic rock. Chrysotile is the most commonly found form, appearing as veins in serpentine rock formations. Asbestiform amphiboles occur in relatively low quantities throughout Earth's crust (ATSDR, 2001). In some localities, erosion of asbestiform rocks leads to naturally occurring asbestos fibres in water sources used for drinking-water. Human activities may also lead to contamination of surface waters.

Asbestos, principally chrysotile, was historically used in a large number of applications, particularly in construction materials. These include roofing materials; asbestos–cement (A/C) sheets and pipe, including pipes carrying drinking-water; electrical and thermal insulation; and friction products, such as brake linings and clutch pads (ATSDR, 2001; IARC, 2012). Crocidolite asbestos appears to have been used in the manufacture of some A/C pipes (Saitoh et al., 1992), although there is uncertainty about the extent of this use in different parts of the world.

Although worldwide production and consumption of asbestos peaked in the 1970s, asbestos minerals were regularly used in the preceding decades. Since then, because of human health concerns, some countries have introduced strict legislation to limit exposure to asbestos – some countries have banned use of asbestos, whereas others continue to use asbestos to varying degrees (IARC, 2012). The longevity of asbestos products and risk management advice to reduce health risks by keeping products in place (rather than attempting to remove or replace them) mean that exposure to products that were installed in the past remains a possibility.

The total worldwide production of asbestos in 2019 was 1.10 million tonnes, with only a small number of countries accounting for this production – China, Brazil, Kazakhstan and Russia (WHO, 2014; Statista, 2020).

A/C water pipes were used extensively around the world in the mid-1900s (Cheng et al., 2020). Although details of the extent and location of use are not generally available, in 1988 it was estimated that approximately 11% (37 000 km) of the total length of water distribution systems in the United Kingdom was A/C pipes (Mordak & Wheeler, 1987), while, in Canada in the 1970s, A/C piping was used in about 19% of water distribution systems (Chatfield & Dillon, 1979). Although in many settings, A/C pipes have likely been replaced with different materials as they reach the end of their operational life due to corrosion (internal, and occasionally external, leaching of calcium), resulting in pipe bursts and fractures, many hundreds and possibly thousands of kilometres of A/C pipes are likely to remain in use globally, particularly in North America and western Europe. It would appear that A/C pipes are still in use in many, and possibly most, parts of the world.

1.5 Environmental fate

The fate of asbestos fibres released into the environment depends on the size and shape of the fibres. In general, asbestos fibres are considered to undergo degradation processes (although very slowly) and transport following release into the environment (US EPA, 2018). They may undergo minor transformation with changes in length or through leaching of minerals from the fibre surface. However, asbestos fibres are nonvolatile and insoluble in water.

Asbestos fibres tend to settle out of air and water to be deposited in soil or sediment (US EPA, 2018). There is evidence to suggest that asbestos fibres that have a small aerodynamic diameter (0.1–1 µm) can be transported considerable distances in air and water. No significant degradation or transformation is considered to occur to asbestos fibres in air, or once deposited in soil or sediment. In water, some dissolution of asbestos fibres, through leaching of magnesium ions from magnesium silicate (as magnesium hydroxide) on the surface of the fibre, may occur at low pH (Clark & Holt, 1960; ATSDR, 2001; US EPA, 2018). In addition, at basic pH, if the magnesium hydroxide layer on asbestos is incomplete (due to naturally occurring defects or chemical leaching), the exposed silanol group becomes accessible and may undergo reactions with a variety of basic species. It is uncertain exactly how these interactions affect the surface properties of asbestos fibres.

2 Environmental levels and human exposure

Although attempts have been made to identify conversion factors to calculate the number of fibres contained in a given mass of asbestos (and vice versa), these largely relate to airborne asbestos and A/C in factory environments (IPCS, 1986). However, conversion factors relevant to drinking-water scenarios have been calculated (Millette, Clark & Pansing, 1979). The mass to number ratio varies because of the differing sizes of the fibres; therefore, these conversion factors cannot be applied without knowing a good deal about the source of the asbestos. Millette, Clark & Pansing (1979) suggested that 10^6 asbestos fibres per litre of water from A/C pipe is equivalent to 0.01 µg of asbestos per litre; however, this value is uncertain because fibre size varies between sources.

Humans can be exposed to asbestos via water, food and air, as discussed below. Due to the ubiquitous nature of asbestos in the environment, a background level of exposure will be present.

2.1 Water

Asbestos fibres are introduced into water from natural and anthropogenic sources, and have been measured in both surface water and groundwater (US EPA, 2014). Dissolution of asbestos-containing minerals and ores is the principal natural source of asbestos fibres in water. Known anthropogenic sources include industrial effluents, air pollution, and corrosion of A/C pipes in water distribution systems. Available information regarding asbestos fibre type and size in different water sources is included in the sections below.

2.1.1 General drinking-water

In 1974, concentrations of optically visible fibres up to 33 million fibres per litre (MFL) were detected in drinking-water supplies in the Netherlands (Montizaan, Knaap & Van Der Heijden, 1989).

Chrysotile was the predominant type of asbestos detected in a national survey of the water supplies of 71 communities in Canada in the 1970s; concentrations ranged from not detectable (<0.1 MFL) to 2000 MFL, and median fibre lengths were in the range 0.5–0.8 µm. It was estimated at the time of this assessment that concentrations were >1 MFL in the water supplies of 25% of the Canadian population, >10 MFL for 5% of the Canadian population and >100 MFL for 0.6% of the Canadian population. Concentrations were higher in raw than in treated water (Chatfield & Dillon, 1979).

A survey carried out between 1977 and 1982 of asbestos levels in United Kingdom waters from 65 locations reported that most drinking-water samples (n = 82, from a total of 144) had fibre concentrations between "nondetectable" and 1.5 MFL; 95% of fibres were <2 µm in length (Conway & Lacey, 1982). The fibres found were predominantly chrysotile, but amphibole fibres were also found at concentrations up to 1 MFL.

In the United States of America, asbestos levels in drinking-water were monitored from 2006 to 2011 as part of the National Contaminant Occurrence assessments conducted to support the third six-year review of the National Primary Drinking Water Regulations by the United States Environmental Protection Agency. The range of detected concentrations was 0.10–6.8 MFL (5th and 95th percentiles, respectively). Concentrations greater than or equal to the regulatory limit (maximum contaminant level) of 7 MFL were reported in systems serving 0.2% of the population; however, the source of asbestos present could not be determined (US EPA, 2016). An earlier study showed that most of the population of the USA (approximately 92%) consumed drinking-water containing asbestos at concentrations below 1 MFL (Millette et al., 1980). Based on studies conducted between 1973 and 1980 in the USA, Millette et al. (1980, 1983) reported that asbestos fibre concentrations of 1–100 MFL were reached in some areas due to erosion of natural deposits, pollution, and/or corrosion of A/C pipes or roofing materials. The distribution of fibre sizes in the water depended on the source of the fibres; the average length of chrysotile fibres found in an A/C distribution system was 4 µm, whereas the average length of chrysotile fibres originating from natural erosion was 1 µm (Millette et al., 1980, 1983).

Ma & Kang (2017) sampled drinking-water in a small number of homes in Korea (n = 6) and Japan (n = 9) to determine asbestos fibre concentrations. They reported average levels of 213.3 and 181.11 fibres/L in these locations, respectively, as the sum of chrysotile, amosite and crocidolite fibres.

2.1.2 Drinking-water in contact with A/C pipes

Release of asbestos fibres from A/C pipes is related to the quality of the water supply, including pH, hardness (calcium and carbonate), and the presence of constituents such as silica, iron, manganese and zinc (Buelow et al., 1980; Schock & Beulow, 1981), and this can be mitigated by coating of distribution pipes. A study concluded that failure of A/C pipes was associated with low pH, low alkalinity, age and whether the internal surface of the pipe was protected with coal tar, bitumen or epoxy resin (Mordak & Wheeler, 1988). The study also concluded that, although structural coatings such as epoxy resin could prevent the release of asbestos fibres, chemical treatment to increase buffering could not prevent the release of fibres from A/C pipes that were already degraded.

Although A/C piping was used in about 19% of water distribution systems in Canada in the 1970s, erosion of such piping appeared to contribute measurably to the asbestos content of water supplies at only two of 71 locations surveyed (Chatfield & Dillon, 1979). In the survey carried out by Conway & Lacey (1982) in locations using A/C pipes for water distribution in the United Kingdom, levels of amphibole asbestos were greater than in areas using non-A/C distribution pipes, but were still considered low (<1 MFL). Levels in samples taken following disturbance of deposits in A/C pipes were considerably higher – up to 58 MFL (Conway & Lacey, 1982). Even higher levels of asbestos fibres (1850 MFL) were recorded in association with the severe deterioration of A/C pipes containing chrysotile and crocidolite in Woodstock, New York (USA) in the late 1980s (Webber, Covey & King, 1989). In a later evaluation, Neuberger et al. (1996) reported that there was no significant elevation in asbestos fibre concentrations from asbestos deposits or A/C pipes in 24 areas of Austria, compared with six control areas. Saitoh et al. (1992) proposed that asbestos fibres in drinking-water in two areas of Japan were due to erosion of the inner wall of the A/C pipes used for water supplies. Levels of 0.027–0.27 MFL and 0.1–0.21 MFL were measured in the two areas, respectively. Crocidolite was the prominent fibre type identified, although chrysotile and a mixture of chrysotile and amosite were also observed. Almost all asbestos fibres detected in the tap water were in the form of thick or sheaved fibres with lengths around 5–10 μm. Their shapes were very different from those of asbestos fibres found in the atmosphere, which were short (around 1 μm) and needle-like. More recently, Fiorenzuolo et al. (2013) evaluated the presence of asbestos fibres in drinking-water in 11 towns in the Marche region of Italy. The area is located near a former asbestos factory and uses A/C pipes in the distribution of drinking-water. In the few samples where asbestos was detected, only one fibre was recorded, which corresponded to levels between 1.8×10^{-3} and 2.7×10^{-3} MFL. This is difficult to interpret given the small volumes and small number of fibres detected, although these levels are considered very low.

Many of the studies described above reported that the majority of asbestos fibres identified in drinking-water were chrysotile-type asbestos of <5 μm in length. This is shorter, and the diameter is greater, than those of asbestos fibres that cause fibrosis and other adverse effects in the lung after inhalation (ATSDR, 2001). In US water supplies, Millette et al. (1980, 1983) determined an average length and width of chrysotile fibres of 1.4 and 0.04 μm, respectively, with an aspect ratio (length to width) generally >10:1. The authors noted, however, that fibre size distribution depended on the source: longer fibres were released from A/C pipes than those

collected from natural erosion of rock. Ma & Kang (2017) reported measured values as the sum of chrysotile, amosite and crocidolite fibres, with the majority being 5–10 μm in length.

2.2 Food

The asbestos fibre content of solid foodstuffs has not been well studied because of a lack of a simple, reliable analytical method. In the 1980s, it was suggested that foods that contain soil particles, dust or dirt probably contain asbestos fibres; crude estimates from that time suggested that the intake of asbestos in food may be significant compared with that in drinking-water (Rowe, 1983). Concentrations of 0.151 MFL and 4.3–6.6 MFL in beer and 1.7–12.2 MFL in soft drinks have been reported (Cunningham & Pontefract, 1971). More recent publicly available data were not identified on asbestos fibre concentrations in food or beverages.

2.3 Air

There is an abundance of literature relating to asbestos exposure via inhalation (e.g. WHO, 2000; ATSDR, 2001; IARC, 2012). The studies discussed below focus on air exposure data that are relevant to drinking-water – for example, exposure through aerosols generated from humidifiers and showers. Occupational exposure to asbestos dust can occur when working to tap into, or remove, A/C pipe if proper precautions are not taken.

The primary exposure route for nonsmoking individuals is from air. For non-occupational settings, inhalation of outdoor air is the primary source of exposure, although indoor air makes a small contribution to total airborne exposure (IARC, 2012). Chrysotile is most frequently detected. Lower concentrations (typically 100-fold) are reported in rural locations (10^{-9} MFL) than at urban sites or in close proximity to industrial sources (10^{-7} MFL) (ATSDR, 2001; IARC, 2012; US EPA, 2018).

Airborne asbestos may be released from tap water in the home. In a study completed in the late 1980s, mean airborne asbestos concentrations (type not specified, from A/C pipes) were significantly higher (1.7 ng/m^3) in three homes with water containing elevated concentrations (>10 billion fibres/L) of asbestos than in three control homes (0.31 ng/m^3); however, the difference in concentration was due primarily to increased numbers of short fibres (<1 μm), which the authors considered to pose little health risk from an inhalation perspective. Moreover, the fibre concentrations found in this limited study were within the range of those measured in indoor air in other investigations (Webber, Syrotynski & King, 1988). Negligible amounts of asbestos fibres (chrysotile type, source unknown) were released to air from water containing 40 ± 10 MFL via a conventional drum-type humidifier (Meranger, Reid & Davey, 1979).

Roccaro & Vagliasindi (2018) compared the release of asbestos fibres from a portable home humidifier and domestic shower. The humidifier was charged with groundwater naturally contaminated with asbestos (not distributed through A/C pipes) at a level of 24 687 fibres/L, and air samples were collected. Fibres longer than 5 μm with a width less than 3 μm and with a length to width ratio greater than 3:1 were counted in accordance with WHO (1997), using 200 counting fields per filter. The authors reported that 0.04–0.07% of fibres were transferred to air through use of the humidifier. This result was comparable to that reported by Hardy et al. (1992), who determined release of asbestos-like fibres from a room humidifier at levels of 0.03–4.7% of the levels present in the charging water. For the domestic shower, Roccaro & Vagliasindi (2018) reported higher levels of transfer than for the humidifier: 4.3–10.8% of fibres from tap water containing natural levels of 8229 fibres/L. Although the higher levels

may have been due to increased water use during showering, the authors also considered that the larger diameter of the released droplets from showers compared with humidifiers (around 3 and 6 µm, respectively) could allow longer fibres to be transferred to air through showering than from use of the humidifier (Hardy et al., 1992; Highsmith et al., 1992; Zhou et al., 2007). However, Roccaro & Vagliasindi (2018) also calculated the lifetime excess cancer risk (LECR) associated with exposure to asbestiform fibres released from the humidifier and shower, and found comparable LECRs for comparable durations of exposure. Although the estimated LECRs were all $>1 \times 10^{-4}$, the authors cautioned that the complex nature of such a risk assessment should be noted – in particular, in modelling the transfer of asbestos from air, which depends on numerous factors.

Some evidence is available on the contribution to inhalation of fibres from exposure through showering with water containing asbestiform fibres. However, extrapolation to assess the risk more generally is not possible because of limited data. Although asbestos fibres are very easily dispersed, asbestos is nonvolatile. Care should be taken when interpreting exposure studies because studies conducted with groundwater naturally contaminated with asbestiform fibres are probably not comparable to studies conducted with water contaminated from A/C pipes. In addition, the fibres that have usually been detected in water (see section 2.1) are predominantly those whose shape and size are considered to be of low risk with regard to adverse health effects via inhalation. For these reasons, showering is unlikely to contribute significantly to exposure to asbestos fibres that are harmful via inhalation.

2.4 Bioaccumulation

Although no data could be identified to assess asbestos fibre concentrations in edible tissues, bioaccumulation of asbestos in the food chain is not expected to occur (ATSDR, 2001).

2.5 Estimated total exposure and relative contribution of drinking-water

The Agency for Toxic Substances and Disease Registry (ATSDR) estimated in 2001 that, over a lifetime (70 years), the general population (non-occupational exposure) would receive a cumulative inhalation dose of asbestos of 0.002–0.4 F-yr/mL (or 600–11 4000 fibres/day, based on an adult inhalation rate of 20 m^3/day) (ATSDR, 2001). The ATSDR estimated intake via drinking-water to be 2–200 million fibres/day, for an adult drinking 2 L/day. However, it is not appropriate to compare or aggregate exposures from the two exposure routes because, as described above, the types of fibres in air and water are different in shape, length and diameter – as a result, they have different toxicological properties.

On the other hand, it should be noted that around 28% of inhaled dust, including asbestos, is transported to the gastrointestinal (GI) tract through mucociliary clearance (Gross et al., 1975). As a result, the GI tract is a major recipient of both inhaled and ingested asbestos fibres (Rowe, 1983; IARC, 2012). In a comparison of the relative source contributions to ingested asbestos in humans, Rowe (1983) stated that exposures through the diet and air are of more significance than exposure through drinking-water. The author tentatively estimated that, for the USA, annual intake of ingested asbestos in drinking-water could range from 9×10^5 to 4×10^{11} fibres, whereas dietary and airborne sources may deliver annual intakes to the gut of 1.2×10^9 to 9×10^{12} and 2.4×10^9 to 1.4×10^{14} asbestos fibres, respectively.

IARC (2012) highlighted that small children may have a higher exposure to asbestos through drinking-water than adults as a result of their higher intake to body weight ratio. However, this does not necessarily translate to a greater risk of adverse health effects, particularly since

asbestos-related toxicity, as reflected in the substantial database on inhalation, requires long-term exposure. No information could be identified that assessed susceptibility to ingested asbestos in children specifically, and this age group is considered by the ATSDR to have the same risk as adults (ATSDR, 2001). The United Kingdom expert Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (COC, 2013) produced a statement on the relative vulnerability of children to exposure to asbestos compared with adults and concluded that there was no evidence for increased inherent susceptibility other than a greater expected risk due to a longer expected lifetime post-exposure (i.e. a longer lag time for an effect to develop).

3 Toxicokinetics and metabolism in humans and laboratory animals

The most likely routes of human exposure to asbestos are through inhalation and ingestion; uptake following dermal exposure is considered to be unlikely (ATSDR, 2001). Information on the toxicokinetics and metabolism of asbestos following inhalation has been well reported and is summarized elsewhere (ATSDR, 2001; Kim et al., 2013; US EPA, 2018). Information on the toxicokinetics and metabolism of asbestos following ingestion, which is of direct relevance to drinking-water, is summarized below. As previously noted, the GI tract is a major recipient of both inhaled and ingested asbestos fibres, due to mucociliary clearance (Gross et al., 1975; Rowe, 1983; IARC, 2012).

3.1 Absorption

Information on the transmigration of ingested asbestos through the GI tract to other tissues is limited. The majority of ingested asbestos fibres are not absorbed by the GI tract in animal studies (ATSDR, 2001). However, there is evidence from human autopsy samples and from several experimental studies that some fibres are able to pass through the wall of the GI tract and reach blood, lymph, urine and other tissues (Cunningham & Pontefract, 1973; Cunningham et al., 1977; Hallenbeck & Patel-Mandlik, 1979; Carter & Taylor, 1980; Sebastien, Masse & Bignon, 1980; Patel-Mandlik & Millette, 1983; Weinzweig & Richards, 1983). The mechanism for this is not clear; however, as the average length of fibres found outside the GI tract following oral exposure is shorter than the average length of fibres ingested, it is suggested that shorter fibres (≤1 µm) could cross the wall of the GI tract via the process of persorption – that is, between the cells (ATSDR, 2001). Transport via a lymphohaematological route from the GI tract to the lungs has also been proposed (Hasanoglu et al., 2008).

It is not possible using currently available data to define the fraction of asbestos fibres absorbed. However, Millette et al. (1983) estimated that around 1 in 1000 ingested asbestos fibres (type and size not specified) could penetrate the digestive tract, based on experimental animal studies. Grosso et al. (2019) also reported the presence of chrysotile asbestos fibres in the liver tissue of Italian patients with cholangiocarcinoma who had environmental and/or occupational exposure to asbestos.

3.2 Distribution

In studies evaluating rats orally exposed to asbestos, fibres were identified in blood and lymph, suggesting that distribution may occur to all organs (ATSDR, 2001). Hasanoglu et al. (2008) reported distribution to the lungs, pleura and spleen of ingested chrysotile asbestos fibres (size range not given), given to rats in drinking-water at extremely high concentrations of 1.5 or 3.0 g/L for up to 9 months. A newly developed Fourier-transform infrared spectroscopy (FT-IR) approach to quantitate asbestos fibres (actinolite, amosite, anthophyllite, chrysotile,

crocidolite and tremolite; size not reported) has been reported. The authors used the approach to monitor the migration of chrysotile asbestos in mice exposed to 1 mg/day (asbestos levels determined by FT-IR) for 5 days via drinking-water (Zheng et al., 2019). They reported that their findings were indicative of asbestos fibres entering the stomach and intestines and being absorbed into the GI mucosa, with some entering the blood. After 60 days following exposure, accumulation of asbestos fibres was noted in the liver, but not in other organs. No indication of the level of absorption from the GI mucosa was provided.

3.3 Metabolism

Asbestos consists of insoluble fibres and, as such, very little metabolism of ingested asbestos fibres occurs in the GI tract. Chrysotile fibres do undergo degradation in simulated gastric fluids through metal ion exchange, leading to alterations in gross structure (ATSDR, 2001).

3.4 Excretion

Ingested asbestos fibres (no further details provided) are mainly excreted in faeces within 48 hours of a single oral dose in rats (ATSDR, 2001). Small numbers of fibres may also be excreted in urine; chrysotile fibres with altered appearance and X-ray diffraction patterns have been detected in the urine of animals (ATSDR, 2001). Zheng et al. (2019) reported that, in mice administered chrysotile fibres (no information on fibre size given) at a dose of 1 mg/day for 5 days via drinking-water, few fibres remained in the stomach, intestines and blood 60 days following cessation of exposure.

4 Effects on humans

The toxicological effects of exposure by inhalation versus ingestion are very different, with ingestion being thought to be of much less concern. Information on the toxicity of asbestos in humans following inhalation has been well reported and comprehensively summarized by a number of authoritative bodies. In brief, ATSDR (2001) and WHO (2014) concluded that the health hazards associated with inhalation of asbestos in the occupational environment have long been recognized. They include asbestosis (scarring and fibrosis), bronchial carcinoma, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx and possible cancer of the GI tract. In the evaluation by the International Agency for Research on Cancer (IARC), it was concluded that exposure to all forms of asbestos causes mesothelioma, and cancer of the lung, larynx and ovary. The conclusions were mainly based on retrospective cohort and case–control studies in workers exposed to asbestos in occupational settings (presumed here to be primarily inhalation exposure). A positive association was reported for cancer of the pharynx, stomach and colorectum (IARC, 2012).

The mechanism of asbestos toxicity to the respiratory system following inhalation has been comprehensively studied in several species. A complex interaction between fibres and cells in vivo has been described, involving mechanisms interacting at several stages of cancer development. Certain physicochemical properties of asbestos fibres have been shown to influence pathogenicity, including surface chemistry and reactivity, surface area, dimensions and biopersistence (ATSDR, 2001; IARC, 2012). The incidence of asbestos-related diseases in humans is related to fibre type, size and dose, and to industrial processing of the asbestos (WHO, 2014). As discussed in section 2.3, the fibres detected in water are of a shape and size that are considered to be of low risk via inhalation; therefore, showering is unlikely to contribute significantly to exposure to asbestos fibres that are harmful via inhalation.

Information on the toxicity of asbestos fibres following ingestion, which is of direct relevance to exposure via drinking-water, is summarized below. The ability of asbestos fibres ingested in drinking-water to migrate through the walls of the GI tract in sufficient numbers to cause adverse local or systemic effects is largely unknown and is the subject of debate. Indeed, currently, no causal association between asbestos exposure via drinking-water and cancer development has been reported for any asbestos fibre type (ATSDR, 2001; IARC, 2012; US EPA, 2018).

4.1 Acute exposure

No studies were identified addressing the toxicity of any asbestos fibre type following acute exposure via ingestion in humans.

4.2 Short-term exposure

No studies were identified addressing the toxicity of any asbestos fibre type following short-term ingestion in humans.

4.3 Long-term exposure

4.3.1 Systemic effects

No studies were identified relating to systemic effects in humans following long-term ingestion of any asbestos fibre type.

4.3.2 Neurological effects

No studies were identified regarding neurological effects in humans following long-term ingestion of any asbestos fibre type.

4.3.3 Reproductive and developmental effects

No studies were identified addressing the reproductive or developmental effects of any asbestos fibre type in humans following long-term oral exposure.

4.3.4 Immunological effects

No studies were identified relating to immunological or lymphoreticular effects in humans following long-term ingestion of any asbestos fibre type.

4.3.5 Genotoxicity and carcinogenicity

4.3.5.1 Genotoxicity

The ATSDR reported that studies on a number of different occupational and non-occupational populations exposed to different types of asbestos (actinolite, amosite, anthophyllite, chrysotile, crocidolite) fibres through inhalation suggest that asbestos is genotoxic. Several genotoxic end-points are proposed, including DNA damage, sister chromatid exchange, chromosomal aberration and gene mutation (ATSDR, 2001). The significance of these findings to humans following oral exposure to asbestos is not currently known.

4.3.5.2 Carcinogenicity

Most notably, findings from a number of occupational and non-occupational epidemiology studies indicate that inhalation of different types of asbestos (actinolite, amosite, chrysotile, crocidolite) fibres may lead to development of the noncarcinogenic end-points of asbestosis (fibrotic lung disease), pleural plaques and thickening and cancer of the lung, pleura and peritoneum.

The IARC has reported a causal relationship between exposure to all asbestos types and cancers of the lung, larynx and ovary, as well as a positive association with cancers of the pharynx, stomach and colorectum; it should be noted that the IARC Working Group was evenly divided as to whether the evidence was strong enough to warrant classification for colorectal cancer as "sufficient" (IARC, 2012). These conclusions were primarily based on retrospective cohort and case–control studies in adults previously exposed to asbestos in occupational settings, most likely via inhalation. The association between asbestos exposure and cancers of the ovary was further supported by cohort studies of women living in a crocidolite asbestos mining town in Australia (Reid et al., 2008) and family members of men who were employed in an A/C factory in Italy (Ferrante et al., 2007). These studies showed positive, although nonsignificant, increases in both ovarian cancer incidence and mortality compared with the general population.

A large proportion of inhaled asbestos fibres are probably removed via mucociliary transport to the GI tract, meaning that this organ may also be directly exposed to fibres, which may increase the risk of development of GI cancers. However, it is expected that the fibres removed by mucociliary transport will be short and thick, compared with the long and thin respirable fibres deposited in the lungs and typically associated with lung toxicity.

The GI tract may also be exposed to asbestos fibres through direct ingestion, including through drinking-water. A number of ecological correlation studies (which do not allow identification of causality, but only provide indications of potential associations) were conducted in the period 1960–1980 in the USA and Canada that suggested an association between asbestos fibres in drinking-water supplies (via both anthropogenic contamination and natural pollution of the springs) and stomach cancers in the populations served by those waters. However, exposure levels were not defined (or at least reported), and any increases were small and may have been confounded by study limitations. These include lack of consideration of lifestyle factors (e.g. cigarette smoking, diet), limited statistical power, uncertainties in exposure levels and duration of exposure, uncertainties as to the asbestos fibre type(s) involved, and limitations in the analytical methods used to measure exposure levels.

Between 1980 and 2005, a number of studies were published in the USA relating to drinking-water with concentrations of asbestos fibres above 1 million/L. Kanarek et al. (1980) conducted an ecological–epidemiological study in the San Francisco Bay area, which indicated a significant association between asbestos in drinking-water and the incidence of GI cancers (Kanarek et al., 1980; Conforti et al., 1981). The study design and data analysis have been criticized as potential confounders – for example, diet, smoking and occupation could not be adequately controlled for (Cantor, 1997). Polissar et al. (1982) calculated population-based and proportional odds ratios for a number of cancers using incidence data from 1974–1977 and mortality data from 1955–1975 for a population in western Washington state (Puget Sound Area), USA. Participants were classified as having high or low mean (± standard deviation) exposures to chrysotile fibre concentrations in drinking-water of 206.5 (± 162.2) $\times 10^4$ and 7.3 (± 12.4) $\times 10^4$ fibres/L, respectively. Fibre lengths were found to be similar: 99.9% of fibres

in high areas and 99.4% of fibres in low areas were <5 µm, and 85.8% of fibres in high areas and 82.5% of fibres in low areas were <1 µm. The authors reported inconsistent findings and concluded that, based on correlational studies, there was no consistent evidence of a cancer risk associated with the ingestion of chrysotile asbestos in drinking-water. In a case–control (interview-based) study that the authors considered to be inherently more sensitive than the previous correlational study (as a result of improved exposure classification), cases in the same geographical area as the previous study were identified using a population-based tumour registry for the period 1977–1980. Interviews were conducted to estimate exposure by four different measures. No statistically significant evidence of an increased risk of cancer following ingestion of chrysotile asbestos in drinking-water was found (Polissar et al., 1982). A similarly negative outcome was observed in a pilot study conducted in Woodstock, New York, USA, where levels of asbestos (chrysotile and crocidolite) ranging between 3.2 and 304.5 MFL were detected (Howe et al., 1989). Using the same cohort and an improved methodology (i.e. prospective study design, individual exposure data), Browne et al. (2005) also reported that there was no increased incidence of GI cancers, respiratory cancers, mesothelioma or all cancers combined; a significant increased pancreatic cancer risk in males was attributed to confounding factors and/or chance occurrence. Kanarek (1983) suggested that the lack of positive results for the Puget Sound Area, compared with the San Francisco Bay Area in California, was attributable to the shorter fibre lengths in the state of Washington than in California.

A comprehensive review and evaluation of 13 epidemiological studies of ingested asbestos (chrysotile or amosite) conducted in five areas of the USA and Canada was reported by Marsh (1983), with a view to developing water quality standards. Eight of the studies described associations between ingestion of asbestos in drinking-water and multiple cancer sites in males or females. However, no individual study or combination of studies was considered adequately strong for use in setting risk-based standards. Cantor (1997) also carried out a systematic review of epidemiological studies investigating the potential relationship between asbestiform fibres (and other contaminants) in drinking-water and cancer incidence in humans. The evidence was insufficient to evaluate cancer risk from exposure to asbestos in drinking-water.

A study examined the incidence of stomach cancer in lighthouse keepers in Norway, whose drinking-water was supplied by rainwater stored in A/C structures (Andersen, Glattre & Johansen, 1993). The drinking-water concentrations of the asbestos fibres were reported to range from 1.7 to 71 MFL (with peaks ≥1 billion fibres/L), without any characterization of the fibres by size, shape or mineralogical indications. A significant excess risk for stomach cancer was found among lighthouse keepers 20 years or more since first exposure, with a standardized incidence ratio (SIR) of 2.4 (95% confidence interval [CI] 1.2–4.3). No excess risk was found for any other type of cancer. The study was limited by uncertainty in the magnitude of the total asbestos exposure and by confounding factors such as diet.

In a similar study, Kjærheim et al. (2005) assessed the incidence of stomach cancer in Norwegian lighthouse keepers (n = 726) exposed to mixed fibre asbestos in drinking-water (runoff from roof tiles comprising 15% asbestos that had suffered significant deterioration). Fibre content ranged between 1800 MFL and 71 000 MFL; 92% of fibres were chrysotile, and a smaller percentage of amphibole fibres were present. Exposure was assumed to have occurred in keepers employed between 1917 to 1967, and individuals were followed up for cancer incidence for the period 1960–2002. Because of a lack of complete work histories, the authors divided the cohort into three subgroups of "certainly exposed" (n = 107), "possibly unexposed"

(*n* = 479) and unknown (*n* = 140). The authors reported an increased risk of stomach cancer in the whole cohort (SIR = 1.6; CI = 1.0–2.3), in the certainly exposed (SIR = 2.5; CI = 0.9–5.5), and in the whole cohort followed up for at least 20 years (SIR = 1.7; CI = 1.1–2.7). Less consistent results were found for colon cancer incidence. Although the authors concluded that the findings support an association between ingested asbestos intake and stomach cancer, several limitations in the study mean that causality cannot be concluded. For example, there is considerable uncertainty in the exposure database (reflected in the wide CI ranges), which may have led to misclassifications of individuals; standardization of the cohort to the rural population was not carried out; covariates (including diet, alcohol intake, smoking habits, isolation and prior exposures) were not accounted for, and so there is high likelihood of confounding; the findings were generally based on low numbers of cases, leading to higher uncertainty; and the database of studies with which to compare findings is poor. It is also unclear whether the high levels of exposure documented are relevant to the general population. The authors noted that the levels of asbestos measured in the study are at the very upper range of those reported by Millette et al. (1983) in water supplies from A/C pipes (0.01–1 000 000 MFL). A better understanding of the biological plausibility of the study findings would assist interpretation of the findings reported by Kjærheim and other oral intake studies for asbestos.

In a further review of evidence from epidemiological, in vivo and in vitro studies, Bunderson-Schelvan et al. (2011) assessed extrapulmonary effects of asbestos exposure, including GI effects. The authors stated that environmental exposure to asbestos is most likely due to chrysotile fibres released from drinking-water pipes. The data in the reviewed publications show that the most likely outcome of exposure to ingested asbestos is the development of stomach cancer, although it is noted that the data are inconsistent and do not allow strong conclusions to be made.

As previously noted, a positive association between exposure to asbestos and stomach and colorectal cancers was reported by the IARC Working Group, However, the Working Group was evenly divided as to whether the evidence was strong enough to warrant classification for colorectal cancer as “sufficient” (IARC, 2012). The conclusions were based on long-term, high-level occupational inhalation cohort studies. No clear conclusions were derived regarding exposure to asbestos through drinking-water and these health end-points (IARC, 2012).

A possible link between non-occupational and environmental exposure to asbestos (including oral exposure through drinking-water) and an increased risk of GI cancers was also evaluated by Kim et al. (2013). The authors noted the inconsistent results from epidemiological studies evaluating the association between asbestos exposure via drinking-water and cancers of the digestive system, and suggested that these inconsistencies could be attributed to varying amounts of asbestos released from water pipelines at various times, differences in the asbestos composition in the water, and methodological differences. In addition, Kim et al. (2013) noted that the evidence for stomach cancer incidence was much stronger for occupational inhalation exposure than for drinking-water exposure.

Di Ciaula & Gennaro (2017) reviewed the available evidence examining a potential relationship between ingestion of asbestos fibres and the risk of GI cancers. However, as a result of the lack of robust epidemiological studies on asbestos ingestion, the authors concluded that it was not possible to derive a risk threshold in non-occupational cohorts, principally because of methodological limitations. In its latest evaluation, the United States Environmental

Protection Agency also concluded that, based on currently available evidence, there is no clear association between asbestos exposure via drinking-water and cancer (US EPA, 2018).

The potential adverse effects associated with ingestion of asbestos have also been evaluated by Cheng et al. (2020) using a weight-of-evidence approach to evaluate human and animal data. The authors reported that, overall, the animal studies did not show definitive GI carcinogenic effects following ingestion of different types of asbestos fibres over a range of doses. A number of epidemiology studies reported statistically significant increases in multiple GI-specific cancers; however, these findings were considered to be potentially confounded due to a number of critical study limitations. Cheng et al. (2020) concluded that, based on the evidence considered, there was "insufficient evidence of causality between the ingestion of asbestos and an increased incidence of GI cancers".

5 Effects on animals and in vitro test systems

5.1 Acute exposure

No studies were identified addressing the toxicity of any asbestos fibre type following acute oral exposure in animals.

5.2 Short-term exposure

Rats administered three doses of crocidolite by oral gavage at 33 mg/kg body weight (bw)/day (numbers of fibres and size range not known) showed increased numbers of aberrant crypt foci, considered to be possible precursors to colon cancer. Increased aberrant foci were also evident following a single dose (assumed by oral gavage) of crocidolite (40 mg/kg bw/day; numbers of fibres and size range not known) and a single dose (assumed by oral gavage) of chrysotile (70 mg/kg bw/day; numbers of fibres and size range not known). No aberrant foci were seen in mice administered either a single dose of chrysotile of 100 mg/kg bw/day or three doses of crocidolite at 50 mg/kg bw/day (numbers of fibres and size range not known) (Corpet, Pirot & Goubet, 1993). However, as no excess of non-neoplastic lesions in the GI epithelium has been noted in a number of other studies in rats and hamsters, the ATSDR concluded that the weight of evidence indicates that ingestion of asbestos is not associated with any significant noncarcinogenic effects in the GI system (ATSDR, 2001).

5.3 Long-term exposure

5.3.1 Systemic effects

No systemic effects have been reported in rats and hamsters exposed – including in lifetime chronic feeding studies – to chrysotile, amosite, crocidolite or tremolite (numbers of fibres and size range not known) in the diet at a level of 1% (estimated by the ATSDR to be equivalent to 500–800 mg/kg bw/day) (Gross et al., 1975; NTP, 1983, 1985, 1988, 1990a,b,c). This supports the view that injury to systemic tissues is likely to be negligible because very few asbestos fibres are able to cross the GI lumen into blood (ATSDR, 2001).

5.3.2 Neurological effects

Histological or clinical evidence of neurotoxicity was not evident in rats and hamsters in a chronic feeding study with exposure to doses of chrysotile, amosite, crocidolite or tremolite (numbers of fibres and size range not known) at 500 mg/kg bw/day (rats) and

830 mg/kg bw/day (hamsters). Acute exposure of rats and mice to crocidolite at doses of 160 mg/kg bw/day (rats) and 50 mg/kg bw/day (mice) or chrysotile at doses of 70 mg/kg bw/day (rats) and 100 mg/kg bw/day (mice) (numbers of fibres and size range not known) was not associated with clinical signs of neurotoxicity (NTP, 1983, 1985, 1988, 1990a,b,c; Corpet, Pirot & Goubet, 1993; ATSDR, 2001).

5.3.3 Reproductive and developmental effects

Rats and hamsters exposed to chrysotile, amosite, crocidolite or tremolite (numbers of fibres and size range not known) individually at doses of 500 mg/kg bw/day (rats) or 830 mg/kg bw/day (hamsters) in the diet during gestation, lactation and throughout life did not show any effects on fertility or histopathology of reproductive organs (NTP, 1983, 1985, 1988, 1990a,b,c).

Administration of 0.3–33 mg/kg bw/day of chrysotile (numbers of fibres and size range not known) to CD-1 female mice on gestational days 1–15 did not affect the survival of the progeny (Schneider & Maurer, 1977).

5.3.4 Immunological effects

No studies were identified addressing potential immunological or lymphorecticular effects in animals following ingestion of any asbestos fibre type.

5.3.5 Genotoxicity and carcinogenicity

5.3.5.1 Genotoxicity

Asbestos fibres (amosite, anthophyllite, crocidolite and chrysotile) were not mutagenic in standard strains of *Salmonella* Typhimurium and *Escherichia coli*; however, positive results were found with *Salmonella* Typhimurium strain TA102, which is sensitive to oxidative substances. In vitro assays carried out for crocidolite and chrysotile using human peripheral lymphocytes and mesothelioma cells have reported variable positive and negative findings, and suggest that crocidolite is a more potent mutagen than chrysotile. Asbestos toxicity shows cell-line specificity in human and animal cells that may be due to differential phagocytic activity – cells with high activity (such as mesothelioma cells) show greater susceptibility to asbestos-induced mutatagenicity than those without such activity (such as lymphocytes) (ATSDR, 2001).

Chromosomal aberrations in Chinese hamster ovary and Syrian hamster embryo cells following exposure to asbestos (amosite, anthophyllite, chrysotile, crocidolite) fibres have been well reported. Aberrations include aneuploidy (usually polyploidy), fragmentation, breaks, rearrangements, gaps, dicentrics, inversions and rings. Similar aberrations have been shown in rat and human mesothelial cells, lymphocytes and amniotic fluid cells, but not in fibroblasts or promyelocytic leukaemia cells. Clastogenic effects may occur as a result of physical interference of the asbestos fibres with chromosome segregation during mitosis (ATSDR, 2001; IARC, 2012).

Other in vitro tests for increased sister chromatid exchange, DNA damage or cell transformation provided both negative and positive findings (ATSDR, 2001).

No in vivo studies were identified examining the genotoxicity of any asbestos fibre type using a standardized protocol. In nonstandard studies, a single oral (gavage) administration of amphibole or crocidolite asbestos (numbers of fibres and size range not known) to rats at 50 mg/kg bw did not increase the frequency of micronuclei formation or sister chromatid exchange in bone marrow samples taken 24 hours following exposure. A single oral (gavage) dose of chrysotile (numbers of fibres and size range not known) of 100 or 500 mg/kg bw did not increase the number of chromosomal aberrations in the bone marrow of rhesus monkeys. In Swiss albino mice, oral (gavage) or intraperitoneal administration of chrysotile (numbers of fibres and size range not known) at doses of 0.4–400 mg/kg bw did not increase the frequency of micronuclei formation in bone marrow (Lavappa, Fu & Epstein, 1975).

In summary, in vivo studies in humans (section 4.3.5) and animals indicate that exposure to the asbestos fibre types tested to date is associated with chromosomal damage (aberrations). In vitro studies with mammalian cells also indicate clastogenicity; however, the findings from in vivo and in vitro gene mutation studies are inconclusive.

5.3.5.2 Carcinogenicity

Although the carcinogenicity of inhaled asbestos in laboratory animals is well established, there is no conclusive evidence that ingested asbestos is carcinogenic (Toft et al., 1984; DHHS, 1987; ATSDR, 2001; IARC, 2012). In a series of extensive investigations involving groups of 250 animals of each sex (McConnell et al., 1983a,b; NTP, 1985), no increases in tumour incidence were observed in Syrian golden hamsters fed (by gavage) 1% amosite (500–800 mg/kg bw/day), or short-range (98% shorter than 10 µm) or intermediate-range (65% longer than 10 µm) chrysotile over their lifetime (no indication of total fibre count per dose was given). Similarly, no increase in tumours was seen in Fischer 344 rats fed the same preparations as evaluated by McConnell et al. (1983a,b) of 1% tremolite or amosite, or short-range chrysotile in the diet over their lifetime (no indication of total fibre count per dose was given). The authors estimated a 1% dose to be around 70 000 times greater than the largest possible human exposure from drinking-water. It should be noted that, although the incidence of benign epithelial neoplasms in the GI tract in male Fischer 344 rats fed 1% intermediate-range chrysotile was significantly increased when compared with pooled controls from lifetime asbestos (chrysotile) feeding studies in the same laboratory, the increase was not statistically significant when compared with concurrent controls and was limited to one sex.

5.4 Mode of action

The mechanistic basis for the carcinogenicity of inhaled asbestos has been well studied and reported. Carcinogenicity is considered to result from several mechanisms interacting at multiple stages of carcinogenesis (IARC, 2012). For example, asbestos fibres can generate free radicals that induce genotoxicity; can interfere with the mitotic apparatus by direct physical interaction, resulting in aneuploidy and polyploidy; can induce macrophage activation and persistent inflammation in animals, leading to the generation of reactive oxygen and nitrogen species, which contributes to tissue injury, genotoxicity and epigenetic alterations; and can lead to conditions of persistent inflammation and chronic oxidative stress. Asbestos fibres are also associated with activation of intracellular signalling pathways, resistance to apoptosis, and stimulation of cell proliferation (IARC, 2012). Of key importance are the surface chemistry and reactivity of the asbestos fibres, surface area, fibre dimensions and biopersistence (IARC, 2012). However, the relevance of these characteristics to asbestos exposure through the oral route has not been determined. At present, there is no conclusive evidence that ingestion of

any asbestos fibre type is associated with carcinogenic risk. It has been proposed that additional studies, both in vitro and in vivo, are needed to determine the role of specific physicochemical characteristics of multiple fibre types in adverse health effects after exposure from all routes to asbestos and related mineral fibres (Gwinn et al., 2011).

As discussed above, although there is general agreement that some types of asbestos are genotoxic in vitro, there is less agreement about the mutagenicity of asbestos fibres, particularly in vivo (Gwinn et al., 2011). Most genotoxicity studies with asbestos have been performed in vitro, and limited in vivo data are available to address this issue. A comprehensive review (Huang et al., 2011) suggests a role for mutagenesis in asbestos-induced neoplastic, but not non-neoplastic, diseases. A mode of action involving inflammation, cellular toxicity and oxidative stress may also be operative.

6 Summary of health effects

Occupational epidemiology studies and supporting animal studies indicate that the major route of human risk from asbestos exposure is inhalation. An extensive evidence base links inhalation exposure to the development of asbestosis, mesothelioma, and cancer of the lungs, larynx and ovary. Although it is feasible that aerosols generated through the use of humidifiers, showering and other domestic uses of water may be inhaled, the shape and size of fibres detected in water suggests that this exposure route is of low risk to human health. Some epidemiological studies have suggested that ingestion of some types of asbestos – for example, through drinking-water – may be linked to an increased risk of GI cancer. However, the current body of evidence from both human and animal studies, including consideration of its limitations, does not support a clear association at present (see section 4). In addition to these limitations, the positive association found in some studies is not reflected in a number of animal cancer bioassays, which do not show carcinogenicity of asbestos following ingestion (US EPA, 2018). The lack of any observed inflammatory lesions and of interstitial fibrosis in orally treated animals supports the low capability of fibres to penetrate the intestinal epithelium; no information is available to indicate whether the gastric environment allows the ingested fibres to maintain their shape, dimensions and surface reactivity, which determine persistence and hazardous features of fibres in the lung.

The database relating to the ingestion of all asbestos types is not as extensive as for the inhalation route and has mainly focused on the carcinogenic end-point. Systemic effects are not considered to be of major concern at present for either inhalation or ingestion, as the number of fibres penetrating either the lung or the GI tract is believed to be very low (ATSDR, 2001).

7 Practical considerations

7.1 Analytical methods and achievability

The method of choice for quantitative determination of asbestos in water has traditionally been transmission electron microscopy (TEM), with identification by energy-dispersive X-ray analysis and selected-area electron diffraction (TEM/SAED). Analysis by TEM/SAED is costly, and preliminary screening with TEM alone, which has a detection limit of below 0.1 MFL in water, is therefore often used (ATSDR, 2001). Scanning electron microscopy (SEM) is the preferred method in Italy, where more drinking-water analysis has been carried out for asbestos than other parts of Europe; SEM is considered more robust than TEM for lower concentrations of fibres.

Phase contrast microscopy (PCM) is a more accessible technique than TEM/SAED, from both technical and cost perspectives. However, PCM cannot differentiate between asbestos and non-asbestos fibres, and does not allow identification of fibres <5 µm in length and 0.2 µm in diameter (Perry, 2004). Li et al. (2019) have recently described the use of PCM and micro-Fourier-transform infrared spectroscopy (micro-FTIR) with scanning electron microscopy and energy-dispersive X-ray spectroscopy for analysing asbestos fibres in drinking-water. Quantitation limits for six types of asbestos fibre types (chrysotile, crocidolite, amosite, anthophyllite, tremolite and actinolite) ranged from 0.0039 to 0.0064 mg/L (information on fibre sizes detected was not reported, and therefore MFL cannot be determined). Analysis using FTIR and inductively coupled plasma optical emission spectroscopy has recently been applied to animal tissue samples to assess the migration of asbestos in mice following ingestion (Zheng et al., 2019). This type of analysis is in the development stage and not yet widely available.

7.2 Source control

Since the main source of asbestos in drinking-water is from the release of asbestos fibres from A/C pipes, efforts to minimize asbestos exposure through drinking-water should focus on materials in contact with drinking-water. In line with WHO's position that all types of asbestos should no longer be used, to most efficiently eliminate asbestos-related disease (WHO, 2006, 2014, 2018), sources of asbestos fibres in drinking-water, such as A/C pipes and storage containers, should not be newly installed, particularly since suitable alternative materials are available. The alternative materials also avoid the potential inhalation hazard to those working with and on A/C pipes.

Where existing A/C pipes are still in active use, as part of water safety plans, suppliers should map and record their location, assess their condition (including related to water aggressiveness) and determine the most appropriate risk reduction strategies. As a precautionary measure, plans should be developed to replace A/C pipes when they fail or deteriorate significantly. For water systems with existing A/C pipes, it is important to ensure that the water is not aggressive (i.e. dissolves the cement component), and provide pH and alkalinity or other adjustments (Schock & Buelow, 1981) to control corrosivity and prevent release of fibres. However, where pipes are already degraded, these actions will not prevent the release of asbestos fibres. It may also be of benefit to consider coating the interior of A/C pipes and storage containers with a structural lining such as epoxy resin or other CIPP (cured in place pipe) polymer coating before the A/C pipes deteriorate significantly (Mordak & Wheeler, 1988). These materials should be approved for use in contact with drinking-water. Where replacement or repair of pipes is required, it is essential that appropriate measures are taken to prevent any worker exposure to asbestos dust, including during transport and disposal.

A report from the Australian Asbestos Safety and Eradication Agency (ASEA, 2018) considered a number of approaches to dealing with A/C water mains. Several techniques are available that do not require removal of the A/C pipes; the circumstances will dictate the most suitable approach. As indicated above, protection of workers and the public from the generation of asbestos dust is a key requirement.

Rainwater harvesting from A/C roofing will most probably result in high numbers of asbestos fibres in the harvested water unless the A/C is sealed with appropriate paint or resin. Where rainwater is collected from A/C roofing, the collected water should be allowed to settle before

use. Similar to A/C pipes, efforts should be made to minimize degradation and release of fibres. This includes avoiding cutting and drilling of asbestos roofs, and avoiding use of high-pressure roof cleaning materials. Where the A/C roof is coated with a suitable paint, this should be maintained. If the A/C roof is to be replaced, the roof catchment area should ideally be replaced with asbestos-free material. Similar to A/C pipes, it is important that appropriate measures are taken to prevent worker and public exposure to asbestos dust. Ideally, re-roofing would be conducted by a licensed professional to avoid exposure of lay workers and contamination of the environment (enHealth, 2013).

7.3 Treatment methods and performance

Where source waters are contaminated with asbestos fibres, coagulation and filtration are very effective in removing both naturally occurring and anthropogenic asbestos fibres. Coagulation and filtration can easily remove more than 99% of fibres if operation is optimized, with a post-filter turbidity of <0.2 nephelometric turbidity units (Lawrence et al., 1975; Logsden, 1979).

Since the main cause of contamination of tap water with asbestos fibres is erosion and peeling of the inner wall of the A/C pipes (Saitoh et al., 1992), it is important to control erosion of the pipe (see section 7.2 for more information).

8 Conclusions

Although asbestos fibres are known human carcinogens by the inhalation route, the data on adverse effects following ingestion are less clear. The overall weight of evidence from epidemiological and animal studies, as described in section 6, does not support the hypothesis that oral exposure to asbestos in drinking-water is associated with an increased risk of developing cancer. Because there is no consistent, convincing evidence for adverse health effects from the ingestion of asbestos fibres in drinking-water, it is considered not appropriate or necessary to establish a guideline value for asbestos fibres in drinking-water at this time. Furthermore, as outlined in section 4, the epidemiological studies have a number of limitations that would preclude their use for deriving a guideline value. However, in view of the uncertainties and limitations of the data, it is appropriate to try to minimise the concentrations of asbestos fibres in drinking-water as far as practical.

The main source of asbestos in drinking-water is A/C materials that are in contact with drinking-water. A/C pipes were used extensively in the past, and are still used in situ for drinking-water distribution in many countries. Rainwater may be harvested from existing A/C roofing, which has been widely used because of its cost and durability. Although there is no consistent evidence for health effects from exposure to asbestos via drinking-water, there are a number of issues associated with A/C pipes and roofs, particularly with regard to maintenance, repairs and the addition of new materials in contact with drinking-water (e.g. connections or roof tiles), which may expose workers to inhaled asbestos fibres. Where A/C materials are used in such situations, degradation and release of fibres should be minimized. Section 7.2 includes information on minimizing levels of asbestos fibres in drinking-water as a result of the use of A/C materials. Further, new sources of asbestos fibres in drinking-water should not be introduced, such as installation of A/C pipes and storage containers.

In view of the limited data available on occurrence of asbestos in drinking-water, it would be useful to conduct investigative monitoring to obtain further information on the contribution of older A/C pipes to fibre numbers, types, size and shape in drinking-water.

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