Asbestos in Drinking-water

Background document for development of
WHO *Guidelines for Drinking-water Quality*

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Version for public review

Preface

To be completed by WHO Secretariat

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Abbreviations used in the text

A/C	Asbestos-cement
ATSDR	Agency for Toxic Substances and Disease Registry (USA)
CHO	Chinese hamster ovary
FT-IR	Fourier-transform infrared spectroscopy
GI	Gastrointestinal
LECR	Lifetime excess cancer risk
MFL	Million Fibres per Litre
PCM	Phase contrast microscopy
SHE	Syrian hamster embryo
SIR	Standardised incidence ratio
F-yr/mL	Total number of fibres in one year per mL of air

(More potential abbreviations)

CI	Confidence interval
NTU	Nephelometric turbidity units
TEM	Transmission electron microscopy
SAED	Selected-area electron diffraction
ROS	Reactive oxygen species

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1.0 EXECUTIVE SUMMARY

To be completed by WHO Secretariat

2.0 GENERAL DESCRIPTION

2.1 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, namely serpentine (i.e. chrysotile, characterised by generally curly fibres) and amphibole (i.e. amosite , crocidolite, anthophyllite, actinolite and tremolite), characterised by generally straight fibres) (Klein et al., 1993; IARC, 2012; WHO, 2014).

2.2 Physicochemical properties

Asbestos minerals are polyfilamentous bundles comprised of long, flexible fibres of small diameter (≤ 3 µm), which are easily separated (IARC, 2012). These fibres are thought of as chemically inert as 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 alkali. 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).

2.3 Organoleptic properties

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

2.4 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 the 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, such as roofing, 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 as to the extent of this use in different parts of the world.

Although world-wide production and consumption of asbestos peaked in the 1970s, asbestos minerals were regularly used in the preceding decades. Due to the longevity of product life and risk management strategies advising to keep products in place rather than to attempt to remove or replace, current exposure to the products remains a possibility. Since that time, due to human health concerns, some countries have introduced strict legislation to limit exposure, some have introduced a ban whilst others have intervened

less and continue to use asbestos to varying degrees (IARC, 2012). The total world-wide production of asbestos in 2019 was 1.10 million metric tonnes, with only a small number of countries accounting for this production (Russia; China; Brazil)[1].

2.5 Environmental fate

In general, asbestos fibres are considered to undergo degradation processes (although very slowly) and transport following release into the environment (US EPA, 2018). Fibres may undergo minor transformation with changes in length or through the leaching of minerals from the fibre surface but are generally non-volatile and insoluble in the environment. The fate of asbestos fibres released into the environment is considered to be dependent on the size and shape of the fibres. 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 if the asbestos fibres have a small aerodynamic diameter (i.e., between 0.1 – 1 µm) they 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 from the surface of the fibre, may occur at low pH (ATSDR, 2001; US EPA, 2018; Clark and Holt, 1960). In addition, at basic pH, for cases where the magnesium hydroxide layer on asbestos is incomplete (due to naturally occurring defects or chemical leaching, as described), the exposed silanol group becomes accessible and may undergo reactions with a variety of basic species. It is uncertain exactly how these interactions impact the surface properties of asbestos fibres.

3.0 ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

While 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 asbestos cement in factory environments (IPCS, 1986). However, conversion factors relevant to drinking water scenarios have been calculated (Millette et al., 1979). The mass to number ratio varies due to the differing size of the fibres; therefore, these conversion factors cannot be applied without knowing a good deal about the source of the asbestos. Millette et al. (1979) suggest that 106 asbestos fibres per litre of water from asbestos cement pipe is equivalent to 0.01 µg of asbestos per litre; however, this value is uncertain as fibre size varies between sources.

3.1 Water

Asbestos fibres are introduced into water from natural and anthropogenic sources and have been measured in both surface and ground waters (EPA, 2014). Dissolution of asbestos-containing minerals and ores is the principal natural source of asbestos fibres in water, with known anthropogenic sources including industrial effluents, atmospheric pollution, and corrosion of A/C pipes in water-distribution systems.

3.1.1 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 et al., 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, while 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

[1] https://www.statista.com/statistics/264923/world-mine-production-of-asbestos/

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 and Dillon, 1979). A survey carried out between 1977 and 1982 of asbestos levels in UK waters from 65 locations reported that most drinking water samples (n=82 of 144 total) had fibre concentrations between 'non-detectable' and 1.5 MFL, with 95% of fibres being < 2 µm in length (Conway and Lacey, 1982). The fibres found were predominantly chrysotile, but amphibole fibres were also found at concentrations up to 1 MFL. In the US, asbestos levels in drinking water were monitored from 2006-2011 as part of the national contaminant occurrence assessments conducted in support of the US EPA's third Six-Year Review of National Primary Drinking Water Regulations (NPDWR). The range of detected concentrations was between 0.10 and 6.8 MFL (5^{th} and 95^{th} percentile respectively). Concentrations ≥ the regulatory limit (maximum contaminant level) of 7 MFL was reported in systems serving 0.2% of the population however, no distinction could be made as to the source of asbestos present (US EPA, 2016). An earlier study showed that most of the population of the USA (approximately 92%) consumed drinking water containing asbestos in concentrations below 1 MFL (Millette et al., 1980). Based on studies conducted between 1973-1980 in the US, Millette et al. (1980; 1983) reported that in some areas asbestos fibre concentrations between 1 and 100 MFL were reached due to erosion of natural deposits, pollution and/or from the corrosion of A/C pipes or roofing materials. The authors stated that the distribution of fibre sizes in the water was dependent on the source of the fibres; the average length of chrysotile fibres found in an A/C distribution system was 4 µm, whilst the average fibre length of chrysotile fibres originating from natural erosion was 1 µm (Millette et al., 1980; 1983). Ma and Kang (2017) sampled drinking water in a number of homes in Korea (n=6) and Japan (n=9) for the determination of asbestos fibre concentrations. The authors reported average levels of 213.3 and 181.11 F/L in each location respectively, as the sum of chrysotile, amosite and crocidolite fibres.

3.1.2 A/C pipe contact with drinking water

Exfoliation of asbestos fibres from A/C pipes is related to the aggressiveness (including low pH and low hardness) of the water supply (Toft et al., 1984) and can be mediated by coating of distribution pipes. A study in the UK reached similar conclusions; failure of A/C pipes were associated with low pH and low alkalinity but also with age and whether the internal surface of the pipe was protected with coal tar, bitumen or epoxy resin (Mordak and Wheeler, 1988). They also concluded that although 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 at the time of the survey (Chatfield and Dillon, 1979). In the survey carried out by Conway and Lacey (1982) in locations using A/C pipes for distribution in the UK, levels of amphibole asbestos were increased over areas using non A/C distribution pipes, but was still considered as low (< 1 MFL). Samples taken following disturbance of deposits in A/C pipes were considerably higher (up to 58 MFL) (Conway and Lacey, 1982). Even higher levels of asbestos fibres (1,850 MFL), were recorded in association with the severe deterioration of A/C pipe containing chrysotile and crocidolite in Woodstock, New York (USA) in the late 1980s (Webber et al., 1989). In a more recent 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, when compared to six control areas. Saitoh et al. (1992) proposed that drinking water in two areas of Japan with asbestos fibres was 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 each area respectively, with crocidolite being 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 possessed the form of thick or sheaved fibres with lengths ranging from ca. 5 to 10μm. Their shapes were very different from those of asbestos fibres found in the atmosphere which were short (ca. 1μm in length) and needle-like. More recently, Fiorenzuolo et al. (2013) evaluated the presence of asbestos fibres in drinking-water in eleven towns in the Marche region of Italy. The area is located near a former asbestos factory and utilises asbestos-cement pipes in the distribution of drinking-water. The authors reported that, in the few samples that detected asbestos, 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 < 5μm in length, therefore with a lower length, and generally with a larger diameter, than the ones causing fibrosis and other adverse effects in the lung after inhalation (ATSDR, 2001). In US water supplies, Millette et al. (1980, 1983) determined average length and width of chrysotile fibres of 1.4 and 0.04 μm respectively, with an aspect ratio generally >10:1. The authors noted however that fibre size distribution was dependant on the source, with longer fibres being released from A/C pipes when compared with those collected from natural erosion of rock. Ma and Kang (2017) reported measured values as the sum of chrysotile, amosite, and crocidolite fibres, with the majority being between 5 and 10 μm.

3.2 Food

The asbestos fibre content of solid foodstuffs has not been well studied because of the lack of a simple, reliable analytical method. In the 1980s, it was suggested that foods that contain soil particles, dust, or dirt probably contained asbestos fibres; crude estimates from that time suggested that the intake of asbestos in food may have been significant in comparison 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 and Pontefract, 1971). More recent publicly available data were not identified on asbestos fibre concentrations in food or beverages.

3.3 Air

There is an abundance of literature relating to asbestos exposure via inhalation (for example, ATSDR, 2001). That discussed below focuses on air exposure data that is relevant to the discussion regarding drinking water.

The primary exposure route for non-smoking individuals is from air. For non-occupational settings, inhalation of outdoor air is the primary source of exposure, although indoor air continues to make a limited contribution to total airborne exposure (IARC, 2012). Chrysotile is most frequently detected, with lower concentrations (typically 100-fold) reported in rural locations (10-9 MFL) when compared to urban sites or close proximity to industrial sources (10-7 MFL) (US EPA, 2018; ATSDR, 2001; IARC, 2012).

Airborne asbestos may be released from tap water in the home. Mean airborne asbestos concentrations (type not specified, from A/C pipes) were significantly higher (1.7 ng/m3) in three homes with water containing elevated concentrations (> 10 billion fibres/L) of asbestos than in three control homes (0.31 ng/m3) in a study completed in the late 1980s; however, the difference in concentration was due primarily to increased numbers of short fibres (<1 μm), which, from an inhalation perspective, the authors considered to pose little

health risk. Moreover, the fibre concentrations found in this limited study were within the range of those measured in indoor air in other investigations (Webber et al., 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 et al., 1979). Roccaro and 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 levels of 24687 f/L and air samples 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 accord to WHO (1997) employing 200 counting fields per filter. The authors reported that between 0.04 to 0.07% of fibres were transferred to air through use of the humidifier, which was noted as being comparable to those reported by Hardy et al. (1992) who determined release of asbestos-like fibres from a room humidifier at levels of 0.03 to 4.7% of that present in the charging water. For the domestic shower, Roccaro and Vagliasindi reported higher levels of transfer, when compared to the humidifier, of between 4.3 to 10.8% of fibres from tap water containing natural levels of 8229 f/L. Although the higher levels may have been due to increased water usage during showering the authors also considered that the larger diameter of the released droplet from showers when compared to 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 and 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 x 10-4 the authors cautioned that in interpreting these, the complex nature of such a risk assessment should be noted and, in particular, in modelling the transfer of asbestos from air which is dependent on a large number of factors.

While there is some limited evidence with regard to the contribution of exposure through showering with water containing asbestiform fibres, to inhalation of fibres, extrapolation to assess this risk more generally is not possible due to limited data. Although asbestos fibres are very easily dispersed asbestos is a non-volatile substance. Care should be taken when interpreting exposure studies as those 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 3.1.) are predominantly those whose shape is considered to be of low risk of adverse health effects via inhalation. For these reasons showering is unlikely to contribute significantly to exposure of asbestos fibres that are harmful via inhalation.

3.4 Bioaccumulation

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

3.5 Estimated total exposure and relative contribution of drinking-water

The 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 between 0.002 – 0.4 F-yr/mL (or 600 – 114000 fibres/day, based on an adult inhalation rate of 20 m^3/day (ATSDR, 2001)) while the Agency estimated for an adult drinking 2L/day, intake via drinking water to be between 2 – 200 million fibres/day. However, it is not correct to compare, nor estimate the aggregate exposure from the two routes of exposures, since, as described above, the types of airborne fibres and those present in water are different in shape, length and diameter and as a consequence 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

tract through mucocilliary clearance (Gross et al., 1975). As such, the gastrointestinal tract is a major recipient of both inhaled and ingested asbestos fibres (IARC, 2012; Rowe, 1983). In a comparison of the relative source contributions to ingested asbestos in humans, Rowe (1983) states that exposure through the diet and air are of more significance than the contribution from drinking water. The authors tentatively estimated that, for the US, annual intake of ingested asbestos in drinking water could range from 9×10^5 to 4×10^{11} fibres, whilst dietary and airborne sources may deliver 1.2×10^9 to 9×10^{12} and 2.4×10^9 to 1.4×10^{14} asbestos fibres to the gut yearly, respectively.

In their evaluation, IARC (2012) highlighted that small children may have a higher exposure to asbestos through drinking water due to their intake to body weight ratio being higher than in adults. However, it should be noted that this does not necessarily translate to having a greater risk of adverse health effects, particularly since asbestos-related toxicity, as reflected by the significant inhalation database, is long-term in nature. 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).

4.0 TOXICOKINETICS AND METABOLISM IN HUMANS AND LABORATORY ANIMALS

The most likely routes for human exposure to asbestos are through inhalation and ingestion, with uptake following dermal exposure considered to be unlikely (ATSDR, 2001). Information on the toxicokinetics and metabolism of asbestos following inhalation has been well reported and is summarised elsewhere by ATSDR (ATSDR, 2001; Kim et al., 2013; US EPA, 2018). Information of the toxicokinetics and metabolism of asbestos following ingestion, which is of direct relevance to drinking water, is summarised below. As previously noted, the gastrointestinal tract is the major recipient of both inhaled and ingested asbestos fibres due to mucocilliary clearance (Gross et al., 1974; IARC, 2012; Rowe, 1983).

4.1 Absorption

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

4.2 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 the distribution 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, to the lungs, pleura and spleen. 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). The authors reported that their findings were indicative of asbestos fibres entering the stomach and intestines and becoming absorbed into the gastrointestinal mucosa, with some entering the blood. After 60 days following exposure, accumulation of asbestos fibres was noted to occur in the liver, but not in other organs. No indication of the level of absorption from gastrointestinal mucosa was provided by the study authors.

4.3 Metabolism

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).

4.4 Excretion

Ingested asbestos fibres (no further details provided) are mainly excreted in faeces within 48 hr of a single oral dose in rats. Small numbers of fibres may also be excreted in urine and 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 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 (no further information of fibre size given).

5.0 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 summarised by a number of authoritative bodies. In brief, ATSDR (2001) and WHO (2014) concluded that the health hazards associated with the inhalation of asbestos in the occupational environment have long been recognised and include asbestosis, bronchial carcinoma, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx and possible cancer of the gastrointestinal tract. In the evaluation by IARC, it was concluded that exposure to all forms of asbestos by inhalation causes mesothelioma and cancer of the lung, larynx and ovary. 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 multiple species. A complex interaction between fibres and cells *in vivo* has been described involving both direct and indirect mechanisms interacting at multiple stages of cancer development. Certain physicochemical properties of 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).

Information of the toxicity of asbestos fibres following ingestion, which is of direct relevance to exposure via drinking-water, is summarised below. It should be noted that the ability of asbestos fibres ingested in drinking-water to migrate through the walls of the gastrointestinal tract in sufficient numbers to cause adverse local or systemic effects remains largely unknown and is the subject of debate. Indeed, at the present time, 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).

5.1 Acute exposure

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

5.2 Short-term exposure

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

5.3 Long-term exposure

5.3.1 Systemic effects

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

5.3.2 Neurological effects

No studies could be identified regarding neurological effects in humans following ingestion of any asbestos fibre type.

5.3.3 Reproductive and developmental effects

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

5.3.4 Immunological effects

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

5.3.5 Genotoxicity and carcinogenicity

5.3.5.1 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 mechanisms 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.

5.3.5.2 Carcinogenicity

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 the development of the non-carcinogenic endpoints of asbestosis (fibrotic lung disease), pleural plaques and thickening. In addition, inhalation exposure to asbestos is linked to cancer of the lung, pleura and peritoneum. IARC has reported a causal relationship between inhalation exposure to all asbestos types and

cancers of the larynx and ovary and a positive association with cancer of the pharynx, stomach and colorectum (IARC, 2012). It is considered that a large proportion of respired asbestos fibres are removed via mucociliary transport to the gastrointestinal tract, meaning that the GI tract is also directly exposed to fibres, which may increase the risk of cancers developing. However, evidence to show increased incidence of cancers of the gastrointestinal tract was not strong (IARC, 2012).

A number of ecological correlation studies (which do not allow identification of causality, but only provide indications of possible associations) were conducted in the period 1960-1980 in the United States and Canada. These suggested an association between asbestos fibres in drinking water supplies (both for anthropogenic contamination and for natural pollution of the springs) with the rates of stomach cancer induced in the population served by those waters. However, exposure levels are not defined (or at least reported) and any increases were small and confounded by lifestyle factors (such as cigarette smoking, diet, etc.). In addition, no consistent increases were noted either within or between studies which may have stemmed from limitations including; statistical power, study design, exposure levels and duration, population size and mobility, differences in asbestos fibre types, or analytical methods used to measure exposure levels.

Between 1980 and 2005, a number of studies were also published in the USA, with concentrations of asbestos fibres above one 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 gastrointestinal cancers (Kanarek et al., 1980; Conforti et al., 1981). The study design and data analysis employed by the study authors has been criticised as potential confounders such as diet, smoking, and occupation could not be adequately controlled (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 (± SD) exposures to chrysotile fibre concentrations in drinking water of 206.5 (±162.2) $x10^4$ and 7.3 (±12.4) x 10^4 fibres/L respectively. Fibre lengths were found to be similar with 99.9 and 99.4% of fibres being < 5 µm and 85.8 and 82.5% being < 1µm in high and low areas respectively. 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 due to 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 the 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, NY, USA where levels of asbestos (chrysotile and crocidolite) ranging between 3.2 – 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 gastrointestinal 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, as compared with the San Francisco Bay Area in California, was attributable to the shorter fibre lengths in the state of Washington as compared with those in California.

A comprehensive review and evaluation of thirteen epidemiology studies of ingested asbestos (chrysotile or amosite) conducted in 5 areas of the USA and Canada was reported by Marsh (1983) with a view to developing water quality standards. The authors reported that eight studies described male or female associations between ingestion of asbestos in drinking water and multiple cancer sites. However, no individual study or combination of studies was considered adequately strong enough for use in setting risk-based standards. Cantor (1997) also carried out a systematic review of epidemiology studies investigating the potential relationship between asbestiform fibres (and other contaminants) in drinking-water and cancer incidence in humans. The author concluded that 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, for which the supply of drinking water was from rainwater stored in asbestos cement structures (Andersen et al., 1993). The drinking water concentrations of the asbestos fibres were reported to range from 1.7 to 71 MFL (with peaks equal to or > 1 billion / L) without any characterization of the fibres by size, shape and mineralogical indications. The overall standardised incidence ratio (SIR) for stomach cancer in the cohort was 1.6 (95% CI: 1.0-2.3). For the subcohort with verified exposure ('certainly exposed' based on work histories) to asbestos the SIR was 2.5 (95% CI: 0.9-5.5); for subjects exposed to asbestos and followed for at least 20 years the SIR was 1.7 (95% CI: 1.1-2.7). The same study, with regards to colon cancer, showed a standardized incidence ratio of 2.5 (95% CI: 0.9-2.2) for the entire cohort, and 0.8 (95% CI: 0.1-2.9) in the subcohort with ascertained exposure to asbestos, and 1.6 (95% CI: 1.0-2.5) among those exposed followed for at least 20 years. Possible confounding factors (diet, smoking, previous occupational exposure) were not controlled.

In a similar study, Kjærheim et al. (2005) assessed the incidence of stomach cancer in Norwegian light-house keepers (n=726) exposed to mixed fibre asbestos in drinking water (run off from roof tiles that were significantly deteriorated, comprising 15% asbestos). Fibre content ranged between 1800 MFL to 71,000 MFL, with 92% of fibres being chrysotile with a smaller percentage of amphibole fibres being 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 to 2002. Due to a lack of complete work histories, the authors divided the cohort into three subgroups of 'certainly exposed' (n = 107), 'possibly exposed' (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 'certainly exposed' followed up for ≥ 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, there are several limitations in the study that do not allow causality to 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, standardisation 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 are generally based on low numbers of cases, leading to higher uncertainty; and the database of studies with which to compare findings is poor. A conclusion based on causality cannot be derived from this study and it is unclear whether the high levels of exposure documented are relevant to the general population. The authors note that the levels measured in the study are at the very upper range of those reported by Millette et al. (1983) in water supplies from asbestos cement pipes (0.01 MFL to 1,000,000 MFL). This highlights a particular need to better understand the biological plausibility of the study findings which would help better

interpretation of the findings reported by Kjærheim, and evidence from other oral intake studies for asbestos.

In a further review of evidence from epidemiology, in vivo and in vitro publications, Bunderson-Schelvan et al. (2011) assessed extrapulmonary effects of asbestos exposure, including gastrointestinal effects. The authors state that environmental exposure to asbestos is most likely due to chrysotile fibres released from drinking water pipes. The review states that the data represented in the reviewed publications show 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 for strong conclusions to be made.

IARC's most recent evaluation (IARC, 2012) included a summary of the evidence of an association between exposure to asbestos and stomach and colorectal cancers. A positive association between exposure to asbestos and stomach and colorectum cancer was reported by their Working Group; the conclusion was based on long-term, high level occupational inhalation cohort studies. As noted earlier, however, the evidence to show increased incidence of cancers of the gastrointestinal tract was not strong (IARC, 2012. 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 gastrointestinal cancers was also evaluated by Kim et al. (2013). The study 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 these inconsistencies could be attributed to varying amounts of the asbestos released from water pipelines at various times, the asbestos composition in the water, and methodologic differences. In addition, Kim et al. (2013) noted that the evidence for stomach cancer incidence was much stronger for occupational inhalation exposure compared to drinking water exposure.

Di Ciaula and Gennaro (2016) reviewed the available evidence examining a potential relationship between ingestion of asbestos fibres and the risk of gastrointestinal cancers. However, due to the lack of robust epidemiological studies concerning asbestos ingestion, the authors concluded it was not possible to derive a risk threshold in non-occupational cohorts, principally due to methodological limitations. In their latest evaluation, the US EPA also concludes that based on currently available evidence there is no clear association for drinking water asbestos exposure and cancer (US EPA, 2018).

6.0 EFFECTS ON ANIMALS AND *IN VITRO* TEST SYSTEMS

6.1 Acute exposure

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

6.2 Short-term exposure

Rats administered three doses of crocidolite by oral gavage at 33 mg/kg bw/day (numbers of fibres and size range not known) showed increased numbers of aberrant crypt foci, considered to be possible precursors of 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 et al. 1993). However, as no excess of non-neoplastic lesions in the gastrointestinal epithelium have 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 gastrointestinal system (ATSDR, 2001).

6.3 Long-term exposure

6.3.1 Systemic effects

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

6.3.2 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 at 500 and 830 mg/kg bw/day respectively (numbers of fibres and size range not known). Acute exposure of rats and mice to crocidolite at doses of 160 and 50 mg/kg bw/day respectively or chrysotile at doses of 70 and 100 mg/kg bw/day respectively (numbers of fibres and size range not known) was not associated with clinical signs of neurotoxicity (NTP 1983, 1985, 1988, 1990a, 1990b, 1990c ; Corpet et al. 1993; ATSDR, 2001).

6.3.3 Reproductive and developmental effects

Rats and hamsters exposed to chrysotile, amosite, crocidolite or tremolite individually at doses of 500 or 830 mg/kg bw/day (numbers of fibres and size range not known) respectively 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; 1990b; 1990c).

Administration of between 0.3 and 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 and Maurer, 1977).

6.3.4 Immunological effects

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

6.3.5 Genotoxicity and carcinogenicity

6.3.5.1 Genotoxicity

No *in vivo* studies examining the genotoxicity of any asbestos fibre type using a standardised protocol were identified. In non-standard studies, a single oral (gavage) administration of 50 mg/kg bw amphibole or crocidolite asbestos to rats (numbers of fibres and size range not known) did not increase the frequency of micronuclei formation or sister chromatid exchange in bone marrow samples taken 24 h following exposure. A single oral (gavage) dose of chrysotile of 100 or 500 mg/kg bw (numbers of fibres and size range not known) did not increase the number of chromosomal aberrations in the bone marrow of Rhesus

monkeys. In Swiss albino mice, oral (gavage) or i.p. administration of chrysotile at doses between 0.4 and 400 mg/kg bw (numbers of fibres and size range not known) did not increase the frequency of micronuclei formation in bone marrow (Lavappa et al., 1975).

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 *S. 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. Crocidolite is reported to be a more potent mutagen than chrysotile, with asbestos toxicity showing cell-line specificity in human and animal cells that may be due to differential phagocytic activity, with those with high activity showing greater susceptibility (ATSDR, 2001, IARC, 2012).

Chromosomal aberrations in Chinese hamster ovary (CHO) and Syrian hamster embryo (SHE) cells following exposure to asbestos (amosite; anthophyllite; chrysotile; crocidolite) fibres have been well reported, with aberrations including aneuploidy (usually polyploidy), fragmentation, breaks, rearrangements, gaps, dicentrics, inversions and rings. Similar aberrations have been shown in rat and human mesothelial cells, lymphocyte and amniotic fluid cells, but not in fibroblasts or promyelocytic leukaemia cells. Clastogenic effects may occur due to 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).

In summary, in vivo studies in humans (section 5.3.5) and animals indicate that exposure to the asbestos fibre types tested to date is associated with chromosomal damage (aberrations). In vitro studies with bacterial cells indicate clastogenicity, however, the findings from in vivo and in vitro gene mutation studies are inconclusive.

6.3.5.2 Carcinogenicity

Although the carcinogenicity of inhaled asbestos in laboratory animals is well established, there is no conclusive evidence that ingested asbestos is carcinogenic (ATSDR, 2001; DHSS, 1987; IARC, 2012; Toft et al., 1984). 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, 1983b) 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 gastrointestinal tract in male Fischer 344 rats fed 1% intermediate-range chrysotile was significantly increased when compared to pooled controls from lifetime asbestos (chrysotile) feeding studies in the same laboratory, the increase was not statistically significant when compared to concurrent controls and was limited to one sex.

6.5 Mode of action

The mechanistic basis for the carcinogenicity of inhaled asbestos has been well studied and reported and considered to result from direct and indirect mechanisms interacting at multiple stages of carcinogenesis. 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 and, at present, there is no conclusive evidence that ingestion of any asbestos fibre type is associated with carcinogenic risk. Additional studies, both in vitro and in vivo, are needed to determine the role of specific physicochemical characteristics from multiple fibre types in adverse health effects after exposure to asbestos and related mineral fibres (Gwinn et al., 2011).

Although there is general agreement that some types of asbestos are genotoxic in vitro, either directly (i.e., fibre interactions with the spindle apparatus) or indirectly (i.e., ROS production), there is less agreement on the mutagenicity of asbestos fibres, particularly in vivo (Gwinn et al., 2011). Most genotoxicity studies with asbestos have been performed in vitro, and therefore 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, and acknowledged that MOA also involving inflammation, cellular toxicity, and oxidative stress may also be operative.

7.0 SUMMARY OF HEALTH EFFECTS

Occupational epidemiology studies and supporting animal studies indicate that the major route of human risk from asbestos exposure is through inhalation. An extensive evidence base exists that links inhalation exposure to the development of asbestosis, lung cancer, mesothelioma and cancer of the larynx and ovary. Some epidemiology studies have suggested that ingestion of some types of asbestos, for example through drinking water, may be linked to an increased risk of gastrointestinal cancer. However, the current body of evidence, including consideration of its limitations, does not support a clear association at the present time (see section 5.0). In addition to these limitations, the positive association found in some studies are not reflected in a number of animal cancer bioassays which do not show the carcinogenesis of asbestos following ingestion (US EPA, 2018). The lack of any observed inflammatory lesions and of interstitial fibrosis in orally treated animals is supportive of the low capability of fibres to penetrate the intestinal epithelium; no information is available to indicate whether or not the gastric environment allows the ingested fibres to maintain their shape, dimensions, and surface reactivity that determines in the lung the persistency and hazardous features.

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 endpoint. Systemic effects are not considered to be of major concern at present for either route of exposure as the number of fibres penetrating either the lung or gastrointestinal tract is believed to be very low (ATSDR, 2001).

8.0 PRACTICAL CONSIDERATIONS

8.1 Analytical methods and achievability

The method of choice for the quantitative determination of asbestos in water is 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).
Phase Contrast Microscopy (PCM) is a more accessible technique, both from a technical and cost perspective than TEM/SAED. However, PCM cannot differentiate between asbestos and non-asbestos fibres, and does not distinguish fibres < 5 µm in length and 0.2 µm in diameter (Perry et al., 2004). Li et al. (2019) have recently described the use of PCM and micro-Fourier-transform infra-red 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 – 0.0064 mg/L (information on fibre sizes detected not reported). have FTIR and inductively coupled plasma optical emission spectroscopy analysis has recently been applied to animal tissue samples to assess the migration of asbestos in mice following ingestion (Zheng et al., 2019). It should be noted that this type of analysis is in the development stage and, as such, not widely available.

8.2 Source control

Since the main source of asbestos in drinking-water is from the release of asbestos fibres from A/C pipes, efforts to minimise asbestos exposure through drinking-water should focus on materials in contact with drinking-water. It may be prudent to not install new sources of asbestos fibres in drinking-water such as A/C pipes and storage containers., particularly since there are suitable alternative materials. 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 however, suppliers should map and record the location of such pipes as part of mapping the distribution system under water safety plans, determine the conditions of the pipes and as a precautionary measure develop plans to replace these when they fail or as they deteriorate. For water systems with existing A/C pipes it is important to ensure that the water is not aggressive and provide pH and alkalinity adjustments to control corrosivity and prevent release of fibres but it should be noted that where pipes are already degraded this will not prevent the release of asbestos fibres. It is important that where replacement or repairs of pipes is required, appropriate measures are undertaken to prevent worker exposure to asbestos dust.

A report from the Australian Asbestos Safety and Eradication Agency (2018) considers 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 but the circumstances will dictate the most suitable approach. However, as indicated above, protection of workers and the public from the generation of asbestos dust is a key requirement.

Where rainwater is collected from A/C roofing, the collected water should be allowed to settle before use. Similar to A/C pipes, effort should be put in place to minimize degradation and release of fibres. This includes avoiding cutting and drilling of asbestos roofs and 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 undertaken 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 (Commonwealth of Australia, 2013).

8.4 Treatment methods and performance

Where source waters are contaminated with asbestos fibres, coagulation and filtration are very good at removing both naturally occurring and anthropogenic asbestos fibres. Coagulation and filtration can easily remove in excess of 99% of fibres if operation is optimised with a post filter turbidity of < 0.2 NTU (Lawrence et al., 1975; Logsden 1979).

Since the main cause of contamination of asbestos fibres in tap water is erosion and peeling of the inner wall of the A/C pipes (Saitoh, 1992), it is important to control erosion of the pipe. (see section 8.3 for more information).

9.0 CONCLUSIONS

Although asbestos fibres are known human carcinogens by the inhalation route, the data on ingestion are unclear and the overall weight of evidence does not support the hypothesis that oral exposure in drinking-water is associated with an increased cancer risk. In addition, extensive feeding studies in laboratory animals have not shown increases in tumours of the gastrointestinal tract. 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.

The main source of asbestos in drinking-water is through the use of A/C materials in contact with drinking-water. A/C pipes were used extensively in the past and there are many countries where A/C pipes are still used in-situ for drinking water distribution. 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 that result 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) where workers may be exposed to inhaled asbestos fibres. Where A/C materials are used in such situations, there is a need to minimize degradation and release of fibres. Section 8.3 includes information to minimize levels of asbestos fibres in drinking-water as a result of the use of A/C materials, including adjusting water conditions to reduce corrosivity to the cement matrix and replacing pipes when they fail or deteriorate. Further, it may be prudent to not install new sources of asbestos fibres in drinking-water such as 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 up to date information on the contribution of older A/C pipes to fibre numbers, types and shape in drinking-water.

10.0 APPENDICES

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