

# Asbestos in drinking water

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## 1 Summary

Health Canada in 1989 provided Guidelines for Asbestos in Canadian Drinking Water. They stated that “there has been no consistent evidence of an association between cancer mortality or incidence and ingestion of asbestos in drinking water”. In addition, “There is also no conclusive evidence from studies in animals that ingested asbestos is carcinogenic.”

Toft & Meek (1983) from the Monitoring and Criteria Division, Bureau of Chemical Hazards, Environmental Health Directorate, Department of National Health and Welfare, Ottawa, Ontario, Canada, concluded that “It is concluded that the risk to health associated with the ingestion of asbestos, at the levels found in municipal drinking water supplies, is so small that it cannot be detected by currently available epidemiologic techniques.”

The WHO (2020) is in the process of updating their assessment of asbestos in drinking water. The WHO states that “However, the current body of evidence, including consideration of its limitations, does not support a clear association at the present time” In addition, “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 US EPA as summarized by the Agency For Toxic Substances And Disease Registry (ATSDR) has established a maximum contaminant level (MCL) for asbestos in drinking water of 7 MFL (million fibers per liter > 10 µm in length). Fubers less than 10 µmin length are not considered.

McRae (1988) reviewed asbestos in drinking water from a UK perspective and concluded that: “It would thus seem highly unlikely that the asbestos- cement pipe distribution system makes any biologically significant contribution to the asbestos content of water passing through it. It follows therefore, in view of the conclusions reached above concerning the likely lack of association between asbestos in drinking water and an excess risk of cancer, that it is highly improbable that asbestos release from asbestos-cement pipe is relevant to to the development of cancer.”

## **2 Health Canada 1989 Guidelines for Canadian Drinking Water Quality Guideline Technical Document – Asbestos**

Chrysotile occurs naturally in numerous locations worldwide. In Canada, the drinking water was analysed in 71 locations across Canada<sup>1</sup>. The results showed that “there was little contamination by amphiboles. Using transmission electron microscopy (TEM) with identification by energy-dispersive X-ray analysis and selected area electron diffraction, chrysotile concentrations ranged from not detectable (<0.1 million fibres/L) to 2000 million fibres/L. In general, median fibre lengths were between 0.5 and 0.8 mm. Based on the results of this survey, which encompassed the water supplies of about 55% of the Canadian population, it was estimated that 5% of the population receives water with chrysotile concentrations higher than 10 million fibres/L and that 0.6% receives water containing more than 100 million fibres/L.”

The Canadian report further summarizes the findings from both epidemiology studies from the United states and Canada and extensive toxicology studies performed by the U.S. National Toxicology Program. They stated that:

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<sup>1</sup> Health Canada 1989 Guidelines for Canadian Drinking Water Quality Guideline Technical Document – Asbestos

“In ecological epidemiological studies of populations in Duluth,<sup>2</sup>10–12 Canadian cities in areas of asbestos deposits,<sup>3</sup>13,14 Connecticut,<sup>4</sup>15,16 Florida<sup>5</sup>17 and Utah<sup>6</sup>,18 there has been no consistent evidence of an association between cancer mortality or incidence and ingestion of asbestos in drinking water. Although there was evidence of an association in an ecological study in the San Francisco Bay area,<sup>7</sup>19,20 reanalysis of the data taking potential confounders into account has undermined the significance of these results.<sup>8</sup>21,22 Moreover, in an analytical epidemiological (case-control) study that was inherently more sensitive than the ecological studies mentioned above, there was no consistent evidence of a cancer risk due to ingestion of asbestos in drinking water in Puget Sound, where levels ranged up to 200 million fibres/L.<sup>9</sup>23 In this study, the minimum risk that could be detected at the 5% significance level with 80% probability was under 2 for each sex for all study sites combined—digestive system, respiratory system, colon and lung.”

“There is also no conclusive evidence from studies in animals that ingested asbestos is carcinogenic. The most extensive animal studies conducted to date have been those of the U.S. National Toxicology

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<sup>2</sup> 10. Levy, B.S., Sigurdson, E., Mandel, J., Laudon, E. and Pearson, J. Investigating possible effects of asbestos on city water: surveillance of gastrointestinal cancer in Duluth, Minnesota. *Am. J. Epidemiol.*, 103: 362 (1976).

11. Mason, T.J., McKay, E.W. and Miller, R.W. Asbestos-like fibres in Duluth water supply: relation to cancer mortality. *J. Am. Med. Assoc.*, 228: 1019 (1974).

12. Sigurdson, E.E., Levy, B.S., Mandel, J., McHugh, R., Michienzi, L.J., Jagger, H. and Pearson, J. Cancer morbidity investigations: lessons for the Duluth study of possible effects of asbestos in drinking water. *Environ. Res.*, 25: 50 (1981).

<sup>3</sup> 13. Wigle, D.T. Cancer mortality in relation to asbestos in municipal water supplies. *Arch. Environ. Health*, 32: 185 (1977).

14. Toft, P., Wigle, D.T., Meranger, J.C. and Mao, Y. Asbestos and drinking water in Canada. In: *Water supply and health*. H. van Lelyveld and B.C.J. Zoeteman (eds.). Elsevier Scientific Publishing Co., Amsterdam. p. 77 (1981).

<sup>4</sup> 15. Harrington, J.M., Craun, G.F., Meigs, J.W., Landrigan, P.J., Flannery, J.T. and Woodhull, R.S. An investigation of the use of asbestos cement pipe for public water supply and the incidence of gastrointestinal cancer in Connecticut 1935–1973. *Am. J. Epidemiol.*, 107: 96 (1978).

16. Meigs, J.W., Walter, S.D., Heston, J.F., Millette, J.R., Craun, G.F., Woodhull, R.S. and Flannery, J.T. Asbestos cement pipe and cancer in Connecticut 1955–1974. *J. Environ. Health*, 42: 187 (1980).

<sup>5</sup> 17. Millette, J.R., Craun, G.F., Stober, J.A., Draemer, D.F., Tousignant, H.G., Hildago, E., Duboise, R.C. and Benedict, J. Epidemiology study of the use of asbestos-cement pipe for the distribution of drinking water in Escambia County, Florida. *Environ. Health Perspect.*, 53: 91 (1983).

<sup>6</sup> 18. Sadler, T.D., Rom, W.N., Lyon, L.T. and Mason, J.O. The use of asbestos-cement pipe for public water supply and the incidence of cancer in selected communities in Utah, 1967–1976. Graduate thesis, University of Utah, Salt Lake City, UT (1981).

<sup>7</sup> 19. Kanarek, M.S., Conforti, P.M., Jackson, L.A., Cooper, R.C. and Murchio, J.C. Asbestos in drinking water and cancer incidence in the San Francisco Bay area. *Am. J. Epidemiol.*, 112: 54 (1980).

20. Conforti, P.M., Kanarek, M.S., Jackson, L.A., Cooper, R.C. and Murchio, J.C. Asbestos in drinking water and cancer in the San Francisco Bay area: 1969–1974 incidence. *J. Chron. Dis.*, 34: 211 (1981).

<sup>8</sup> 21. Conforti, P.M. Effect of population density on the results of the study of water supplies in five California counties. *Environ. Health Perspect.*, 53: 69 (1983).

22. Tarter, M.E., Cooper, R.C. and Freeman, W.R. A graphical analysis of the interrelationships between waterborne asbestos, digestive system cancer and population density. *Environ. Health Perspect.*, 53: 79 (1983).

<sup>9</sup> 23. Polissar, L., Severson, R.K. and Boatman, E.S. Cancer risk from asbestos in drinking water: summary of a case-control study in Western Washington. *Environ. Health Perspect.*, 53: 57 (1983).

Program involving treatment groups of 250 animals of each sex.<sup>10</sup> No treatment-related increases in tumour incidence were observed in Syrian golden hamsters fed 1% amosite or short-range or intermediate-range chrysotile in the diet over their lifetime. Similarly, no treatment-related effects were observed in Fischer-344 rats fed 1% tremolite or amosite in the diet over their lifetime. The incidence of benign epithelial neoplasms in the gastrointestinal tract in male Fischer-344 rats fed 1% intermediate-range chrysotile (65% longer than 10 mm) was significantly increased in comparison with pooled controls from contemporary lifetime asbestos feeding studies in the same laboratory. However, the increase was not statistically significant in comparison with concurrent controls and was limited to one sex. Moreover, no increase in tumour incidence was observed in Fischer-344 rats ingesting short-range chrysotile (98% shorter than 10 mm) that was composed of fibre sizes more similar to those found in drinking water.”

### **3 WHO 2020 Asbestos in Drinking-water Background document for development of WHO Guidelines for Drinking-water Quality (14 December 2020 Version for public review)**

The WHO in summarizing the health effects from asbestos in drinking water stated that “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 WHO document has summarized the current literature on asbestos in drinking water and the issue of A/C pipe contact with drinking water as stated below.

#### **“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

<sup>10</sup> McConnell, E.E., Shefner, A.M., Rust, J.H. and Moore, J.A. Chronic effects of dietary exposure to amosite and chrysotile asbestos in Syrian golden hamsters. *Environ. Health Perspect.*, 53: 11 (1983).

McConnell, E.E., Rutter, H.A., Ulland, B.M. and Moore, J.A. Chronic effects of dietary exposure to amosite asbestos and tremolite in F344 rats. *Environ. Health Perspect.*, 53: 27 (1983).

National Toxicology Program (NTP). Toxicology and carcinogenesis studies of chrysotile asbestos in F344 rats. National Institutes of Health Publication No. 86-2551, Technical Report No. 295, Public Health Service, U.S. Department of Health and Human Services (1985).

lengths were in the range 0.5–0.8  $\mu\text{m}$ . 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 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  $\mu\text{m}$  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 (5th and 95th percentile respectively). Concentrations  $\geq$  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  $\mu\text{m}$ , whilst the average fibre length of chrysotile fibres originating from natural erosion was 1  $\mu\text{m}$  (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  $1.8 \times 10^{-3}$  and  $2.7 \times 10^{-3}$  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.”

#### **4 ATSDR 2018: AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY CASE STUDIES IN ENVIRONMENTAL MEDICINE (CSEM) Asbestos Toxicity**

As stated by the ATSDR, the US EPA has set the following maximum contaminant level (MCL) for asbestos in drinking water. It should be noted that this limit is for “million fibers per liter > 10 µm in length”. Fibers less than 10 µm in length are not included.

“EPA has established a maximum contaminant level (MCL) for asbestos in drinking water of 7 MFL (million fibers per liter > 10 µm in length) in drinking water [EPA 2011]. Asbestos in drinking water comes from two main sources:

1. Decay of water mains constructed of asbestos-containing cement, and
2. Erosions of naturally occurring asbestos deposits into watersheds [EPA 2012a].”

**5 MacRAE (1988) from the Department of Medical Statistics, Charing Cross and Westminster Medical School, London reviewed Asbestos in drinking water and cancer**

MacRAE (1988) reviewed asbestos in drinking water from a UK perspective and concluded that:

“It would thus seem highly unlikely that the asbestos- cement pipe distribution system makes any biologically significant contribution to the asbestos content of water passing through it. It follows therefore, in view of the conclusions reached above concerning the likely lack of association between asbestos in drinking water and an excess risk of cancer, that it is highly improbable that asbestos release from asbestos-cement pipe is relevant to to the development of cancer.”