### **GUIDELINES FOR DRINKING-WATER QUALITY**

- Shirachi DY, Tu SH, McGowan JP. Carcinogenic potential of arsenic compounds in drinking water. Research Triangle Park, NC, US Environmental Protection Agency, 1986 (EPA-600/S1-86/003).
- Buchet JP, Lauwerys RR. Evaluation of exposure to inorganic assenic. Cahiers de midecine du travail, 1982, 19:15.
- Wagner SL et al. Skin cancer and assenical intoxication from well water. Archives of dermatology, 1979, 115:1205-1207.
- 44. Feinglass EJ. Arsenic intoxication from well water in the United States. New England journal of medicine, 1973, 288:828-830.
- Murphy MJ, Lyon LW, Taylor JW. Subacute assenic neuropathy: clinical and electrophysiological observations. *Journal of neurology, neurosurgery and psychiatry*, 1981, 44:896-900.
- Wesbey G, Kunis A. Arsenical neuropathy. Illinois medical journal, 1981, 150:396-398.
- Fennell JS, Stacy WK. Electrocardiographic changes in acute assenic poisoning. Irith journal of medical science, 1981, 150:338-339.
- Tseng WP. Effects of dose-response relationship of skin cancer and blackfoot disease with assenic. Environmental health perspectives, 1977, 19:109-119.
- Tseng WP et al. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. Journal of the National Cancer Institute, 1968, 40:453-463.
- Borgoño JM, Greiber R. Epidemiological study of arsenicism in the city of Antofagasta. In: Hemphill DD, ed. Trace substances in environmental health, V. A symposium, Columbia, University of Missouri Ptess, 1972:13-24.
- Zaldivar R. A morbid condition involving cardio-vascular, broncho-pulmonary, digestive and neural lesions in children and young adults after dietary arsenic exposure. Zentralblatt für Bakteriologie und Hygiene, Abteilung I: Originale, 1980, B170:44-56.
- Zaldivar R, Ghai GL. Clinical epidemiological studies on endemic chronic arsenie poisoning in children and adults, including observations on children with high- and low-intake of dietary arsenic. Zentralblatt für Bakteriologie und Hygiene, Abteilung I: Originale, 1980, B170:409-421.
- Valentine JL et al. Assenic effects on human nerve conduction. In: Gawthorne JM, Howell JM, White CL, eds. Proceedings of the 4th International Symposium on Trace Element Metabolism in Man and Animals, Perth, Western Australia, 11-15 May 1981. Beelin, Springer-Verlag, 1982:409.

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- Cebrian ME et al. Chronic assenic poisoning in the north of Mexico. Human toxicology, 1983, 2:121-133.
- Hindmarsh JT et al. Electromyographic abnormalities in chronic environmental arsenicalism. Journal of analytical toxicology, 1977, 1:270-276.
- 56. Lu FJ. Blackfoot disease: arsenic or humic acid? Lancer, 1990, 336(8707):115-116.
- Wu MM et al. Dose-response relation between arsenie concentration in well water and mortality from cancers and cardiovascular diseases. American journal of epidemiology, 1989, 130:1123-1132.
- Chen CJ, Wang CJ. Ecological correlation between assenic level in well water and age-adjusted mortality from malignant neoplasms. Cancer τesearch, 1990, 50:5470-5474.
- Chen CJ et al. Malignant neoplasms among residents of a blackfoot disease-endemic area in Taiwan: high-arsenic artesian well water and cancers. Cancer research, 1985, 45:5895-5899.
- Chen CJ et al. A retrospective study on malignant neoplasms of bladder, lung and liver in blackfoot disease endemic area of Taiwan. British journal of cancer, 1986, 53:399-405.
- Aschengrau A, Zierler S, Cohen A. Quality of community drinking water and the occurrence of spontaneous abortion. Archives of environmental health, 1989, 44:283-290.
- International Agency for Research on Cancer. Overall evaluations of carcinogenicity: an updating of IARC Monographs volumes 1-42. Lyon, 1987:100-106. (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Suppl. 7).
- Joint FAO/WHO Expert Committee on Food Additives. Toxicological evaluation of certain find additives and contaminants. Cambridge, Cambridge University Press, 1989:155-162 (WHO Food Additives Series, No. 24).

### 13.5 Asbestos

13.5.1 General description

**Identity** 

Asbestos is a general term for fibrous silicate minerals containing iron, magnesium, calcium, or sodium. These can be divided into two main groups, namely serpentine (e.g. chrysotile) and amphibole (e.g. amosite, crocidolite, and tremolite)

# Physicochemical properties

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 to it a number of characteristics, including high tensile strength, durability, flexibility, and resistance to heat and chemicals (1).

#### Major uses

Asbestos, particularly chrysotile, is used in a large number of applications, particularly in construction materials, such as asbestos-cement (A/C) sheet and pipe, electrical and thermal insulation, and friction products, such as brake linings and clutch pads (1).

# 13.5.2 Analytical methods

The method of choice for the quantitative determination of asbestos in ambient air and water is transmission electron microscopy (TEM) with identification by energy-dispersive X-ray analysis and selected-area electron diffraction (TEM/SAED). However, TEM/SAED is costly, and preliminary screening with TEM alone (2), which has a detection limit of below 0.1 million fibres per litte (MFL) in water (3), is therefore often used.

# 13.5.3 Environmental levels and human exposure

#### Air

Mean chrysotile concentrations at 24 locations in southern Ontario (Canada) ranged from <2 to 11 fibres longer than 5 µm per litre. Concentrations at 10 remote rural locations were all below the detection limit in this study (<2 fibres/litre) (1, 4). Levels in samples from downtown and suburban locations in Stockholm (Sweden) were in the range 1-3 fibres longer than 5 µm per litre (1, 4).

Airborne asbestos may be released from tapwater in the home. Mean airborne asbestos concentrations were significantly higher (1.7 ng/m<sup>3</sup>) in three homes with water containing elevated concentrations of asbestos than in three control homes (0.31 ng/m<sup>3</sup>); however, the difference in concentration was due primarily to increased numbers of short fibres (<1 µm), which are considered to pose little health risk. Moreover, all the fibre concentrations found in this limited study were within the range of those measured in indoor and outdoor air in other investigations (5). Negligible amounts of asbestos fibres were released to air from water containing 40 ± 10 MFL via a conventional drum-type humidifier (6).

#### Water

Asbestos is introduced into water by the dissolution of asbestos-containing minerals and ores as well as from industrial effluents, atmospheric pollution, and A/C pipes in water-distribution systems. Exfoliation of asbestos fibres from A/C pipes is related to the aggressiveness of the water supply (3). Although A/C piping is used in about 19% of water-distribution systems in Canada, erosion of such piping appeared to contribute measurably to the asbestos content of water supplies at only two of 71 locations surveyed (7). In contrast, high levels of asbestos have been recorded in association with the severe deterioration of A/C pipe containing chrysotile and crocidolite in Woodstock, New York (USA) (8).

Chrysotile was the predominant type of asbestos detected in a national survey of the water supplies of 71 communities in Canada; concentrations varied 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 that concentrations were >1 MFL in the water supplies of 25% of the population, >10 MFL for 5% of the population, and >100 MFL for 0.6% of the population. Concentrations were higher in raw than in treated water (7).

The results of a number of surveys indicate that most of the population of the USA consumes drinking-water containing asbestos in concentrations below 1 MFL (9). In 1974, concentrations of optically visible fibres up to 33 MFL were detected in drinking-water supplies in the Netherlands (10). The results of a survey of asbestos concentrations in raw and treated waters in the United Kingdom suggest that most drinking-waters contain asbestos fibres in concentrations varying from not detectable up to 1 MFL (11).

#### Food

The asbestos content of solid foodstuffs has not been well studied because of the lack of a simple, reliable analytical method. Foods that contain soil particles, dust, or dirt probably contain asbestos fibres; crude estimates suggest that the intake of asbestos in food may be significant in comparison with that in drinking-water (12). 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 (13).

13.5.4 Kinetics and metabolism in laboratory animals and humans Information on the transmigration of ingested asbestos through the gastrointestinal tract to other tissues is contradictory (1, 3). Available data indicate that penetration, if it occurs at all, is extremely limited.

# 13.5.5 Effects on laboratory animals and in vitro test systems Reproductive toxicity, embryotoxicity, and teratogenicity

Administration of 4–400 mg of chrysotile per kg of body weight to CD-1 mice on days 1–15 of pregnancy did not affect the survival of the progeny. In vitro administration did not interfere with implantation on transfer of exposed blastocysts to recipient females but did result in a decrease in post-implantation survival. The authors concluded that asbestos was not teratogenic in these studies (14).

## Mutagenicity and related end-points

Although not mutagenic, all types of asbestos have induced chromosomal aberrations in in vitro studies (15). In in vivo studies, a single oral administration of chrysotile did not increase the frequency of micronuclei in mice, and there was no increase in chromosomal aberrations in monkeys following oral administration of chrysotile by gavage (10).

# Carcinogenicity

Although the carcinogenicity of inhaled asbestos is well established, there is no conclusive evidence that ingested asbestos is carcinogenic (1, 3, 16). In a series of extensive investigations involving treatment groups of 250 animals of each sex (17–19), no treatment-related increases in tumour incidence were observed in Syrian golden hamsters fed 1% amosite or short-range (98% shorter than 10 µm) or intermediate-range (65% longer than 10 µm) chrysotile, or in Fischer 344 rats fed 1% tremolite or amosite or short-range chrysotile in the diet over their lifetime. Although the incidence of benign epithelial neoplasms in the gastrointestinal tract in male Fischer 344 rats fed 1% intermediate-range chrysotile was significantly increased as compared with that in pooled controls from contemporary lifetime asbestos feeding studies in the same laboratory, the increase was not statistically significant in comparison with the data for concurrent controls and was limited to one sex.

#### 13.5.6 Effects on humans

The health hazards associated with the inhalation of asbestos in the occupational environment have long been recognized and include asbestosis, bronchial carcinoma, malignant mesothelioma of the pleura and peritoneum, and possibly cancers of the gastrointestinal tract and larynx. In contrast, little convincing evidence has been found of the carcinogenicity of ingested asbestos in epidemiological studies of populations supplied with drinking-water containing high concentrations of asbestos (1, 15, 19-26). Moreover, 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 is the subject of considerable disagreement (1, 27, 28).

In ecological population studies (1, 20, 22–25) (i.e. studies in which individual exposures were not estimated and population mobility was not adequately addressed), no consistent evidence was found of an association between cancer mortality or incidence and the ingestion of asbestos in drinking-water. In an analytical epidemiological (case-control) study that was inherently more sensitive than the ecological studies, there was no consistent evidence of a cancer risk associated with the ingestion of asbestos in drinking-water in Puget Sound, where levels up to 200 MFL were observed (26).

### 13.5.7 Conclusions

Although asbestos is a known human carcinogen by the inhalation route, available epidemiological studies do not support the hypothesis that an increased cancer risk is associated with the ingestion of asbestos in drinking-water. Moreover, in extensive feeding studies in animals, asbestos has not consistently increased the incidence of tumours of the gastrointestinal tract. There is therefore no consistent, convincing evidence that ingested asbestos is hazardous to health, and it is concluded that there is no need to establish a guideline value for asbestos in drinking-water.

#### References

- Asbestor and other natural mineral fibres. Geneva, World Health Organization, 1986 (Environmental Health Criteria, No. 53).
- Pitt R. Asbestos as an urban area pollutant. Journal of the Water Pollution Control Federation, 1988, 60:1993-2001.
- Tost P et al. Asbestos in drinking-water. Ottawa, Ontario, Canada, Department of National Health and Welfare, Health Protection Branch, 1984 (CRC critical reviews in environmental control).
- Chatfield E. Measurement of asbestos fibre concentrations in ambient atmospheres. Report prepared for the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. Toronto, Ontario, Canada, 1983.
- Webber JS, Syrotynski S, King MV. Asbestos-contaminated drinking water; its impact on household air. Environmental research, 1988, 46:153-167.
- Metanger JC, Reid WW, Davey ABC. The transfer of asbestos from water to alr via a
  portable drum-type home humidilier. Canadian journal of public health, 1979,
  70:276-278.

- Chatfield EJ. Dillon MJ. A national survey for asbestos fibres in Canadian drinking water supplies. Ottawa, Canada, Department of National Health and Welfare, 1979 (Environmental Health Directorate Report 79-EHD-34).
- Webber JS, Covey JR, King MV. Asbestos in drinking water supplied through grossly deteriorated A-C pipe. Journal of the American Water Works Association, 1989, 81:80.
- Millette JR et al. Asbestos in water supplies of the United States. Environmental health perspectives, 1983, 53:45-48.
- Montizaan GK, Knaap AG, van der Heijden CA. Asbestos: toxicology and risk assessment for the general population in The Netherlands. Food chemistry and toxicology, 1989, 27:53-63.
- Conway DM, Lacey RF. Asbestos in drinking water. Results of a survey. Medmenham, Water Research Centre, 1984 (Technical Report TR202).
- Rowe JN. Relative source contributions of diet and air to ingested asbestos exposure. Environmental health research, 1983, 53:115-120.
- Cunningham HM, Pontefract RD. Asbestos fibres in beverages and drinking water. Nature (London), 1971, 232:332-333.
- Schneider U, Maurer RR. Asbestoš and embryonic development. Teratology, 1977, 15:273-279.
- International Agency for Research on Cancer. Overall evaluations of carcinogenicity: an updating of LIRC Monographs valumes. I-42. Lyon, 1987:106-116 (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Suppl. 7).
- IG. DHHS Working Group. Report on cancer risks associated with the ingestion of asbestos. Environmental health perspectives, 1987, 72:253-265.
- McConnell EE et al. Chronic effects of dietary exposure to amosite and chrysotile asbestos in Syrian golden hamsters. Environmental health perspectives, 1983, 53:11-25.
- McConnell EE et al. Chronic effects of dietary exposure to amosite asbestos and tremolite in F344 rats. Environmental bealth perspectives, 1983, 53:27-44.
- National Toxicology Program. Toxicology and carcinogenesis studies of chrysotile aubestos in F344 rats. Research Triangle Park, NC, US Department of Health and Human Services, 1985 (NIH Publication No. 86-2551; Technical Report No. 295).
- 20. Toft P et al. Asbestos and drinking water in Canada. Science of the total environment, 1981, 18:77-89.

### 13. INORGANIC CONSTITUENTS AND PHYSICAL PARAMETERS

- Conforti PM et al. Asbestos in drinking water and cancer in the San Francisco Bay area; 1969-1974 incidence. Journal of chronic diseases, 1981, 34:211-224.
- 22. Sigurdson EE et al. Cancer morbidity investigations: lessons from the Duluth study of possible effects of asbestos in drinking water. *Environmental research*, 1981, 25:50-61.
- Meigs JW et al. Asbestos cement pipe and cancer in Connecticut 1955-1974. Journal
  of environmental health, 1980, 42:187.
- Millette JR et al. Epidemiology study of the use of asbestos-cement pipe for the distribution of drinking water in Escambia County, Florida. Environmental health perspectives, 1983, 53:91-98.
- Sadler TD et al. The use of asbestos-cement pipe for public water supply and the incidence of cancer in selected communities in Utah. Journal of community health, 1984, 9:285-293.
- Politsar L, Severson RK, Boatman ES. A case control study of asbestos in drinking water and cancer risk. American journal of epidemiology, 1984, 119:456-471.
- Boatman ES et al. Use of quantitative analysis of urine to assess exposure to asbestos
  fibres in drinking water in the Puget Sound region. Environmental health perspectives,
  1983, 53:131-139.
- Carter RE, Taylor WF. Identification of a particular amphibole asbestos fibre in tissues of persons exposed to a high oral intake of the mineral. Environmental research, 1980, 21:85-93.

### 13.6 Barium

13.6.1 General description

### Identity

Barium is present as a trace element in both igneous and sedimentary rocks. Although it is not found free in nature (1), it occurs in a number of compounds, most commonly barium sulfate (barite) and, to a lesser extent, barium carbonate (witherite).