WHO/SDE/WSH/03.04/02
English only

Asbestos in Drinking-water

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

Originally puiblished in **Guidelines for drinking-water quality**, 2nd ed. Vol. 2. *Health criteria and other supporting information.* World Health Organization, Geneva, 1996.

Preface

One of the primary goals of WHO and its member states is that "all people, whatever their stage of development and their social and economic conditions, have the right to have access to an adequate supply of safe drinking water." A major WHO function to achieve such goals is the responsibility "to propose regulations, and to make recommendations with respect to international health matters ...."

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

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

Since the first edition of the GDWQ, WHO has published information on health criteria and other supporting information to the GDWQ, describing the approaches used in deriving guideline values and presenting critical reviews and evaluations of the effects on human health of the substances or contaminants examined in drinking-water.

For each chemical contaminant or substance considered, a lead institution prepared a health criteria document evaluating the risks for human health from exposure to the particular chemical in drinking-water. Institutions from Canada, Denmark, Finland, France, Germany, Italy, Japan, Netherlands, Norway, Poland, Sweden, United Kingdom and United States of America prepared the requested health criteria documents.

Under the responsibility of the coordinators for a group of chemicals considered in the guidelines, the draft health criteria documents were submitted to a number of scientific institutions and selected experts for peer review. Comments were taken into consideration by the coordinators and authors before the documents were submitted for final evaluation by the experts meetings. A "final task force" meeting reviewed the health risk assessments and public and peer review comments and, where appropriate, decided upon guideline values. During preparation of the third edition of the GDWQ, it was decided to include a public review via the world wide web in the process of development of the health criteria documents.

During the preparation of health criteria documents and at experts meetings, careful consideration was given to information available in previous risk assessments carried out by the International Programme on Chemical Safety, in its Environmental Health

Criteria monographs and Concise International Chemical Assessment Documents, the International Agency for Research on Cancer, the joint FAO/WHO Meetings on Pesticide Residues, and the joint FAO/WHO Expert Committee on Food Additives (which evaluates contaminants such as lead, cadmium, nitrate and nitrite in addition to food additives).

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

Acknowledgements

The work of the following coordinators was crucial in the development of this background document for development of WHO *Guidelines for drinking-water quality*:

J.K. Fawell, Water Research Centre, United Kingdom
(inorganic constituents)
U. Lund, Water Quality Institute, Denmark
(organic constituents and pesticides)
B. Mintz, Environmental Protection Agency, USA
(disinfectants and disinfectant by-products)

The WHO coordinators were as follows:

Headquarters:
H. Galal-Gorchev, International Programme on Chemical Safety
R. Helmer, Division of Environmental Health

Regional Office for Europe:
X. Bonnefoy, Environment and Health
O. Espinoza, Environment and Health

Ms Marla Sheffer of Ottawa, Canada, was responsible for the scientific editing of the document.

The efforts of all who helped in the preparation and finalization of this document, including those who drafted and peer reviewed drafts, are gratefully acknowledged.

The convening of the experts meetings was made possible by the financial support afforded to WHO by the Danish International Development Agency (DANIDA), Norwegian Agency for Development Cooperation (NORAD), the United Kingdom Overseas Development Administration (ODA) and the Water Services Association in the United Kingdom, the Swedish International Development Authority (SIDA), and the following sponsoring countries: Belgium, Canada, France, Italy, Japan, Netherlands, United Kingdom of Great Britain and Northern Ireland and United States of America.

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*).

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). Analysis by TEM/SAED is costly, and preliminary screening with TEM alone (*2*), which has a detection limit of below 0.1 million fibres per litre (MFL) in water (*3*), is therefore often used.

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^3) in three homes with water containing elevated concentrations of asbestos than in three control homes (0.31 ng/m^3); however, the difference in concentration was due primarily to increased numbers of short fibres (<1 µm), which 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 77 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*).

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.

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.

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*).

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 for asbestos in drinking-water.

REFERENCES

1. *Asbestos and other natural mineral fibres*. Geneva, World Health Organization, 1986 (Environmental Health Criteria, No. 53).
2. Pitt R. Asbestos as an urban area pollutant. *Journal of the Water Pollution Control Federation*, 1988, 60:1993-2001.
3. Toft 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).
4. 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.
5. Webber JS, Syrotynski S, King MV. Asbestos-contaminated drinking water: its impact on household air. Environmental research, 1988, 46:153-167.
6. Meranger JC, Reid WW, Davey ABC. The transfer of asbestos from water to air via a

portable drum-type home humidifier. *Canadian journal of public health*, 1979, 70:276-278.
7. 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).
8. 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.
9. Millette JR et al. Asbestos in water supplies of the United States. *Environmental health perspectives*, 1983, 53:45-48.
10. 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.
11. Conway DM, Lacey RF. *Asbestos in drinking water. Results of a survey*. Medmenham, Water Research Centre, 1984 (Technical Report TR202).
12. Rowe JN. Relative source contributions of diet and air to ingested asbestos exposure. *Environmental health research*, 1983, 53:115-120.
13. Cunningham HM, Pontefract RD. Asbestos fibres in beverages and drinking water. *Nature (London)*, 1971, 232:332-333.
14. Schneider U, Maurer RR. Asbestos and embryonic development. *Teratology*, 1977, 15:273-279.
15. International Agency for Research on Cancer. *Overall evaluations of carcinogenicity: an updating of IARC Monographs volumes 1–42*. Lyon, 1987:106-116 (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Suppl. 7).
16. DHHS Working Group. Report on cancer risks associated with the ingestion of asbestos. *Environmental health perspectives*, 1987, 72:253-265.
17. 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.
18. McConnell EE et al. Chronic effects of dietary exposure to amosite asbestos and tremolite in F344 rats. *Environmental health perspectives*, 1983, 53:27-44.
19. National Toxicology Program. *Toxicology and carcinogenesis studies of chrysotile asbestos 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.
21. 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 for the Duluth study of possible effects of asbestos in drinking water. *Environmental research*, 1981, 25:50-61.
23. Meigs JW et al. Asbestos cement pipe and cancer in Connecticut 1955-1974. *Journal of environmental health*, 1980, 42:187.
24. 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.
25. 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.
26. Polissar 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.
27. 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.
28. 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.