

**ASBESTOS CEMENT DRINKING WATER PIPES AND  
POSSIBLE HEALTH RISKS  
REVIEW FOR DWI**

**PUBLISHED MAY 2002**

[http://www.dwi.gov.uk/research/completed-research/reports/DWI70\\_2\\_135\\_asbestos%20cement%20pipes.pdf](http://www.dwi.gov.uk/research/completed-research/reports/DWI70_2_135_asbestos%20cement%20pipes.pdf)

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**Report for Contract 70/2/135**

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## **Asbestos Cement Drinking Water Pipes and Possible Health Risks**

### **Summary Statement**

The possibility of health effects from asbestos fibres in drinking water has been widely studied but with little evidence for any concern.

The World Health Organisation considered asbestos in drinking water arising from asbestos cement pipe in their 1993 edition of the Guidelines for Drinking Water Quality. The guidelines state “Although well studied, there has been little convincing evidence of the carcinogenicity of ingested asbestos in epidemiological studies of populations with drinking water supplies containing high concentrations of asbestos. Moreover in extensive studies in laboratory species, asbestos has not consistently increased the incidence of tumours of the gastrointestinal tract. There is therefore no consistent evidence that ingested asbestos is hazardous to health and thus it was concluded that there was no need to establish a health-based guideline value for asbestos in drinking water”.

Although many countries throughout the world, including many European countries, still have asbestos cement water pipes, there appears to be no concern for health of consumers receiving the water and no programmes to specifically replace asbestos cement pipe for this reason.

## **Health issues Arising from the Use of Asbestos Cement Pipes for Drinking Water – Executive Summary**

Although measurement of asbestos fibres in drinking water is technically difficult, research has indicated that most waters, whether or not distributed through asbestos cement pipes, contain asbestos fibres. This is because asbestos is widely found in the environment as a consequence of natural dissolution of asbestos-containing minerals. Asbestos cement pipes can give rise to an increase in the numbers of asbestos fibres in drinking water, particularly when first installed. The risks to health from ingestion of asbestos fibres in food and drinking water have been extensively studied by both epidemiology and by experiments in laboratory animals.

Most epidemiological studies found no association with any specific gastrointestinal cancers, although a small number of studies did find a weak positive association. The studies considered the best did not provide evidence for a link between asbestos in drinking water and cancer. Of the 8 long-term animal studies, only one suggested a possible statistically significant increase in benign tumours in one sex, when compared to historical control animals but not the control animals used in the study.

There is potential for exposure to asbestos fibres in drinking water by inhalation of aerosol droplets or from fibres that are trapped on clothing during washing and which are subsequently released into the atmosphere. This has been studied and except in an extreme case there was no measurable increase in the number of fibres in the indoor atmosphere of houses. In addition, the fibres in drinking water consist almost entirely of short fibres, which are considered to contribute little or no risk to public health.

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Asbestos cement pipes have been widely used for drinking water distribution and there are many kilometres to be found all over the world. Although few countries still install asbestos cement pipe, primarily because of issues with handling, there appears to be no concern for health of consumers receiving the water and no programmes to specifically replace asbestos cement pipe for this reason.

## **1. Introduction**

Asbestos cement pipes have been widely used in many countries for many years, although installation of new asbestos cement water pipes has declined significantly over the past 10 years because of difficulties in handling, public perception and the availability of better materials. The health effects resulting from chronic inhalation of asbestos dusts are well established and include asbestosis and mesothelioma, a highly characteristic cancer of the lung. There is a very good qualitative understanding of the effects of inhaled asbestos fibres and the physical characteristics of the fibres are considered to be very important in determining their ability to cause disease. Concern that ingested asbestos fibres could also lead to possible health effects has resulted in this topic having been widely studied by both epidemiology and by studies in laboratory animals.

## **2. Occurrence of asbestos fibres in drinking water**

Measurement of asbestos fibres in food and water is extremely difficult and requires the use of highly specialised techniques involving electron microscopy. Analysis for asbestos fibres in drinking water is not, therefore, carried out on a routine basis. However, research has indicated that most waters, whether or not distributed through asbestos cement pipes, contain asbestos fibres. Asbestos cement pipes do give rise to an increase in the numbers of asbestos fibres in drinking water in some circumstances (Chatfield and Dillon 1979, Webber et al 1989 Millette et al 1983, Conway and Lacey 1984), although studies from Italy showed that the numbers of fibres in water passing through asbestos cement pipes was apparently independent of the aggressiveness of the water. This was considered to be due to inorganic deposits and organic slimes on the surface of the pipes that acted as a physical barrier to fibres entering the water. The Italian studies also indicated that the highest number of fibres appeared just after the installation of new pipes but this decreased rapidly (ISS 1993).

As indicated above, the physical characteristics, including fibre dimensions and surface properties, are an important factor in the pathogenicity of asbestos. In general, the fibres that are considered to be of significantly greater risk are long thin fibres of greater than 8  $\mu\text{m}$  length and less than 1.5  $\mu\text{m}$  in diameter. In the WRc studies, the fibre size was predominantly less than 5  $\mu\text{m}$  in length and that only one of 13 drinking water sites showed greater than 1 million fibres per litre. In the ISS study in Italy the maximum mean levels, from any supply, of fibres greater than 10 microns in length was 1.1 million per litre while the maximum mean concentration of fibres between 5 and 10  $\mu\text{m}$  in length was 2.8 million per litre (ISS 1993). A national survey of Canada estimated that median fibre lengths were between 0.5 and 0.8  $\mu\text{m}$  and that 25% of the population were exposed to greater than 1 million fibres per litre, with a small proportion exposed to greater than 100 million fibres per litre (Chatfield and Dillon 1979). A study from Japan (Saitoh et al 1992) found that asbestos fibres in drinking water arising from asbestos cement pipes were thick and quite different to the shape and structure of fibres considered to be of high risk by inhalation.

## **3. Epidemiological Studies**

Epidemiological studies of the possible risks to health of asbestos fibres in drinking water have been carried out in various parts of the United States and in Canada. The majority of studies have been of the ecological design and all suffer from some inadequacies. The supply in Duluth was known to contain high numbers of asbestos fibres. Epidemiological studies were published in 1974, 1976 and 1981 (Mason et al

1974, Levy et al 1976 and Sigurdson et al 1981). The first study, which found higher rates of cancer of the stomach and rectum in males and females and of the pancreas in females, was considered to be flawed because important confounders such as race and occupation were not considered. The two later studies revealed no association between the ingestion of asbestos fibres through drinking water and cancers of the gastrointestinal tract.

Studies carried out in the San Francisco Bay area of California found some positive associations with gastrointestinal, peritoneal and lung cancer (Kanarek et al 1980) and a follow up study by Conforti et al (1981) found a significant excess of colon cancers in males and peritoneal cancers in females. There were, however, a number of serious flaws in these studies, not least of which was inappropriate statistical analysis and the fact that population mobility in the area was particularly high, so there was considerable uncertainty regarding the exposure period. There was also a lack of control for several important confounding variables.

A study in Quebec (Wigle 1977, Toft et al 1984) found that in areas of very high drinking water asbestos, there was an increase in overall cancer mortality in men, slight increases in stomach cancer in men and pancreatic cancer in women but no excess of overall cancers of the gastrointestinal tract. The authors concluded that the excess in males was probably due to occupational exposure. There were a number of flaws due to the lack of control for a significant number of confounding variables.

Epidemiological studies were also carried out in two areas where asbestos cement pipes had been extensively used. In Connecticut, studies by Harrington et al (1978) and Meigs et al (1980) found no consistent patterns of cancer associated with ingestion of asbestos from drinking water. However, there were limitations to both studies. In Florida, a study in Escambia County by Millette et al (1983) concluded that there was no observed association between asbestos cement pipe and cancer mortality but noted that the study would not be sufficiently large to identify small changes.

In Washington State there have been studies of the impact of naturally occurring asbestos fibres in drinking water in the Puget Sound area. The first by Polissar et al (1982) found consistent associations with cancer of the small intestine but these were not statistically significant. A number of other positive and negative associations were also observed but none of these was consistent throughout the study groups. In 1984, Polissar et al published a case control study as a follow up to the original ecological study. This second study was more sensitive than the ecological studies and was considered to be the best study to date. This found no consistent evidence of an increased risk of cancer from ingestion of asbestos fibres in drinking water.

#### **4. Animal Studies**

A number of long-term studies have been carried out using laboratory hamsters and rats.

One of the topics of investigation has been whether asbestos fibres can penetrate the gastrointestinal tract. The overall results of these studies remain contradictory and uncertain. The method used is that of electron microscopy but the potential for external cross contamination is significant (WHO 1996).

The question also remains as to whether the fibres retain the surface properties that are considered to be an important feature in their pathogenicity. Fibres that are inhaled may have very different surface properties to fibres that have been

transported in water and that have passed through the stomach and gastrointestinal tract with the potential for chemical change at the surface.

There have been three studies in which hamsters have been given asbestos fibres by the oral route. Smith et al (1980) gave amosite tailings in water for 650 days at concentrations of up to 50 mg/l. There were no significant differences between the groups in terms of survival, body weight, histopathology or numbers of gastrointestinal tumours. The National Toxicology Programme in the United States carried out 2 lifetime studies in which hamsters were given asbestos in their diet (NTP 1983, 1990). In the first study in which 1% amosite was incorporated into the diet, milling to improve homogeneity of the fibre size gave rise to a higher chromium content in the test diet. The treated animals actually survived better than the control animals but there were no differences between the groups in the tumours at any of the sites. It is not clear whether the milling process would have resulted in significant changes to the surface properties. The later study used unmilled, chrysotile asbestos incorporated into diets at 1%. This included a differentiation between a group fed primarily short fibres and a second group fed predominantly long fibres, described in the study as intermediate fibres. Although there was an increase in adrenal tumours in males and females fed the long fibres this was only significant when compared to pooled controls. NTP concluded that neither fibre size was carcinogenic in the hamster.

There have been a number of long-term studies in rats carried out by different groups. The earliest studies (Gross et al 1974) with chrysotile were too short to be considered for carcinogenicity but a study by Cunningham et al (1977) in which rats were fed diets containing 1% chrysotile for up to 920 days gave results that were inconclusive. In another study in which rats were fed 10% chrysotile in the diet for 32 months (Donham et al 1980), tumours of the colon were observed in both treated and control groups but there was no statistically significant difference.

In a UK study, chrysotile, crocidolite or amosite asbestos were administered to rats in their diet at a level of 5 mg/gm diet, taking care to reduce the risk of inhalation exposure, for a period of 2 years (Bolton et al 1982). There appeared to be no damage to the gastrointestinal mucosa as a consequence of prolonged exposure to such high concentrations and there were no increases in malignant tumours in the treated groups. The chrysotile treated group appeared to have more benign tumours but this was not significantly different from controls.

NTP carried out 2 rat studies with one examining the effects of 1% of amosite or tremolite in the diet for a lifetime and the other 1% of chrysotile (NTP 1985). As with the hamster studies two lengths of chrysotile fibres were studied. There was no indication of an increase in cancer in any group except those animals receiving the longer chrysotile fibres. There was an increased incidence of benign adenomatous polyps in the large intestine in males, which was significantly greater than the incidence in pooled historical controls but not the concurrent controls from the study. NTP considered that this was evidence for the carcinogenicity of ingested asbestos but most other authorities dispute this in view of the small numbers of polyps, the fact that these are benign and there was no evidence of an increase in females.

In another study (Truhaut and Chouroulinkov cited in IARC 1989) a mixture of fibres administered in palm oil at doses from 10 to 360 mg per day for 2 years gave no evidence of any increase in tumours as a consequence of the treatment.

Between 1996 and 1999 a series of studies of the possible co-carcinogenicity and co-genotoxicity, by the oral route of exposure, of asbestos fibres with benzo(a)pyrene

(BaP) were published (Varga et al 1996 a,b, 1998, 1999). In the initial studies, crocidolite or anthophyllite asbestos was soaked in an aqueous solution containing 250 to 2500 µg/litre of BaP. Since the water solubility of BaP is given as 3.8 µg/litre at 25°C and the log  $K_{oc}$  is 6.1 at the same temperature, it would be expected that significant amounts of BaP would adsorb onto the asbestos fibres. The fibres were tested in short-term genotoxicity tests with untreated asbestos fibres as a control. Fibres were administered to rats by gavage with 50 mg/kg of the treated or untreated fibres. Concentrated urine and serum samples were tested in the Ames Salmonella mutagenicity assay and bone marrow was examined for micronuclei and SCE. There appeared to be no positive mutagenicity in the *in vitro* tests. There was a marginal increase in micronuclei in animals treated with the asbestos soaked at 1000 µg/litre and a dose dependent increase in SCE. Since there are no data to indicate that asbestos fibres penetrate the gut and are transported to the bone marrow, these data appear to indicate that BaP can be removed from the surface of asbestos fibres in the gastro-intestinal tract.

In a subsequent study, no mutagenicity was detected in *S. typhimurium* strains TA98 and TA100 when concentrated samples of urine and serum were tested following exposure of rats as described above. In studies using the Comet assay, significant strand breaks were detected in samples of cells from the intestine and omentum but not in peritoneal macrophages. There are number of questions that arise from these data, not least of which relate to the penetration of asbestos fibres into cells. There is no evidence to indicate that the effects detected were not entirely due to BaP.

In addition the concentrations of BaP used were unrealistic when compared to drinking water in which the concentrations rarely exceed 10 ng/litre. Should asbestos cement pipe have been coated with coal tar, this would have provided circumstances in which asbestos fibres could have been exposed to high concentrations of BaP and other PAH. However, the coal tar lining would have had to have deteriorated to a sufficient extent to that the asbestos was exposed to the water. Under such circumstances it is conceivable that the asbestos fibres might have carried higher concentrations of BaP than would be encountered dissolved in water. There appears to be no evidence that such circumstances existed.

## **5. Inhaled Asbestos**

It is possible that asbestos fibres in drinking water could be released to the atmosphere and could result in increased exposure by inhalation. Webber et al (1988) examined the impact of waterborne asbestos fibres on household air and observed that fibre concentrations in air samples correlated with water concentrations. However, the fibres were predominantly less than 1 µm in length, which is considered to be of minimal hazard to health. It must also be noted that the concentrations in drinking water in this study were significantly higher than has been recorded in the UK and the concentration recorded in air were in the range of airborne fibres recorded in other studies of indoor and outdoor air.

## **6. Replacement of Asbestos Cement Pipes**

Asbestos cement pipes have been widely used in the past in many parts of the world, including North America, Japan and Europe. Currently there appear to be no countries that are still installing asbestos cement pipes. This appears to be as a consequence of three factors, of which the problems of handling asbestos cement pipes seems to be predominant. Enquiries have revealed no countries that are



currently planning a programme of replacement of asbestos cement pipes because of concerns over ingested asbestos. When asbestos cement pipes need to be replaced, the material that is removed needs to be treated as a special waste in the UK because of the concern over inhaled asbestos.

## **7. Conclusions**

Inhaled asbestos is a known human carcinogen and considerable care is required in handling asbestos products, including asbestos cement water pipes, to prevent the inhalation of asbestos fibres. The tumours caused by asbestos are mesotheliomas and are considered to be characteristic of asbestos exposure. The evidence that inhaled asbestos can cause tumours at any other site in the body is, at best, equivocal. However, the evidence with regard to mesotheliomas strongly supports the contention that fibre size and surface characteristics are important in the pathogenicity of asbestos. Fibres greater than 8 µm in length and less than 0.25 µm in diameter are the greatest concern with very short fibres of less than 1 µm considered to be of low risk. Asbestos fibres from drinking water are either in this latter category or are of much greater diameter than those of greatest concern and so the risk to health from inhalation of such fibres is considered to be low.

That asbestos cement pipes can contribute to fibre levels in drinking water is not in doubt but asbestos fibres from natural sources are found in the great majority of waters, whether or not they have passed through asbestos cement pipes. Asbestos in drinking water is not, therefore, solely a function of asbestos cement pipe.

The evidence from epidemiological studies and from laboratory animal feeding studies does not provide support for the view that asbestos from drinking water is of concern. WHO concluded in their 1993 Guidelines for Drinking Water Quality "Although well studied, there has been little convincing evidence of the carcinogenicity of ingested asbestos in epidemiological studies of populations with drinking water supplies containing high concentrations of asbestos. Moreover in extensive studies in laboratory species, asbestos has not consistently increased the incidence of tumours of the gastrointestinal tract. There is therefore no consistent evidence that ingested asbestos is hazardous to health and thus it was concluded that there was no need to establish a health-based guideline value for asbestos in drinking water".

The WHO Drinking Water Committee did not regard asbestos as necessary for reconsideration in the current phase of the rolling revision.

Asbestos cement pipes have been widely used for drinking water distribution and there are many kilometres to be found all over the world, including many European countries. Few countries still install asbestos cement pipe, primarily because of issues with handling, but there appears to be no concern for the health of consumers receiving the water and there appear to be no programmes to replace asbestos cement pipe for this reason.

## **8. References**

1. Chatfield EJ, Dillon MJ. (1979) *A national study for asbestos fibres in Canadian drinking water supplies*. Ottawa, Canada, Department of National Health and Welfare, 1979. (Environmental Health Directorate Report 79-EHD-34).
2. Webber JS, Covey JR, King, MV. (1989) Asbestos in drinking water supplied through grossly deteriorated A-C pipe. *Journal of the American Water Works Association*, 18:80.

3. Millette JR et al. (1983) Asbestos in water supplies of the United States. *Environmental Health Perspectives*, 53:45-48.
4. Conway DM, Lacey RF. (1984) *Asbestos in drinking water. Results of a survey*. Medmenham, Water Research Centre, 1984 (Technical Report TR202).
5. ISS (1993) Research Projects of the Istituto Superiore di Sanita. Environment, ISS, Rome
6. Mason TJ, McKay FW, Miller RW. (1974) Asbestos-like fibres in Duluth water supply. *Journal of the American Medical Association*, 228:1019-1020.
7. Levy BS et al. (1976) Investigating possible effects of asbestos in city water: Surveillance of gastrointestinal cancer incidence in Duluth, MN. *American Journal of Epidemiology*, 103:362-368.
8. Sigurdson EE, Levy, BS, Mandel J, Landon E, Pearson J. (1981) Cancer morbidity investigations: Lessons from the Duluth study of possible effects of asbestos in drinking water. *Environmental Research*, 25:50-61.
9. Kanarek MS, Conforti PM, Jackson LA, Cooper RC, Murchio JC. (1980) Asbestos in drinking water and cancer incidence in the San Francisco Bay area. *American Journal of Epidemiology*, 112:54-72.
10. Conforti PM, Kanarek, M, Jackson, LA, Cooper RC, Murchio JC. (1981) Asbestos in drinking water and cancer in the San Francisco Bay Area; 1969-1974 incidence. *Journal of chronic diseases*, 34:211-224.
11. Wigle DT. (1977) Cancer Mortality in relation to asbestos in municipal water supplies. *Archives of environmental health*, 32:185-190.
12. Toft P, Meek ME, Wigle DT, Meranger JC. (1984) Asbestos in drinking water. *CRC Critical Reviews in Environmental Control*, 14(2):151-197.
13. Harrington JM, Craun G, Meigs JW, Landrigan PJ, Flannery JT, Woodhull RS. (1978) An investigation of the use of asbestos cement pipe for public water supply and the incidence of gastrointestinal cancer in Connecticut, 1935-1973. *American Journal of Epidemiology*, 107:96-103.
14. Meigs JW, Walter S, Heston J, Millette JR, Craun GF, Woodhull RS, Flannery JT. (1980) Asbestos cement pipe and cancer in Connecticut 1955-1974. *Journal of environmental health*, 42:187.
15. Millette JR, Craun GF, Stober JA, Kraemer DF, Tousignant HG, Hildago E, Duboise RL Benedict J. (1983) Epidemiology study of the use of asbestos-cement pipe for the distribution of drinking water in Escambia County, Florida. *Environmental health perspectives*, 53:91-98.
16. Polissar L, Severson RK, Boatman ES, Thomas DB. (1982) Cancer incidence in relation to asbestos in drinking water in the Puget Sound Region. *American Journal of Epidemiology*, 116(2):314-328.
17. Polissar L, Severson RK, Boatman ES. (1984) A case control study of asbestos in drinking water and cancer risk. *American Journal of Epidemiology*, 119:456.
18. WHO (1996) Guidelines for Drinking Water Quality Second Edition Volume 2. WHO, Geneva.
19. Saitoh K, Takizawa Y, Muto H, Hirano K. (1992) [Concentration and form of asbestos fibres in tap drinking water contaminated from a water supply pipe with asbestos cement] In Japanese. *Nippon Eiseigaku Zasshi*, 47(4): 851-860.
20. Smith WE, Hubert DD, Sobel HJ, Peters ET, Doerfler TE. (1980) Health in experimental animals drinking water with and without amosite and other mineral particles. *Journal of Environmental Pathology Toxicology*, 3:277-300.
21. National Toxicology Program. (1983) Lifetime Carcinogenesis studies of amosite asbestos (CAS No. 121-72-73-5) in Syrian Golden hamsters (feed studies). NTP TR 249, 1983, NI Publication No. 84-2505.
22. Gross P et al. (1974) Ingested mineral fibres. Do they penetrate tissues or cause cancer? *Archives of Environmental Health*, 29:341-347.
23. Cunningham HM et al. (1977) Chronic effects of ingested asbestos in rats. *Archives of Environmental Contamination and Toxicology*, 6:507-513.

24. Donham KJ, Berg JW, Woll LA, Leininger JR. (1980) The effects of long term ingestion of asbestos on the colon of F344 rats. *Cancer*, 45:1073-1084.
25. Bolton RE, Davis JMG, Lamb D. (1982) The pathological effects of prolonged asbestos ingestion in rats. *Environmental Research*, 29:134-150.
26. National Toxicology Program. (1985) Lifetime Carcinogenesis studies of chrysotile asbestos. (CAS No. 12001-29-5) in F344/N rats (feed studies). *NTP TR* 295, 1985,
27. Truhaut and Chouroulinkov Cited in IARC (1989) Non Occupational Exposure to Mineral Fibres. J Bignon, J Peto, R Saracci Eds. Scientific Publications Series No 90. IARC, Lyon.
28. Varga C, Pocsai Z, Horvath G, Timbrell V. (1996a) Studies on genotoxicity of orally administered crocidolite asbestos in rats: implications for ingested asbestos induced carcinogenesis. *Anticancer Research*, 16(2): 811-814.
29. Varga C, Horvath G, Timbrell V. (1996b) In vivo studies on genotoxicity and cogenotoxicity of ingested UICC anthophyllite asbestos. *Cancer Letters*, 105(2): 181-185.
30. Varga C, Horvath G, Pocsai Z, Timbrell V. (1998) On the mechanism of cogenotoxic action between ingested amphibole asbestos fibres and benzo(a)pyrene: I. Urinary and serum mutagenicity studies with rats. *Cancer Letters*, 128(2): 165-169.
31. Varga C, Horvath G, Timbrell V. (1999) On the mechanism of cogenotoxic action between ingested amphibole asbestos fibres and benzo(a)pyrene: II. Tissue specificity studies using comet assay. *Cancer Letters*, 139(2): 173-176.
32. Webber JS, Syrotynski S, King MV. (1988) Asbestos-contaminated drinking water: its impact on household air. *Environmental research*. 46:153-197.