

Health Problems Related to Environmental Fibrous Minerals

GUNNAR HILLERDAL, M.D.

Departments of Lung Diseases

Karolinska Hospital, Stockholm, and Akademic Hospital, Uppsala, Swedens

Introduction

Very early in the history of mining and with the industrial applications of asbestos, it was known that a variety of mineral fibers were hazardous to health. By the 1940s, the potential risk of lung cancer, in addition to the fibrosis disorder asbestosis, one of the pneumoconioses, was described. Within twenty years, another malignant disease, mesothelioma, cancer of the tissues that surround the lung, was ascribed to asbestos exposure. It is now common knowledge that inhalation of certain mineral fibers can cause disease (Skinner et al. 1988). Because the fibers are inhaled, the lung and surrounding tissues are the primary targets, but there may be subsequent reactions in many other parts of the body. The information on disease related to fibrous materials emanates from studies of occupational environments where the dose or exposure is likely to be high and continued over long periods of time. However, it has been increasingly realized that domestic or general environmental exposure is also possible and can pose grave dangers.

For a mineral fiber to be inhalable it should be less than one micron in diameter, but the length can be 10 microns or greater because the particle can align with the air stream in the bronchi and penetrate far into the lung. The ratio between length and diameter of the fiber is critical. The most dangerous fibers are very thin

(one tenth of a micron in diameter or less) with a high length-diameter ratio.

Another important factor is biodurability. Typically, the dangerous fibers are not broken down at all or only very slowly, with half-lives in the body of many years. They may remain in situ throughout life and can be found at autopsy.

There are many varieties of fibers in the environment today, both naturally occurring and man-made. Only a few, however, fulfill the above criteria and occur in amounts where human exposure is possible. The problem fibers are collectively known as asbestos and the fibrous zeolite, erionite. There are many other fibers (Skinner et al. 1988), but their contributions to human disease are not recognized.

Mineral Fibers of Medical Interest

Asbestos is not a mineralogical but a commercial term. There are six individual minerals classified as asbestos when they occur in fibrous form (Skinner et al. 1988). The chemically distinct minerals that form straight fibers are from the amphibole mineral group. The mineral names are crocidolite ("blue asbestos"), amosite ("brown asbestos"), tremolite, actinolite, and anthophyllite. However, the most common and widely used type is chrysotile, or white asbestos, which has

Table 18.1: Mineral fibers of medical interest

<i>Name</i>	<i>Relative Size</i>	<i>Biodurability</i>	<i>Use</i>	<i>Diseases</i>
ASBESTOS:				
AMPHIBOLES (straight fibers)				
Crocidolite	Very thin and long	Very high	Now little	Very high risk mesothelioma Lung Cancer, Plaques
Tremolite plaques	Thin, long	High	None Contaminant	High risk mesothelioma, lung cancer,
Amosite	As tremolite	High	Limited	As for tremolite
Anthophyllite	Thicker fibers	High	Now none	Very low risk mesothelioma Median risk lung cancer Very high incidence plaques
CHRYSOTILE (curly fibers)	Thin and short Can be very long	Fairly low	> 90% of commercial asbestos	Not certain that it can cause disease except lung cancer (see text)
ZEOLITES:				
Erionite	Extremely thin Long	High	None	Extremely high risk for mesothelioma; lung cancer unknown; plaques occur

curly fibers, and is a member of the serpentine mineral group. All these fibers may occur with different diameters and lengths, and also in their ability to resist breakdown in biological tissues (Table 18.1). Chrysotile has the fastest clearance from the body, and diseases seem to be mainly associated with exposure to the amphiboles (Churg 1982).

Whether chrysotile can cause mesothelioma has been hotly debated in the last few years. Because this asbestos variety has relatively short survival in the body, only rarely can one find high levels at autopsy even in chrysotile miners. The hypothesis is that one of the other amphibole asbestos varieties is a more likely culprit. Mesothelioma, an uncommon cancer, even in miners, has been associated with the amphibole group fibrous mineral crocidolite. The indictment that tremolite, a very common mineral in non-fibrous or blocky morphology, may contaminate commercial chrysotile ore, and be responsible for any disease, has not been proved. Fibrous, or asbestiform tremolite, occurs very rarely.

Crocidolite is the most dangerous of the asbestos fibers and is no longer mined. This fiber has a high resistance to acids, which made it a very useful industrial substance. Amosite is also rarely used nowadays. Tremolite has been mined only to a small extent but is a common contaminant in talc mines as well as chrysotile and also many other ores, such as nickel and iron. Tremolite is one of the most common asbestos minerals, occurring in many rocks all over the world. Anthophyllite has been mined, for example, in Finland and Japan, but today has no industrial use.

Erionite, finally, is not a very common mineral but is formed under certain conditions in volcanic areas of the world and is found with other zeolite minerals often in thick layers.

Diseases and Radiological Findings Caused by Asbestos and Erionite

These can be divided into malignant and benign. The first described was asbestosis, or pulmonary fibrosis, which can occur with all types of asbestos. The

lung becomes fibrotic and stiff and gas exchange dramatically decreases. It is a dose-related disease, and a fairly high exposure is necessary to cause the clinical manifestations: the patient gets more and more short of breath. Once the process has started, it continues to worsen. However, asbestosis is rare with environmental exposure but can occur after many years of slight exposure.

The other benign lesions associated with asbestos are pleural plaques. In the 1950s, it became clear that plaques were the most common manifestation of asbestos inhalation. Macroscopically, they are shining white elevations with sharp borders on the inside of the chest wall and consist of fibro-hyaline connective tissue containing very few cells. There is no inflammation within the plaques, but small cellular aggregates can be seen at their periphery, indicating low-grade inflammation there.

Over 80% of strictly defined pleural plaques are due to occupational exposure to asbestos (Hillerdal 1978, Hillerdal 1994, Karjalainen 1994). In the general population there are no "endemic" plaques but the occasional appearance without clinical symptoms may occur in individuals who had only low-level or sporadic exposure.

The relation between dose and response for pleural plaques is much weaker than that for asbestosis. The mean number of asbestos fibers or bodies in persons with plaques is as a rule higher than in the normal population (Karjalainen et al. 1994, Kishimoto et al. 1989, Warnock et al. 1982), but there is a fairly large variation and a number of persons with plaques may have values little or no different from the general population. In other words, plaques are associated with a wide range of asbestos burdens that overlaps with that found in any control population.

Plaques are more related to time after exposure than to the dose (Hillerdal 1978). Very few plaques will be seen earlier than 15 years after the first exposure to asbestos, and most will appear only after 30 years. In areas where the population is exposed from birth, the first pleural changes appear after age 30 and the incidence then increases with age. Thus, occurrence of plaques is dependent on cumulative exposure and time since first exposure (Järholm 1992). Many plaques are not

seen until long after exposure has ceased. Once seen, they will slowly grow larger over the years, and with time many will calcify (Hillerdal 1978).

The malignant diseases due to asbestos exposure are mainly lung cancer and malignant mesothelioma. Other tumors in the body have been claimed to be more abundant in asbestos workers, but the scientific proof varies and they can be disregarded in this review.

Lung cancer is mainly a disease caused by smoking. However, exposure to asbestos increases the risk in a dose-related manner. Most data point to a multiplicative effect. For example, it can be estimated that smoking 20 cigarettes a day will increase the risk of lung cancer at least 10 times. If this smoker has an "average" occupational exposure to asbestos – say, he has been a construction worker – the risk will double to 20 times. Thus, asbestos exposure will more likely result in smokers developing lung cancer. In a non-smoker exposed to asbestos, the risk of developing a lung cancer is very small. In other words, most "asbestos lung cancers" are also "smokers' cancers." Lung cancer is thus a difficult disease to use as a marker of low dose exposure to mineral fibers, since smoking is overwhelmingly the primary cause.

Malignant mesothelioma, a very rare cancer, has no correlation to smoking. Apart from a small basal incidence due to unknown causes (and some other, very rare causes such as radiation or old pleural scars), all cases can be considered to be due to exposure to asbestos (or erionite). The tumors grow slowly in the pleura, restricting the action of the lung and causing the death of the patient often within a year of diagnosis. No curative treatment exists, though some cytostatics seem to have effect on the tumor. The disease is dose-related (Hillerdal 1999), but even a slight exposure can be enough. Like pleural plaques, the appearance is usually more than 30 years after exposure. Rarely, the disease starts in the peritoneum.

Thus, two findings — pleural plaques and malignant mesothelioma — are useful as "sentinel" diseases, indicating that asbestos (or erionite) exposure has occurred.

ficult, as in the erionite case in Turkey, where the problem is not any particular use of the substance but rather that it occurs in the ground. The only solution is then to move the village. Sometimes things can go wrong, as in the example of Karain Village. The press, including of course the Turkish, obtained the story and headlines such as "Village of Death" were seen on articles worldwide. This caused not only villagers to have difficulties marrying outside the village but even such problems as people refusing to buy vegetables or other products from the area. Naturally, such problems will sour the relations with researchers. One must be careful and try to avoid such unintended results of any intervention. Cooperation between the inhabitants and the medical profession and officials is very important and it must never be forgotten that the goal is to help the population.

References

- Baris YI, Saracci R, Simonato L et al. Malignant mesothelioma and radiological chest abnormalities in two villages in central Turkey. An epidemiological and environmental investigation. *Lancet* 1981; I, p.984-987.
- Baris YI, Bilir N, Artvinli M et al. An epidemiological study in an Anatolian village environmentally exposed to tremolite asbestos. *Brit J Industr Med* 1988; v.45, p.838-840.
- Churg A. Fiber counting and analysis in the diagnosis of asbestos-related disease. *Hum Pathol* 1982; v.13, p.381-392.
- Constantopoulos SH, Saratzis N, Kontogiannis D et al. Tremolite whitewashing and pleural calcifications. *Chest* 1987; v.92, p.709-712.
- Epler GR, Gerald MXF, Gaensler A, Carington CB. Asbestos-related disease from house-hold exposure. *Respiration* 1980; v.39, p.229-240.
- Hillerdal G. Pleural plaques in a health survey material. Frequency, development, and exposure to asbestos. *Scand J Respir Dis* 1978; v.59, p.257-263.
- Hillerdal G. Pleural plaques and risk for bronchial carcinoma and mesothelioma. A prospective study. *Chest* 1994; v.105, p.144-50.
- Hillerdal G. Pleural plaques: Incidence and epidemiology, exposed workers and the general population. A review. *Inddor Built Environ* 1997; v.6, p.86-95.
- Hillerdal G. Mesothelioma: casdes associated with non-occupational and low-dose exposures- *Occup Environ Med* 1999; v.56, p.505-13.
- Järholm B. Pleural plaques and exposure to asbestos: a mathematical model. *Int J Epidemiol* 1992; v.21, p.1180-1184.
- Karjalainen A, Karhunen PJ et al. Pleural plaques and exposure to mineral fibers in a male urban necropsy population. *Occup Environ Med* 1994; v.51, p.456-460.
- Kilburn KH, Lilis R, Anderson H et al. Asbestos disease in family contavts of ship yard workers. *Am J Public Health* 1985; v.75, p. 615-617.
- Kishimoto T, Ono T, Okada K, Ito H. Relationship between number of asbestos bodies in autopsy lung and pleural plaques on chest X-ray film. *Chest* 1989; v.95, p.549-552.
- Kiviluoto R. Pleural calcification as a roentgenologic sign of non-occupational endemic anthophyllite asbestosis. *Acta Radiol* 1960; Suppl 194.
- Metintas M, Özdemir N, Hillerdal G et al. Environmental asbestos exposure and malignant pleural mesothelioma. *Respir Med* 1999; v.93, p.349-55.
- Meurman LO. Pleural fibrocalcific plaques and asbestos exposure. *Environ Res* 1968; v.2, p.30-46.
- Moorcroft JS, Duggan MJ. Rate of decline of asbestos fibre concentration in room air. *Ann Occup Hyg* 1984; v.28, p.453-457.
- Puffer JH, Germine M, Hurtubise DO et al. Asbestos distribution in the central serpentine district of Maryland - Pennsylvania. *Environ Res* 1980; v.23, p.233-246.
- Raunio V. Occurrence of unusual pleural calcification in Finland. Studies on atmospheric pollution caused by asbestos. *Ann Med Int Fenniae* 1966; v.55 Suppl. 47.
- Rohl AN, Langer AM, Selikoff IJ, Nicholson WJ. Exposure to asbestos in the use of consumer spackling, patching, and taping compounds. *Science* 1975; v.189, p.551-553.
- Skinner, H.C.W., Ross, Malcolm and Frondel, Clifford, 1988, *Asbestos and other fibrous materials: mineralogy, crystal chemistry and health effects*. Oxford University Press., New York.
- Warnock ML, Prescott BT, Kuwahara TJ. Numbers and types of asbestos fibers in subjects with pleural plaques. *Am J Pathol* 1982; v.109, p.37-46.
- Whysner J, Covello VT, Kuschner M et al. Asbestos in the air of public buildings: a public health risk? *Prev Med* 1994; v.23, p.119-125.
- Yazicioglu S, Ilcayto R, Balci B et al. Pleural calcification, pleural mesotheliomas and bronchial cancers caused by tremolite dust. *Thorax* 1980; v.35, p.564-569.