Polish Journal of Environmental Studies Vol. 9, No. 6 (2000), 443-454

Review

Sources and Effects of Asbestos and Other Mineral Fibres Present in Ambient Air

L. Wachowski, L. Domka

Faculty of Chemistry, Adam Mickiewicz University,
Grunwaldzka 6, 60-780 Poznań, Poland

Received: June 15, 2000
Accepted: July 10, 2000

Abstract

This review describes the structure, physicochemical properties and sources of asbestos as well as other mineral fibres in the natural environment. The diversity of character and methods for the determination of airborne inorganic fibres are discussed with particular attention to the biological effects on humans health. It also demonstrates the practical significance of asbestos.

This article also shows some possibilities transformation of asbestos containing materials into harmless products.

Keywords: mineral fibres, structure, physicochemical properties and application of asbestos, environmental exposure to mineral fibres, methods modification into harmless products

Introduction

Asbestos belongs to a group of ten substances established to have the greatest carcinogenic potential for malignant cancers such as two types of lung cancer (lung tissue and mesothelioma), pharynx cancer, larynx cancer, colon cancer and stomach cancer [1-7]. It is listed as the third most abundant pollutant in the global scale [4, 5]. The health risk associated with exposure to asbestos depends on the type of mineral used in the product, concentration of asbestos dust, size of inhaled fibres and time of exposure [7-9]. From many factors enhancing negative effects of asbestos fibre presence in ambient air the most important is cigarette-smoking [8]. The possibility of exposure to commonly applied asbestos and other mineral fibres has led to the introduction of legal regulations significantly restricting or completely forbidding their use [10-12]. Also, the use of materials containing asbestos is strictly controlled. In Poland asbestos has been listed in a group of 5 substances whose use is heavily fined. Much effort has been made to replace asbestos by environmental-friendly materials and to devise effective methods of its elimination.

Structure of Silicates

So far over 200 varieties of natural silica (SiO_2) have been identified and the number of names assigned to them by geologists exceeds 100 [13-20]. Together with different metal oxides silica is the main component of lava which on pouring onto the earth crest forms different complex compounds known as silicates [13, 17-19]. X-ray structural studies of this group of compounds have provided explanations of their structure and helped establish the rules of their system [9, 13]. The fundamental structural units of all silicates are SiO_4 tetrahedrons of a silica atom surrounded by four oxygen atoms, Fig. 1. The tetrahedrons can undergo condensation, which leads to the formation of a polymer chain structure with the structural unit of a co-ordination polyhedron SiO_3^{-2}. The proportion of their occurrence determines the kinds of connections among the polyhedrons and between the polyhedrons and metals. Assuming as a criterion the kind of connections among the co-ordination polyhedrons, the silicates have been divided into groups characterized by 5 typical arrangements of the elementary tetrahedrons SiO_4 [13, 15].

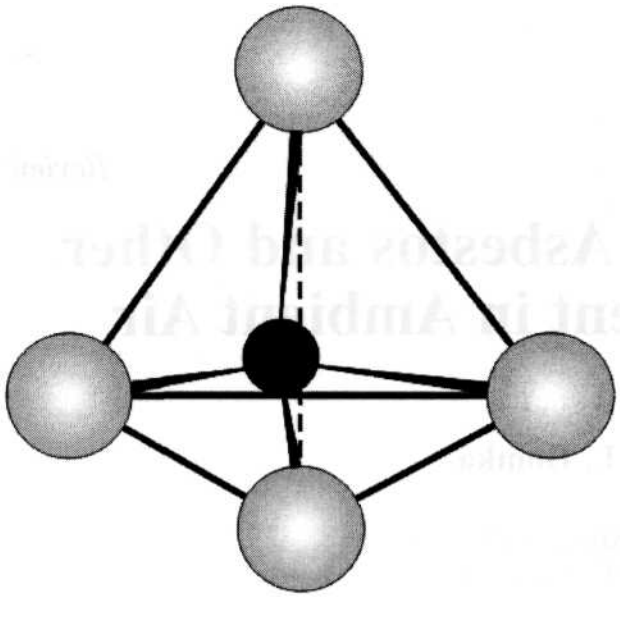

Fig. 1, SiO_4 tetrahedron.

The most important of the arrangements are: the chain structure made of individual polymer chains in which $(SiO_3)^{2-}{}_n$ anion is the group repeated along the z axis throughout the whole crystal (Fig. 2a), and the ribbon structure in which two polymer chains are joined by free corners while the main structural unit is the $(Si_4O_{11})^{6-}{}_m$ anion (Fig. 2b) [9, 13, 14].

The oxygen-silica chains, both single and double, do not occur as individual units but as double layer packets. They are composed of silicon-oxygen layer deposited on the metal-oxygen layer made of the oxygen octahedrons with different cations (mostly magnesium) inside (Fig. 3a) [6, 7]. These packets are neutral macromolecules, which are arranged into the crystal structure [9, 13, 14, 20].

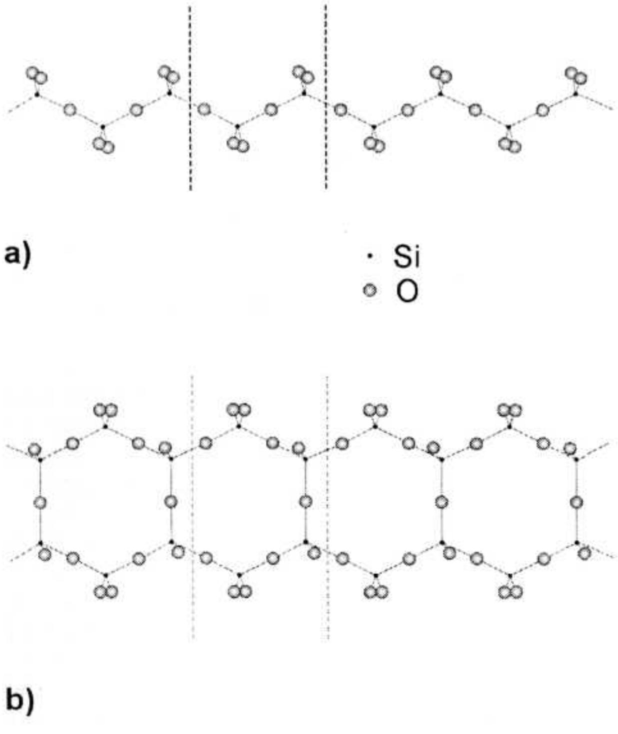

Fig. 2. Oxygen-silica complexes: a) $(SiO_3)^{2-}{}_n$ chains and b) $(Si_4O_{11})^{6-}{}_m$ ribbons.

Structure of Asbestos and Other Natural Mineral Fibres

Asbestos, meaning indestructible or unquenched in Greek, is the commercial name applied to a group of crystalline and relatively insoluble silicates of chain or ribbon structure. The group includes 6 minerals one of which occurs in the silicates of the serpentite group characterized by the chain structure [7, 13,14, 19, 20], and the others belong to amphiboles with ribbon anions [6, 9, 13-16]. A representative of the first group is chrisotile (Fig. 3), and the other includes crocidolite, amosite, anto-phyllite, tremolite and actinolite, Fig. 4, [7, 9, 18, 19, 21-24].

The bonds within the silica-oxygen chains in the two above-mentioned silicate structures are relatively strong,

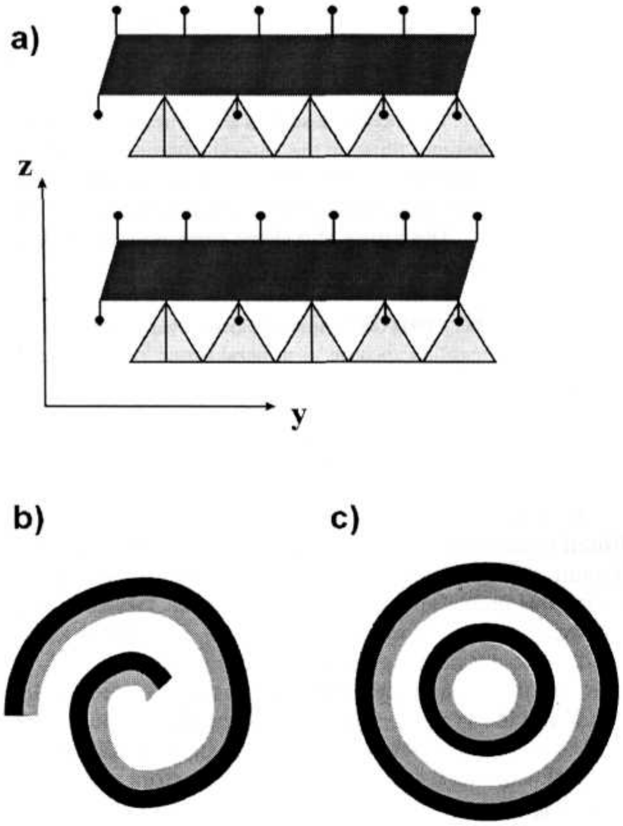

Fig. 3. The structure of chryzotile, mineral from the group of serpentine, as an example of the chain structure; a) the ideal structure of chrisotile viewed along the x axis (the triangles represent SiO_4 tetrahedrons, whereas the darkened band stands for the layers made of the co-ordination octahedral of $[Mg(OH)_6]$), b) chrisotyle structure in the form of a roll darker bonds stand for the layer of octahedrons and c) chrisotile structure in the form of the coaxial empty pipes.

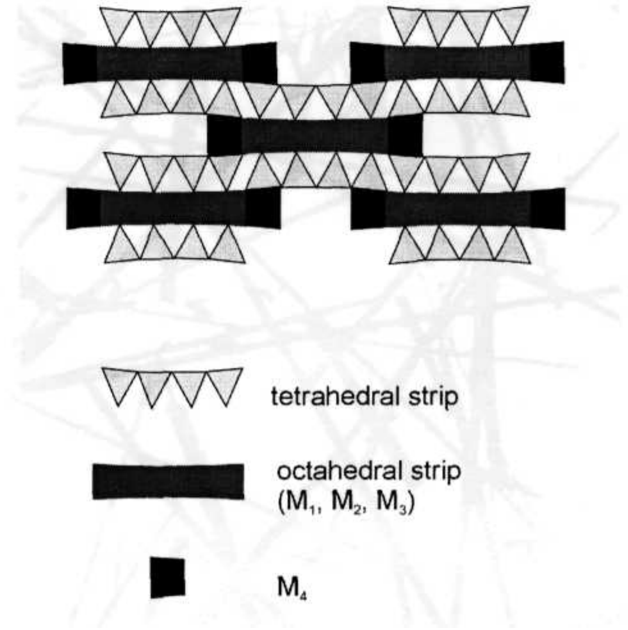

Fig. 4. The ideal structure of amphiboles along the z axis as an example of ribbon-type structure; the triangles denote the SiO_4 tetrahedrons, the darkened band is the metal-oxygen layer made of octahedrons with the cationic positions (Mi, M_2 and M_3) occupied by Mg^{2+}, Fe^{2+} and Fe^{3+} ions, and M_4 positions dominated by Na^+ ions (reprinted from Ref. 20).

whereas the connections between the chains are based on weak electrostatic interactions [14, 7, 9, 18, 19, 20]. Because of the character of the spatial lattice, the asbestos minerals cleavage mainly in the parallel to the x axis, so have fibrous structure made of aggregations of relatively long (up to 30 cm) thin (diameter 0.01 to 0.40 mm) and elastic fibres resembling glass fibres. They can be arranged into bundles, warps or clusters [9, 16, 17, 23].

Chemical and Physical Characterization of Asbestos and Other Natural Mineral Fibres

According to the chemical composition of asbestos minerals, they can be classified as hydrated silicates of magnesium, iron, calcium, sodium and other elements [6, 9, 14, 18, 22]. These minerals differ by the content of the main components (SiO_2, $A1_2O_3$, Fe_2O_3, FeO, MgO, CaO, Na_2O and H_2O), colour (from white to pale green, yellow, pink, blue, pale grey, pale brown, dark green), density, melting point (from 1200 to 1500°C) resistance to acids and bases, arrangement of fibres (i.e. texture - elastic, silk-like and hard) and mechanical properties (resistance to stretching and rotation) [7, 9, 16-20, 23].

Commercially available asbestos minerals include: - chrisotile (white asbestos) $Mg_3[Si_2O_5](OH)_4$, one of the minerals most commonly met in the earth crust; its melting point 1500°C, poor conductor of heat, electricity and sound; resistant to high temperatures because of a high concentration of magnesium oxide; this mineral is characterized by uniform chemical composition and its structure is composed of relatively long (1-20 mm, exceptionally 100 mm) and elastic fibres whose elementary fibriles (from 0.02 to 0.025 mm in diameter) can be woven or felted; the fibres resemble coils (Fig. 3b) or empty pipes (Fig. 3c), and usually form bundles of the size determined by the kind and degree of mechanical processing (Fig. 5a) [14, 21-24].

- crocidolite (blue asbestos), $Na_2Fe^{III}{}_2(Fe^{II},Mg)_3Si_8O_{22}(OH)_2$,
- amphiboles of variable chemical composition and the melting point of 1200°C, it is built of shorter fibres of smaller diameter, which easily break into smaller ones (it is brittle) and show high resistance to acids. The contribution of this mineral in the total amount of mined asbestos reaches 4% (see Fig. 5b.);
- amosite (brown asbestos), $(Fe^{II},Mg)7Si_8O_{22}(OH)_6$, belongs to the group of amphiboles, its melting point is 1400°C (see Fig. 5c.) [9, 14, 21-24, 15].

The other much rarer minerals from the group of amphiboles characterized by ribbon anions are: anthophylite $(Mg,Fe)_7[Si_4O_{11}]_2(OH)_2$, tremolite $Ca_2Mg_5[Si_5O_{11}]_2(OH)_2$ and actinolite $Ca_2(Mg,Fe)_5[Si_4O_{11}]_2(OH)_2$, [18, 21, 25]. Other natural mineral fibres, which are potentially hazardous for human health are:

- erionite, $NaK_2MgCa_{15}[Al_8Si_{28}O_{72}] \bullet 28H_2O$, which belongs to a group of over 30 natural crystalline aluminosilicates known as zeolites [9, 17,18], the fibres of this mineral are of a size similar to that of asbestos (see Fig. 6.) [14, 21, 26];
- wolastonite, $Ca_3[(Si_3O_9)]$, which belongs to fibrous silicates and is considered one of the potential mineral replacements of asbestos [7, 9, 14, 21, 24];
- attapulgite (polygorskite), $Mg_5[Si_4O_{11}]2(OH)_4(H_2O) \bullet 4H_2O$, is characterized by the chain-ribbon structure close to that of montmorillonite and is built of fibres of up to 5000 µm resembling empty pipes or rods [7, 9, 24];
- sepiolite, $[Mg_8Si_{12}O_{30}](OH)_4(H_20)_4 \bullet 8H_2O$, belongs to the class of clay minerals, which can occur in the form of fibres or floccules, e.g. sepiolite (sea-foam) is a compact form of this mineral [7, 9, 13, 21].

The properties of the asbestos minerals which determine its stability in the natural environment and its biological aggressiveness are related to their fibrous structure, in particular to the length and diameter of the fibres [7, 9]. These properties include: high durability (they do not evaporate and are insoluble in water so do not change in time), are strongly resistant to mechanical stress and temperature (refractory), show very low electrical and sound conduction, are resistant to chemical reagents and to corrosion, are very elastic, show low thermal conductivity and specific sorptive and insulating features [7, 9, 21-23].

Asbestos in the Environment and Its Practical Significance

Asbestos minerals are common in many geological formations as accompanying minerals and are widespread throughout the world [6, 9, 15, 17]. Archaeological studies show that 4000 years ago asbestos was used by man for production of ceramic dishes to make them

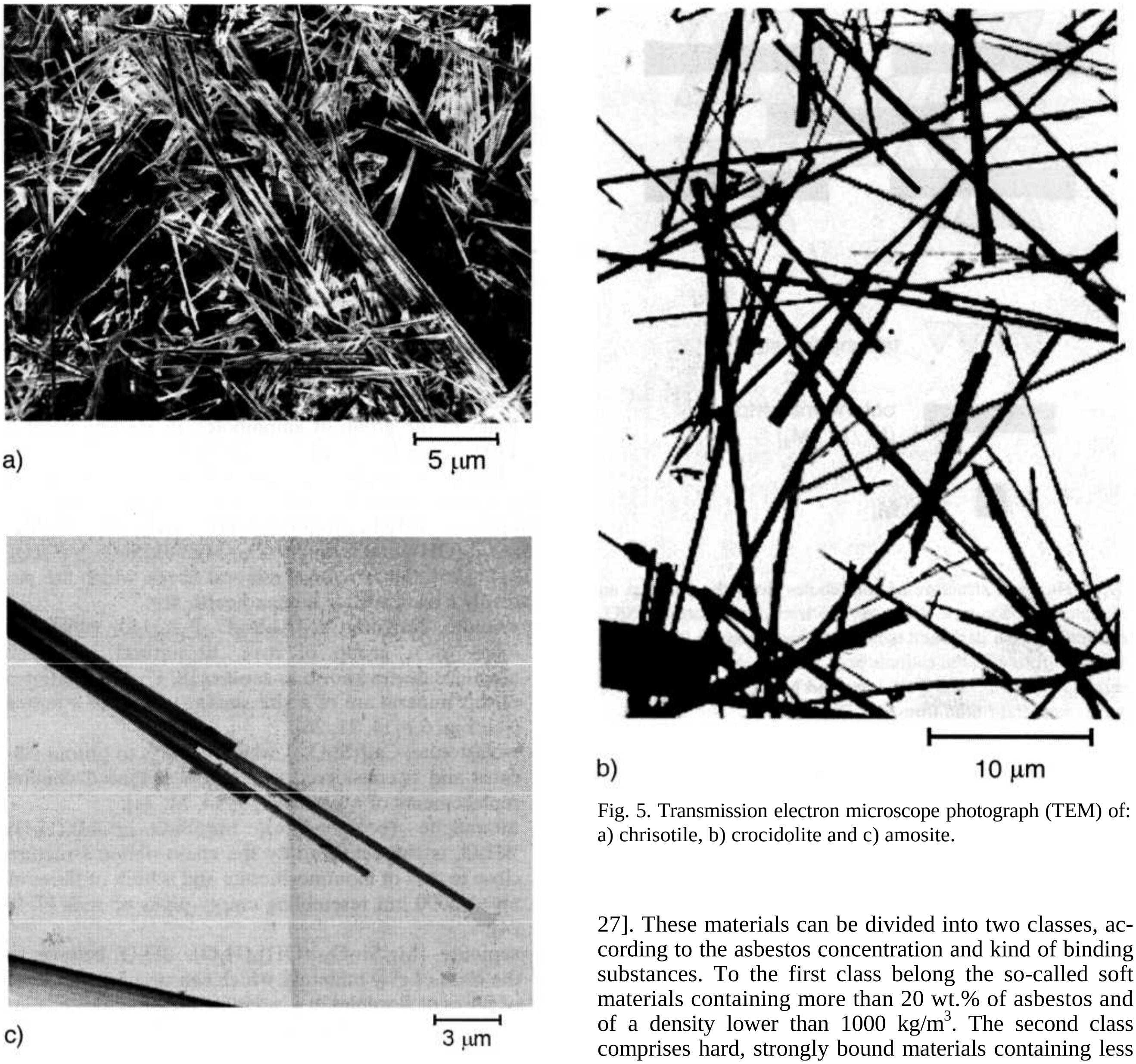

Fig. 5. Transmission electron microscope photograph (TEM) of: a) chrisotile, b) crocidolite and c) amosite.

more mechanically and thermally resistant. The ancient Greeks used asbestos for production of fabrics, e.g. for special wear for dead notables, tablecloths and oil lamp wicks [7, 25, 27].

The first quarries which performed systematic excavation of chrisotile were established in the Ural Mountains in about 1700 and in Quebec province in Canada in 1870. The exploitation of amosite began in Australia in 1916, and crocidolite in South Africa in 1980 [7, 25]. The chrisotile deposits suitable for exploitation are mostly found in Canada, South Africa and Zimbabwe. Until recently this mineral was excavated in over 40 countries. The asbestos minerals from the amphibole group are limited to Australia and South Africa. The main producers of asbestos in the world are the former USSR countries and Canada [7, 21, 22, 25].

Owing to the above-mentioned properties of asbestos minerals, they have been commonly used for production of asbestos-containing materials (ACM), in which their contribution varies from 1 to 100% wt. [7, 19, 25, 27]. These materials can be divided into two classes, according to the asbestos concentration and kind of binding substances. To the first class belong the so-called soft materials containing more than 20 wt.% of asbestos and of a density lower than 1000 kg/m^3. The second class comprises hard, strongly bound materials containing less than 20 wt.% of asbestos. The latter are more mechanically resistant and thus are less hazardous for the natural environment [28].

The asbestos-cement industry, the largest consumer of asbestos minerals, started developing in 1850. The commercial production of thermal insulating materials began in 1879. In Great Britain at the beginning of the 20th century asbestos was used for production of bed-mattress recommended as the best. In the USA asbestos was used since the beginning of the 20th century, mainly as thermal insulation for steam engines. With the development of technology, asbestos found increasing use in many fields, mainly in the building industry [6, 7, 9, 11]. According to data from 1983, annual world production of asbestos was about 5 mln tons, which was used for production of over 3000 kinds of products [7, 11]. The first on the list of asbestos consumers was the Democratic Republic of Germany - 3 kg per head. Poland, using 1.5 kg of asbestos minerals per head was fifteenth. The most important from the point of view of industrial applications was chrisotile,

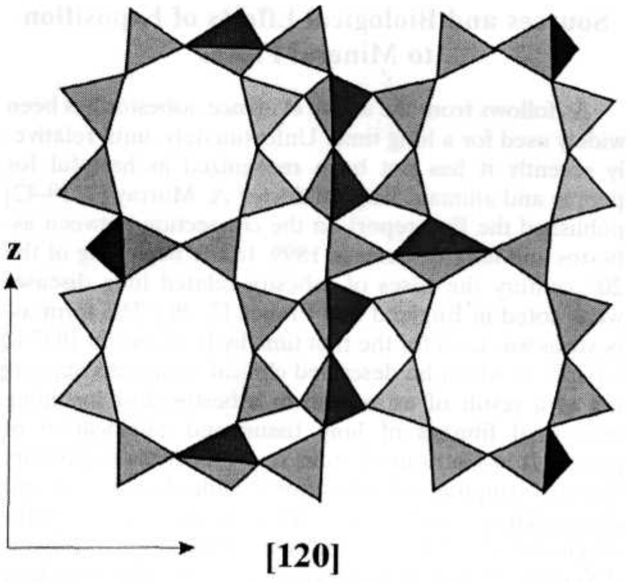

Fig. 6. The ideal structure of erionite along the x-axis (reprinted from Ref. 9).

making 95% of the mass of the asbestos used [5, 9]. The other varieties of this mineral are not treated as raw material for industry but can be chrisotile admixtures [6, 14, **17]**.

The commercial products based on asbestos minerals can be classified into the three following groups:

- the materials made of loosely bound fibres of pure asbestos and different materials of inorganic origin such as cement, gypsum, diatomaceous earth, hydrated calcium silicate, basic magnesium carbonate etc.;
- strongly bound compositions of asbestos fibres with inorganic materials (Portland cement, hydrated calcium silicate) and organic materials (elastomers, plastic mass of different kinds, bituminous mass);
- fabrics containing asbestos [7].

The largest amounts of asbestos materials have been used for production of:

- asbestos-cement composites for building industry e.g. asbestos cement, asbestos cardboards, large-size sewage and water-main pipes, in which the concentration of asbestos is relatively low e.g. of 10-15% wt.;
- refractory plates and plates resistant to chemical agents, insulating plasters, containing from 25 to 40% wt of asbestos, insulating materials and asbestos-rubber plates for production of packings, containing from 25 to 85% wt of asbestos;
- frictional materials (over 200 types of brake and gear linings) containing up to 70% wt. of asbestos;
- vinyl-asbestos tiles and floor coverings containing up to 7.5% wt. of asbestos;
- paper, cardboard and felt in which the content of asbestos varies from 25 to 98% wt.;
- yarn and insulating fabrics for protective suits, gloves, refractory blankets and electric insulation, containing from 25 to 98% wt. of asbestos [11, 25].

Fibrous zeolites as e.g. the above-mentioned erionit are used in the ion-exchange processes for retaining nitrogen in fertilizers and in production of concrete [7, 11, 29, 31].

Transport of Mineral Fibres in Natural Environment

The effect of atmospheric factors on asbestos minerals (e.g. wind erosion of geological materials leading to chemical changes) as well as the use of asbestos containing products lead to disintegration of the macroscopic fibres into individual fibres and their release into the environment, which means that it is everywhere. The released fibres are of a diameter usually smaller than 0.3 µm (a few times thinner than a human hair, see Fig. 7.) easily float in the air (a low rate of sedimentation) and can hardly be noticed with the naked eye [6,7,11,30, 31].

The microfibres are transported and dispersed mainly by water and air [6, 7, 12]. In the air asbestos as well as other mineral fibres form a stable suspension (atmospheric aerosol) which can move over significant distances from the source of emission. The rate and direction of their dispersion are determined by the status of the atmosphere (cloudiness, precipitation, strength and direction of wind, temperature, humidity etc.) in the lower parts of the troposphere. As shown by the relevant calculations, the concentration of minute size fibres in the air very slowly decreases with increasing distance from the point sources [6, 7, 12, 32-34].

Into water ecosystems asbestos penetrates as a result of dissolution of minerals and ores in which it occurs, with precipitation from the air, with industrial wastes and

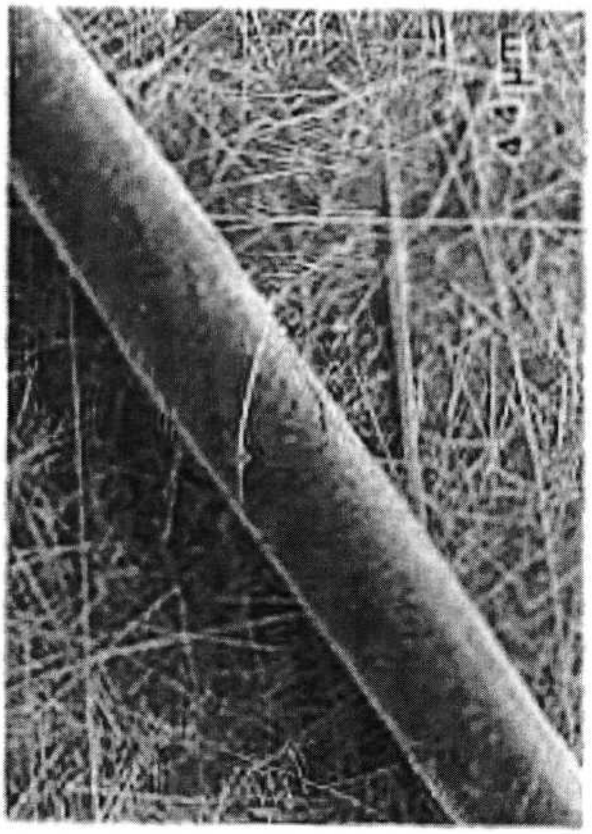

Fig. 7. Microphotograph (TEM) a human hair against a background of chrosotile fibres.

contact of water with sewage and water-main pipes [6, 7, 12]. The presence of asbestos in drinking water was for the first time detected in 1971 in Canada [7]. Through water ecosystems asbestos fibres are dispersed over significant distances from the source of emission [6].

Identification and Quantification of Mineral Fibres in Ambient Air

The presence of asbestos minerals in ambient air can be detected via pertinent colour tests dying asbestos fibres in shades of blue or red [7, 35]. In contrast to the majority of harmful substances in ambient air, the presence of inorganic fibres can be easily determined (abundance and kind) by optical and electron microscopy observing the deposits on membrane filters after air filtration [35-37].

The methods for determination of asbestos fibres differ in the mode of sample collection, instruments used and techniques of fibre counting [7, 11, 35-38].

The fibres deposited on membrane filters can be performed by the following methods.

- using a phase-contrast optical microscope (PCM), for the first time applied in 1966, assuming as dangerous the fibres of the length to diameter ratio > 3:1, which corresponds to a length greater than 5 µm and a diameter smaller than 3 µm; the fibres represent the biologically active part of respirable fraction (penetrating the air sacks) [32, 36, 37];
- transmission electron microscope (TEM) [7, 32, 35, 38], equipped with an energy dispersive X-ray analyzer (EDXA) [9, 35, 36];
- X-ray diffractometer (XRD) [7, 36, 37];
- scanning electron microscope (SEM) [7, 32, 38], coupled with an energy dispersive X-ray diffractometer (EDXA) [7, 35, 36];
- selected area electron diffraction (SAED) [7, 35, 36];
- laser microprobe mass spectrometry (LMMS) [7, 32, 35, 38];
- polarized light microscopy (PLM) [7, 36, 37].

Quantitative determinations of the contents of asbestos fibres in the air have been performed using a phase-contrast microscope equipped with a (grid, grating) eyepiece [37]. With this microscope the concentration of asbestos fibres in the air at a work-place expressed as numerical concentration (e.g. the number of fibres F present in 1 cm^3 or in 1 m^3), the concentration of asbestos fibres in ambient air is expressed as the number of fibres in 1 dm^3, 1 m^3 or as weight concentration in mg/m^3 or ng (1 ng = 10^{-9} g) in 1 dm^3 or 1 m^3 of air. In biological material the concentration of fibres is expressed as the number of fibres F or the content of fibres in µg per 1 g of dry tissue [7, 37].

The use of electron microscopes equipped with auxiliaries permit differentiation between the fibrous and non-fibrous particles, identification of the type of asbestos minerals and their structure and determination of particular elements in a sample of fibrous material [6, 35-38].

Sources and Biological Effects of Exposition to Mineral Fibres

As follows from the above evidence, asbestos has been widely used for a long time. Unfortunately, until relatively recently it has not been recognized as harmful for people and animals. English doctor A. Murray [7, 39-42] published the first report on the connection between asbestos and lung diseases in 1899. In the beginning of the 20th century the cases of asbestos-related lung diseases were noted in England and France [7, 39]. The term asbestosis was used for the first time by B. Cooke in 1927 in a paper in which he described clinical symptoms appearing as a result of exposition to asbestos dust including interstitial fibrosis of lung tissue and calcification of pleura. It is a serious chronic, non-cancerous respiratory disease. Symptoms of asbestosis include short breath and dry crackling sound in the lungs while inhaling. The pathological studies have proven that the progress of fibrosis development is slow, but it continues even after breaking the exposition to asbestos dust [26, 40, 41]. In 1935 the Americans B. Lynch and J. Smith [7, 39] described the first case of asbestosis leading to lung cancer, while the first documented case proving a relationship between exposure to asbestos and development of malignant cancer of pleura or peritoneum mesothelioma was reported by J.C. Wagner in 1960 [43, 44]. A result of exposure to asbestos can also be a rare malignant cancer known as mesothelioma (in the USA about 200 cases annually) initiated by all asbestos minerals [17, 22, 36]. The first case of mesothelioma was described by Weiss in 1953 [7, 31, 45-47].

The following types of exposure to asbestos dust have been distinguished:

- occupational (indirect and direct) related to employment in asbestos mining, treatment of raw asbestos (milling), production of asbestos containing products, transportation of asbestos, building conservation (exchange of asbestos packing), repairs or demolition of buildings constructed with the use of asbestos materials, transformation of asbestos into harmless products [7, 33, 48-52];
- non-occupational (environmental exposition), related to living in the neighbourhood of asbestos mines or plants producing asbestos products, or living in one household with persons of occupation related exposure to asbestos dust [7, 50, 53];
- general of the whole population related to the presence of asbestos pollution in ambient air [6, 7, 58-62].

The processes of asbestos mining, conversion and application, along with natural erosion of asbestos minerals, may cause a significant increase in the concentration of asbestos fibres in ambient air. The amount and size of respirable asbestos fibres released into ambient air depends on the kind of product and its processing. Erosion of natural sources of asbestos and release of asbestos fibres from sewage pipes and water-main pipes depend on the aggressiveness of water determined by the content of carbon dioxide, pH value, general basicity etc. [6, 7]. The data collected so far prove that the most important threat is inhalation of single dry fibres in closed premises [7, 21, 48]. The fibres absorbed via the alimentary track (in drinking water or food products) can be responsible for the increasing number of cancer cases in a population under a long-term exposure to asbestos dust [56, 57].

The period of latency, that is the time between the first contact with asbestos and the appearance of the first symptoms, is long and may reach from 10 to 30 years [7, 24, 30, 40]. The health risk related to exposure to asbestos and other mineral fibres depends on many factors, including: mineral composition, size of fibres, Mg/Si proportion, degree of fibre condensation, concentration of asbestos dust, time of deposition in the lungs, time of exposition, mode of penetration into organism (inhalation, intake, skin), other accompanying substances (e.g. cigarette smoke, cement dust) and individual features of the organism (age, sex, genetic inclinations, style of life and health status) [7, 21, 40].

According to epidemiological data, the cancer rate in the population of men smoking cigarettes is 12-fold higher than in non-smokers, while for women it is 7.7-fold higher in smokers than in non-smokers [8, 58-61].

The number and complexity of factors affecting biological aggressiveness of asbestos makes it impossible to give a simple assessment of the health risk or determine a dose-response relation [7, 61]. Another factor making it difficult is the long latency time [39, 40].

An important factor in aetiology of asbestos-related diseases is the size of the fibres, in particular their diameter [6, 7, 38, 62, 63]. It has been shown that fibres longer than 8 µm and of a diameter smaller than 1.5 µm are the most potent cause of malignant cancers of the human respiratory system [7, 22, 39, 62, 63]. Moreover, it has been established that the smaller the fibre diameter the greater the number of fibres per a mass unit of asbestos dust, which increases the chance of their inhalation and penetration into deeper parts of the lungs [7, 9, 39, 60]. The fibres deposited in air sacks are mainly 1-10 µm, long and as many as 90% of them are fibres shorter than 5 µm [62-65]. Fibres shorter than 1 µm are easily inhaled but their retention in the lungs is insignificant, whereas fibres longer than 10 µm deposit on mucous membrane or other first elements of the respiratory tract and do not reach the air sacks [62, 63].

Environmental Exposure to Mineral Fibres

Asbestos fibres occurring in the atmospheric air make a relatively small fraction of the fibrous aerosol. It has been assessed that the amount of fibres from natural sources is probably higher than that from man-made sources [4, 9, 13, 32]. A study of the chemical composition of the minerals in cores collected from ice cover on Greenland has shown the presence of chrisotile fibres long before man started to mine and process this mineral [6, 7, 21, 62].

The common use of asbestos for production of boards for thermal and acoustic insulation at schools and in other public buildings (hotels, office buildings) has substantially increased the number of people exposed to the hazardous effects of asbestos dust. For example, data published by the Environmental Protection Agency (EPA) revealed the hazardous health conditions due to exposure to asbestos fibres in 107,000 schools and 733,000 public buildings in the USA [30, 59]. It has been estimated that assuming as 1.0 the concentration of asbestos fibres in the air in asbestos-free buildings, their concentration in ambient air is equal 1.95, in the air in the buildings constructed with asbestos-containing materials in good state it is 2.95, whereas in the buildings constructed with asbestos-containing materials in which they had eroded the concentration of asbestos fibres reaches 3.65 [29, 30, 40].

The level of the occupational and environmental hazard shows considerable local fluctuations depending on the distance from the sources of mineral fibres. For example at a distance of 300 m, with the wind, from a plant producing asbestos-containing materials the concentration of the fibres in the air was 2200 F/m^3, at a distance of 700 m it dropped to 800 F/m^3, while at a distance of 1000 m it dropped to 600 F/m^3 [6, 7, 21, 26, 40, 48]. In the air at remote villages the concentration of asbestos fibres longer than 5 µm is usually lower than 3 F/m^3, while in the air of the urban areas the mean values of the fibre concentrations can be 100 times higher (3-300 F/m^3) [7, 37, 52, 62]. In the air near high traffic roads the concentration of mineral fibres is elevated, but dominant there are chrisotile fibres shorter than 5 µm, which are not taken in regard to estimation of the dose-response relation in people [7, 40, 43, 61-65].

As shown by numerous studies, exposure to asbestos fibres indoors (indoor air) is usually greater than in open air. The level of their concentration in the air depends on the ventilation systems, type of asbestos-containing materials, kinds of processing of asbestos materials (grinding, cutting, conservation procedures, construction, etc.) types of asbestos-containing products (thermal insulation, floor tiles, plaster etc.) and its technical status. The study conducted in different public buildings in New York proved an elevated level of asbestos fibre concentration [59]. The mean concentration of fibres in public buildings was diverse and varied from 2000 to 510 and 190 F/l m^3 of ambient air [29, 54, 62].

The eroding asbestos-containing insulating layers (boards) were identified as the main source of asbestos fibres [35, 49, 52, 53, 62].

It has been estimated that in the USA about 1.3 mln workers are exposed to elevated level of asbestos mineral fibres in ambient air [30, 36, 59]. This concerns in particular the workers employed at construction or demolition of buildings or devices in which asbestos or asbestos products have been used [33].

Biological Effect of Inhaled Mineral Fibres

The health risk of exposure to dust containing asbestos and other natural fibres is related to the fact that the dust can be inhaled to the lungs, where fibres are arrested. The fibre remaining in the lungs can become a site of deposition of iron-containing proteins which form club-like iron bodies [7, 66-74], Usually the cores of these bodies are the amphibole fibres. Their prolonged presence in the lungs can induce various organic changes, sometimes many years after the first exposure [41].

The most important factors affecting the development of various diseases of the respiratory system include:

- the time of exposure (when the contact with asbestos-containing dust is intense asbestosis can develop in a relatively short time of 5 years, in average exposition in current environmental conditions it is rarely detected in time shorter than 20 years after the first contact [7, 11, 12, 62, 64, 65];
- time after the first contact with asbestos dust [7, 53, 62],
- cigarette smoking [62];
- type of fibres we are exposed to (for example tremolite, amosite and in particular crocodolite can be more biologically aggressive than chrisotile); moreover, the probability of deposition in the lungs is lower for the fibres of symmetric structure than for the asymmetric ones [40, 47, 49, 63];
- the size of fibres; results of previous studies suggest that the fibres longer than > 5 µm and of diameter lower than 3 µm are more dangerous than the short fibres (shorter than 1 µm), the fibres of diameter greater than 3 µm cannot penetrate the air sacks in the lungs (are not respirable), and fibres shorter than 5 µm can be removed from the lungs through microphages (i.e. small cells being elements of the reticuloendothelial system [7, 61, 65].

The diagnostic means most often used for detection of asbestosis is roentgenography. Although this method cannot detect the presence of asbestos fibres directly as it only provides the information of the changes in the lungs, which can be caused by different fibres, it is still indicative of the effects caused by exposure to asbestos [24].

Prolonged exposure to asbestos-containing dust can lead to development of the following changes:

- asbestosis, a chronic disease causing disseminated interstitial pulmonary fibrosis and thickening and pleural plaque appearance; fibrosis develops slowly and proceeds even after cessation of exposure [7, 20, 24, 64];
- malignant lung cancer (mesothelioma of pleura, cancer of the lungs, cancer of the larynx and pharynx); the risk of developing cancer of the lungs and mesothelioma is greater the longer and thinner the fibres inhaled [7, 31, 26, 39-42];
- cancer of the intestines [7, 56];
- impairment of the ventilatory function of the lungs [6, 7];
- undesirable influence on the course and treatment of some other diseases of the respiratory system such as (chronic bronchiolitis, chronic upper respiratory tract infection);
- circulatory-respiratory failure [7].

Results of the epidemiological studies unambiguously prove that people whose occupation involves contact with asbestos fibres (exposure to asbestos fibres) much more often develop malignant cancer of the lungs than people exposed to different other kinds of dust [7, 24, 23, 34]. From among all asbestos minerals the most potent as cancer etiological factor is amphibolic asbestos, mainly crocidolite [7, 24, 62].

The fibres of asbestos are a few times thinner than human hair and thus are hard to notice with the naked eye. They easily float in the air. A significant part of the fibres inhaled with the air is deposited in the upper respiratory tract lined with epithelium covered with cilia and from there the fibres can be removed or penetrate further inside, even getting to the circulatory system. In the lung chrisotile fibres are cleaved into fibrils and partly dissipated through lung fluid [40-45, 78]. The amphibole fibres do not divide into fibrils and do not break, they are less soluble in the lung fluid and remain longer in the lungs.

Mechanism of Fibre Carcinogenesis

The first reports on the development of lung cancer as a result of inhalation of asbestos-containing dust prompted intense studies aimed at an explanation of the mechanism of carcinogenic effect of asbestos fibres. Although it has been established that the potential of the carcinogenic effect of the fibres depends on their chemical stability and size, not one of the hitherto proposed mechanisms has not been commonly accepted [7, 69]. The reason is the lack of convincing experimental evidence from cell tests which would give a direct connection between the initiation of cancer changes and the interaction of asbestos fibres with DNA [20, 58, 73, 74]. One of the proposed mechanisms described as the hypothesis of a carrier explains the appearance of cancer changes by the fact that asbestos fibres can adsorb and carry polycyclic aromatic hydrocarbons (PAH) to the lungs [63]. Results of many studies prove that heavy metals substituted in crystalline structures of asbestos, mainly iron, [20, 31, 39, 62, 65, 66, 75, 76] and PAH adsorption [7, 69, 72] are particularly dangerous for man when an air sack in his lungs has been pricked by microfibres of asbestos carrying PAHs or metal ions [20,58, 69, 77]. The fibres of asbestos can also interact with the proteins of red blood cell membrane leading to dissolution of red blood cells (haemolysis) [63, 65, 69]. The hemolytic activity of asbestos is determined by the type of the mineral, the ratio Mg:Si and the size of the fibre area. The greatest biological activity is believed to characterize chrisotile fibres [7, 42, 77].

There is increasing evidence that iron from asbestos may cause cancer as a result of radical induced damage. Many mechanisms have been proposed as the potential reactions of iron to catalyze the oxidation of DNA [20, 78-80], lipid [58, 82-85] and protein [58, 86-88]. Analysis suggests that different chemical reactions catalyzed by asbestos and other mineral fibres, e.g. erionite, are responsible for their pathological influence [20, 26, 30, 45, 62].

Legal Solutions Concerning Asbestos Production and Use

The above-presented facts prompted intensive studies in many countries on all aspects related to exposure of man and animals to asbestos and other mineral fibres. The excavation and processing of asbestos, as well as the use of asbestos-containing products, have been gradually limited [12, 22, 30, 41, 42, 59]. The majority of countries have introduced legal regulations in order to protect the workers whose occupation was related to exposure to increased level of asbestos dust. However, legal limitations on the emission of asbestos to atmospheric air has been introduced in only a few countries: USA, Germany, France, Sweden and Poland [12, 28, 32, 33, 59].

In the beginning the ban on processing and using asbestos-containing products concerned only crocidolite characterized by the highest potential of initiation of cancer changes [40, 62, 65]. In a few countries like Great Britain and Scandinavian countries, this ban has been extended over all asbestos minerals from the group of amphibole [72]. For example in the USA the Environmental Protection Agency (EPA) banned the production and use of asbestos-containing materials in 1989 [30, 59]. Moreover, regulations were introduced specifying the admissible level of asbestos fibres in school premises and other public buildings and the rules were given to be followed during removal or conservation of earlier installed asbestos, its transportation and storage [59].

In the 1970s Poland belonged to the most important producers and users of asbestos-containing products. At nine plants about 100 thousand tonnes of asbestos minerals were processed annually [12, 25, 26, 50, 52, 89]. The documented health risk related to exposure to asbestos resulted in a gradual reduction of its processing so that in 1991 the amount of asbestos processed in Poland decreased to 30 thousand tonnes [12, 20, 32, 33, 39]. Restrictions were imposed as to the use of asbestos-containing materials and a list of products which may not contain asbestos was compiled [90]. Finally, a ban on using asbestos-containing products was introduced in June 1997 [91]. Moreover, regulations concerning safe use and conditions of removal of asbestos-containing products were issued [93]. Also, the list of asbestos-containing products admitted for production or import was prepared [93].

Methods of Elimination of Mineral Asbestos and their Modification into Harmless Products

The first measures taken to eliminate or limit exposure to asbestos consisted in wet separation and ventilation of the rooms containing sources of its emission, appropriate organization of such activities as removal, transportation and storage of asbestos waste and conservation works [20, 23, 26, 28, 32]. The simplest measures aimed at elimination or limitation of emission of asbestos dust were mechanization of all kinds of handling of asbestos-containing materials, which should be wet processes if possible. Emission of asbestos dust can essentially be limited if the materials containing asbestos are wetted with surfactants whose use also limits the use of water [11, 20, 26, 89]. For example, an anionic surfactant, such as linear alkyl sulphonate, sodium lauryl sulphates, polyalkoxy carboxylates, or an non-anionic surfactants, such as alcohol alkoxylates, alkyl phenol ethoxylates, polyoxoethylene esters sand polyalkene oxide block copolymers can be added to the treating composition in conventional amounts, e.g. up to about 5% by weight. The problems of wet removal techniques include the requirements of physical removal and handling of the wet asbestos-containing material. In addition, the removed material must be further treated to destroy the remaining asbestos component if the material is to be discarded as a non-asbestos containing material.

Another technique preventing the release of asbestos fibres to the atmosphere consists in using different binding media in the processes of solidification or cementation [28].

In Poland a technology of neutralization of asbestos has been devised based on impregnation of asbestos-containing materials with a preparation whose composition has been patented [94]. The interaction of this preparation with asbestos leads to its partial destruction or modification of its structure, which significantly limits the possibility of releasing asbestos fibres to the atmosphere. After this processing the material can be additionally solidified by a binding agent such as hydraulic binder or organic polymer. The technology developed at the EKO-PIK in Sroda Wielkopolska (Poland) allows the use of the processed material as support for road construction [94].

In 1998, the construction firm W.R. Grace Products from Boca Raton in Florida as the first firm in the USA got the licence for commercial application of its new asbestos treatment product. This product was obtained on the basis of a new technology of neutralization of materials containing chrisotile, protected by 6 patents [95-100] developed in co-operation with the Department of Energy's Brookhaven National Laboratory. The studies also led to invention of a new quantitative analytical method that detects chrysotile asbestos fibres in material containing as little as 0.1% of the fibres, also protected by a patent [97].

This technology permits chemical elimination of asbestos contained in installed fireproofing without first removing the fireproofing. This method is referred to as Asbestos Digestion Process and is abbreviated as DMA®. An important advantage of the above-mentioned technology is the possibility of destroying asbestos without diminishing the existing fire-resistive capabilities of the fireproofing material on columns and beams. The development of the asbestos-destroying process uses a foamy solution sprayed directly onto asbestos-containing material. The foam chemically digests asbestos fibres, dissolving them into harmless minerals. It has been established that spraying of the foamy solution on the material containing chrisotile would reduce asbestos to less than 1% after 24 h [96], which according to the Environmental Protection Agency's definition makes them non-asbestos materials [11]. The cost of neutralization of asbestos by the DMA® method has been estimated at about 4 times lower than by traditional method [95]. Recently a technology was developed allowing simultaneous destruction of chrisotile and amosite [101]. Additionally, elaborated new composition digestion solution, which contains a component capable of inhibiting corrosion of metals in contact therewith [102]. It has been found that thiourea and alkylpyridinium halides (e.g. dodecylpyridinium chloride, dodecylpyridinium bromide at a concentration of at least 0.001M) provide corrosion inhibition in the applications contemplated above. The thiourea corrosion inhibitors when used, should be present in the treating composition at a concentration of at least about 0.0001M.

The asbestos digestion process may be presented as a sequence of the following reactions:

chrysotile-MgO + H_3PO_4 = magnesium phosphate(V) + H_2O

$Me_2[SiF_6]$ or $Me[BF_4]$ = H_2O + F^-

(where: Me - an alkali metal or alkaline earth metal)

$F^- + \text{chrysotile-}SiO_2 = SiF_6^{2-}$

$SiF_6^{2-} + H_2O = SiO_2 \text{ (amorphous)} + F^-$

$F^- + \text{chrysotile-}SiO_2 = SiF_6^{2-}$

$SiF_6^{2-} + H_2O = \text{etc., etc.}$

As follows from the above sequence of reactions, there are two pathways leading to chemical destruction of asbestos. The first involves removal of MgO, or to be exact brucite $Mg(OH)_2$ from the crystal lattice of chrisotile as a result of etching by phosphoric(V) acid, while the second is based on generation of the possibly lowest amount of fluoride ions forming HF acting as a catalyst in the reaction of removal of SiO_2 from the crystal lattice.

Results of the study carried out on animals have proved that the product obtained as a result of asbestos etching is non-toxic [97-100].

Substitutes for Asbestos

To meet the requirements specified by the act banning the use of asbestos-containing products [59, 72, 91] and accompanying regulation [93] the producers of such products were forced to look for asbestos substitutes safe for people and animals. The materials considered to be such substitutes were:

- natural fibres e.g. cellulose, hemp [103];
- synthetic fibre e.g. polyacrylnitrile, carbon [104, 105];
- mineral fibres e.g. glassy, ceramic, alsifer, wollastonite [106];
- metallic fibres e.g. steel fibres [20, 89].

This substitution seemed very simple, however after some testing they have been proved ineffective for the following reasons.

- the products with substitutions do not meet the techno logical demands, e.g. the boards;
- made without asbestos undergo cyclic changes in the size depending on the humidity level, which leads to their deformation;
- the products are characterized by poorer stability be cause of the susceptibility to the effect of bacteria and fungi (e.g. cellulose fibres) and it is difficult to obtain a really uniform material using e.g. glass fibres or nets;
- the products with asbestos substitutions are usually more expensive, especially carbon fibres [20, 89].

Nowadays the asbestos substituents are short synthetic fibres, e.g. polyacrylnitrile, polyamide and aramide, for production of nets and fabrics - polyacrylnitrile, polypropylene or glass fibres of elevated level of circonium, or mineral wool [89]. Although glass fibres and mineral wool are not so hazardous as asbestos, they cannot be considered as absolutely safe and their inhalation can lead to different diseases [106].

References

1. DOLL R., PETO J., Asbestos effects on health of exposure to asbestos, Stationary Office, London **1985.**
2. Consumer Product Safety Commission Reports to the U.S. CPCC by the Chronic Hazard Advisory Panel on Asbestos, Washington D.C. **1983.**
3. AMANDUS H.E, Brit. **J.** Ind. Med., **4,** 526, **1986.**
4. Air Quality Guidelines for Europe, WHO Regional Publica tions, European Ser. No. 23, Copenhagen **1987.**
5. FUBINI B., GIAMELLO E., VOLANTE M., BOLIS V., Toxicol. Ind. Health, **6,** 571, **1990.**
6. SCHRIEIER H., Asbestos in the natural environment, Elsevier Science Pub. Co., New York **1989.**
7. INDULSKI JA., Ed., in: ser.: Kryteria zdrowotne srodowiska, torn 53, Azbest i inne wlokna naturalne, Panstwowy Zaklad Wydawnictw Lekarskich, Warszawa **1990.**
8. COFFIN D.L.,PALEKAR L.D, COOK P.M., CREASON J.P., in: Biological interaction of inhaled mineral fibres and cigarette smoke, Ed. A.P. Wehner, Batelle Press, Seattle WA **1989.**
9. VERBLEN D.R., WYLIE A.G., in: Reviews in Mineralogy, Eds. G.D. Guthrie, B.T. Mossman, Brookcrafters Inc., Chel sea **MI, 28,** 61, **1993.**
10. Theresold Limit Values for Chemical Substances and Physi cal Agents and Biological Expose Indices, ACGIH, **1994-1995.**
11. ZOLTAI T., in: Proceedings of Workshop on Asbestos, Definitions and Measurement methods, National Bureau of Standards Special Publication No 506, Eds. C.C. Gravatt, P.D. LaFleur, K.F.J. Heinrich, U.S. Government Printing Office, Washington **1978.**
12. WOZNIAK H., WIECEK E., SZYMCZAK W., w: Ocena narazenia zawodowego na WWA i azbest w wybranych galeziach przemyslu, Instytut Medycyny Pracy, Lodz **1995.**
13. CHOJNACKI J., Krystalografia chemiczna i fizyczna, Panstwowe Wydawnictwo Naukowe, Warszawa **1961.**
14. CHOJNACKI J., Elementy krystalografii chemicznej i fizycznej, Panstwowe Wydawnictwo Naukowe, Warszawa **1971.**
15. MASLANKIEWICZ K (Ed.), Ziemia, Wiedza Powszechna, Warszawa **1970.**
16. GHIO A.J. ZHANG J., Piantadosi C.A., Arch. Biochem. Biophys, **298,** 646, **1992.**
17. POLANSKI A, SMULIKOWSKI K, Geochemia, Wydawnictwa Geologiczne, Warszawa **1969.**
18. BIELANSKI A., Podstawy chemii nieorganicznej, Panstwowe Wydawnictwo Naukowe, Warszawa **1987.**
19. BLIEFERT C, Umweltchemie, Wiley-VCH, Weinhaim **1997.**
20. HARDY **J.A.,** AUST A.E., Chem. Rev., **95,** 97, **1995.**
21. CARR D.D., HERZ N., Eds. Concise Encyclopaedia of Mineral Resources; MIT Press, Cambridge, MA **1989.**
22. HOCHELLA M.F., in: Reviews in Mineralogy, Eds. D.G. Guthrie, B.T. Mossman; Brookcrafters Inc., Chelsea, MI **1993.**
23. **ALLISON** AC, HARINGTON J.S., BADAMI D.V., Phar macol. Chemother., **12,** 291, **1975.**
24. DAVIS J.M.G., in: Reviews in Mineralogy, Eds. G.D. Guth rie, B.T. Mossman; Brookcrafters Inc., Chesea, MI **1993.**
25. COMMINS B.T., The Significance of asbestos and other mineral Fibres, Environmental Ambient Air Health Criteria, Report Maidenhead Commins. Ass., **1985.**
26. POOLEY F.D., in: Dusts and Disease, Eds. R. Lemen, J. Dement; Pathotox, Park Forest, IL **1979.**
27. MOSSOP M.B., Ph.D. Thesis, Nottingham Polytechnic (GB), **1992.**
28. DOMKA L., KOZAK M., KOZAK A., Materialy Ogolnopolskiej Konferencji Szkoleniowej, Unieszkodliwianie i utylizacja komunalnych odpadow niebezpiecznych, Wista, Poland, 11-13.04.2000, pp. 143-154.
29. HATFIELD E.J., in: Proceedings Asbestos Fibres Measure ments in Buildings Atmosphere, Ed, E.J. Hatfield, Ontario Research Foundation, Toronto, Canada **1982.**

30. REYNOLDS T., J.Natl. Cancer Inst., **84,** 560, 1992.
31. SIMONATO L, BARIS L, SARACCIK R., SKIDMORE J., WINKELMAN M, IARC Sci. Publ., **90,** 398, **1989.**
32. LIS D.O., PASTUSZKA J.S., Ochrona powietrza i problem odpadow, **29,** 99, **1995.**
33. STROSZEJN-MROWCA G, SZCZEPANIAK M. WIECEK E, Medycyna Pracy, **17,** 343, **1991.**
34. PASTUSZKA J.S., KABALA-DZIK A, Sci. Total Environ, 229,131, **1999.**
35. FLETCHER R.A, SMALL J.A, in: Aerosol Measurement Principles, Techniques and Applications, Ed. Van Nosytrand Reinhold, New York **1993.**
36. Proc. 6lh American Conference of Governmental Industrial Hygienists (ACGIH), Asbestos identification and measurement, Cincinati, Ohio USA **1979.**
37. American Society for Testing Materials (ASTM), Draft Report D 22.05, Philadelphia, PA USA **1990.**
38. FRIEDRICHS K.H, in: Physical and chemical characterisation of individual airborne particles, Ellis Horwood Ltd, Chichester, England, **1986.**
39. SELIKOFF, LILLIS R, NICHOLSON W.J, Ann. N.Y. Acad. Sci. **331,** 295, **1980.**
40. DOLL R. PETO J. Asbestos effects on health of exposure to asbestos, Ed. N.M. Stationary Office, London **1985.**
41. PEZERAT H, in: Mechanisms in Fibre Carcinogenesis, Eds. R.C. Brown, J.A. Hoskins, N.F. Johnson, Plenum Press, New York **1991.**
42. FOURNIER J, GUIGNARD J, NEIJARI A, ZALMA R, PEZERAT H, in: Mechanisms in Fibre Carcinogenesis, Eds. R.C. Brown, J.A. Hoskins, N.F. Johnson, Plenum Press, New York **1991.**
43. WAGNER J.C, BERRY G, SKIDMORE J.W, TIMBRELL V, Br. J. Cancer, **29,** 252, **1974.**
44. BARIS Y.I, SIMONATO L, ARTVINLY M, POOLEY P, SARRACCI R, SKIDMORE J, WAGNER C. Int. J. Cancer Inst. **39,** 10, **1987.**
45. BARIS Y.I, Bull. Med. Surg, **8,** 165, **1975.**
46. BARIS Y.I, ARTVINL M, SAHIN A.M., Ann. N.Y. Acad. Sci, **330,** 423, **1979.**
47. NEWHOUSE ML., BERRY G, Br. **J.** Ind. Med, **33,** 147, **1976.**
48. POOLEY F.D, Environ. Rev, **12,** 281, **1976.**
49. FINKELSTEIN M.M, Br. **J.** Ind. Med, **40,** 138, **1983.**
50. KIEC-SWIERCZYNSKA M, Polish J. Occup. Med, **2,** 200, **1989.**
51. PETO J, SEIDMAN H, SELIKOFF I.J, Br. J. Cancer, **45,** 124, **1982.**
52. WIECEK E, WOZNIAK H, STRODZEJN-MROWCA G, Higiena Pracy, **3,** 5, **1994.**
53. National Research Council, Asbestos Form Fibres: nonoccupational health risk, National Academy Press, Washington DC, **1984.**
54. BURDETT G.J, in: A workshop on asbestos; Police for the Future, Part II, Newcastle-upon Tyre, England **1986.**
55. ACHESON E.D., GARDNER M.J, in: Final Report of the Advisory Committee on Asbestos, Ed. Health and Safety Commission, Vol. 2, pp. 7-83, London **1983.**
56. HAMMOND E.C, Natl. Cancer Inst. Monogr, **19,** 127, **1966.**
57. WARNOCK M.L, CHURG A.M., Cancer, 35, 1236, **1975.**
58. SPURNY K.R, in: Mechanisms Fibre Carcinogenesis, Eds. R.C. Brown, J.A. Hoskins, N.F. Johnson, Plenum Press, New York **1991.**
59. AMANDUS H.E, **J.** Ind. Med, **43,** 526, **1986.**
60. MOSSMAN B.T, Environ. Carcinog. Rev, **C4,** 151, **1988.**
61. BERRY G. WAGNER J.C, Inst. J. Cancer, **17,** 477, **1976.**
62. National Research Council, Asbestos form fibres: nonoccupational health risks, National Academy Press, Washington DC. **1984.**
63. STANTON M.R, J. Natl. Cancer Inst, **48,** 797, 1972.
64. LIDDELL F.D.K, HANLEY J.A, Br. J. Ind. Med, **42,** 389, **1985.**
65. **LIDDELL** F.D.K, HANLEY J.A, Zur Beurteilung der Krebs Gebsgefahr durch Asbest, Medicin Verlag, Munich **1984.**
66. STEVENS R.G, BEASLEY R.P.BLUMBERG B.S, J. Natl. Cancer Inst.,76, 605, **1986.**
67. SELBY J.V, FRIEDMAN G.D, Int. J. Cancer. **41,** 677, **1988.**
68. STEVENS R.G, JONES D.Y, MICOZZI M.S., TAYLOR P.R, Natl. Engl. J. Med, **319,** 1047, **1988.**
69. AUST A.E., LUND L.G, in: Mechanisms in Fibre Carcinogenesis, Eds. R.C. Brown, J.A. Hoskins, N.F. Johnson; Plenum Press, New York **1991.**
70. ASHROFT T, HEPPELESTON A.G., J.Clin. Pathol, **26,** 224, **1973.**
71. AUERBACH O, HAMMOND E.C, SELIKOFF I.J, PARKIS V.R, KOSLOW H.O, GARFINKEL L, Environ. Res, **14,** 286, **1977.**
72. BOATMAN E.S, MERRILL T, O'NEILL A, POLISSAR L, Mitette J.R, Environ. Health Perpect, 53,131, 1983.
73. PONKA P, SHULMAN H.M., WOODWORTH R.C, Iron Transport and Storage, Ed. CRS Press, Boca Raton US **1990.**
74. MILLER D.M, BUETTNER G.R, AUST S.D., Free Radical Biol. Med, **8,** 95, **1990.**
75. GULAMIAN M, van WYK J.A, in: Mechanisms in fibre carcirogenesis, Eds. R.C. Brown, J.A. Hoskins, N.F. Johnson, Plenum Press, New York **1991.**
76. CRICHTON R.R., Inorganic biochemistry of iron metabolism, Ellis Horward Limited, Chichester, England **1991.**
77. NOLAN **R.P.,** LANGER A.M., in: Reviews in Mineralogy, Eds. G.D. Guthrie, Mossman B.T, Bookcrafters Inc., Chelsea **MI 1993,** vol. 28, pp.309-325.
78. PHAM Q.T.GAERTNER M, MIUR J.M, BRAUN, GABIANO M, SADOUL P., Respir. Dis, **64,** 534, **1983.**
79. NIEDERAU C, FISHER R, SONNENSBERG A, STREMMEL W, TRAMPISH J, STROHEMEYER G.N, Engl. J. Med, **313,** 1256, 1985.
80. STEVENS R.G, KALKWARF D.R, Environ. Health Prospect, **87,** 291, **1990.**
81. PRYOR W.A, Free Radical Biol. Med, 4, 219, 1988.
82. BUCHER J.R, TIEN M, AUST S.D., Biochem. Biophys. Res. Commun, **111,** 777, 1983.
83. MINOTTI G, AUST S.D, Free Radical Biol. Med, **3,** 379, **1987.**
84. BRAUGHTER J.M, DUNCAN L.A, CHASE R.L,J. Biol. Chem, **261,** 10282, **1986.**
85. MINOTTI G, AUST S.D, J. Biol. Chem, **262,** 1098, **1987.**
86. STADTMAN E.R., Science, **257,** 1220, 1992.
87. STADTMAN E.R, BERLETT B.S, J. Biol. Chem, **266,** 17201, **1991.**
88. STADTMAN E.R, OLIVER C.N, J. Biol. Chem, 266, 2005, **1991.**
89. SKALMOWSKI W, Chemia materialow budowlanych, Arkady, Warszawa **1971.**
90. Dz.U. RP nr 133, poz.690, **1994.**
91. **Dz.U.** RP nr 101, poz. 628, **1997.**
92. Dz.U. RP nr 45, poz. 628, **1997.**

93. Dz.U. **RP nr** 15, poz. 188, **2000.**
94. Pat. RP 174819, 1998.
95. US Pat. 5,743,841, **1998.**
96. US Pat. 5,753,035, **1998.**
97. US Pat. 5,753,034, **1998.**
98. US Pat. 5,753,033, **1998.**
99. US Pat. 5,753,032, **1998.**
100. US Pat. 5,753,031, **1998.**
101. US Pat. 6,005,158, **1999.**
102. US Pat. 5,741,358, **1998.**
104. PIELICHOWSKI J.A, PUSZYNSKI, Technologia tworzyw sztucznych, Wydawnictwo Naukowo Techniczne,Warszawa **1991.**
105. SZLEZYNGIER W., Tworzywa sztuczne, tom 3, Fosze, Rzeszow **1999.**
106. VALLYATHAN V., ROBINSON V., Am. Rev. Respir. Dis., **129,** A 1178, **1984.**