Slate dust

Health-based Reassessment of Administrative Occupational Exposure Limits

Committee on Updating of Occupational Exposure Limits,
a committee of the Health Council of the Netherlands

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1 Introduction

The present document contains the assessment of the health hazard of slate dust by the Committee on Updating of Occupational Exposure Limits, a committee of the Health Council of the Netherlands. The first draft of this document was prepared by AAE Wibowo, Ph.D. (Coronel Institute, Academic Medical Centre, University of Amsterdam, Amsterdam, the Netherlands).

In June 1999, literature was searched in the databases Medline, Embase, and Chemical Abstracts starting from 1966, 1988, and 1970, respectively, and using the following key word: slate. HSELINE, CISDOC, MHIDAS, and NIOSHTIC (covering the period 1985/87 up to 1998), databases available from CD-ROM, were consulted as well. The final search was carried out in Medline and Toxline in October 2002.

In April 2003, the President of the Health Council released a draft of the document for public review. The committee received no comments.

2 Identity

Slate is a very fine-grained, splittable rock. It has a leaden-greyish, reddish or greenish colour. It has a high calcium carbonate content; it also contains silicates (mica, chlorite, hydrosilicates), iron oxides, and amorphous and crystalline silica. The crystalline silica (quartz) content of slate dust ranged from less than 10% to more than 50%. Slade dust is a mixture of minerals, the composition of which depends on the geological characteristics of the geographic location where the slate stones are quarried. For example, in North-Wales (United Kingdom) quarries, respirable slate dust contains between 13% and 32% of quartz. Microscopic examination showed fine crystalline structures made of calcites, mica and particles of quartz of 2 to 30 μm in size (Don83). Higher quartz content (>50%) was reported in respirable slate dust retained from India (Sai85a, Sai85b). However, Fulekar and Khan (Ful95) reported that the quartz content in airborne total dust generated by different manufacturing units in the production of slate pencil in India varied from 6.7 to 29.1% (mean 15.3%). The typical chemical composition of slate dust collected from areas of Mandsaur in India was reported as follows: Si 45.8%, Mn 15%, Fe 3.4%, Mg 3.2%, K 0.7%, Ti 0.65%, Ba 950 ppm, Zr 190 ppm, Rb 160 ppm, Ce 105 ppm, Sr 70 ppm, La 40 ppm and Mb 10 ppm (Kha89). Craighead et al. described slate as a metamorphic rock made up of several minerals. The mineral composition of slate, retained from Vermont and New York, USA, was: muscovite (white mica) 38-40%, quartz 31-45%, chlorite 6-18%, haematite 3-6%, carbonate 1-5%, and magnetite 1-2% (Cra92). Because of its variable mineral composition, no CAS number has been given for slate dust.

3 Physical and chemical properties

Of the major components, quartz (CAS No: 14808-60-7) is a colourless, odourless, non-combustible solid with a molecular weight of 60 and boiling and melting points of 2230 and 1610ºC, respectively. Muscovite (CAS No: 12001-26-2) is an odourless, non-flammable, non-fibrous, water-insoluble silicate from the mica group of chemicals with a molecular weight of 797 and no known additional physical chemical data.

4 Uses

Slate slabs are used for roofing, stair treads, door, window and porch casements, flooring, fireplaces, billiard tables, electricity switch panels, school blackboard, pencils, etc. Powdered slate has been used as a filler or pigment in rust-proofing or insulating paints, in mastics, and in paints and bituminous products for road surfacing.

5 Biotransformation and kinetics

In vitro experimental studies showed that about 3.8% of the silica in slate dust binds with proteins in rat plasma, and 2.4% with purified bovine serum albumin within 96 hours. Binding of silica to protein occurs most rapidly in the first 24 hours, and reaches a plateau after 48-72 hours. It was hypothesised that when slate dust particles come into contact with blood, a proportion of the silica is dissolved, and in turn combines with blood protein and possibly with other biomacromolecules. The results indicate that in mammalian blood, natural silica may exist in protein-bound form and that exposure to silica dust could enhance the level. In rat lung, silica-binding protein was found to be a glycoprotein (Sin84). The authors commented that besides a normal physiological role, silica-binding protein could function as a biochemical mechanism for dust clearance in subjects exposed to slate dust, apart from clearance through lymphatics and coughing (Sin84).

6 Effects and mechanism of action

Human data

In slate workers, a disease called 'slate-worker's pneumoconiosis' has attracted attention since early 19th century. This disease is primarily respiratory in nature and has a relatively slow progression. Silicotic lesions become apparent after about 15 years of exposure, and nodular forms are seen only after a period of exposure exceeding 20 years (Don83).

A study was conducted on the lungs of 12 slate workers who developed pneumoconiosis while employed in the quarries of Vermont (US) and adjacent areas of New York. Perivascular and peribronchial lesions, accompanied by interstitial fibrosis and macules, were scattered diffusely in the lungs. The lesions were associated with a variable number of silicotic nodules. Scanning electron microscopy combined with energy-dispersive X-ray spectrometry, demonstrated aluminium- and silica-containing particles with variable cationic constituents, and silica alone in a pattern typical of free crystalline quartz. By X-ray diffraction analysis, the majority of mineral particulates were free crystalline quartz and muscovite. The author concluded that slate workers are exposed to respirable airborne dust that has the capacity to induce a pneumoconiosis that differs from classic silicosis in that slate workers reveal a diffuse interstitial pulmonary disease differing from the nodular lesions characteristic of silicosis (Cra92).

Three studies have been reported on the effects of slate-dust exposure on the lungs of slate quarry workers in North Wales, United Kingdom. In the first study, a health survey was conducted on 2432 male workers. The objective of the study was to ascertain the prevalence of pneumoconiosis purely by examination of chest X-ray film of these workers, as read by 3 independent clinicians. The level of exposure to slate dust of these workers was not reported. The results showed that 69% of the workers had normal radiographs, 30% were diagnosed with pneumoconiosis from grade 1 to grade 3, and 0.6% of the workers had a progressive massive fibrosis. It was also found that 27 out of 219 workers who were referred to the chest clinic for further assessment were also suffering from pulmonary tuberculosis (Jar57). The second study was a cross-sectional study, conducted in 1975 on 725 workers and ex-workers who had been occupationally exposed to slate dust, but not to any other industrial dust. Another group of 530 men from the same area had never been exposed to any industrial dust, and served as a control group. The fraction of respirable quartz in respirable slate dust was between 13% and 32%. Evidence of pneumoconiosis was found in one-third of the slate workers. The prevalence of respiratory symptoms was high, and there was evidence of an effect of both simple and complicated pneumoconiosis on lung function, additional to that of age. No exposure data were reported (Glo80). In a follow-up study, the mortality during a 6-year period (1975 till 1981) was investigated in the above 2 groups of workers. At date of interview in 1975, the workers' age, smoking habits, radiological score derived from the radiological classification of pneumoconiosis, anthropometric data, forced vital capacity (FVC), and 1-second forced vital capacity (FEV_1) were collected. The complete data set was available for 89% of the slate workers and for 87% of the controls. In slate workers, 129 out of 725 (17.8%) died during the survey, compared with 73 out of 530 (13.8%) of the controls. Eight slate workers died from pneumoconiosis. In non-smokers, there was no significant excess mortality associated with exposure to slate dust, but in smokers, there was a 26% excess. In slate workers, the risk for smokers was on average 76% greater than for non-smokers, and increased with severity of pneumoconiosis. In the control group, the risk was 50% greater for smokers than non-smokers. In slate workers, the risk of death of the ex-smokers, but not of the smokers or non-smokers, was higher in those with a lower FVC and FEV_1. Exposure data to slate dust were not reported (Old86).

A medical survey was undertaken on 151 workers (144 males, 7 females) engaged in the production of slate pencils in Mandsaur, India, to investigate the prevalence of silicosis. The age of 124 workers was below 30 years. The silica content of the dust was 69% (type of silica not reported). Exposure levels were not reported. Cough, dyspnoea, and pain in the chest were the most important respiratory symptoms. Symptoms were correlated with length of employment in slate-pencil production. All workers experienced cough and dyspnoea after 15 years of employment. Chest X-ray films were taken of 145 workers. In 82 radiographs (57%), there was evidence of silicosis. The incidence and severity of pneumoconiosis as judged by chest X-ray films were found to be directly proportional to the duration of employment (Jai77).

In a health survey of 593 slate-pencil workers in Mandsaur, India, the workers were divided into 3 groups. Group 1 comprised 405 males ('cutters'), who were engaged in cutting of slate pencils, and who were directly exposed to the dust; group 2 comprised 117 males ('non-cutters'), who were engaged in separating, counting, and packing of the pencils, and who worked about 1.5-3 meters from the cutters; group 3 consisted of 71 females ('non-cutters'), who frequently took the work to their homes to carry out separating, counting, and packing of the pencils. Air concentrations of dust were measured in 6 factories.

Dust samples were collected from the breathing zone of the cutters and from the different workplaces of the factories (sampling times not given). The mean dust exposure level of the cutters was 46 mg/m^3 for total dust (range: 11-177 mg/m^3) and 10.4 mg/m^3 for respirable dust (range: 4.3 - 18.4 mg/m^3). The mean total mg/m^3 for total dust (range: 7.4-97 mg/m^3) and 5.5 mg/m^3 for respirable dust (range 3.7 - 8.8 mg/m^3). The mean free silica (quartz) content of the dust was 56%. The mean duration of employment for cutters, male non-cutters, and female non-cutters was 7.3, 7.6, and 14.7 years, respectively. There was a higher proportion of smokers among the cutters (85%) than the non-cutters (males: 60%, females: 0%). Chest radiographic films of 324 workers (54.6%) showed silicosis. Of these, 17.7% had progressive massive fibrosis (PMF). In the group of cutters, prevalences of silicosis and PMF were 60% and 22%, respectively. In the group of male non-cutters, 41% had silicosis and 11% PMF, and in the group of female non-cutters, 47% had silicosis and 7% PMF. The prevalence of silicosis and PMF was related with the duration of employment in all 3 groups. All 29 cutters, who had been employed in the slate pencil industry for 16-20 years, and all 6 non-cutters, who had been employed for more than 21 years, had developed silicosis (Sai85a). The same authors conducted a follow-up study, 16 months after the initial survey. During this period, 23 out of the 593 workers (3.9%) had died. All those died were males, 20 were cutters and 3 were non-cutters, and they had been diagnosed as having PMF in the initial survey. Their mean age was 34.7 years (range 18-55 years), and they had been employed in the slate-dust industry for on average 12 years (range 3-20 years). In the follow-up study, 279 out of the 570 remaining workers participated. The cutters comprised 204 men and the non-cutters 47 men and 28 women. The prevalences of silicosis at the initial examination were 59%, 40%, and 29%, and were increased after the 16-months follow-up to 96%, 62%, and 43%, respectively. The prevalence was slightly higher among smokers as compared to non-smokers in the same group (Sai85b).

Testing of silicate dusts for cytotoxicity has been done in *in vitro* model systems. *In vitro* haemolysis of human erythrocytes in isotonic conditions was used as a test system for the prediction of the relative cytotoxicity of different samples of slate dust from Mandsaur, India. The haemolytic index of slate dust, i.e., the dust concentration needed to induce 50% haemolysis of human erythrocytes, was relatively high (1.5 mg of dust per mL of human erythrocyte suspension in normal saline) and comparable to that of chrysotile asbestos, indicating potentially high cytotoxicity (Sin83a, Sin83b, Sin87).

Animal data

Irritation and sensitisation

The committee did not find data from eye- or skin-irritation, or on skin-sensitisation studies of slate dust

Acute toxicity

The committee did not find data from acute lethal toxicity studies of slate dust.

Several studies have been conducted to study the biochemical changes in the lung of rats, treated with single intratracheal doses of slate dust.

Female albino rats (n = 70) were given a single intratracheal dose of 50 mg of slate dust suspended in saline, and biochemical changes in the lung lavage and in the blood were monitored up to 90 days after treatment. A control group of 70 rats was given only saline solution. A several-fold increase of free cell population (initially macrophages) was elicited by the dust. The effect was at its maximum after 4 days, and was 2-fold higher by the end of 90 days. An increase in acid phosphatase activity was seen over the whole observation period in the acellular fraction, but the activity gradually decreased in the cellular fraction. The phospholipid content varied both in cellular and acellular fractions, indicating altered turnover of membrane lipids and surfactants. At advanced periods of the study, sialic acid was found to be released into the acellular fraction, indicating membrane damage. These results indicate a cytotoxic action of slate dust *in vivo* (Kha83). In a subsequent study, rats were monitored for 150 days after intratracheal treatment with 50 mg of slate dust. Biochemical changes in the lung included a turnover of collagen after 90 days, reaching substantially higher values at 150 days. A concurrent increase of hexosamine and sialic acid content was also observed. The phospholipid content in whole lung tissue, as well in the mitochondria, was generally higher in the dust-treated rats. The mitochondrial cytochrome *c* oxidase and glutamate dehydrogenase activities were increased, whereas monoamine oxidase was marginally affected, compared with control animals (Kha84). In another experiment, 6 female albino rats were treated intratracheally with ^{14}C-acetate, 4 and 40 days after intratracheal installation of 50 mg of slate dust, to investigate the incorporation of radioactivity into lung lipids. The results indicated that slate dust caused an enhanced synthesis of pulmonary surfactant and other lung lipids and, therefore, had an effect on the metabolism of type II alveolar epithelial cells (Kha89).

The committee did not find data from repeated-dose toxicity, including carcinogenicty and reproduction toxicity, or mutagenicity and genotoxicity studies of slate dust.

7 Existing guidelines

The current administrative occupational exposure limit (MAC) for slate dust in the Netherlands is 10 mg/m^3, 8-hour TWA, as inhalable dust.

The committee could not find occupational exposure limits for slate dust on lists of the European Commission (EC03), European countries such as Denmark (Arb02), Germany (DFG02, TRG00), Sweden (Swe00), and the UK (HSE02), or the USA (ACG03a, ACG03b).

8 Assessment of health hazard

The major route of occupational exposure to slate dust is by inhalation of the respirable particles. There are numerous human data that show that the lung is the target organ after long-term exposure and that pneumoconiosis is the critical effect. The induction of 'slate worker's pneumoconiosis' is known since the 19th century. Recent epidemiological data show that the prevalence of pneumoconiosis due to slate dust in workers is dependent on the exposure level, the duration of exposure, as well as on smoking habits.

Health hazard assessment of exposure to slate dust is hampered by the complexity of the material. Slate dust is a mixture of minerals. The composition of the minerals is dependent on the geologic characteristics of the area where the slate stones are quarried. The principal deposits are in France, Belgium, the United Kingdom, the United States, and Italy (Don83).

From the induction of pneumoconiosis observed in humans, the committee concludes that quartz appears to be responsible for the lung effects of slate dust. The quartz content of slate dust ranges from less than 10% to more than 50% by weight. Other components should not be underestimated though, especially when quartz content is low. For example, manganese can make up to 15% of the slate dust and muscovite up to 40% (Cra92, Kha89).

In the Netherlands, the current statutory occupational exposure limit for crystalline silicon dioxide (or 'free silica'), which covers quartz, cristobalite, and tridymite is 0.075 mg/m^3, 8-hour TWA, for respirable particles (SZW02). This limit is in agreement with the health-based occupational exposure limit recommended by the Dutch Expert Committee on Occupational Standards (DECOS), a committee of the Health Council of the Netherlands (DEC92).

Moreover, the Dutch Ministery of Social Affairs and Employment of the Netherlands classified quartz dust as carcinogenic (SZW02). Recently, DECOS concluded that crystalline silica inhaled in the form of quartz or cristobalite from occupational sources is carcinogenic to humans (DEC98). In its evaluation, this committee of the Health Council concluded that the tumours are induced by quartz by a non-genotoxic mechanism, i.e., caused by long-term irritation of the target tissue. This means that there is a threshold level justifying recommendation of a health-based occupational exposure limit.

The current statutory occupational exposure limit (MAC) for manganese in the Netherlands is 1 mg/m^3, 8-hour TWA (SZW02).

Because of the varying composition of slate dust, the committee considers that recommendation of a health-based occupational exposure limit for slate dust is not justified. To protect workers from work-related diseases caused by exposure to slate dust, the committee advises the authorities to uphold the existing occupational exposure limits of individual components in the dust of which quartz appears to be the major one.

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