Scand J Work Environ Health 1985;11(2):65-74
https://doi.org/10.5271/sjweh.2250
Issue date: Apr 1985

Mica pneumoconiosis--a literature review.
by Skulberg KR, Gylseth B, Skaug V, Hanoa R

Corrections

See 1985;11(6):500 for a correction.

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/3890162

Print ISSN: 0355-3140 Electronic ISSN: 1795-990X

Scand J Work Environ Health 11 (1985) 65—74

Mica pneumoconiosis — A literature review

by Knut R Skulberg, BSc,[1] Bjørn Gylseth, PhD,[1] Vidar Skaug, MD,[1] Rolf Hanoa, MD, MSc[2]

SKULBERG KR, GYLSETH B, SKAUG V, HANOA R. Mica pneumoconiosis — A literature review. *Scand J Work Environ Health* 11 (1985) 65—74. Sixty-six cases of mica pneumoconiosis have been reported in the literature. Twenty-six of the cases suggest that pneumoconiosis may be caused by pure mica alone. In only six cases the diagnosis was based on clinical examination, radiography, and lung biopsy or autopsy results. In one of these six, doubt was raised by the authors about the purity of the mica exposure. Seven epidemiologic studies have been performed among mica-processing workers, and these studies are all cross-sectional. In addition 30 experimental investigations have been carried out. However, there are no controlled inhalation studies among them. The results from the intratracheal instillation studies do not give a unanimous conclusion as to whether pure mica is fibrogenic or not. Present knowledge suggests that pure mica is moderately toxic and may induce pneumoconiosis. Exposure to mica is usually associated with exposure to other minerals such as quartz and feldspar.

Key terms: biotite, mixed dust pneumoconiosis, muscovite, sericite.

Introduction

In 1932, Ferguson (18) reported that exposure to mica might represent a health hazard. In 1933, Jones (24) presented the hypothesis that sericite might cause silicosis (29 cases). This suggestion initiated several experimental investigations on mica. Four epidemiologic studies were performed in the 1940s and 1950s. Later several case reports appeared. These were followed in the 1970s by further experimental studies. The latest report on mica pneumoconiosis was published in 1983.

This literature review is based on a broad computer search of the literature. Efforts were also made to include the literature from the mica mining countries India and the Soviet Union. Sixty-one articles were relevant for this study.

The aim of the present investigation was to review and reevaluate the literature on the adverse effects of mica to humans and experimental animals.

Occurrence, production and use of mica

Mica minerals belong to the phyllosilicate group, which comprises nine different entities, whereof muscovite (sericite), biotite, phlogopite, paragonite, and lepidolite are commercially the most important. All mica minerals belong to the monoclinic crystal system. They are flaky structures with a perfect basal cleavage (6).

Mica is one of the most common minerals in the earth crust; it occurs in granitic pegmatite (muscovite), gneisses and schists (sericite, biotite), and metamorphosed limestones (phlogopite).

Unmanufactured mica is classified either as scrap and flake mica or as sheet mica. Scrap and flake mica are produced in India, Korea, the United States, and the Soviet Union. Sheet mica is produced in India, Brazil, and some African countries.

Mica was previously used as a filler in pharmaceuticals and for decoration purposes. Ground mica is now used as a filler in paints, cement, and asphalt and as insulation material in electric cables. It is also applied as a component of drilling muds in the oil industry. Sheet mica is used in the electrical industry in vacuum tubes and condensators. A special product called micanite is used as electrical insulation material. Micanite is composed of small mica sheets and a binder and exists in a variety of shapes.

The industrial use of mica has increased considerably during this century. The annual world production was 2 600 t in 1905, 44 200 t in 1937, 234 000 t in 1974, and 350 000 t in 1981 (8, 9, 11).

Human beings are exposed to mica dust in mines, mills, agricultural and construction work, and in factories which either process mica products or apply mica in their production.

The increased industrial use of mica, partly due to it being more recently used as a substitute for asbestos, has raised the question of possible adverse health effects due to inhalation of its dust. As mica often occurs together with other minerals, for example, with quartz, both in nature and in industrial use, the question of biological interaction has also been raised. Those reports in which the authors conclude that mica exposure alone or above other agents may be responsible for lung diseases are the main objects of the present investigation.

[1] Institute of Occupational Health, Oslo, Norway.
[2] University of Bergen, 5014 Bergen, Norway.

Reprint requests to: Mr KR Skulberg, Institute of Occupational Health, PO Box 8149 Dep, Oslo 1, Norway.

Hygienic standards

The American Conference of Governmental Industrial Hygienists (ACGIH) (2) has recommended a threshold limit value (TLV) of 20 millions of particles per cubic foot for mica dust containing less than 1 % quartz. In the Federal Republic of Germany mica is classified as a nuisance dust, and the hygienic standard is 8 mg/m^3 for the respirable dust fraction defined by the Johannesburg convention. In Norway the corresponding standard is 6 mg/m^3 for total dust and 4 mg/m^3 for dust less than 5 μm in particle size.

Experimental studies

General survey

In table 1 we have listed the experimental studies which have been carried out on the biological effects of mica.

Of the 30 studies 15 were performed prior to 1950, 6 between 1950 and 1970, and the remaining 9 after 1970. In 19 of the studies intratracheal instillation was applied. The inhalation model was used in three and intraperitoneal injection in three. The remaining five applied various methods such as intravenous and subcutaneous injections.

The exposure and observation time has to be sufficient to allow experimental fibrosis to occur. The latency period for pneumoconiosis in man is long, often more than 15 years. The length is dose dependent. Less is known about the latency period in experimental animals. With regard to the highly fibrogenic dust quartz, mature collagen formation may be observed after one month. Less fibrogenic dusts need a longer observation time. Of the 30 previously mentioned studies only three had an observation period exceeding 12 months. In 18 of the studies the observation period was between 6 and 12 months. The remaining nine studies had an observation period of less than six months.

The reports on the experiments performed prior to 1950 present little information on the chemical and physical characteristics of the dust. This lack significantly limits the value of the studies. Proper dust analysis includes chemistry, crystallography, and determination of particle size and shape, all of which may be of biological importance. The reports prior to 1950 do not provide any suggestion of a possible relationship between dust particle characteristics and fibrogenic effect.

Various species have been used in the experimental studies. One species may be more prone to lung fibrosis than others, and thus comparison of experimental results may be difficult. In older studies rabbits, dogs, rats, and guinea pigs were used, whereas more recent studies have used various breeds of rats. It is difficult to induce fibrosis in the lungs of mice by silica and mica (46).

Only one study (14) has looked into the interaction of mica and resin, which are the two components of micanite. Dianova et al (14) concluded that resin reduced the fibrogenic effect of mica.

Intratracheal instillation

Intratracheal instillation has been applied in 19 studies.

Kaw & Zaidi (26), Lemon & Higgins (33), and Sahu et al (46) all reported an acute inflammatory reaction in the lung and the formation of reticulin fibers one week after instillation of a suspension of mica dust. Lemon & Higgins (33) also reported transient atelectasis and consolidation of the lung tissue as an initial event accompanied by alveolar macrophage necrosis.

Between 14 and 90 d after intratracheal instillation Lemon & Higgins (33) and Sahu et al (46) reported an additional proliferation of fibroblasts and reticulin fibers. In this period proliferation of fibroblasts and reticulin fibers was also reported by King et al (27). Le Bouffant et al (32) and Martin et al (36) reported collagen formation in this period, but far less than induced by quartz.

After three to six months' observation further proliferation of reticulin fibers was observed by Kaw & Zaidi (26) and Lemon & Higgins (33). In this period a granulomatous and interstitial lung fibrosis, not further specified, was reported by Krasnopeeva (29).

After eight to nine months' observation Kaw & Zaidi (26) noted isolated dust cell granulomata containing reticulin fibers and a substantial amount of collagen fibers, but typical nodules as in silicosis were not observed.

Le Bouffant et al (32) reported formation of collagen fibers after three months. They did not find any progression of the collagen formation at 12 months. Sahu et al (46) found no progression of reticulin fiber formation between 150 and 210 d. This result was also observed by King et al (27); however, mica treated with hydrochloric acid induced a nodular reaction within five weeks with increasing reticulin formation up to 15 months later.

Shanker et al (50) used mica particles with a maximum length of less than 5 μm in their experiment and demonstrated that these particles were transported to the tracheobronchial lymph nodes. The lymph nodes showed thick reticulin fibers. These results are supported by those of Sahu et al (46).

Inhalation studies

The inhalation model was used in three studies. Brambilla et al (7) studied lung pathology and the mineral dust content in lung tissue of 100 environmentally exposed mammals and birds in the San Diego Zoo. Fifteen percent of the animals had mild fibrosis, while 5 % had severe fibrosis at autopsy.

Table 1. Experimental studies on the biological effect of mica. (NI = not indicated)

Author	Year	Animal species	Number of animals	Methods	Dust	Dose (mg)	Particle size (μm)
Drinker et al (16)	1934	Dogs	2	Injection via lymphatics	Sericite	NI	NI
Policard (43)	1934	Rats	NI	Inhalation	Muscovite	NI	< 6
Fallon & Banting (17)	1935	Rabbits	3	Subcutaneous injection	Sericite	50	< 5
Fallon & Banting (17)	1935	Rabbits	3	Intratracheal instillation	Sericite	150	< 5
Lemon & Higgins (33)	1935	Rabbits	NI	Intratracheal instillation	Sericite	NI	< 5
Miller & Sayers (38)	1936	Guinea pigs	NI	Intraperitoneal injections	Sericite + quartz	100 and 200	<43
Cummins (12)	1937	Rabbits	4	Subcutaneous injection	Sericite	20	Coarse particles, many exceeding 10
Cummins (12)	1937	Rabbits	1	Intratracheal instillation	Sericite	1 000	<10
Selter & Weiland (49)	1937	Guinea pigs	20	Inhalation	Sericite + andesin	NI	< 2
Gardner (19)	1938	Rabbits	>4	Intravenous injection	Muscovite Biotite Sericite	1 000	< 3
Gardner (19)	1938	Guinea pigs	>5	Intraperitoneal injection	Muscovite Biotite Sericite	NI	< 3
Simpson & Strachan (52)	1940	Rabbits	4	Intravenous injection	Muscovite	284	67 % <1 100 % <5
Belt & King (4)	1945	Rats	36	Intratracheal instillation	89 % sericite, kaolin, quartz	200	< 1
Belt & King (4)	1945	Rats	36	Intratracheal instillation	Muscovite	200	< 5
King et al (27)	1947	Rats	53	Intratracheal instillation	Sericite	50	< 1
Vorwald (60)	1960	Rats	NI	Intratracheal instillation	Biotite Muscovite	NI	NI
Krasnopeeva (29)	1964	Rats	NI	Intratracheal instillation	Muscovite Phlogopite	50	80 % <5 20 % >5
Tripsa & Rotaru (58)	1966	Rats	15	Intratracheal instillation	Mica	50	1—3
Tripsa & Rotaru (58)	1966	Rats	15	Intratracheal instillation	Mica	50	1—6
Tripsa & Rotaru (58)	1966	Rats	15	Intratracheal instillation	Mica	50	1—25
Goldstein & Rendall (20)	1970	Rats	20—30	Intratracheal instillation	Mica + magnetite	NI	< 5
Starkov et al (56)	1971	Rats	NI	Intratracheal instillation	Mica with 45 % silica	50	NI
Kaw & Zaidi (26)	1973	Rats	47	Intratracheal instillation	Muscovite	50	< 5
Pott et al (44)	1974	Rats	40	Intraperitoneal injection	Biotite	100	< 5
Shanker et al (50)	1975	Guinea pigs	36	Intratracheal instillation	Muscovite	75	< 5
Dianova et al (14)	1976	Rats	NI	Intratracheal instillation	Muscovite, phlogopite and resin	50 20	NI
Martin et al (36)	1977	Rats	10	Intratracheal instillation	Muscovite	50	NI
Sahu et al (46)	1978	Mice	80	Intratracheal instillation	Mica	NI	< 5
Brambilla et al (7)	1979	Mammals, birds	100	Inhalation	Mica, quartz	NI	<10
Le Bouffant et al (32)	1980	Rats	10	Intratracheal instillation	Muscovite	NI	NI

Mineral analysis showed that the dust consisted of 90—95 % silicates (whereof 70 % was mica) and 5—10 % quartz. Selter & Weiland (49) studied the combined effects of tubercle bacilli and mica dust. They found an increased morbidity among animals exposed to both, compared with those exposed to only one. Policard (43) exposed rats (3 to 15 d) to an atmosphere heavily charged with muscovite. He found pulmonary granulomas, some of them containing giant cells.

Intraperitoneal injection

In three experimental studies intraperitoneal injection has been applied. Miller & Sayers (38) and Gardner (19) reported biological effects similar to those of nuisance dusts. In the study of Pott et al (44) the primary objective was to look for tumorigenic effects. The mica applied (biotite) did not produce malignant tumors in the animals.

Other administration routes

Mica has also been injected to lymphatics, subcutaneously or intravenously (12, 16, 17, 19, 52). All five of these studies are rather old; they do not give relevant knowledge of the biological effects of mica particles on lung tissue. In the studies the lesions caused by mica were compared with lesions caused by quartz. Four studies (12, 17, 19, 52) showed that mica induced considerably less fibrosis than quartz.

Mica exposure and pneumoconiosis in man

This literature review comprises 368 cases of pneumoconiosis associated with mica exposure. Table 2

Table 2. Cases of pneumoconiosis associated with mica exposure reported through 1983. (NI = not indicated)

Author	Year	Number of cases	Type of dust	Type of workplace or workprocess	Diagnostic methods	Author's view of the causation of the disease
Ferguson (18)	1932	3	Mica, no details	NI	Clinical examination, radiography	Mica pneumoconiosis
Jones (24)	1933	29	Sericite, quartz, others	21 underground workers in collieries, 8 others	Mineral analysis of silicotic lungs	Pneumoconiosis, probably caused by sericite
Dreessen et al (15)	1940	9	Pure mica	Mica grinding	Clinical examination, radiography	Mica pneumoconiosis
Dreessen et al (15)	1940	1	Pure mica	Mica factory/ mica grinding	Clinical examination, radiography	Mica pneumoconiosis
Dreessen et al (15)	1940	23	Mica, quartz, feldspar	Mica miners/ pegmatite millers	Clinical examination, radiography	Silicosis
Vestal et al (59)	1943	7	Pure mica	Mica grinding	Clinical examination, radiography	Mica pneumoconiosis
Vestal et al (59)	1943	2	Pure mica	Mica grinding	Clinical examination, radiography	Borderline mica pneumoconiosis
Vestal et al (59)	1943	12	Mica, quartz, feldspar	Mica mine	Clinical examination, radiography	Pneumoconiosis, no expressed opinion
Vestal et al (59)	1943	22	Mica, quartz, feldspar	Mica mine	Clinical examination, radiography	Borderline pneumoconiosis, no expressed opinion
Heimann et al (22)	1953	112	Mica with 11—67 % quartz	Mica mine	Clinical examination, radiography	Silicosis
Vorwald (60)	1960	1	Biotite, probably along with talc	Rubber factory	Clinical examination, radiography, autopsy, X-ray diffraction	Diffuse pulmonary fibrosis caused by mica or other inhaled agents
Vorwald et al (61)	1962	1	Biotite	Rubber factory	Clinical examination, radiography, autopsy, X-ray diffraction	Mica pneumoconiosis
Krasnopeeva (29)	1964	NI	Mica, no details	Mica factory	Clinical examination, radiography	Mica pneumoconiosis
Podnebesnaya (42)	1965	66	Mica with 25—30 % quartz	Mica mine	Clinical examination, radiography	Pneumoconiosis, no expressed opinion
Kleinfeld (28)	1966	1	Pure muscovite	Sawing and sanding mica	Clinical examination, radiography	Mica pneumoconiosis, calcified pleural plaques
Michailov & Berova (37)	1968	1	Mica, asbestos	Asbestos and mica curing factory	Clinical examination, radiography	Asbestosis
Kajita et al (25)	1972	1	Sericite, quartz	Latex factory	Clinical examination, radiography, autopsy	Mixed dust pneumoconiosis

(continued)

Table 2. Continued.

Author	Year	Number of cases	Type of dust	Type of workplace or workprocess	Diagnostic methods	Author's view of the causation of the disease
Rüttner et al (45)	1972	1	Mica, kaolin, feldspar	Electric insulation factory	Clinical examination, radiography, autopsy	Diffuse "asbestosis-like" interstitial fibrosis due to the dust exposure
Misra & Jain (39)	1973	1	Mica, no details	Manufacture of tazias	Clinical examination, radiography, autopsy	Progressive massive fibrosis, caused by the mica exposure
Dianova et al (14)	1976	7	Muscovite, phlogopite and resin	Mica goods factory	Radiography	Mica pneumoconiosis
Berry et al (5)	1976	NI	Quartz, mica, clays	NI	Electron diffraction and electron probe microanalysis of dust from lungs	Silicosis
Berry et al (5)	1976	NI	Talc, cristobalite, chlorites, mica, spinelles	NI	Electron diffraction and electron probe microanalysis of dust from lungs	Pneumoconiosis, no expressed opinion
Sedov et al (48)	1977	31	Phlogopite with 2—71 % free silica	Mica mine	Clinical examination, radiography, biopsy	Pneumoconiosis, no expressed opinion
Pimentel & Menezes (41)	1978	1	Pure muscovite	Mica grinding	Clinical examination, radiography, autopsy, X-ray diffraction	Mica pneumoconiosis
Sherwin et al (51)	1979	7	Silicates (mostly micas), quartz	Farm workers	Clinical examination radiography, autopsy	Interstitial inflammation and fibrosis related to the dust exposure or toxic soil additives
Hayashi (21)	1980	1	Quartz, sericite	Miner	Analytical electron microscopy of pulmonary dust	Pneumoconiosis, no expressed opinion
Li Weizu (34)	1980	16	Mica with 36—55 % quartz	Mica mine	Clinical examination, radiography	Mica mine silicon lung
Seaton et al (47)	1981	4	Mica, quartz, kaolin	Shale mine	Clinical examination, radiography, autopsy (3 cases)	Shale miners pneumoconiosis
Lapenas et al (31)	1982	1	Pure mica	Exposed via husband (grinder)	Clinical examination, radiography, biopsy, mineral analysis by scanning electron microscopy	Interstitial fibrosis, probably caused by the mica exposure
Lapenas et al (31)	1982	1	Mica, probably along with quartz	Slate quarrying	Clinical examination, radiography, biopsy, mineral analysis by scanning electron microscopy	Interstitial fibrosis and fibrotic nodules, probably caused by the mica exposure
Lapenas et al (31)	1982	1	Kaolinite, mica, pigments	Wire insulation	Clinical examination, radiography, biopsy, mineral analysis by scanning electron microscopy	Interstitial fibrosis, probably caused by the mixed dust exposure
Lapenas et al (31)	1982	1	Talc, mica	Exposed via use of body talc	Clinical examination, radiography, biopsy, mineral analysis by scanning electron microscopy	Interstitial fibrosis, probably caused by the talc
Davies & Cotton (13)	1983	1	Pure muscovite	Mica grinding	Clinical examination, radiography	Mica pneumoconiosis
Davies & Cotton (13)	1983	1	Pure muscovite	Mica grinding	Clinical examination, radiography, autopsy, X-ray diffraction	Mica pneumoconiosis
Lankester (30)	1983	2	Mica and/or talc	Rubber tire factory	Clinical examination, radiography	Diffuse interstitial shadowing due to the dust exposure
Sinha (53)	1983	NI	Mica, no details	Mica factories	Radiography	Lung dust disease
Total		368				

lists these cases through 1983. Three studies (5, 29, 53) did not state the number of observed cases; the cases of these studies are accordingly not counted. The surveys by Dreessen et al (15) and Vestal et al (59) were carried out in the same geographic area; thus some cases may occur in both studies.

Table 3. Reported cases of pneumoconiosis associated with mica exposure classified according to the author's view of the causation.

Classification according to the author's view of the causation	Number of cases
Definitely mica pneumoconiosis	35
Probably mica pneumoconiosis	31
Mixed-dust pneumoconiosis	1
Silicosis	135
Pneumoconiosis, not specified; other views	166
Total	368

Table 4. Reported cases of mica pneumoconiosis according to type of dust and workplace.

Workplace	Dust			
	Pure mica	Mica + quartz + others	Mica, but no details	Total
Grinding and packing	21	—	—	21
Other mica production	2	—	8	10
Mining	—	21	—	21
Other	3	8	—	11
No information	—	—	3	3
Total	26	29	11	66

Table 5. Reported cases of mica pneumoconiosis according to diagnostic methods.

Methods	Number of cases
Radiography	7
Clinical examination, radiography	24
Clinical examination, radiography, biopsy	2
Clinical examination, radiography, autopsy	4
Autopsy, mineral analysis	29
Total	66

Table 6. Reported cases of mica pneumoconiosis according to number of years of exposure.

Number of years of exposure	Number of cases
<5	—
5—9	3
10—14	6
15—19	3
≥20	16
No information	38
Total	66

Six epidemiologic studies have been carried out among mica miners (15, 22, 34, 42, 48, 59). All were cross-sectional. Four of them presented information on the quartz content of the dust in the work atmosphere; the quartz content varied from 2 to 71 %. Pneumoconiosis among mica miners has thus been named silicosis (15, 22), mica mine silicon lung (34), or pneumoconiosis (42, 48, 59). A total of 282 cases of pneumoconiosis are reported in the six studies. In five of them tuberculosis was also observed among the patients; the prevalence ranged from 6 % (34) to 50 % (42).

Seven epidemiologic studies (14, 15, 18, 23, 34, 55, 59) have been carried out among mica processing workers. Three of the studies (23, 34, 55) did not report any case of pneumoconiosis among these workers. Some of the earliest epidemiologic studies do not fulfill standard methodological demands for epidemiologic surveys of today (18, 55). Adequate information on Sinha's study (53) is not available, and for this reason it is not classified as an epidemiologic study in our review.

Table 3 presents the 368 reported cases according to the author's view on the causation of the pneumoconiosis. Tables 4, 5, and 6 comprise only the 66 cases which the authors presented in their reports as definitely or probably caused by mica (ie, mica pneumoconiosis).

Table 4 shows the distribution of the cases of mica pneumoconiosis by type of dust and type of workplace. One of the older investigations (18) presented little information on the type of dust and workplace. In nearly half the cases [all of them reported by Jones (24)] quartz was found in the dust. Twenty-one of the 26 cases who were exposed to pure mica had been working in grinding and packing operations (13, 15, 41, 59).

In table 5 the reported cases of mica pneumoconiosis are presented according to the diagnostic methods applied. In the six cases in which clinical examination, radiography, and autopsy or biopsy were performed, interstitial fibrosis was observed (13, 31, 39, 41, 61). Fibrotic nodules were observed in three cases (13, 31, 39). Vorwald et al (61) noted that the diffuse fibrosis had also fused into irregular massive lesions. Jones (24) performed mineral analysis of 29 lungs obtained at autopsy.

In table 6 the reported cases of mica pneumoconiosis are distributed by the number of years of exposure. Information on the length of the exposure period and the level of exposure is often scarce. In 38 of the 66 cases no such information was given. Of the remaining cases 16 had an exposure time exceeding 20 years.

The recent study of Davies & Cotton (13) is of great value although it only reports two cases. In this report a detailed occupational history is given. The patients have been investigated by clinical examination and chest radiographs. In one of the cases

autopsy and microanalysis of the particles retained in the lung tissue were performed. That patient worked from 1951 to 1964, except for three years, grinding and packing mica. After seven years' exposure small opacities were discovered by chest radiography. New radiographs taken in 1964 showed an increased number of small opacities. Later radiographs showed that the nodular and linear shadows had increased in size. Dyspnea and dry cough were recorded in 1972 and 1975. The patient was a smoker. In 1973 the patient contracted myocardial infarction and died from heart failure in 1977. At autopsy, ill-defined fibrotic nodules, more pronounced in the lower lobes, and diffuse interstitial fibrosis in all lobes were found along with deposits of birefringent crystals. Some histiocytes and foreign body giant cells were found, but there were no granulomas. The authors came to the conclusion that there was a direct correlation between the concentration of crystalline material and the extent of the fibrosis. Electron microscopy and X-ray microanalysis of dust extracted from the tissue revealed thin mineral sheet particles varying from less than 1 μm to more than 50 μm. X-ray diffractometry of the particles identified them as muscovite mica.

The second patient worked from 1957 to 1974 grinding and packing powdered mica. Radiographic evidence of pneumoconiosis was observed after six years' exposure to mica dust. The radiographic abnormalities progressed up until 1978. The changes have been classified as category 2, simple pneumoconiosis. On examination a moderate number of crackles at the lung bases and a restricted lung function were recorded.

Other reports on biological effects of mica dust

Seven cases of pleural plaques among mica workers have been reported (28, 55). All had been engaged in sawing and sanding mica sheets. In two of the seven cases other occupational dust exposure was excluded.

In a study of 69 cases of malignant mesothelioma Chahinian et al (10) reported one person with peritoneal mesothelioma who had been exposed to mica. The authors stressed that there was no evidence so far that mica is carcinogenic. Mica has also been observed along with other minerals in the lung tissue in cases of lung cancer (3, 47).

Michailov & Berova (37) observed that many employees in the asbestos and mica curing factory suffered from skin lesions caused by the dust.

Discussion

Experimental studies

Intratracheal instillation is the most frequently employed administration technique. This method is easily carried out but is considered "nonphysiological." When one large dose is instilled, a nonspecific inflammatory reaction may occur and may give a foreign body reaction with cholesterol clefts in the alveoli (26).

Transportation of mica particles to the tracheobronchial lymph nodes (26, 46, 55) is one of the mechanisms which slowly reduces the concentration of mica in the lungs. Le Bouffant et al (32), Sahu et al (46), and Shanker et al (50) found no progression of collagen and/or reticulin formation between 6 and 12 months after exposure. This result might have been due to the efficient clearance of the single dose given by intratracheal instillation. Inhalation studies are therefore important for the evaluation of long-term effects of mica.

The lung tissue response to a certain dose of dust may vary with the particle size distribution. In most studies it is indicated that the particle size has been less than 5 μm. Whether it is the true physical diameter or the aerodynamic diameter (considering shape and density) is only indicated in one report (20). Tripsa & Rotaru (58) claimed that mica particles of 10—25 μm were more pathogenic than particles between 1—6 μm in size. They found granulomas similar to foreign-body granulomas. In the study by Krasnopeeva (29), who also reported granuloma formation and diffuse fibrosis, 20 % of the particles were larger than 5 μm. Three studies (26, 32, 36) reported collagen fibrosis. Two of them (32, 36) did not indicate particle size.

The purity of the mica used in the experimental studies is also important. In most studies detailed chemical and mineralogical analyses are lacking. In earlier investigations light microscopy and chemical analysis have been used for characterizing the dust. However, compared with methods used today, the techniques provide limited information.

In a review of experimental studies of mica (20, 26, 27, 38, 58, 60), Parkes (40) concluded that there is no evidence that mica induces pneumoconiosis.

The results from the experimental studies do not give a unanimous conclusion as to whether pure mica is fibrogenic or not. It is necessary to perform new experimental studies with pure and well-characterized mica samples to determine whether mica induces collagen formation.

Only one experimental report illuminates whether mica is carcinogenic (44).

Case studies

Of the 29 cases described by Jones (24), 21 had worked in collieries. He demonstrated that the "silicotic" lungs contained mainly sericite. He also demonstrated that the incidence of "silicosis" was related to the sericite rather than the quartz content in the rock in gold-bearing quartz areas of South Africa and India and in the anthracite coal fields of Wales and Scotland. Jones (24) concluded that quartz was not the main cause of the pneumoconiosis among the colliers but rather sericite. The question of sericite as

a causative agent in coal worker's pneumoconiosis was again raised in 1982 (54).

Pimentel & Menezes (41) reported one case of mica pneumoconiosis. The autopsy showed extensive areas of diffuse pulmonary fibrosis, emphysematous foci, and honeycombing. Microscopy demonstrated increased numbers of histiocytes, fibroblasts, reticulin, and collagen fibers in the interalveolar septa. In addition, sarcoid-type granulomas were observed in the liver. Mica was found in the sarcoid-type granulomas, and the authors suggested that this observation excludes the differential diagnosis sarcoidosis. The patient had been working for only seven years grinding and packing mica before dying of respiratory failure. The diagnosis of primary generalized sarcoidosis cannot definitely be excluded since granulomas in general may accumulate dust as a secondary phenomenon.

Lapenas et al (31) reported microanalytical findings in lung biopsies from four cases with interstitial lung fibrosis associated with mica exposure. One of them had worked 12 years in a slate quarry. The authors were not able to draw any conclusion as to whether the fibrosis resulted from pure mica or mica in interaction with small amounts of quartz (not detected by the mineral analysis). Another of the cases was exposed to mica dust while laundering her husband's clothing. The diagnosis of pneumoconiosis in this case of pulmonary fibrosis was not considered likely prior to the biopsy and particle analysis. This case demonstrates the importance of a proper dust analysis along with the occupational history. An analysis of minerals from the lungs may be performed by electron microscopy and X-ray microanalytical techniques. These methods, as applied in pneumoconiosis studies, have been described by Abraham (1), Berry et al (5), and Hayashi (21).

Davies & Cotton (13) found mica particles of up to 50 μm in the lung tissue of one of their reported cases. Usually only particles less than 5 μm are expected to reach the lung. Tomb & Corn (57) found that mica particles tested in a horizontal elutriator did not assume a preferred orientation during settling and that the orientation influenced the settling velocity. A reduction in the settling velocity would permit larger particles to be deposited at places where only smaller particles would be expected. These findings can explain the rather large mica particles found in the lung tissue of the exposed patient (13).

Epidemiologic studies of mica-processing workers

Seven epidemiologic studies of mica-processing workers have been published (14, 15, 18, 23, 34, 55, 59). They are all cross-sectional. The prevalence rates vary from 0 % (23, 34, 55) to up to 25 % (18). The variation in prevalence may be explained both by methodological differences and by real differences in the prevalence of mica pneumoconiosis.

In the studies of Ferguson (18) and Smith (55) sufficient information on occupational history and exposure time was not given. Thus the etiology of the disease in these cases might only be a matter of assumption. Smith (55) and Dianova et al (14) used radiography alone as the standard examination method. Ferguson (18) examined only 12 individuals. Thus these studies are of limited value.

Heimann et al (23) examined 61 workers of whom 44 % had abnormal lung radiographs. They classified these findings as early events in the development of dust-induced lesions but not really as pneumoconiosis. The ACGIH *Documentation of the Threshold Limit Values* (2) presents the findings of Heimann et al (23) as mild pneumoconiosis.

In the report by Heimann et al (23) only a few workers had exposure periods exceeding five years. On the other hand Li Weizu (34) examined 302 workers, of whom 90.7 % had worked more than 15 years in mica processing. No cases of mica pneumoconiosis were found, and he concluded that the toxic potential of mica is low. Only three studies give information on the level of dust exposure (15, 23, 34). All of them report mica exposure that is far above the present Norwegian hygienic standard.

Twenty-one of the 66 reported cases of mica pneumoconiosis had worked grinding and packing mica. Parkes (40) maintains that, when crude mica is milled, quartz is not separated until the later stages in the refining process. Dreessen et al (15) have shown that the highest levels of dust exposure occur during the drying and packing process, and the authors claimed that the exposure consists of nearly pure mica. Heimann et al (23) found that during mica processing the mica dust in the work atmosphere contains less than 1 % free silica.

In a review of human studies (15, 23, 28, 41, 55, 59) Parkes (40) concludes that there is no evidence that pure mica can cause pneumoconiosis in man. Lusis (35) points out in his review that respirable mica particles act as inert foreign bodies in the lung, and as such they induce scar tissue formation. After these conclusions were drawn, another three papers (13, 31, 32) have been published demonstrating collagen fibrosis related to exposure to mica.

Present knowledge does not exclude the possibility that pure mica may cause pneumoconiosis in man. Probably there is a causal relationship, but a definite such relationship is difficult to establish. This is due to (i) the long latency period (the disease occurs late in life), (ii) often scarce symptoms, and (iii) coexposure to other types of dust such as quartz, feldspar, and/or asbestos. Mica may also occur in mixed-dust pneumoconiosis. The interaction of mica and other minerals should be further studied with regard to both pneumoconiosis and malignant disease.

Acknowledgments

We thank Ms AE Eide for typing the manuscript.

Thanks are also due to the Norwegian Council for Science and the Humanities and the European Science Foundation for financial support.

References

1. Abraham JL. Documentation of environmental particulate exposure in humans using SEM and EDXA. Scanning Electron Microsc 11 (1979) 751—766.
2. American Conference of Governmental Industrial Hygienists. Documentation of the threshold limit values. Fourth edition. Cincinnati, OH 1980.
3. Anttila S, Sutinen S, Pääkkö P, Alapieti T, Peura R, Sivonen SJ. Quantitative x-ray microanalysis of pulmonary mineral particles in a patient with pneumoconiosis and two primary lung tumours. Br J Ind Med 41 (1984) 468—473.
4. Belt TH, King EJ. Chronic pulmonary disease in South Wales coalminers: III Experimental studies. Med Res Counc Spec Rep Ser 250 (1945) 29—68.
5. Berry JP, Henoc P, Galle P, Pariente R. Pulmonary mineral dust. Am J Pathol 83 (1976) 427—456.
6. Berry LG, Mason B. Mineralogy: Concepts, descriptions, determinations. WH Freeman and Company. San Francisco and London 1959, pp 499—517.
7. Brambilla C, Abraham J, Brambilla E, Benirschke K, Bloor C. Comparative pathology of silicate pneumoconiosis. Am J Pathol 96 (1979) 149—170.
8. Bureau of Mines. Mineral yearbook 1976. Washington, DC 1978, pp 823—831.
9. Bureau of Mines. Mineral yearbook 1981. Washington, DC 1983, pp 593—601.
10. Chahinian P, Pajak TF, Holland JF, Norton L, Ambinder RM, Mandel EM. Diffuse malignant mesothelioma: Prospective evaluation of 69 patients. Ann Int Med 96 (1982) 746—755.
11. Chowdhury RR, ed. Handbook of mica. Chemical Publishing Company, Inc, Brooklyn, NY 1941, p 332.
12. Cummins CL. Sericite and silica: Experimental dust-lesions in rabbits. Br J Exp Pathol 18 (1937) 395—401.
13. Davies D, Cotton R. Mica pneumoconiosis. Br J Ind Med 40 (1983) 22—27.
14. Dianova AV, Kochetkova TA, Rumyantsev GI. The problem of the combined action of the dust of mica and resins used in the production of micanite articles [in Russian]. Gig Sanit 41 (1976): 7, 33—37.
15. Dreessen WC, Dallavalle JM, Edwards TI, Sayers RR, Eason HF, Trice MF. Pneumoconiosis among mica and pegmatite workers. US Public Health Services, Washington, DC 1940. (Public health bull no 250).
16. Drinker CK, Field ME, Drinker P. The cellular response of lymph nodes to suspensions of crystalline silica and to two varieties of sericite introduced through lymphatics. J Ind Hyg 16 (1934) 296—299.
17. Fallon JT, Banting FG. Tissue reaction to sericite. Can Med Assoc J 33 (1935) 407—411.
18. Ferguson TA. Note on mica dust and the lung condition of some mica workers. Unpublished memo to the Commission on Industrial Pulmonary Disease of the Medical Research Council (1932). Cited by Middleton EL. Industrial pulmonary disease due to the inhalation of dust. Lancet 2 (1936) 59—64.
19. Gardner LU. Reaction of the living body to different types of mineral dusts with and without complicating infection. American Institute of Mining and Metallurgical Engineers, New York, NY 1938. (Technical publication no 929).
20. Goldstein B, Rendall REG. The relative toxicities of the main classes of minerals. In: Shapiro HA, ed. Pneumoconiosis: Proceedings of the international conference, Johannesburg. Oxford University Press, London 1970, pp 429—434.
21. Hayashi H. Analytical electron microscopy in the study of pneumoconiosis. Environ Res 23 (1980) 410—421.
22. Heimann H, Moskowitz S, Iyer CRH, Gupta MN, Mankiker NS. Silicosis in mica mining in Bihar, India. Arch Ind Hyg Occup Med 8 (1953) 420—435.
23. Heimann H, Moskowitz S, Iyer CRH, Gupta MN, Mankiker NS. Note on mica dust inhalation. Arch Ind Hyg Occup Med 8 (1953) 531—532.
25. Kajita A, Hayashi H, Mashiko S. An autopsy case of mixed dust pneumoconiosis. Acta Pathol Jpn 22 (1972) 739—753.
26. Kaw JL, Zaidi SH. Effect of mica dust on the lungs of rats. Exp Pathol 8 (1973) 224—231.
27. King EJ, Gilchrist M, Rae MV. Tissue reaction to sericite and shale dusts treated with hydrochloric acid: An experimental investigation on the lungs of rats. J Pathol Bacteriol 59 (1947) 324—327.
28. Kleinfeld M. Pleural calcification as a sign of silicatosis. Am J Med Sci 251 (1966) 215—224.
29. Krasnopeeva LF. Experimental pneumoconiosis caused by the mica dust [in Russian]. Gig Tr Prof Zabol 8 (1964): 11, 3—8.
30. Lankester J. Ten patients with different types of pneumoconiosis due to mica, talc and asbestos in one rubber tyrefactory. In: Abstracts of communications, VIth International pneumoconiosis conference, Bochum. International Labour Office, Geneva 1983, posters p 46.
31. Lapenas DJ, Davis GS, Gale PN, Brody AR. Mineral dusts as etiological agents in pulmonary fibrosis: The diagnostic role of analytical scanning electron microscopy. Am J Clin Pathol 78 (1982) 701—706.
32. Le Bouffant L, Daniel H, Martin JC. The value and limits of the relationship between cytotoxicity and fibrogenicity of various mineral dusts. In: Brown RC, Chamberlain M, Davies R, Gormley IP, ed. The in vitro effect of mineral dust. Academic Press, London, New York, Toronto, Sydney, San Francisco 1980, pp 333—338.
33. Lemon WS, Higgins GM. The tissue reactions of the lung to the intratracheal injection of particulate sericite. Am Rev Tuberc 32 (1935) 243—256.
34. Li Weizu. A preliminary report on the conditions of the Mou mica mine dusts causing lung disease [in Chinese]. Chin J Prev Med 14 (1980) 177—178.
35. Lusis I. Review of the health effects of micas. Ind Miner (London) October (1980) 45—55.
36. Martin JC, Daniel H, Le Bouffant L. Short- and long-term experimental study of the toxicity of coal-mine dust and of some of its constituents. In: Walton WH, ed. Inhaled particles IV. Pergamon Press, Oxford 1977, pp 361—370.
37. Michailov P, Berova N. Professional injuries during production and work with asbestos and mica [in Bulgarian]. Hyg Zdraveopazvane 11 (1968): 2, 145—150.
38. Miller JW, Sayers RR. The physiological response of peritoneal tissue to certain industrial and pure mineral dusts. Public Health Rep 51 (1936) 1677—1689.
39. Misra NP, Jain SC. Progressive massive fibrosis following mica inhalation (a case report). J Ind Med Assoc 21 (1973) 457—459.
40. Parkes WR. Occupational lung disorders. Butterworths, London 1982, pp 315—316.
41. Pimentel JC, Menezes AP. Pulmonary and hepatic granulomatous disorders due to the inhalation of cement and mica dusts. Thorax 33 (1978) 219—227.
42. Podnebesnaya NI. The course of pneumoconiosis in underground workers of Mamsk mica mines [in Russian]. Gig Tr Prof Zabol 9 (1965): 12, 30—33.

43. Policard A. The action of mica dust on pulmonary tissue. J Ind Hyg 16 (1934) 160—164.
44. Pott F, Huth F, Friedrichs KH. Tumorigenic effect of fibrous dusts in experimental animals. Environ Health Perspect 9 (1974) 313—315.
45. Rüttner JR, Spycher MA, Sticher H. Diffuse "asbestos-like" interstitial fibrosis of the lung. Pathol Microbiol 38 (1972) 250—257.
46. Sahu AP, Shanker R, Zaidi SH. Pulmonary response to kaolin, mica and talc in mice. Exp Pathol 16 (1978) 276—282.
47. Seaton A, Lamb D, Brown WR, Sclare G, Middleton WG. Pneumoconiosis of shale miners. Thorax 36 (1981) 412—418.
48. Sedov KR, Sljosar RD, Starkov MV. Clinical and morphological characteristics of the pneumoconiosis caused by dust in the mica mine in Irkutsk region. Gig Tr Prof Zabol 21 (1977): 2, 48—49.
49. Selter H, Weiland P. Staub und Tuberkulose. Arch Gewerbepathol Gewerbehyg 8 (1937) 71—82.
50. Shanker R, Sahu AP, Dogra RKS, Zaidi SH. Effect of intratracheal injection of mica dust on the lymph nodes of guinea pigs. Toxicology 5 (1975) 193—199.
51. Sherwin RP, Barman ML, Abraham JL. Silicate pneumoconiosis of farm workers. Lab Invest 40 (1979) 576—582.
52. Simson FW, Strachan AS. A study of experimental tissue reactions following intravenous injections of silica and other dusts. Publ S Afr Inst Med Res 45 (1940) 95—122.
53. Sinha JK. Pneumoconiosis amongst mine workers in India. In: Abstract of communications, VIth international pneumoconiosis conference, Bochum. International Labour Office, Geneva 1983, posters p 2.
54. Slater D, Durrant T. Quartz and pneumoconiosis in coal miners. Lancet 1 (1982) 45—46.
55. Smith AR. Pleural calcification resulting from exposure to certain dusts. Am J Roentgenol 67 (1952) 375—382.
56. Starkov MV, Javerbaum PM, Vjatchina EA. Biochemical and morphological evaluation of the formation of collagen in the lungs in experimental pneumoconiosis, caused by mica dust and mixed dust from the mica occurrence of the Irkutsk region. Gig Tr Prof Zabol 15 (1971): 8, 42—43.
57. Tomb TF, Corn M. Comparison of equivalent spherical volume and aerodynamic diameters for irregularly shaped particles. Am Ind Hyg Assoc J 34 (1973) 13—24.
58. Tripsa R, Rotaru G. Recherches experimentales sur la pneumoconiose provoquee par la poussiere de mica. Med Lav 57 (1966) 492—500.
59. Vestal TF, Winstead JA, Joilet PV. Pneumoconiosis among mica and pegmatite workers. Ind Med Surg 12 (1943) 11—14.
60. Vorwald AJ. Diffuse fibrogenic pneumoconiosis. Ind Med Surg 29 (1960) 353—358.
61. Vorwald AJ, MacEwen JD, Smith RG. Mineral content of lung in certain pneumoconiosis. Arch Pathol 74 (1962) 267—274.

Received for publication: 4 February 1985