Perlite

(CAS No: 93763-70-3)

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 perlite 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 JAGM van Raaij, Ph.D., WK de Raat, Ph.D., and J Krüse, Ph.D. (OpdenKamp Registration & Notification, The Hague, the Netherlands).

In December 1998, literature was searched in the on-line databases Toxline, Medline, and Chemical Abstracts covering the period 1965-1966 until January 1999, and using the following key word: perlite. Information published by the American Conference of Governmental Industrial Hygienists (ACGIH) was also used (ACG99). The final search was carried out in Toxline and Medline in October 2002.

It is stated by the ACGIH that perlite is essentially a non-crystalline silicate. However, it is also stated that the free crystalline silica content in some perlite ores and in expanded perlite ranged from <1% to 5.8% (ACG99, Coo75). Crystalline silica is, in contrast to amorphous silica, considered to be hazardous to the human respiratory tract (DEC92). Therefore, the short- and long-term toxic effects of free crystalline silica, in particular with respect to the effects on the respiratory tract, have been taken into account to assess the risk of perlite exposure of humans. To this end, the committee applied a review on the risk of silica substances to humans from the Dutch Ministry of Social Affairs and Employment (DEC92).

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

name	:	perlite
synonyms	:	-
molecular formula	:	-
structural formula	:	-
CAS number	:	93763-70-3

Data from ACG99.

3 Physical and chemical properties

molecular weight	:	-
boiling point	:	-
melting point	:	>1093°C
vapour pressure	:	-
solubility in water	:	very slightly soluble (<1%)
log $P_{octanol/water}$	:	-
conversion factors	:	not applicable

Data from ACG99.

Commercial perlite is a naturally occurring glass of volcanic origin, which, when heated, expands to form a product of low density, high surface area, and low thermal conductivity. The ore is essentially an amorphous, hydrated glassy volcanic rock consisting of fused sodium potassium aluminium silicate of variable composition. The chemical composition depends on the particular ore body in which it is found. The colour of crude perlite ranges from transparent light grey to glossy black (ACG99). Perlite exists as crude and as expanded material (Mic71). High-temperature treatment of the crude material in furnaces where temperatures are maintained in the range of 760 to 1100°C yields the expanded form. Expanded perlite is a white powder with a bulk weight of 55-220 kg/m^3, available in different sizes, e.g., 4-8 mesh and finer (ACG99). There is no evidence that the heat treatment causes the formation of cristobalite as happens when kieselgur is calcined (Elm87).

Perlite ores and expanded perlite are about 75% non-crystalline silicate. Both may contain small amounts (<1% to 5.8%) of quartz, which complicates the evaluation of the potential health hazards (Coo75). In two material safety data sheets, commercial perlite was stated to contain less than 0.1% α-cristobalite and tridymite and 0.01-0.05 % α-quartz (Red98, Sch01).

4 Uses

The commercial production of perlite started in 1946. Heat-expanded perlite is used in thermal insulation, in lightweight plasters, as a filter aid, as an inert carrier and filler, as concrete aggregate, and as soil improver. It is applied in the

chemical, foundry, steel, and food industries and in horticulture (ACG99, Elm87).

5 Biotransformation and kinetics

The committee did not find data on the biotransformation or kinetics of perlite.

6 Effects and mechanism of action

Human data

To investigate whether long-term exposure of workers to perlite dust is associated with the development of pulmonary effects, pneumoconiosis in particular, 3 studies have been reported by the same author, over the period 1975-1986 (Coo75, Coo76, Coo86). In the first study, conducted in 1972/73, chest roentgenograms were examined of 240 men who had worked in the perlite industry in the United States, producing crude or expanded perlite, for 1 to 23 years. The last available film for each worker was used. Since the films studied were from men who had terminated work in the past as well as men still at work, the last film available for each man had been taken in the period between 1955 and 1974. Of the 240 workers, 231 showed films without pulmonary changes consistent with pneumoconiosis, 7 showed films classified as doubtful, and 2 showed films classified as positive. The 2 subjects with pneumoconiosis and 2 out of 7 subjects who had films classified as 'doubtful' had prior histories of working with diatomaceous earth. None of the 28 workers who had been working in the perlite industry for more than 15 years developed pneumoconiosis. Limited exposure data suggest that the population had had exposures to perlite dust probably exceeding the nuisance dust levels of 10 mg/m^3. The author's conclusion was that in this worker population, there was no evidence of pneumoconiosis associated with perlite exposures (Coo75).

In the second study, conducted in 1975, possible pulmonary effects were examined of 117 men engaged in the mining and processing of perlite in 3 plants in the major perlite-producing area of the United States for periods up to 23 years. Of these, 38 had been employed for 10 years or more, 18 for 15 years or more, and 4 men for 20 years or more. The most recent chest roentgenograms of the 117 perlite workers were examined. Two out of the 117 workers showed films classified as positive. However, the pulmonary changes were not interpreted as pneumoconiosis. Pulmonary function was measured during a 4-day period by determination of the forced vital capacity (FVC) and the 1-second

forced expiratory volume (FEV_1). An increase in FVC, but a decrease in FEV_1 and in the ratio FEV_1/FVC was found that correlated with length of employment, after effects of cigarette smoking had been taken into account. However, none of these changes were statistically significant. The authors concluded that there was no evidence of pneumoconiosis in men who had worked in the perlite industry for periods up to 23 years. Exposure levels and free silica content of the perlite were not reported (Coo76).

The third study, conducted in 1983, was a follow-up of the 1975 study in workers employed in the same 3 plants. The study population comprised 152 workers with at least 5 years of employment and for whom chest films were available. For current employees, new films were requested, if none had been taken within the past year. For former perlite workers, the most recent film was used. Of the 152 workers, 60 had perlite employment for 5-9 years, 73 for 10-19 years, and 19 for 20-29 years. Most of the workers were exposed to crude perlite rather than the expanded product. Airborne exposure levels to perlite dust in representative work areas were nearly all below the nuisance dust concentration of 10 mg/m^3. The amount of quartz in the perlite was not reported. Chest radiography did not show any positive film, consistent with pneumoconiosis, in any of the workers. Pulmonary function was conducted in 120 out of the 152 workers during a 4-day period by determination of FVC and FEV_1. Of these 120 workers, 66 had also been examined in 1975. Both FVC and FEV_1 were negatively correlated with years of employment in the perlite industry, but the association was not statistically significant. In contrast, smoking was found to be the most important factor influencing the pulmonary function in the perlite workers. This was also seen in the 66 workers who were tested in 1975 and again in 1983: the greater mean decrease in FVC observed in workers compared with a non-smoking reference population was not due to perlite exposure, but could be explained by the severe smoking habits of 28 of the workers. However, the mean decrease in FEV_1 in the workers compared with the reference population was less than expected, which was explained by the very small average decrease in FEV_1 in the groups of light smokers and non-smokers. The authors concluded that there was no evidence of perlite-induced pneumoconiosis, and that perlite should continue to be treated as a nuisance dust when it is not contaminated with quartz dust above 1% (Coo86).

The findings of Cooper (Coo75, Coo76, Coo86) are in agreement with the statement of Elmer (Elm87) that dust particles from perlite (expanded) are unlikely to survive long enough in the lung to cause fibrosis after prolonged exposure to acceptable levels.

Pulmonary function tests (FVC; FEV_1; FEV_1/FVC; $FEF_{50\%}$ and $FEF_{75\%}$, i.e., forced expiratory flow when 50 or 75%, respectively, of forced expiratory vital capacity has been exhaled; T_L, CO, i.e., diffusing capacity of the lung for carbon monoxide; and K_{CO}*, i.e., carbon monoxide transfer coefficient) in conjunction with chest radiograms were carried out in 9 non-smoking and 27 smoking perlite-exposed male workers and 22 unexposed male office workers (all smokers) of a Turkish perlite plant in 1992, after an average of 12 years of work, and 1996. In 1991 and 1994, perlite concentrations in processing area dust samples were on average 7.5 and 12.83 mg/m^3, respectively, and in personal respirable dust samples 4.56 and 9.89 mg/m^3, respectively. In the non-smoking workers, the pulmonary function test values observed in 1992 for FEV_1, T_L, CO, and K_{CO} were statistically significantly higher than predicted values. In 1996, observed values for FEV_1 and FEV_1/FVC were significantly higher than predicted values. In comparison to 1992, a significant decrease was found in K_{CO} in 1996. While T_L, CO and K_{CO} values were higher than predicted values in 8/9 and 9/9 workers, respectively, in 1992, these figures changed to 5/9 and 3/9 workers, respectively, in 1996. For 4/9 workers, T_L, CO values were 81-100% of predicted values (vs. 1/9 in 1992); K_{CO} values were 81-100 or 61-80% of predicted values in 2/9 and 4/9 workers, respectively. In contrast, the percentage of workers with higher than predicted values did not change for FEV_1 or FVC and increased for FEV_1/FVC. According to Polatli et al., the change in diffusing capacity suggests that 4-year exposure to relatively high dust levels (i.e., above the 'permissible' dust level of 5 mg/m^3) may affect the condition of the interstitial tissue, which may be interpreted as an early indicator for respiratory airway effects. In the smoking perlite-workers, the pulmonary function test values observed in 1992 were similar to the predicted values except for a statistically significantly lower $FEF_{75\%}$. In 1996, observed values were similar to predicted values, except for significantly lower $FEF_{75\%}$ and K_{CO}. Both these parameters and FEV_1/FVC and T_L, CO were statistically significantly decreased in comparison to 1992. Similar changes as found in the non-smoking and the smoking perlite workers were observed in the smoking control group. According to Polatli et al, damage to small airways, parenchymal and interstitium, caused by smoking might be worsened by synergistic action of perlite exposure. There was no correlation between duration of work in perlite areas and pulmonary function test parameters whereas a significant correlation was found between smoking history in smoking perlite and control workers, T_L, CO, and K_{CO}. Although chest radiography showed abnormalities in 10/27 smoking perlite

* Or T_L, CO/V_A (V_A: alveolar volume).

workers, large opacities or higher profusion categories were not observed. These abnormalities included small rounded opacities in profusion (doubtful) in 3, possible tuberculosis in 3, pleural calcification in 2, and probable asbestosis due to environmental exposure in 2 workers. In non-smoking perlite workers, all chest radiographs were normal (Pol01). Polatli et al summarised results of other in Turkish published studies on perlite-exposed workers. In one study, significant decreases in $FEF_{50\%}$ and $FEF_{75\%}$ were found in a group of 27 smoking and non-smoking workers (average age: 33 years) exposed to (unreported) perlite dust levels for 4 years. As numbers were small, non-smokers were not evaluated separately. In another study on 53 perlite workers, small airway obstruction was determined. The decrease in flow rate in small airways was concluded to be associated with smoking because 70% of the workers had smoking histories up to 17.9 pack-years. A significant association between smoking period and respiratory functional decrease was found (Pol01).

In 2 additional, unpublished studies, reported in 1990 and 1994, it was concluded that the workers studied were 'free of any evidence of a silicosis risk, or indeed, any measurable adverse respiratory effects of perlite exposure' (not any other information available) (Sch02).

Animal data

Irritation and sensitisation

The committee did not find data from irritation or sensitisation studies with perlite.

Acute toxicity

The committee did not find data on the acute respiratory or dermal toxicity of perlite. The acute oral LD_{50} in mice was >12,960 mg/kg bw. No signs of toxicity were reported (Ohk72, Ito81). In 2 other unpublished studies, the oral LD_{50} in rats was stated to be greater than 10,000 mg/kg bw, the highest dose tested (Sch02).

Short-term toxicity

In an inhalation study, guinea pigs (n=5/sex/group) were exposed to perlite (non-crystalline) dust at mean airborne concentrations of 0 or 6600 mg/m^3, 30 minutes/day, 5 days/week, for 24 weeks. The mass mean aerodynamic diameters

(MMAD) were between 1.7 and 1.9 µm. Effects observed were minor respiratory distress, increased locomotion, and shaking and brushing their faces with their paws in an attempt to prevent dust from entering airways. These symptoms disappeared 1-2 hours following cessation of exposure. No body weight loss or change in food intake was observed in treated animals compared with the controls. After 4 weeks, tidal volume and respiratory rate were measured by fastening a small pneumograph around the chest of the animal. Parameters were recorded before perlite exposure, at 15 and 30 minutes during exposure, and at 30 minutes following termination of exposure. Tidal volume and respiratory rate were decreased by 60% and 40%, respectively, at the end of the 30 minutes exposure period, compared with pre-exposure values. However, values returned to pre-exposure levels at the end of the 30-minute recovery period. Gross examination of the lungs revealed oedematous and haemorrhagic foci. Microscopic changes of the lung included lymphoid aggregates in the adventitia of blood vessels and terminal bronchioles, and thickened interalveolar septa. Dust particles were present in phagocytes and were free in the alveoli and terminal bronchioles. However, no evidence of pulmonary fibrosis or extensive destruction of parenchymal tissues could be demonstrated. The authors concluded that perlite particles are more than just a nuisance substance (McM78, McM83).

Rats given a single intratracheal instillation of 50 mg perlite dust showed pulmonary fibrosis at 12 to 18 months following administration. The perlite composition was not specified (Liu88). Intratracheal infusing of 5 mg perlite dust in 5% alcohol did not result in a strong pulmonary fibrinogenic reaction in rats, at 12 weeks after administration. No more details were provided (Ued78). In another study, a single 50-mg suspension of crude or expanded perlite was administered intratracheally to albino rats. Nine months after administration, it was found that expanded perlite had caused more lung fibrosis in the sacrificed animals than the crude perlite. The degree of fibrosis was not reported. The increased fibrosis from the expanded material was attributed to the higher cristobalite and tridymite content (Bor67). A single intratracheal instillation of a 75 mg/mL saline dose of perlite (18-30% quartz) produced a ‘foreign body reaction’ in white male rats, but no pulmonary fibrosis was observed. The post-exposure observation period was not reported (Tim65). In an earlier study, 9 perlite products were tested in guinea pigs, by weekly intratracheal injection of 0.5 ml of a 5% perlite suspension in saline for 3 weeks. At 4 to 12 months after the last injection, no evidence of pulmonary fibrosis was shown (Vor53).

CRJ:ICR mice (n=21/sex/group) were fed perlite via the diet at doses of 0, 1500, 15,000, or 30,000 mg/kg bw/day, for 28 weeks. The powder

(characterised as 'crystalline' by the authors) consisted mainly of SiO_2 (71.74%), Al_2O_3 (14.94%), and K_2O (6.86%). No effects on mortality, food intake, or behaviour were observed in the treated groups compared with the controls. A slight reduction of growth rate was seen in the males that were fed the 2 highest doses. No abnormalities were observed in organ weights and in clinical chemical and haematological parameters when compared with controls. Microscopic examination did not reveal treatment-related changes in any of the organs investigated. The NOAEL was 1500 mg/kg bw/day of perlite in the diet, based on a slight reduction of growth rate at 15,000 mg/kg bw/day (Sak85).

Long-term toxicity and carcinogenicity

In an 18-month inhalation study, guinea pigs and rats (numbers, strain, or sex not given) were exposed to perlite dust at a concentration of 226 mg/m^3. No significant pulmonary reaction, including fibrosis, was observed. No further details were given. The authors concluded that perlite acted as inert or nuisance dust (Vor53).

Mutagenicity and genotoxicity

The committee did not find data from mutagenicity or genotoxicity studies with perlite.

Reproduction toxicity

The committee did not find data from reproduction toxicity studies with perlite.

7 Existing exposure limits

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

In other European countries, no occupational exposure limit for perlite has been established. Existing occupational exposure limits in the USA are summarised in the annex.

8 Assessment of health hazard

Workers can be exposed to crude or heat-expanded perlite through inhalation of the respirable fraction of the substance. The committee could not find data of the percentage uptake of perlite through the lungs, or of the biokinetics or metabolism of the compound following absorption.

In 3 long-term occupational studies in the USA, conducted in the period 1972-1983 on groups of 450, 117, and 152 workers, no evidence of a relationship between working in the perlite industry and the development of lung fibrosis (pneumoconiosis) was found. Tests included chest radiography and lung function parameters FVC and FEV_1. No data on personal air exposures were reported, but workplace monitoring in the plants showed that airborne concentrations were close to the nuisance dust level of 10 mg/m^3. No data were provided on the percentage of quartz in the perlite, which may vary from <0.1% to about 6%. In 2 additional, unpublished studies, reported in 1990 and 1994, no evidence of a silicosis risk or any other adverse respiratory effect was stated to have been found.

In experimental animals, the committee could not find data on eye or skin irritation, or on skin sensitisation. One acute oral lethal toxicity study revealed that the compound did not cause harm after ingestion. A number of short-term studies and one long-term study have been reported. These comprised 2 inhalation studies, 5 intratracheal-instillation studies and one oral (feeding) study. Only the latter included the usual range of toxicological endpoints. The other studies were all focussed on the effects of perlite on the lungs. Adverse local effects on the respiratory tract (lymphoid aggregation, perivascular inflammatory response), but no evidence of pulmonary fibrosis or extensive destruction of parenchymal tissues, were demonstrated in guinea pigs, exposed to an air concentration of 6600 mg/m^3, 30 minutes/day, 5 days/week, for 24 weeks. Because of the unrealistically high air concentration, the committee considered this experiment not relevant to the occupational situation. A long-term inhalation study (18 months) in rats or guinea pigs, exposed to a much lower air concentration (226 mg/m^3), did not reveal any effects on the respiratory tract. In 3 out of 5 studies, adverse local effects (fibrosis) were found, 9 to 18 months after single intratracheal instillation at doses of 5 to 50 mg of perlite. The committee is of the opinion that fibrosis might have been caused by the presence of quartz in the perlite samples tested. The committee also comments that the relevance of the intratracheal route of exposure for risk assessment of inhalation exposure to perlite is uncertain. The 28-week oral study in mice did not provide

relevant information on local effects on the respiratory tract. The NOAEL was 1500 mg/kg bw/day, based on a slight reduction of growth rate at 15,000 mg/kg bw/day.

Based on the above data, the committee takes the NOAEL of 226 mg/m^3 in rats or guinea pigs (18-month inhalation study) as a starting point in deriving a health-based recommended occupational exposure limit (HBROEL). For extrapolation to a HBROEL, an overall assessment factor of 9 is established. This factor covers the following aspects: intra- and interspecies variation. Applying this factor and the preferred value approach would lead to a health-based occupational exposure limit of 20 mg/m^3, which is still higher than the usual occupational exposure limit for nuisance dust (10 mg/m^3).

As an alternative, the committee takes the occupational studies of Cooper as a starting point in deriving a HBROEL, in spite of lack of personal exposure data. However, in the absence of such data, the author gives useful information on workplace exposures, which were close to the occupational exposure limit for nuisance dust. More importantly, it has been demonstrated that long-term exposure to perlite does not lead to lung effects. Therefore, perlite can be considered as a nuisance dust, for which a health-based occupational exposure limit of 10 mg/m^3 is recommended.

However, perlite may contain varying amounts of crystalline silica. The Dutch Expert Committee on Occupational Standards (DECOS), another committee of the Health Council of the Netherlands, has recommended a health-based occupational exposure limit for crystalline forms of silicon dioxide (quartz, including cristobalite and tridymite) of 0.075 mg/m^3 as respirable dust, 8-hour TWA. This concentration should prevent adverse chronic effects on the respiratory system (DEC92). Thus, when perlite contains ca. 0.7% or more crystalline silica, the MAC value for crystalline forms of silicon dioxide will be exceeded.

The committee recommends a health-based occupational exposure limit for perlite (containing <0.7% crystalline silica) of 5 mg/m^3 as respirable dust and 10 mg/m^3 as inhalable dust, as 8-hour time-weighted averages (TWA). A skin notation is not deemed necessary. For perlite containing 0.7% crystalline silica, the MAC value for crystalline silica should be applied.

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Annex

Occupational exposure limits for perlite in various countries.

country - organisation	occupational exposure limit		time-weighted average	type of exposure limit	note[a]	reference[b]
	ppm	mg/m^3				
the Netherlands						
- Ministry of Social Affairs and Employment	-	10[c]	8 h	administrative		SZW03
Germany						
- AGS	-	-				TRG00
- DFG MAK-Kommission	-	-				DFG02
Great Britain						
- HSE	-	-				HSE02
Sweden	-	-				Swe00
Denmark	-	-				Arb02
USA						
- ACGIH	-	10[d]	8 h	TLV	A4[e]	ACG03b
- OSHA	-	15[f]	8 h	PEL		ACG03a
	-	5[g]	15 min			
- NIOSH	-	10[f]	10 h	REL		ACG03a
	-	5[g]	15 min			
European Union						
- SCOEL	-	-				EC03

[a] S = skin notation, which means that skin absorption may contribute considerably to body burden; sens = substance can cause sensitisation.

[b] Reference to the most recent official publication of occupational exposure limits.

[c] As inhalable dust.

[d] The value is for particular matter containing no asbestos and <1% crystalline silica.

[e] Classified in carcinogen category A4, i.e., not classifiable as a human carcinogen: agents which cause concern that they could be carcinogenic for humans but which cannot be assessed conclusively because of lack of data. *In vitro* or animal studies do not provide indications of carcinogenicity which are sufficient to classify the agent into one of the other categories.

[f] As total dust.

[g] As respirable dust.