POSSIBLE HEALTH HAZARDS FROM DIFFERENT TYPES OF AMORPHOUS SILICAS - SUGGESTED THRESHOLD LIMIT VALUES

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necessary also to make an x-ray analysis of the sample. (Contrary to what is stated in the NIOSH Manual of Analytical Methods, second edition Volume 1, page 110-2, amorphous silica does indeed interfere with the infrared determination of quartz and other chrystaline forms of silica). If the potassium bromide method has been used for the infrared analysis, the tablet may be dissolved in water and the particles collected on 25 mm diameter Nuclepore filter which then may be used directly for x-ray difraction analysis, see Altree-Williams[1] or Edholm, Gylseth and Nicholson[2]. A detailed description of the x-ray difraction method is given by Edholm and Nicholson[3].

HEALTH HAZARDS TO HUMAN BEINGS

Diatomaceous earth pneumoconiosis has been described by Sander[4] in the second edition of Patty's "Industrial hygiene and toxicology." (This section has been removed in the third edition). In a medical study, 869 workers from five plants were x-rayed and 786 had complete medical examinations and laboratory studies. The main conclusions were that 9 % of the 869 x-rayed workers showed changes consistent with a diagnosis of pneumoconiosis, and that those at work in the mills had a higher risk than those employed in the quarries. The quartz-content in both crude and milled diatomite averaged 2 %. Workers with massive confluent lesions showed reduced pulmonary ventilatory function, but those with only linear or nodular changes (on the x-ray film) showed no consistent pattern of disturbed lung function. This work was carried out by United States Public Health Service. Sanders also sites a work by Smart and Anderson[5] who concluded that inhalation of crude diatomite dust caused only benign linear fibrosis with no symptoms or disability. Heavy exposure to calcined diatomite, however, often caused rapidly progressive fibrosis.

There are conflicting reports on the risk of silicosis among ferro-silicon operators. Fehnel[8] did not find a single case of silicosis at American ferro-silicon plants. Panchery[9] found slight fibrosis, but no silicosis among the workers at ferro-silicon plants. Re-examining Pancherys material, Radica[10] was also only able to show reticulation, which had increased in two cases. Neither Drees and Jung[11] nor Roberts[12] have detected silicosis among ferro-silicon workers, in spite of partly heavy exposure to amorphous silica.

Broch[13] claimed that he had found 29 cases of silicosis and 30 suspect cases among 208 workers at Norwegian plants. Five of the cases had worked in a quartz quarry or in the quartz crushing department. One person had worked for 8 years in a foundry before working 8 years in the ferro silicon furnace-house. Unfortunately, the dust samples collected were only analysed chemically, so it is unknown whether the dust contained crystalline silica.

Glømme and Swensson[14], studied 865 workers from Norwegian and Swedish ferro silicon-smelting plants. They found 17 cases where the change in lung tissue corresponded to silicosis. Of these, four had worked in the atmosphere of the furnace-house. These four had slight changes on the lungs, and the authors conclude that the risk of lung dust disease due to the furnace-house atmosphere itself seems to be moderate.

Bruce[15] was first to claim the occurence of silicosis in the manufacture of silicon alloys. Among a total 64 workers in two small smelting plants he claimed to have found 10 cases of silicosis. These cases were thoroughly re-examined by Swensson, Kvarnstrøm, Bruce, Edling and Glømme[16] with the result that the diagnosis of silicosis could be maintained only in one of the tissue reaction in the lungs, which afterwards did not show any tendency to progress. These particles were substantially smaller than the quartz particles, 0.15μm as against 1.2μm. The reaction to amorphous particles from a ferro-silicon smelting furnace was considerably smaller than that from quartz, fused silica and the silica produced by combustion of the silicon-halogen compound.

Renewed animal experiments by Glømme and Swensson[14] with dust from ferro-silicon smelting furnace confirmed that this type of dust caused considerably less fibrotic changes than quartz with particle size smaller than 5μm (mean value 1.2μm), while quartz with particle size less than 0.3μm (mean value 0.12μm) was somewhere in between. Some of the results are illustrated in figures 1 and 2, (App. 2) giving the weight of respectively lungs and lymph nodes as a function of time after injection. There seems to be a good correlation between the weight of these organs and the degree of fibrosis.

Prochazka[19] concluded that amorphous silica from electro-chemical high temperature prosesses not only caused major damage to cells (in vitro experiments), it also caused silicotic tissue reactions in animal tests. He does not, however, say anything about the degree of the silicotic effect and adds that further tests are necessary and that a justified occupational health judgement of the dust conditions in the ferro-silicon industry was not possible at the time being.

Swensson[20] has investigated the fibroginetic effect of a special amorphous silicon dioxide obtained as a by-product in the production of aluminium fluoride. Animal experiments showed that the particles had an acute irritative effect and made it difficult for the animals to breathe during the first 24 hours after injection. This is an unusual reaction are in many cases extremely old and have been subjected to considerable pressure, may be also some temperature. This could probably explain the reported cases of silicosis from mining and milling of diatomaceous earth, as the inferior amount of crystalline silica, normally about 2 %, does not seem sufficient to explain these cases unless the exposure had been extreme.

The available data is not adequate for setting up definite threshold limit values for the various amorphous silicas. For this purpose it would be necessary to have the lifetime dose, for instance calculated as suggested by Jahr[20] coupled with definite medical diagnosis for each of many persons not exposed to other fibrinogenic dusts. We are, however, able to make farely good "guesstimates" as given in Appendix 3. If these values are not exceeded, the risk of contracting silicosis seems to be extremely small.

10) Radica, U.: "Contributo alla studio sulla pneumoconiosi da ferrosilico", Rass Med. Industri. 3(25): pp. 181-185 (1956).

11) Drees and Jung, cited by Swensson et al., see 16).

12) Roberts, W.C.: "The ferroalloy industry hazards of the alloys and semi-metals", part II, J. Occup. Med. 7, pp. 71-77 (1965).

13) Broch, C.: "Silicosis caused by the smoke dust in a ferrosilicon and ferro-chromium smelting-plant", (In Norwegian, with English summary and English text for tables and figures), Olaf Norlis Forlag, Oslo 1953.

14) Glømme, J. and Swensson, Å.: "Risikoen for støvlungesykdom i ferrisilicium-smelteverk", Del I - IV, Yrkeshygienisk institutt, Oslo and Karolinska sjukhuset, Stockholm, 1965 - 66. Stencil.

15) Bruce, T.: "The occurence of silicosis in the manufacture of silicon alloys", J. Indust. Hyg. Toxicol. 19, pp. 155-162 (1937).

16) Swensson, Å., Kvarnstrøm, K., Edling, N.G.P. and Glømme, J.: "Pneumiconiosis in Ferrosilicon Workers - A Follow-Up Study", J. Occup. Med. 13, pp. 427-432 (1971).

17) Vitums, V.C., Edwards, M.J. and Niles, N.R.: "Pulmonary Fibrosis from Amorphous Silica Dust; a Product of Silica Vapor". Arch. Environ, Health, pp. 62-67, March/April 1977.

Trace element, PAH and SiO_2 in amorphous silica from silicon and ferro-silicon production.

From production of Si-metal

	Institute of Occupational Health 1978				E - S 1976
Sample prep.	Extraction with HNO_3		Complete		(Not the same sample)
Component	Parallels %	Mean %	Parallels %	Mean %	%
Cd	0,00004 0,00005	0,00005	0,00064 0,00043	0,0005	< 0,0001
Ni	0,00001 0,00001	0,00001	< 0,009 < 0,009	< 0,009	0,001 0,002
Pb	0,0016 0,0016	0,0016	0,0052 0,0060	0,006	0,001 0,002
Cr	< 0,002 < 0,002	< 0,002	< 0,004 < 0,004	< 0,004	
As	0,00091 0,00087	0,0009	0,031 0,036	0,034	
V	0,000057 0,000057	0,00006	0,0007 0,0007	0,0007	
Co	< 0,0002 < 0,0002	< 0,0002	0,0044 0,0044	0,004 0,004	0,001 0,002
PAH		< 0,0002			C= 0,2 - 1,3
Total SiO_2	-	-	-	-	94 - 98

From production of 75 % FeSi

Component	Parallels %	Mean %	Parallels %	Mean %	E - S %
Cd	0,00012 0,00012	0,0001	0,00043 0,00022	0,0003	< 0,0001
Ni	0,00046 0,00040	0,0004	0,004 0,003	0,004	0,02 0,04
Pb	0,012 0,012	0,012	0,022 0,023	0,023	0,005 0,006
Cr	0,0016 0,0016	0,0016	0,0066 0,0067	0,0067	
As	0,0105 0,0113	0,011	0,14 0,14	0,14	
V	0,000080 0,000084	0,00008	0,0009 0,0005	0,0007	
Co	< 0,0002 < 0,0002	< 0,0002	0,0022 0,0033	0,003	0,05 0,06
PAH *		< 0,0011			2,0 - 4,0% C
Total SiO_2	-	-	-	-	86 - 90

* 3 samples from a closed furnace: 0.37, 0.24 and 0.65 % PAH.

Free crystalline silica could not be detected by x-ray analysis in any of the samples analysed by Institute of Occupational Health.

Reproduced from "Risikoen for støvlungesykdom i ferrosilisiumverk, del II. Dyreeksperimentelle undersøkelser" by Jon Glømme (†). 14)

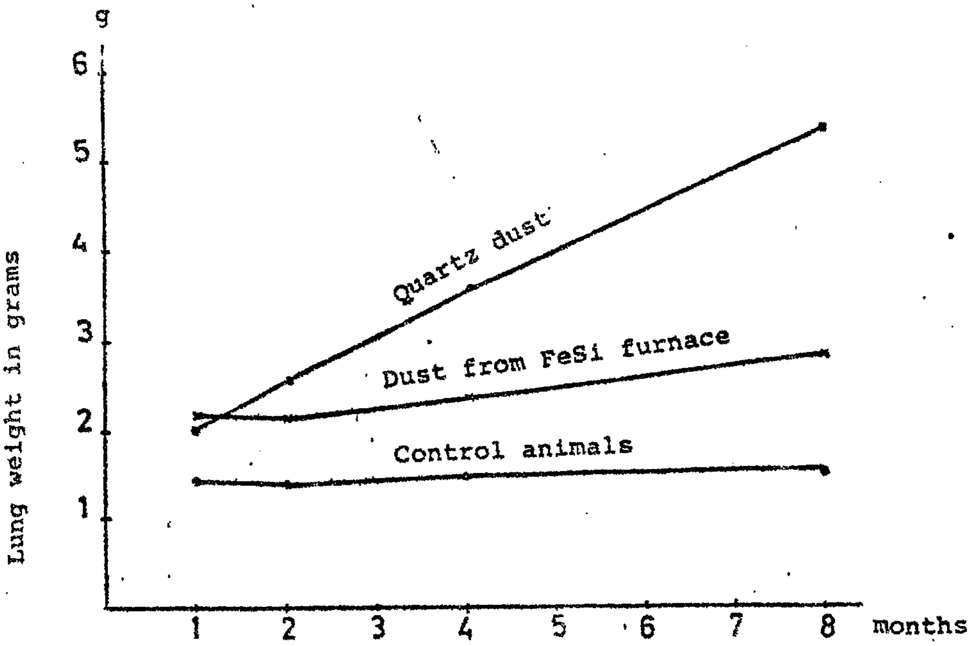

Fig.1. Weight of the lungs as a function of time after a single intratracheal injection of 40 mg quartz and dust from FeSi furnaces and for untreated control animals.

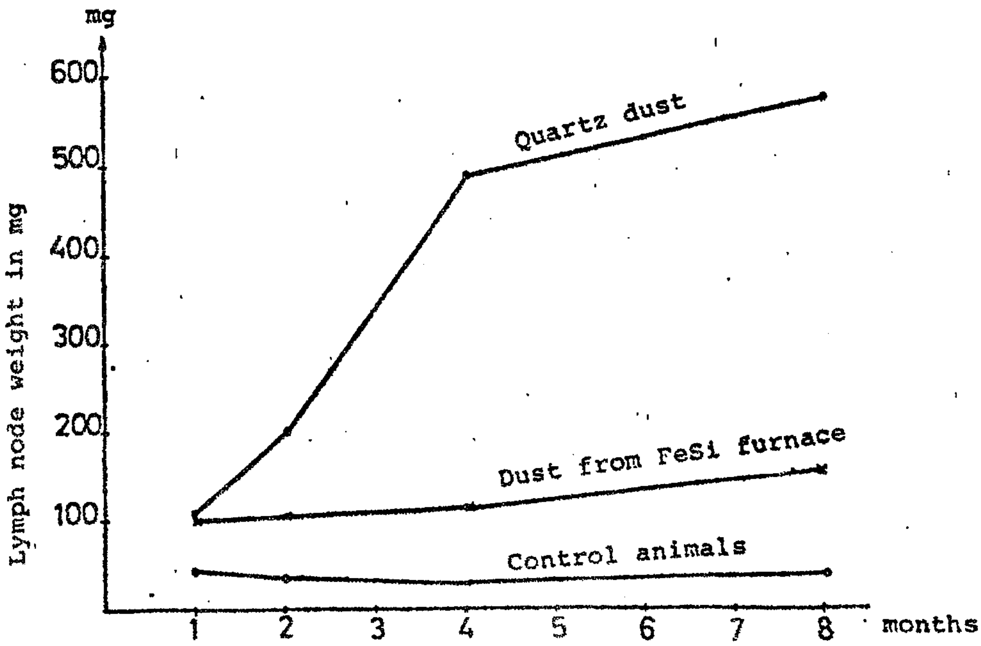

Fig.2. Weight of lymph nodes as a function of time after a single intratracheal injection of 40 mg quartz and dust from FeSi furnace, and for untreated control animals.

SUGGESTED TLV's FOR DIFFERENT SILICAS WITH PARTICLE SIZE < 5μm.

Type	Note	TLV mg/m^3
Cristobalite (C)		0.1
Tridymite (T)		0.1
α-Quartz (Q)		0.2
Amorphous silica from production of AlF_3 (A)	TLV set on basis of irritating effect	1.0
Diatomeaceous earth (Kieselguhr) (D)	With crystalline silica ≤2 %	1.5
Amorphous silica from FeSi and Si smelter (S)		2.0
Precipitated amorphous silica (P)		2.0
Inert dust (I)	Nuisance dust	5.0

For mixtures:

$$TLV = \frac{1}{\frac{\%C + \%T}{10} + \frac{\%Q}{20} + \frac{\%A}{100} + \frac{\%D}{150} + \frac{\%S + \%P}{200} + \frac{\%I}{500}}$$