

TOXICITY DATA
Silica

LAM 026428

ABS-044362

Published by AsbestosClaims.Law
asbestosclaims.law/library/17086/1

SILICA October 28, 1954

Mr. R. B. Mosely of the Petroleum Refining Department consulted me concerning the possible hazard of silica encountered in the catalytic refining operations. He stated that certain of his laboratory assistants had raised questions concerning possible health hazards from breathing the duts which are encountered in transferring and handling the silica and magnesium powders which are present in a finely-divided state.

A discussion was held with Mr. Mosely and it was found that the period of exposures seldom exceeded 5 to 10 minutes, two or three times a day, and that the quantities involved were very small. The average particle size was stated to be above 10 micron. The significance of the etiology of silicosis and the lack of hazard due to such minimal exposures were explained to Mr. Mosely's satisfaction. He will convey the information to his laboratory personnel.

LAM 026429

ABS-044363

For Information Only - Not For Publication

API TOXICOLOGICAL REVIEW

SILICA

Note: This review summarizes the best available information on the properties, characteristics, and toxicology of Silica. It offers suggestions and tentative recommendations pertaining to medical-treatments, medical examinations, and precautionary measures for workers who are exposed to Silica. It was prepared at the Harvard School of Public Health, Boston, Mass., under the direction of Professor Philip Drinker. Anyone desiring to submit additional information or proposed changes for consideration prior to re-issuance of this review is requested to send them to the American Petroleum Institute.

Prepared by: Marshall Clinton, M. D.

Accepted by: Medical Advisory Committee (April 1950).

Edited by: Chairman, Subcommittee on Toxicological Reviews (September 1951).

.....

NOTE: This review has not been submitted to the General Committee, API Division of Refining, nor to the Safety Committee of the Board of Directors for approval.

AMERICAN PETROLEUM INSTITUTE
Department of Technical Services
50 West 50th Street
New York 20, New York

LAM 026430

ABS-044364

For Information Only - Not For Publication

TOXICOLOGICAL REVIEW OF SILICA

I. Substance

Silica

Formula: SiO_2

Molecular Weight = 60.06

Synonym: Silicon dioxide

II. Properties and Characteristics (1,2)

Silica is the most widely occurring mineral; it is the chief component of ordinary sand. It is chemically most inert and inactive; minerologically it is widely encountered both in the free state and in combination with other substances as silicates. The physical characteristics of silica vary, depending on the form in which it is encountered. In general, the following characteristics apply:

Melting point = 1600°- 1670°C (2912 - 3038°F).

(M.P. of cristobalite estimated at 1710°C ± 10°. Pure silicon dioxide shows no definite melting point in the absence of catalysts, but softens gradually (as does glass) and becomes plastic at 1500°C. (2732°F).)

Boiling Point = 2230°C (4046°F)

Specific gravity = 2.66

Specific gravity (amorphous silica) = 1.9 - 2.3

Hardness (Mohr's scale) = 7 (Diamond being 10 and talc 1 by comparison. Amorphous silica has hardness of 5.5 - 6.5).

Indices of refraction (birefringent) W = 1.544 E = 1.553

Silica is encountered in both crystalline and amorphous forms. The former are more common and have more uses. The commonest crystalline forms are the three minerals, quartz, tridymite and cristobalite. The crystalline and vitreous forms of silica are acted on only slightly by any mineral acid except HF. They are less resistant to alkalis, but are virtually unaffected by exposure. Silica is soluble in water only to the extent of 0.016% at 25°C (77°F) and 0.0214% at 90°C (194°F).

Union Carbide trial exhibit UC-2064: Memo on Silica Hazard Consultation with R. B. Mosely, with API Toxicological... Published by AsbestosClaims.Law
ACL Doc 17086 · Page 4 of 11 asbestosclaims.law/library/17086/4

Fused silica is readily attacked, however, by H_3PO_4 and alkalis. "Silica, although chemically inactive at ordinary temperatures, acts as a powerful acid anhydride at high temperatures, combining with many bases and metallic oxides to form more or less fusible silicates. Thus, sodium carbonate, when fused with silica, will give off CO_2 and form sodium silicate. This is due to the fact the CO_2 and most of the other acid anhydrides volatilize at much lower temperatures than silica, hence will be removed from competition for the base by volatilization."

III. Toxicology

a. General considerations. Silica dust is toxic to man because it is capable of bringing about a specific proliferative pathological change in the lungs, called silicosis, which may progress sufficiently to interfere with normal respiratory function and lead to the development of incapacitating emphysema and dyspnea. This process generally takes many years. An understanding of the development of silicosis can best be arrived at by a consideration of the manner in which inhaled dust is ordinarily eliminated from the alveoli, and the manner in which the ordinary processes of dust elimination fail in the case of silica dust, leading to the development of silicosis.

In general, only dust particles below 5 or 10 micra in size reach the alveoli of the lungs in any quantity. The larger particles are deposited in the nasopharynx or on the mucosal lining of the tracheobronchial tree, from whence they are eliminated by means of ciliary action, which carries them upward toward the mouth, where they are either swallowed or expectorated. Dusts below 5 or 10 micra in size are less apt to be trapped on the mucous membranes, however, so the particles reach the alveoli in considerable quantity. Dusts which are freely soluble in body fluids, such as gypsum, magnesium silicate or limestone are dissolved and carried away in the lymph. These soluble dusts do not cause any bodily harm, unless the dissolved material happens to be capable of producing systemic poisoning, as in the case of many metallic dusts. Dusts which are insoluble in body fluids are removed from the alveoli in quite a different manner. They are engulfed by phagocytic cells, which then migrate through the lymph channels in the lungs to the regional lymph nodes at the lung hilus, where they deposit the material. Large quantities of inert dusts such as coal dust or silicon carbide are disposed of by the body in this manner without producing any ill health. Silica dust is also practically insoluble in body fluids, so is also removed from the alveoli by phagocytosis. However, as the phagocytic cells bearing free silica particles progress through the lymphatic channels

Union Carbide trial exhibit UC-2064: Memo on Silica Hazard Consultation with R. B. Mosely, with API Toxicological... Published by AsbestosClaims.Law
ACL Doc 17086 · Page 5 of 11 asbestosclaims.law/library/17086/5

the silica in some way poisons them, so they do not all reach the regional lymph nodes, but instead become fixed in the lymphatic channels in the lung, where they tend to collect. As this process continues, a specific proliferative tissue reaction takes place. These changes at first produce only a generalized linear or perivascular increase in pulmonary density, and are not accompanied by any symptoms. As the inhalation of silica dust continues, more dust collects in the lymphatics and the tissue reaction to the previously inhaled dust continues, and small nodules of silica and proliferating tissue ranging in size from 2 to 6 micra in diameter become thickly and diffusely scattered throughout the lung fields. At this state the x-ray picture is fairly characteristic, although other conditions may give a similar appearance. This process goes on gradually, and the nodules slowly increase in size. Eventually, however, they attain sufficient size to interfere with normal pulmonary function, leading to the dyspnea and emphysema characteristic of the more advanced stages of the disease.

It should be emphasized that silicosis except in rare instances takes many years to develop, and that the effects of repeated inhalations of silica dust are cumulative. Furthermore, the pathological changes in the lungs occurring as a result of the reaction of the body to inhaled silica do not become arrested when exposure is stopped, but continue to progress for months or even years after the last exposure. This fact is reflected in the latent period of about 5 years between the time new dust control measures are introduced and a significant drop in the incidence of silicosis is observed in a given stable community of workers (4).

The most serious and unfortunately one of the commonest complications of silicosis is tuberculosis. Whereas simple silicosis generally produces only moderate disability unless it is very far advanced and seldom if ever produces death, silicosis complicated by tuberculosis produces a disease syndrome characterized by coalescence of nodules, nodulation with massive fibrosis or nodulation with cavitation. The condition progresses rapidly to extreme disability and death. The worker with simple silicosis can generally look forward to a long if somewhat restricted life, whereas the silicotic who is unfortunate enough to develop superimposed tuberculosis will probably develop massive fibrosis or cavitation or both, and be dead within a few months (4,5).

b. Acute effects. As already noted, the inhalation of extreme concentrations of finely divided silica particles of 1 micron or less may lead to a diffuse fulminating lung fibrosis in a few months. Such cases are extremely rare (6).

Union Carbide trial exhibit UC-2064: Memo on Silica Hazard Consultation with R. B. Mosely, with API Toxicological... Published by AsbestosClaims.Law
ACL Doc 17086 · Page 6 of 11 asbestosclaims.law/library/17086/6

c. Chronic effects. The development of the usual chronic type of silicosis has already been described. The rate at which the disease develops may take from many months to many years, and the pulmonary fibrosis may proceed for years after exposure is terminated. The rate at which the disease develops depends primarily on the duration and intensity of exposure to dusts containing silica particles of 5 microns or less in size.

d. Safe limits. There is a considerable variation of opinion as to the safe limits of exposure to silica-containing dusts. The following formula for expressing maximum permissible concentrations of silica in the air breathed is generally accepted, however, for dusts of mixed composition:

Multiply the percentage of free silica by the total dust count in particles per cubic foot of air. If the result is under 5 million, the concentration may be considered permissible. If the result is over 5 million, the concentration may be considered too high. For example, 10 per cent free silica with an average total dust concentration of 30 million particles per cubic foot would give 0.10 times 30 million, which equals 3 million (good practice); 30 per cent with an average total dust concentration of 50 million, would equal 0.3 times 50, or 15 million (unsatisfactory). This formula is not applicable to any dust containing less than 5 per cent free silica (7).

New York State has adopted a system for expressing the maximum allowable dust concentration which considers the free silica content of the parent rock. Their industrial code specifies that there be no dissemination of injurious silica dust concentrations in the atmosphere, and defines an injurious dust concentration as a dust concentration in excess of one hundred million particles per cubic foot of air if the parent rock is composed of less than ten per cent free silica by weight and ten million particles per cubic foot of air if the parent rock is composed of more than ten per cent free silica by weight. (Counts based on lightfield impinger technique) (8).

IV. Treatment

There is no entirely satisfactory treatment for silicosis once the disease has developed. Uncomplicated silicosis is not especially disabling unless it becomes far advanced, however, so that no special therapy is necessary. The most important consideration in the management of minimal or moderately advanced silicosis is the avoidance of tuberculosis. Some benefit may result from the inhalation of aluminum or aluminum hydroxide gel, although its therapeutic

Union Carbide trial exhibit UC-2064: Memo on Silica Hazard Consultation with R. B. Mosely, with API Toxicological...

Published by AsbestosClaims.Law

ACL Doc 17086 · Page 7 of 11

asbestosclaims.law/library/17086/7

status is not entirely clear. The status of aluminum inhalation as a means of preventing the development of silicosis is much sounder than the use of aluminum to treat already existing silicosis (9). The use of prophylactic aluminum dusting is no substitute for careful engineering control of exposure, although it may be a useful adjunct when engineering control is incapable of completely eliminating exposure.

V. Examinations

a. Preplacement examinations. All workers who will be exposed to silica dust should receive a detailed preplacement examination which should include a complete medical and occupational history, careful physical examination and chest roentgenogram. Employment at any job which presents a potential silicosis risk should be denied any man with any evidence of tuberculosis, either active or arrested, except that a calcified Ghon complex should not disqualify. Similarly, workers who show any chronic disease of the heart or lungs or who have any anatomical abnormalities of the chest or respiratory tract which affects pulmonary ventilation should be eliminated (10).

b. Periodic examinations. Periodic chest roentgenograms should be taken on all workers exposed to a potential silicosis hazard. The frequency of these examinations should be determined by the severity of the exposure. Under most circumstances re-examination should be conducted at yearly intervals, even if exposure is relatively slight, in order to detect the development of tuberculosis. The discovery of tuberculosis at a periodic examination certainly requires immediate withdrawal from exposure and contact with other workers. The victim should be referred to a sanitarium for proper care. The discovery of simple silicosis in a worker exposed to silica dust does not necessarily indicate withdrawal from exposure, however, if the disease is not advanced and the degree of exposure is reasonably well controlled. A worker with mild silicosis can be better controlled if allowed to continue at his job. Transfer to other types of work may be satisfactory in the case of unskilled labor, but certainly is not desirable if it works an economic hardship on a skilled worker (10).

VI. Precautionary Measures:

Two types of safety precautions are generally employed in controlling exposure to silica dust -- proper engineering and design control and the use of protective equipment. The exact type of safety precautions and protective equipment can be determined only after a detailed study of existing conditions, and a delineation of all possible control measures is beyond the scope of this review. In general, it can be said that design and engineering control should provide:

Union Carbide trial exhibit UC-2064: Memo on Silica Hazard Consultation with R. B. Mosely, with API Toxicological... Published by AsbestosClaims.Law
ACL Doc 17086 · Page 8 of 11 asbestosclaims.law/library/17086/8

a. Segregation of dust-producing operations.
b. Adequate ventilation to collect dust at its source.
c. Good housekeeping
d. Elimination or reduction of dust production at the source by wetting down or use of wet processes.
e. Substitution of materials producing biologically inert dusts (11).

In addition, personal protective equipment should be provided which assures the wearer a safe atmosphere. If conditions are highly hazardous, such as in sand-blasting, then nothing short of a supplied-air helmet and protective suit will suffice.

Finally, it is necessary to determine whether the ventilation equipment is functioning adequately and personal protective equipment used in the manner intended, so that it provides adequate protection (12).

The degree of exposure to silica dust can best be determined by proper sampling, counting and analysis of the dust. At present, it is common practice to determine dustiness at the breathing level of exposed workers by collecting dust samples with a Greenburg-Smith or Bureau of Mines impinger (13). A variety of such impingers are now commercially available. The dust in an impinger sample can then be determined by microscopic count, gravimetrically, and by indirect photometric method. In evaluating dust samples, one should consider the composition and particle sizes as well as the total amount of dust. The particle size can best be determined by direct estimation in the counting chamber or by elutriation, while the composition can best be determined by petrographic analysis or x-ray diffraction supplemented, if need be, by chemical determinations (13). A detailed discussion of dust analysis is beyond the scope of this review.

VII. Bibliography

1. Mellor, J. W. A Comprehensive Treatise on Inorganic Theoretical Chemistry 6: 245. Longmans, Green and Co., New York, 1925.

2. Jacobs, M. B. The Analytical Chemistry of Industrial Poisons, Hazards and Solvents. p. 135, Interscience Publishers, New York, 1944.

3. Harrington, D. and Davenport, S. J. Review of literature on effects of breathing dusts with special reference to silicosis. Part I. United States Bureau of Mines J. C. 6835, March, 1935.

4. Lanza, A. J. ed. Silicosis and Asbestosis. Oxford University Press, New York, 1938.

Union Carbide trial exhibit UC-2064: Memo on Silica Hazard Consultation with R. B. Mosely, with API Toxicological... Published by AsbestosClaims.Law
ACL Doc 17086 · Page 9 of 11 asbestosclaims.law/library/17086/9

5. Riddell, A. R. The clinical aspects of simple silicosis and silicosis with tuberculosis. Am. Rev. of Tuberculosis 29: 36, 1934.

6. Hayhurst, E. A. Silicosis. Ohio State University Engineering Experiment Station News. Vol. 8, No. 1. February, 1936.

7. United States Department of Labor, National Silicosis Conference Report on Medical Control: Final report of the committee on the prevention of silicosis through medical control. Bull. No. 21, Part 1, 1938.

8. State of New York, Department of Labor. Rules relating to the control of silica dust on stone crushing operations. Industrial Code Rule No. 34, July, 1942.

9. Gardner, L. U., Dworski, M. and Delahant, A. B. Aluminum therapy in silicosis - an experimental study. J. Ind. Hyg. and Toxicol. Vol. 26: 211, 1944.

10. Sander, O. A. The silicosis problem; some medical social phases. Wisconsin Med. J. April, 1936.

11. United States Department of Labor, National Silicosis Conference, Report on Engineering Control. Final report of the committee on the prevention of silicosis through engineering control. Bull. No. 21, Part 2, 1938.

12. United States Public Health Service, the impinger dust sampling apparatus as used by the United States Public Health Service, Public Health Reports Vol. 37, No. 2, 1932.

13. Salazar, A. and Silverman, L. A new method for the determination of free silica in industrial dusts. J. Ind. Hyg. and Toxicol. Vol. 25, p. 139, 1943.

5/19/52

Union Carbide trial exhibit UC-2064: Memo on Silica Hazard Consultation with R. B. Mosely, with API Toxicologica... Published by AsbestosClaims.Law

ACL Doc 17086 · Page 10 of 11 asbestosclaims.law/library/17086/10

Published by AsbestosClaims.Law
asbestosclaims.law/library/17086/11