criteria for a recommended standard . . . .

OCCUPATIONAL EXPOSURE TO CRYSTALLINE SILICA

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PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health and safety of workers exposed to an ever-increasing number of potential hazards at their workplace. The National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices, to provide relevant data from which valid criteria for effective standards can be derived. Recommended standards for occupational exposure, which are the result of this work, are based on the health effects of exposure. The Secretary of Labor will weigh these recommendations along with other considerations such as feasibility and means of implementation in developing regulatory standards.

It is intended to present successive reports as research and epidemiologic studies are completed and sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this report on crystalline silica by members of my staff and the valuable constructive comments by the Review Consultants on Crystalline Silica, by the ad hoc committees of the American Industrial Hygiene Association and the Society for Occupational and Environmental Health, by Robert B. O'Connor, M.D., NIOSH consultant in occupational medicine, and by Edwin C. Hyatt, NIOSH consultant on respiratory protection. The NIOSH recommendations for standards are not necessarily a consensus of all the consultants and

professional societies that reviewed this criteria document on crystalline silica. Lists of the NIOSH Review Committee members and of the Review Consultants appear on the following pages.

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CRITERIA DOCUMENT: RECOMMENDATIONS FOR AN OCCUPATIONAL EXPOSURE STANDARD FOR CRYSTALLINE SILICA

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I. RECOMMENDATIONS FOR A CRYSTALLINE SILICA STANDARD

The National Institute for Occupational Safety and Health (NIOSH) recommends that employee exposure to crystalline silica in the workplace be controlled by adherence to the following sections. The standard is designed to protect the health and safety of workers for up to a 10-hour workday, 40-hour workweek, over a working lifetime. Compliance with the standard should prevent adverse effects of crystalline silica on the health and safety of workers. The standard is measurable by techniques that are valid, reproducible, and available to industry and government agencies and are attainable with existing technology. The criteria and the standard recommended in this document will be subject to review and revision as necessary.

Crystalline silica, hereafter referred to in this document as free silica, is defined as silicon dioxide (SiO_2). "Crystalline" refers to the orientation of SiO_2 molecules in a fixed pattern as opposed to a nonperiodic, random molecular arrangement defined as amorphous. The three most common crystalline forms of free silica encountered in industry are quartz, tridymite, and cristobalite. Micro- and crypto-crystalline varieties of free silica, also included in the recommended standard, are composed of minute grains of free silica cemented together with amorphous silica and include tripoli, flint, chalcedony, agate, onyx, and silica flour. Other forms of free silica which, upon analysis, are found to have a crystalline structure as part of their composition are also subject to the recommended standard.

"Exposure to free silica" means exposure of the worker to an airborne concentration of free silica greater than half of the recommended environmental level in the workplace. Worker exposure at lower environmental concentrations will not require adherence to the following sections.

Section 1 - Environmental (Workplace Air)

(a) Concentration

Occupational exposure shall be controlled so that no worker is exposed to a time-weighted average (TWA) concentration of free silica greater than 50 micrograms per cubic meter of air (50 μg/cu m; 0.050 mg/cu m) as determined by a full-shift sample for up to a 10-hour workday, 40-hour workweek.

(b) Sampling, Calibration, and Analysis

Exposure to free silica shall be determined by a personal (breathing zone) sample.

Procedures for sampling, calibration of equipment, and analysis of environmental samples shall be as provided in Appendices I and II, or by methods shown to be equivalent in sensitivity, accuracy, and precision to the method specified.

Section 2 - Medical

(a) Medical examinations shall be made available to all workers subject to "exposure to free silica" prior to employee placement and at

least once each 3 years thereafter. Examinations shall include as a minimum:

(1) A medical and occupational history to elicit data on worker exposure to free silica and signs and symptoms of respiratory disease.

(2) A chest roentgenogram (posteroanterior 14" by 17" or 14" by 14") classified according to the 1971 ILO International Classification of Radiographs of Pneumoconioses. [ILO U/C International Classification of Radiographs of Pneumoconioses 1971, Occupational Safety and Health Series 22 (rev). Geneva, International Labor Office, 1972]

(3) Pulmonary function tests including forced vital capacity (FVC) and forced expiratory volume at one second (FEV 1) to provide a baseline for evaluation of pulmonary function and to help determine the advisability of the workers using negative- or positive-pressure respirators. It should be noted that pulmonary function tests may vary significantly in various ethnic groups. For example, the average healthy black male may have an approximately 15% lower FVC than a healthy caucasian male of the same body build.

(4) Body weight.

(5) Height.

(6) Age.

(7) Initial medical examinations for presently employed workers shall be offered within 6 months of the promulgation of a standard incorporating these recommendations.

(b) Medical Management

An employee with or without roentgenographic evidence of silicosis who has respiratory distress and/or pulmonary functional impairment should be fully evaluated by a physician qualified to advise the employee whether he should continue working in a dusty trade.

(c) These records shall be available to the medical representatives of the Secretary of Health, Education, and Welfare, of the Secretary of Labor, of the employee or former employee and of the employer.

(d) Medical records shall be maintained for at least 30 years following the employee's termination of employment.

Section 3 - Labeling (Posting)

(a) The following warning shall be posted to be readily visible at or near entrances or accessways to work areas where there is potential exposure to free silica.

WARNING!

FREE SILICA WORK AREA

Unauthorized Persons Keep Out

(b) The following warning shall be posted in readily visible locations in any work area where there is potential exposure to free silica.

WARNING!

FREE SILICA WORK AREA

Avoid Breathing Dust

May Cause Delayed Lung Injury (Silicosis)

The posting required under sections 3(a) and 3(b) shall be printed both in English and in the predominant language of non-English-speaking workers, unless they are otherwise trained and informed of the hazardous areas. Illiterate workers shall receive such training.

(c) The following warning label, in addition to or in combination with labels required by other statutes, regulations, or ordinances, shall be affixed to all new materials, mixtures, and other products containing more than 5% free silica, or to their containers.

WARNING!

CONTAINS FREE SILICA

DO NOT BREATHE DUST

May Cause Delayed Lung Injury (Silicosis)

Section 4 - Personal Protective Equipment and Work Clothing

Engineering controls shall be used to maintain free silica dust exposures below the prescribed limit. Subsection (a) shall apply whenever a variance from the standard recommended in Section 1 is granted under provisions of the Occupational Safety and Health Act, or in the interim period during the application for a variance. When the limits of exposure

to free silica prescribed in paragraph (a) of Section 1 cannot be met by limiting the concentration of free silica in the work environment, an employer must utilize, as provided in subsection (a) of this section, a program of respiratory protection to effect the required protection of every worker exposed.

(a) Respiratory Protection

Appropriate respirators, as prescribed in Table I-1, shall be provided and used when a variance has been granted to allow respirators as a means of control of exposure to routine operations and while the application is pending. Administrative controls may also be used to reduce exposure. Respirators shall also be provided and used for nonroutine operations (occasional brief exposures above the environmental standard and for emergencies); however, for these instances a variance is not required but the requirements set forth below continue to apply. Appropriate respirators as described in Table I-1 shall only be used pursuant to the following requirements:

(1) For the purpose of determining the type of respirator to be used, the employer shall measure the atmospheric concentration of free silica in the workplace when the initial application for variance is made and thereafter whenever process, worksite, climate, or control changes occur which are likely to affect the free silica concentration. This requirement shall not apply when only atmosphere-supplying positive pressure respirators are used. The employer shall ensure that no worker is exposed to free silica in excess of the standard because of improper respirator selection, fit, use, or maintenance.

(2) Employees experiencing breathing difficulty while using respirators shall be evaluated by a physician to determine the ability of the worker to wear a respirator.

(3) A respiratory protective program meeting the requirements of Section 1910.134 of the Occupational Safety and Health Standards shall be established and enforced by the employer. [29 CFR 1910.134 published in the Federal Register, vol 39, page 23671, dated June 27, 1974, as amended]

(4) The employer shall provide respirators in accordance with Table I-1 and shall ensure that the employee uses the appropriate respirator.

(5) Respiratory protective devices in Table I-1 shall be those approved either under 30 CFR 11, published March 25, 1972, or under the following regulations:

(A) Filter-type dust, fume, and mist respirators--30 CFR 14 (Bureau of Mines Schedule 21B)

(B) Supplied air respirator--30 CFR 12 (Bureau of Mines Schedule 19B)

(6) A respirator specified for use in higher concentrations of free silica may be used in atmospheres of lower concentrations.

(7) Employees shall be given instruction on the use of respirators assigned to them, on cleaning respirators, and on testing for leakage.

TABLE I-1

REQUIREMENTS FOR RESPIRATOR USAGE AT CONCENTRATIONS ABOVE THE STANDARD

Concentrations of Free Silica in Multiples of the Standard	Respirator Type*
Less than or equal to 5x	Single use (valveless type) dust respirator.
Less than or equal to 10X	Quarter or half mask respirator with replaceable dust filter or single use (with valve) dust respirator.
	Type C, demand type (negative pressure), with quarter or half mask facepiece.
Less than or equal to 100X	Full facepiece respirator with replaceable dust filter.
	Type C, supplied air respirator, demand type (negative pressure), with full facepiece.
Less than or equal to 200X	Powered air-purifying (positive pressure) respirator, with replaceable applicable filter.**
Greater than 200X	Type C, supplied air respirator, continuous flow type (positive pressure), with full facepiece, hood, or helmet.

*Where a variance has been obtained for abrasive blasting with silica sand, use only Type C continuous flow, supplied air respirator with hood or helmet.

**An alternative is to select the standard high efficiency filter which must be at least 99.97% efficient against 0.3 μm dioctyl phthalate (DOP).

(b) Work Clothing

Where exposure to free silica is above the recommended environmental limit, work clothing shall be vacuumed before removal. Clothes shall not be cleaned by blowing or shaking.

Section 5 - Informing Employees of Hazards from Free Silica

(a) Each employee exposed to free silica shall be apprised at the beginning of his employment or assignment to a free silica exposure area of the hazards, relevant symptoms, appropriate emergency procedures, and proper conditions and precautions for safe use or exposure. The employee shall be instructed as to availability of such information including that prescribed in (b) below. Such information shall be kept on file and shall be accessible to the worker at each place of employment where free silica is involved in unit processes and operations. Workers shall also be advised of the increased risk of impaired health due to the combination of smoking and free silica dust exposure.

(b) Information, to the extent applicable to free silica, as specified in Appendix III shall be recorded on US Department of Labor Form OSHA-20, "Material Safety Data Sheet" (see Appendix III) or on a similar form approved by the Occupational Safety and Health Administration, US Department of Labor.

Section 6 - Work Practices and Control Procedures

(a) Substitution

(1) Wherever a hazard of silicosis can be eliminated by a reasonable substitution of other less toxic materials for free silica, the substitution shall be made unless the silica sand has been so processed before use to make it nonrespirable such as by washing to remove fine particles. Examples of such substitution are the use of alumina instead of

flint for china placing in potteries, and the substitution of a quartz-free grit in abrasive blasting.

(2) Uncontrolled abrasive blasting with silica sand is such a severe silicosis hazard that special attention must be given to this problem. Silica sand, or other materials containing more than 1% free silica, should be prohibited as an abrasive substance in abrasive blasting cleaning operations.

(b) Dust suppression

Moisture shall be added where such addition can substantially reduce the exposure to airborne respirable free silica dust.

(c) Ventilation

Where a local exhaust ventilation and collection system is used, it shall be designed and maintained to prevent the accumulation or recirculation of free silica dust into the workplace. The total system shall be inspected periodically for efficiency of operation. In addition, necessary measures shall be taken to ensure that discharge outdoors will not produce a health hazard to humans, animals, or plants.

(d) General Housekeeping

(1) Cleaning by blowing with compressed air or dry sweeping shall be avoided and dustless methods of cleaning such as vacuuming or washing down with water shall be substituted.

(2) Emphasis shall be placed upon cleanup of spills, preventive maintenance and repair of equipment, proper storage of materials, and collection of dusts containing free silica. Sanitation shall meet the requirements of 29 CFR 1910.141 as amended.

Section 7 - Monitoring and Recordkeeping Requirements

Work environments where it has been determined, on the basis of a professional industrial hygiene survey or by the judgment of a compliance officer, that the workers' exposure does not exceed half of the standard shall not be considered to have exposure to free silica. Records of these surveys, including the basis for concluding that air levels are at or below half of the standard shall be maintained. Surveys shall be repeated when any process change indicates a need for reevaluation or at the discretion of the compliance officer. Requirements set forth below apply to areas in which there is "exposure to free silica."

Employers shall maintain records of the workers' exposure to free silica based upon the following sampling and recording schedules:

(a) In all monitoring, samples representative of the exposure in the breathing zone of employees shall be collected. An adequate number of samples shall be collected to permit construction of a full-shift exposure for every operation or process. The minimum number of time-weighted average determinations for an operation or process shall be based on the number of workers exposed as provided in Table I-2 or as otherwise indicated by a professional industrial survey.

(b) The first work environment (breathing zone) sampling shall be completed within 6 months of the promulgation of a standard incorporating these recommendations.

(c) Work environment (breathing zone) samples shall be taken within 30 days after installation of a new process or process changes.

TABLE I-2

SAMPLING SCHEDULE

Number of Employees Exposed	Number of Time-weighted Average Determinations
1-20	50% of the total number of workers
21-100	10 plus 25% of the excess over 20 workers
over 100	30 plus 5% of the excess over 100 workers

(d) Samples shall be collected and analyzed at least every 6 months in accordance with Appendices I and II for the evaluation of the workers' exposure with respect to the recommended standard.

(e) When monitoring of the workers' exposure indicates a free silica concentration in excess of the recommended standard, suitable controls shall be initiated to reduce the exposure level to or below the recommended standard. In such cases monitoring shall continue at 30-day intervals until 2 consecutive surveys indicate the recommended standard is no longer exceeded. Periodic review and evaluation of environmental and medical data shall be performed to determine the effectiveness of control measures.

(f) Records shall be maintained of medical examinations and all sampling schedules to include the sampling and analytical methods, type of personal protection devices, if any, in use at the time of sampling and the determined free silica dust concentration. Records shall be maintained for at least 30 years following termination of workers' employment. Each employee shall be able to obtain information on his exposure.

II. INTRODUCTION

This report presents the criteria and the recommended standard based thereon which were prepared to meet the need for preventing occupational diseases arising from exposure to crystalline variants of free silica. The document fulfills the responsibility of the Secretary of Health, Education, and Welfare, under Section 20 (a)(3) of the Occupational Safety and Health Act of 1970 to "... develop criteria dealing with toxic materials and harmful physical agents and substances which will describe... exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience."

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultation with others, formalized a system for the development of criteria upon which standards can be established to protect the health of workers from exposure to hazardous chemical and physical agents. It should be pointed out that any recommended criteria for a standard should enable management and labor to develop better engineering controls resulting in more healthful work practices and should not be accepted as a final goal.

These recommendations for a standard for free silica are part of a continuing series of criteria being developed by NIOSH. The recommended standard applies to the processing, manufacture, and use of free silica as applicable under the Occupational Safety and Health Act of 1970.

These recommendations are not developed for the population-at-large and any extrapolation beyond general occupational exposure is not warranted. They are intended to assure that the standard based thereon will (1) protect against development of acute and chronic fibrogenic disease (silicosis) or functional incapacities arising from inhalation of free silica; (2) be measurable by techniques that are valid, reproducible, and available to industry and governmental agencies; and (3) be attainable with existing technology.

Criteria presented in this document do not pertain to amorphous, noncrystalline forms of silica.

III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure/Hazard

The chief source of free silica is quartz, a mineral found in most classes of rock.

Because of their composition, quartz and the other silicon dioxide (SiO_2) minerals have always been considered with the oxides, but physical properties and crystal structures of the oxides are more in accord with those of the silicate group. Basic differences are defined in Pough's [1] and Hurlbut's [2] manuals of mineralogy.

Of all the minerals, quartz is most nearly a pure (46.7% silicon and 53.3% oxygen) chemical compound and its physical properties are constant. [1] Spectrographic analyses show, however, that even the most "perfect" quartz crystals contain traces of lithium, sodium, potassium, aluminum, ferric iron, divalent manganese, or titanium. [2] Quartz can be categorized into 2 groups on the basis of appearance: crystalline and microcrystalline. Usually, the 2 classes cannot be differentiated without the aid of a microscope. Quartz may be colorless, or it may be white, smoky, rose, violet, brown, or almost any hue, depending on the impurities. Luster and fracturability are typical, and hardness is greater than that of most minerals. [2]

Quartz can occur almost anywhere and is an important constituent of those igneous rocks having an excess of silica, such as granite and pegmatite. It is extremely resistant to both mechanical and chemical alteration; thus the breakdown of igneous rock containing it yields quartz

grains that may accumulate and form the sedimentary rock, sandstone. In rocks quartz is associated chiefly with feldspar and muscovite. Quartz occurs in large amounts as sand in stream beds and seashores, and as a constituent of soils. It is an important industrial material from many standpoints. Silica sand is used in the manufacture of glass and silica brick, in mortar, and as an abrasive. In powdered form (silica flour) it is used in paints, porcelain, scouring soaps, and as a wood filler. The clear rock crystal is of great value for electronic equipment. The colored varieties are valued as gems or are used as ornamental material (amethyst, onyx, rose quartz, agate). [2]

Two minerals chemically identical to quartz are tridymite and cristobalite. [1] They differ from quartz in crystalline form. Tridymite forms from quartz above 870 C into white or colorless platy orthorhombic crystals while cristobalite forms above 1470 C into white cubic-system crystals. The distinguishing crystalline structures of the two minerals can be recognized by microscopic examination, X-ray diffraction, and infrared spectrophotometry. Tridymite and cristobalite usually occur together as abundant high-temperature silicate minerals in the volcanic rock of California, Colorado, and Mexico. [1,2]

In addition to being natural mineral constituents of rock and soil encountered in the mining industry, cristobalite and tridymite are formed when quartz or amorphous (noncrystalline) silica are heated, as in the calcining of diatomaceous earth or in the silica brick industry. These minerals are used extensively as filtering and insulating media, and as siliceous refractory materials for furnace linings and silica bricks.

In the United States, occupational exposures to free silica, mostly quartz, occur in mining, manufacturing, construction, and agriculture. Traditional industries with free silica exposure and their 1970 employment as recorded in the US Bureau of Census Statistical Abstracts for 1971 [3] are given in Table III-1.

TABLE III-1

EMPLOYMENT IN INDUSTRIES HAVING POTENTIAL EXPOSURE TO FREE SILICA, 1970

Metal mining	76,000
Coal mining	125,000
Nonmetallic minerals, except fuels	95,000
Stone, clay, and glass products	507,000
Iron and steel foundries	188,000
Nonferrous foundries	69,000
Total	1,060,000

The National Institute for Occupational Safety and Health estimates that 1,200,000 workers are exposed to free silica.

By no means are all of the listed production workers exposed to free silica. The proportion varies from most of the metal miners to perhaps 20% of the stone, clay, and glass product workers. The total omits substantial numbers of the 2.5 million people employed in agriculture, 0.6 million employed in production of chemicals and allied products, and the 0.6 million workers in heavy construction, many of whom may receive exposure to free silica.

Studies by Trasko [4-6] and Doyle et al [7] focus on the socioeconomic and disease prevalence aspects of silicosis. Available statistics from Trasko's 1964 report [6] are an example of the magnitude of these aspects. In the period from 1950-64, 27,000 claims for pneumoconioses, amounting to approximately 132 million dollars, were settled by workmen's compensation agencies in only 18 states. The report also states that the accumulated data undoubtedly underestimate the true situation for such compensation.

Historical Reports

The pneumoconioses have probably existed since man began to dig into the earths' crust. Silicosis, of all the pneumoconioses, is identified as claiming the largest number of victims, either alone or with tuberculosis, with which it is frequently associated. [8] Silicosis, a nodular pulmonary fibrosis caused by inhalation and pulmonary deposition of particles of free silica [9] has also been known as dust consumption, ganister disease, grinders' asthma, grinders' consumption, grinders' rot, grit consumption, masons' disease, miners' asthma, miners' phthisis, potters' rot, rock tuberculosis, stonehewers' phthisis, and stonemasons' disease. [8]

Among the many historical reviews of silicosis are those of Hunter [8] and Zaidi. [10] Hunter stated that "Probability suggests that the starting-point of human progress, prehistoric man's manufacturing of flint implements, was associated with at least one form of silicosis. Hippocrates speaks of the metal digger as a man who breathes with

difficulty, and Pliny mentions the use of respirators to avoid dust inhalation.

"The first account of the pathology of what is now called silicosis came in 1672 from van Diemerbroeck who described how several stone cutters died of asthma. At necropsy he found that to cut their lungs was like cutting a mass of sand. Ramazzini (1713) describes how stone cutters breathe in small splinters and turn asthmatic and consumptive.

"The starting-point of the study of silicosis in modern times was a paper by Johnstone in 1796, calling attention to the high mortality among needle pointers at Redditch, England. Thackrah (1831) knew of the danger from sandstone dust in mining and of the harmlessness of limestone dust. He had noted that bricklayers and limeworkers were long-lived and that sandstone masons usually died before they reached the age of 40. In 1843, Calvert Holland described the conditions of work amongst Sheffield grinders. He discovered on examination that among 97 men, about 30 were suffering in varying degree from grinders' asthma. T.B. Peacock and E.H. Greenhow established between 1860 and 1866 the existence of miners' disease as an entity and distinguished it clinically from pulmonary tuberculosis. In the Transactions of the Pathological Society of London (1860-66) are to be found excellent clinical and pathological descriptions of the disease which was later to be called silicosis by Visconti in 1870.

"The work of J.S. Haldane in tin miners (1904), Hay in granite workers (1909) and Wheatley (1911) in sandstone-quarry workers, and the report of a Commission on Miners' Phthisis in South Africa (1912) opened

the twentieth century, the early part of which has been notable for a gradually extending and now world-wide interest in silicosis."

Despite the vast body of facts from recent research concerning the pathogenesis of silicosis, Zaidi [10] concluded that "there are still many hiatuses in our knowledge." The manner in which the human lesion develops is still not precisely known; the relationship of the crystal structure of silica to silicosis needs more investigation; the size of the dust particles with reference to pathogenicity, and the correlation between retained dust load and the degree of tissue reaction also require further explanation.

It seems reasonable to presume that modern-day industry (including mines and foundries) with increased mechanization and speed of production has created problems of increased dustiness and greater incidences of dust diseases unless properly controlled.

Effects on Humans

Of the numerous theories that have been proposed to explain the mechanism by which inhaled free silica particles cause tissue damage, many are based on one or more of the four main characteristics of these particles: their physical shape, their solubility, their cytotoxicity to macrophages, or their crystalline structure. [10] At this time silicotic fibrosis cannot be fully explained by any single theory.

The clinical signs of silicosis are not unique. Symptoms may be progressive with continued exposure to quantities of dust containing free silica, with advancing age, and with continued smoking habits. Symptoms

may also be exacerbated by pulmonary infections and cardiac decompensation. [11]

Pulmonary symptomatology usually begins insidiously. Symptoms include presence of cough, dyspnea, wheezes, and repeated nonspecific chest illnesses. Impairment of pulmonary function may be progressive. In individual cases there may be little or no decrement when simple discrete nodular silicosis is present, but when nodulations become larger or when conglomeration occurs, recognizable cardiopulmonary impairment tends to occur. [11]

As is true of any of the pneumoconioses, the various stages of progression of the silicotic lesions are related to the degree of exposure to free silica (exposure concentration), the duration of exposure, and the duration of time which the retained dust is permitted to react with the lung tissue. Because there are very few symptoms, very little is known about the early lesions resulting from moderately high exposures to free silica. Occasionally, exposures to very high concentrations occur in short periods of time in occupations such as sandblasters and tunnel workers. In these cases of "acute or rapidly-developing" silicosis there can be severe respiratory symptoms and death. Roentgenographic examination of the lungs usually does not reveal typical silicotic nodulation. [12-15]

Other factors, chemical or biological, can influence the rate of reaction of the free silica with the tissue and can create problems in diagnosis. One of the most frequent complications in the past was the occurrence of tuberculosis with silicosis, in which case the disease was called silicotuberculosis or tuberculosilicosis. [16-18]

The most common criteria used in diagnosing silicosis (and other occupational respiratory diseases) are the results of pulmonary function tests, chest roentgenograms, and occupational exposure histories. [13,19] Pulmonary function tests are objective indicators of respiratory dysfunction. However, there is no pulmonary function test specific for silicosis. The chest roentgenogram is a moderately good indicator of the degree of tissue reaction to exposure to free silica. Unfortunately, several other disease entities can produce the same roentgenographic pattern as free silica. Hamlin [19] has found more than 20 conditions or diseases that cannot be differentiated from silicosis by X-ray alone. In some cases, as with dust particles of iron, tin, and barium, nodular densities are produced by aggregates of particles alone without any fibrosis. A history of exposure to free silica is necessary before the other two criteria (pulmonary function and chest roentgenogram) can be utilized in making a diagnosis of silicosis. Apparently the only single method at the present time that can unequivocally demonstrate the unique pulmonary effects of exposure to free silica is a lung tissue examination.

The generally accepted diagnostic lesion of silicosis is a firm nodule composed of concentrically arranged bundles of collagen. [9,13] These nodules usually measure between 1 and 10 mm in diameter and appear in lymphatics around blood vessels and beneath the pleura in the lungs. The presence of 1 or more of these characteristic nodules in a lung is indicative of an exposure to free silica. These nodules may also occur in the mediastinal lymph nodes. Fusion of the nodular lesions in the

silicotic lung is frequently referred to as progressive massive fibrosis (PMF).

According to Gloyne [20] and Pendergrass, [13] the severity of the exposure and presumably the severity of the disease can be determined by the numbers and sizes of silicotic nodules in whole lungs. Frequently the lumina of the blood vessels in the silicotic nodules become narrow and obliterated by fibrous tissue. Another common finding around the nodule is perifocal emphysema, ie, destruction of alveolar walls with a concomitant increase in the sizes of alveolar sacs and ducts. These pathologic features decrease the blood flow and ventilation in the lung.

In mixed dust exposures, eg, free silica with iron ore or coal dust, a more varied pathologic appearance is typical. [10,11] The majority of pulmonary lesions caused by mixed dusts are not the classical silicotic nodules. The principal lesion in coalworker's pneumoconiosis (CWP) is the coal dust macule. [21] This is a dense aggregate of coal dust (and birefringent crystals) around respiratory bronchioles and alveolar ducts. Varying amounts of collagen are present in this macule, but the bundles of collagen are arranged haphazardly and not concentrically as in the silicotic nodule. Whether or not the free silica present in the dust is primarily responsible for producing the excess collagen or whether it is some other ingredient is not clear. However, Naeye and Dellinger [22] found that the collagen content of coal dust macules increased with increasing concentrations of free silica as seen by polarized light microscopy. This use of polarizing light microscopy can provide the pathologist with a means for more accurate diagnosis of the early stages of

developing silicosis and of the "rapidly-developing" variety. Naeye and Dellinger [22] also found a few small silicotic nodules in the lungs of 44% of 175 Appalachian bituminous coal miners. It would seem logical, based upon this evidence, to suggest that free silica inhaled with coal dust contributes to the production of coal worker's pneumoconiosis.

Roentgenological studies are of primary importance in the diagnosis of the pneumoconioses. However, as stated by the Council on Occupational Health, [11] "... there is a great gap between demonstrable microscopic evidence of lung changes and clearly defined pneumoconiosis as seen in the roentgenogram". Routinely, a posteroanterior (PA) view of the chest is taken at full inspiration and with a technique designed to demonstrate parenchymal lesions of the lung as classified by the ILO U/C classification. [23] Despite the limitations of the roentgenographic examinations, they are routinely used, alone or in combination with pulmonary function tests, for diagnosing silicosis. Thus, roentgenographs can provide information as to the current status of the disease in addition to providing a reference for evaluating both retrospective and prospective progression of silicotic lesions.

Epidemiologic Studies

Despite an awakening of interest in the early 1900's in the health problems of American dusty trades, epidemiologic studies of those industries accountable for the most prominent occupational respiratory disease--the pneumoconioses, and more particularly, silicosis--have been limited. This is especially true of the last decade despite the

disability, compensation costs, and economic loss resulting from earlier uncontrolled worker exposures to free silica dusts.

It has long been recognized that workers engaged in hard-rock mining of nonferrous metal ores are subject to diseases of the lungs. Studies in the early 1900's of lead-zinc miners in the tri-state area of Missouri, Kansas, and Oklahoma [16,18] and of copper mines in Butte, Montana, [24] revealed serious problems of pulmonary disease.

The first major silicosis study of the hard-rock mining industry was conducted by the Public Health Service (PHS) and the Bureau of Mines in 1913-15. [16,18] The study showed that of the 720 miners examined, 433 or 60.4% were reported to be suffering from diseases of the lungs directly due to the mine rock-dust exposures. Chemical analysis of mine drill cuttings showed a content of siliceous residue ranging from 71-95%. Mine-air dust samples collected with a Draeger liter bag-granulated sugar filter apparatus [18] were as high as 2,200 mg/cu m of air with an average concentration of 30-50 mg/cu m. [16] In the light of present day technology for dust sampling (impinger and personal samplers) and analysis (X-ray diffraction), these concentrations cannot be readily related to those reported in later studies of the mines.

An investigation by Harrington and Lanza in 1916-19 [24] of health conditions of copper miners revealed a 42.4% incidence of dust injury to the lungs of 1,018 miners. Of the 432 cases of what was defined as miners' consumption, 194 cases (44.9%) were in the early stages of the disease, 128 cases (29.6%) were moderately advanced, and 110 cases (25.5%) far advanced. Mine air dust concentrations were reported only by a statement that "mines

in general were more dusty than the dangerously dusty mines of the Joplin district, Missouri." [24] The dust in the Joplin mines was considered more toxic since it contained approximately 90% silica, practically all of which was free as contrasted with the dust of the Butte Copper mines which contained only 75% silica of which 50-60% was free silica.

Data from a representative group of metal mines studied by Dreessen et al [25] in 1939 showed 66 cases (9.1%) of first- or second-stage silicosis among a group of 727 mine workers studied. An additional 42 cases were diagnosed as borderline silicosis. Dust exposure concentrations for all mine occupations ranged from 2-37 million particles per cubic foot (mppcf) of air. Eighty-six percent of the workers were exposed to dust concentrations between 6 and 30 mppcf. Free silica content of the mine dusts ranged from 1-99%. The median dust particle size determined from impinger samples was 0.94 μm. No cases of silicosis were observed in workers whose exposures did not exceed an average of 18 mppcf and whose employment did not exceed 10 years. The severity of pulmonary fibrosis among cases of silicosis increased greatly with increasing length of employment.

The silicosis problem among these metal mine workers was found to be most severe for those working principally at the face of the mines where a combination of high dust concentrations (10-23 mppcf) and a free silica content of 20-40% were encountered. This combination resulted in an incidence of silicosis in a fourth of the workers who had been exposed for more than 6 years.

Flinn et al [26] reported on an extensive study of silicosis in metal mines in the United States conducted by the US Public Health Service and the Bureau of Mines between 1958 and 1961. The environmental study included 67 underground mines employing 20,500 persons. The medical study included 14,076 employees from 50 of the mines. Dust concentrations as determined from 14,480 impinger samples ranged from a reported weighted average of 0 to more than 50 mppcf. Quartz content of settled and airborne dust samples ranged from approximately 2-95%.

Medical examinations included medical history and symptoms, occupational histories, chest roentgenograms, and pulmonary function tests. Of the 14,076 metal miners in the study group, chest roentgenograms of 476 (3.4%) were classified as consistent with a diagnosis of silicosis. The prevalence rate varied greatly, ranging from zero in 7 mines to 12.9% in one mine. This rate increased rapidly with increasing years of work within the metal mining industry. No cases were observed among workers with less than 5 years of dust exposure and only a 0.2% incidence occurred among workers with 5-9 years exposure. After 10 years of exposure, the incidence rose rapidly in 10-year increments with an average of 16.6% in those workers with 30 or more years of exposure.

Renes et al [27] reported on a medical study of silicosis made by the US Public Health Service and the Illinois Department of Public Health in 1948-49 in 18 ferrous foundries. A 9.2% incidence of pulmonary fibrosis as determined by chest roentgenograms and historical and clinical data was found in 178 of the 1,937 foundrymen examined. Among foundry workers with

20 or more years in their occupation a 25.8% incidence of pulmonary fibrosis was reported.

Environmental studies found that 90% or more of the airborne dust in the 18 foundry atmospheres was 3 μm or less in size. The amount of free silica in the dust varied with the operation and ranged from 13-29%. Free silica content in settled dust was higher, averaging 30% throughout the foundries. Mean dust levels at molding, pouring, and coremaking operations were under 3 mppcf except for sand-slinger molding where a concentration of about 19 mppcf was determined. Mechanical shakeout operations showed mean dust levels ranging from 10-75 mppcf. Manual shakeout and sand conditioning produced mean dust levels under 7 mppcf. Despite some of these relatively high dust concentrations, the operational dust levels at the various foundry activities were much lower than those reported in an earlier investigation of foundries by McConnell and Fehnel. [28] Renes and his co-workers [27] found that the frequency distribution of dust concentrations from all the various foundry activities they investigated showed that 82% of the samples were below 6.9 mppcf. They suggested that "there is good reason to believe that dust conditions in general in the foundry industry have improved in the past 10-20 years. ...thus, it is likely that the pulmonary fibrosis observed was due in great part to higher dust concentrations which probably existed 10, 15, or more years previously in foundries."

Flinn et al [29] studied 9 West Virginia potteries during 1936-37. Physical examinations supplemented by roentgenological studies of the chest were made of 2,516 workers actively engaged in the manufacture of pottery

products. Of this population, 189 (7.8%) were diagnosed as being silicotic: 123 (4.5%) were classified as first stage silicotic, 60 (2.4%) as second stage, and 6 as third stage. The data presented in Table III-2, taken from the Flinn study, [29] show the relation between dust concentration, length of employment in the pottery industry, and the percentage of workers affected by silicosis.

TABLE III-2

RELATION OF DUST CONCENTRATION AND LENGTH OF EMPLOYMENT IN THE POTTERY INDUSTRY TO SILICOSIS*

Dust concentration, million particles/cu ft	Years in pottery industry 0-9	10-19	20-29	30-39	Over 40
0-3.9:					
Cases of silicosis	-	1	1	-	-
Workers exposed	481	223	65	21	8
Percentage	0	0.4	1.5	0	0
4-7.9:					
Cases of silicosis	1	6	26	27	29
Workers exposed	321	198	110	53	34
Percentage	0.3	3	24	51	85
8-15.9:					
Cases of silicosis	-	8	5	10	10
Workers exposed	176	119	25	17	14
Percentage	0	7	20	59	71
Over 16:					
Cases of silicosis	13	33	10	5	4
Workers exposed	363	174	21	7	5
Percentage	4	19	48	71	80

*Includes 1st, 2nd, and 3rd stage cases

Two cases of silicosis out of a total of 189 were found at a dust concentration lower than 4 mppcf. Both cases were first-stage silicosis, the diagnosis resting largely on X-ray evidence, and "reasonable doubt exists in each case whether a higher value might not be a more appropriate measure of that individual's dust exposure." However, closer evaluation of the other cases of silicosis related to relatively low dust concentrations showed 9 cases between 4-5 mppcf, 9 cases between 5-6 mppcf and 76 cases between 6-7 mppcf. These data suggest that even at these low dust concentrations pottery workers may be at some risk of developing silicosis.

Impinger samples of the workroom air were collected at the breathing level of workers engaged in representative occupations. In the areas where silicosis was found, estimated quartz content of settled dust samples (analyzed petrographically) ranged from 1-39% of the collected dust. Weighted average total dust concentrations for all occupations ranged from 3 mppcf to 440 mppcf. Particle size analysis of impinger dust samples indicated a particle diameter of 1.2 μm. No data on respirable free silica were reported. The authors [29] suggested that "if the dust concentration in potteries could be brought below 4 million particles per cubic foot (of air) new cases of silicosis would not develop."

Rajhans and Budlovsky's [30] recent (1970-71) investigation of dust conditions in 10 brick and tile plants of Ontario found no silicotics among 1,166 production workers examined. Free silica content of total airborne dusts varied from 13.2-24.8%. Raw materials (clay and shales) contained from 22-32% free silica. Free silica content of the respirable dust was approximately 13%. Workplace dust concentrations by impinger counts ranged

from 9-2464 mppcf (means ranged from 12-1026 mppcf). Average respirable dust concentrations collected with the Hexlet sampler were between 1.05-4.26 mg/cu m. Length of service or length of exposure of workers examined was from 1-30 years in the industry. These findings are similar to those reported by Keatinge and Potter [31] who investigated health conditions of workers in 3 British brick plants. Minimal "dust" changes were observed on chest films and it was concluded that brick making did not involve excessive occupational hazards. It was suggested by Rajhans and Budlovsky [30] that the combined alumina content (14%) of the clays and slates used in manufacturing the brick and tile of their study may inhibit the progression of the silicotic process. This may account for the absence of silicosis among the workers exposed to high free silica dust concentrations in those industries. [30,31] This phenomenon has been observed by others; it is discussed later.

A comprehensive study of 4 representative plants of the silica brick industry in Pennsylvania was reported by Fulton et al [32] in 1941. Quartz content of the quartzite rock used in manufacturing the silica bricks averaged 97.2% free silica, the remainder (2.8%) being oxides of calcium, iron, and aluminum. Analyses of representative dust encountered in operations before the bricks had been burned showed it contained 75% quartz. Dusts from the unfired (green) brick departments contained 88% quartz with traces of cristobalite and tridymite whereas the burned brick dust consisted of 80% free silica as cristobalite and tridymite with only a trace of quartz. Particle size of the dusts ranged from approximately 1.85 - 2.03 μm. Average dust concentrations in separate silica brick

manufacturing departments ranged from 16 - 83 mppcf. Average dust concentrations for individual operations within the 7 brick manufacturing departments ranged from 0.9-726 mppcf. Environmental data prior to the 1939 survey were not available.

In sharp contrast with the findings of Rajhans and Budlovsky [30] and Keatinge and Potter, [31] Fulton and his co-workers [32] found that of the 1,035 exposed workers they examined, 538 (52%) were silicotic (classified as 1st, 2nd, and 3rd stage silicosis). Sixty-nine of these had been previously exposed to pneumoconiosis-producing dust and were excluded from the study. A high prevalence rate (ranging from 41-58%) was found among the workers who were grouped in 3 exposure classifications 0-9.9, 10-19.9, and 20 or above mppcf. An average of 17.9 years was required to produce stage 2 silicosis among the green-brick men whose exposure was restricted to green-brick dust (chiefly quartz) while the men in the burned brick department (cristobalite and tridymite) were found to have stage 3 silicosis with the same length of exposure (17.9 years). Fourteen of 65 workers whose average exposure was 2-4 mppcf were silicotic. No silicosis was found among workers whose average exposure was less than 2 mppcf.

Silicosis in the Barre, Vermont, granite monument industry has long been a major source of data for permissible dustiness in the United States. [17,33,34] As a manufacturing operation conducted year after year in the same facilities and with a stable population of more than 1,000 workers in the same geographic vicinity, this industry maintains a degree of stability not found in occupations such as mining and quarrying. In spite of extensive technical changes in the industry, some operations are still

conducted as they were in the 1920's except for the introduction of controls at the dustier jobs. Most importantly, the Division of Industrial Hygiene of the Vermont State Board of Health as well as other groups have conducted periodic inspections and dust evaluations in this industry for more than 30 years. These factors have made this location and occupation highly useful for repeated investigations into the environmental and medical factors associated with silicosis.

The early history of the granite-working sheds was typical of other uncontrolled operations (mining, foundries, ceramic industry) involving exposures to quartz-containing dust. The advent of pneumatic tools around the turn of the century caused dust concentrations to increase by factors of 10 to 100 as compared with manual operations. Discomfort and, after a few years, dust disease caused initiation of rudimentary dust reduction measures, such as placing surfacing machines outdoors and wetting the stone before cutting. But when groups of workers were examined, most showed some evidence of silicosis. Silicotuberculosis was the usual cause of death of granite cutters, and few lived to be 60 years of age. [17,34]

Russell et al [17] reported that of 972 men in 14 granite sheds studied, 614 were exposed to dust concentrations averaging from 37-59 mppcf. In this group, the first case of early silicosis appeared after approximately 2 years of service, and prevalence of the disease was 100% after 4 years. Of 108 men in occupations where dustiness averaged between 3-9 mppcf, two cases of early silicosis occurred after 10 years' exposure and one case of moderately developed silicosis after 6 years' exposure. The average dust concentration at the time of the Russell study was about

20 mppcf. Unknown dust concentrations to which the workers in this lowest exposure group may have been exposed early in their occupational history may have been a factor in the development of their silicosis. Russell et al [17] concluded that on the basis of these findings, for this type of work, a presumptive safe limit of dustiness for rock dust containing 35% free silica lay somewhere between 9-20 mppcf in the size range under 10 μm.

A restudy by the US Public Health Service in 1937-1938 [33] of 116 of the workers examined in the 1924-26 study [17] confirmed the findings of the original study. Progression of silicosis was marked in the highly exposed cutters in contrast with workers exposed to the lower concentrations of dust, emphasizing that the differences in reaction to the dust hazard are in direct proportion to the intensity of dustiness. The author [33] concluded that where the average dust concentration was 6 mppcf (range of 2.5-9.0 mppcf) there was no indication of any unfavorable effects on health, either from the physical examinations, the sickness records, or such mortality data as were applicable to the less than average dustiness exposed group. Russell [33] stated that "What appears to stand out most clearly is that a maximum of dust exposure, falling somewhere about 10 mppcf of air for the dust-making operations, for a dust which contains from 25 to 35 percent of free silica in the form of quartz, is a desirable limit". Although not officially adopted by the Vermont Department of Health, this airborne level has been in use in the Vermont granite sheds since 1937 as the desirable upper limit for dustiness.

From the environmental and medical data associated with the various granite shed occupations Russell [33] suggested the "tentative thresholds

of dust tolerance" shown in Table III-3 based upon classification of the industry into three dust-count concentration groups.

TABLE III-3

TENTATIVE THRESHOLD DUST TOLERANCES
FOR BARRE, VERMONT, GRANITE INDUSTRY

Occupation	Avg. Dustiness mppcf	% Free silica	Tentative threshold mppcf	Hazard - actual and potential
Above average dustiness (Granite cutters)	42	30-35	Less than 10	Silicosis
Average plant dustiness (Mechanics, laborers, cranemen)	20	30-35	Less than 10	Moderate fibrosis Silicosis
Less than average dustiness (Office workers, sawyers, blacksmiths)	6	30-35	Less than 10	Slight fibrosis

Derived from references 17,33

An environmental study and a review of medical records conducted by the US Public Health Service and the Vermont State Board of Health in 1955 [34] of the Vermont granite industry indicated that few exposures in the granite sheds studied exceeded 5 mppcf. A total of 1,112 workers in the study group started work in the industry before 1937 and 1,134 during or after 1937. Prevalence of silicosis, as determined by chest roentgenographic surveys of the workers, had decreased from 45% in 1937-38 to 15% in 1956. The average years of employment for the men with silicosis was 32.4 and for the men with no silicosis 26.3. Only one new case of

silicosis, and this one doubtful, was found in the group of 1,134 men who started work in the granite industry in 1937 or later. Quartz content of settled and airborne dust samples averaged 22-25% as compared to an earlier analysis of 25-35%. [33]

The report [34] concluded that "Insofar as the chest X-ray records show, progress observed thus far in the prevention and elimination of silicosis in the Vermont granite industry is indeed gratifying." The authors recognized however that "...the number of men working under complete dust control and over long enough periods is relatively small, so that it may take some time before the adequacy of present day control methods can be ultimately determined". In addition, "the prolonged effects of the uncontrolled working conditions will be felt for many years to come".

In 1964 Ashe and Bergstrom [35] published the results of a medical study which reported no cases of silicosis among workers in the Vermont granite industry whose span of employment began after the 1937 dust controls were started. Chest roentgenograms of the 1,478 granite workers studied were interpreted on two occasions by a panel of three readers. Of the granite workers studied, 855 had work experience limited to the 26-year period of dust control. Based on the pre-1937 prevalence rates of silicosis, at least 146 cases of silicosis could have been expected in this group of workers had they been employed prior to 1937. This study confirms earlier findings reported by Ashe [36] which showed a similar absence of silicosis in granite workers exposed over an 18-year span (1937-55). Dust concentrations averaged about 5 mppcf for the 1955 study and 3 mppcf for

the 1964 survey. Quartz dust content of the airborne granite dust was reported to be approximately 25% for both studies.

Data from these two studies suggest that dust control measures incorporated in 1937 have successfully reduced the exposure concentrations of quartz-bearing granite dust to a level sufficient to prevent silicosis in workers exposed to quartz for 26 years. The authors concluded that continued environmental and medical surveillance is necessary to determine the ultimate efficacy of the dust control measures.

In 1969 the Harvard School of Public Health in cooperation with the Vermont Division of Industrial Hygiene began a comprehensive study of the relationship between the exposure to granite dust, its quartz content and lung disease among granite shed workers in Vermont. [37-39]

In considering these latest studies of the Vermont granite shed industry it must be noted that in the last decade major changes have occurred in methods for sampling and analyzing dusts. Personal breathing zone sampling has replaced fixed location sampling, providing a better estimate of the quantity of dust breathed by the worker. This personal sampling method now provides respirable mass dust concentration (mg/cu m) data rather than the count concentration (mppcf) obtained from the impinger sampling technique used earlier. Another factor which must be considered is that the characteristics of the inhaled dust have changed over the years. Such changes have resulted from the introduction of new technology in the granite shed operations, eg mechanization of cutting and of grinding and polishing operations. The quartz content of the dust has also changed as a result of dilution from ambient air pollutants, silicon carbide, and

other particulates from wet cutting operations, and dusts from other activities. [37]

To estimate the current dust exposure in the granite sheds, [37] 784 personal respirable mass dust samples were collected from 13 occupational groups in 49 granite sheds. Of these samples 486 (61%) were analyzed for quartz content by infrared spectrophotometry. [40] Occupations within the granite shed were classified in the same manner as reported by Russell et al [17,33] and Hosey et al. [34] In comparing the degree of dustiness for a given occupational classification, this latest study [37] showed a significant reduction in the "above average" and "average" plant dustiness classifications. Concentrations were reduced from 42 to 7 and 20 to 3 mppcf. Quartz content of settled dust and the nonrespirable fraction of dust collected with a size-selective personal sampler were 30% and 28%, respectively. On the other hand, the quartz content of the respirable dust samples was 9% as analyzed by infrared spectrophotometry. [40]

On the basis of employment records and utilizing the results of the present and past dust sampling studies in the 49 granite sheds, each granite worker studied was assigned a lifetime weighted respirable dust exposure. [37] A granite dust-year was defined as exposure for 40 hours/week for one year to an average dust concentration of 523 μg/cu m for all occupations in the granite sheds. One quartz-year was defined as an exposure of 40 hours/week for one year to the average quartz concentration of 50 μg/cu m. Seven hundred and ninety-two active granite shed workers were studied for pulmonary function alterations, [38] and 784 of the same workers for chest roentgenographic changes. [39]

The population studied included workers who had been employed before completion of dust control measures in the Vermont granite industry in 1937. Three control groups were utilized: one of 69 workers from the granite shed population who supposedly had no dust exposure; another utilized the results published as "normal values" from studies of Kory et al [41]; and a third control consisted of marble workers employed in an industry similar to the granite sheds but without exposure to granite dust. Measures of lung function included forced vital capacity (FVC), forced expiratory volume in one second (FEV 1), total lung capacity (TLC), and residual volume (RV). Results of the pulmonary function tests showed [38,39] that granite dust and quartz dust caused a decrease in FVC, FEV 1, and TLC but not in RV. This decrease was estimated by multiple regression analysis at 2 ml/dust-year. This decrease for every year of exposure at an average concentration of 523 μg/cu m of granite dust and 50 μg/cu m of quartz-dust was considered significant by the authors. [38]

Chest roentgenograms of the 784 workers were divided by one physician according to the UICC/Cincinnati classification [42] into two groups: 551 normal readings and 233 abnormal readings. Films showing opacities compatible with pneumoconiosis were classified as abnormal. Workers with abnormal roentgenograms were exposed on the average to 2.3 times more dust than those with normal roentgenograms. The increase in dust exposure also correlated with increase in size of rounded opacities and profusion. Comparison of the group with normal film readings and those with abnormal films revealed that individuals with opacities, after standardization for age, height, and smoking habits, had statistically

significantly (p = less than 0.02) lower forced vital capacities. A dose-response curve relating the effects of granite dust exposures on ventilatory function and chest roentgenograms suggested that there was a delay of about 13.5 dust-years between the appearance of pulmonary function alterations and the finding of abnormal roentgenograms. A total of 32.5 dust-years of exposure was necessary to affect the ventilatory function of 50% of the workers while it took 46 dust-years to produce opacities on roentgenograms. The difference of 13.5 dust-years between appearance of changes in the two responses measured would indicate that pulmonary function measurements are more sensitive indicators of the effects of exposure to granite dust.

Few epidemiologic studies relate directly to industrial exposures to forms of free silica other than quartz. In 1948 Vigliani and Mottura [43] reported on 20 workers exposed to calcined diatomite used in manufacturing filter candles. The X-rays of 13 of these workers showed some stage of rapidly developing silicosis. The majority of those exhibiting radiological signs indicative of silicosis had worked for 4 or more years in the candle-turning department. The material employed in manufacturing the candles contained 80% calcined diatomaceous earth. This microcrystalline free silica gave the X-ray diffraction pattern of cristobalite which was considered responsible for the cases of silicosis reported. Dust exposures were estimated at 11-14 mppcf of air. Particle size ranged from 0.5-2 μm.

The study described earlier of Fulton et al [32] of the Pennsylvania silica brick industry included data on cristobalite- and tridymite-bearing

dusts evolved from the "burned brick" manufacturing process. Results of physical examinations of 1,035 silica brick workers showed 538 (52%) to have silicosis. An average length of exposure of 17.9 years was required to produce stage 3 silicosis among the burned-brick department employees whose exposure was to cristobalite and tridymite while only stage 2 silicosis had developed in green-brick workers who had the same length of exposure to quartz only.

A 1953-54 study by the Public Health Service [44] in five diatomite plants in the Western states included roentgenographic examination of 869 diatomite workers. Of this number 78 or 9% showed changes interpreted as consistent with a diagnosis of pneumoconiosis; doubtful changes were found in an additional 9% of the workers.

Data presented in the report also suggested that nearly all presumptive abnormal chest roentgenograms were associated with employment where workers could be exposed to calcined diatomite containing 15-61% cristobalite. Airborne dust concentrations for all plant operations ranged from 1-66 mppcf with a median particle size of 1.1 μm.

The report concluded that the extent and severity of pneumoconiosis, as evidenced by roentgenographic changes, appeared to correlate with the cristobalite content of the dust and length of exposure. It was recommended that exposures to cristobalite containing dusts be kept under 5 mppcf.

Two subsequent studies of this diatomite industry have been made. [45,46] Cralley et al [45] in a 10-year followup study reported no new cases of pneumoconiosis in any of 253 employees who had joined the work

force between 1953-1963. There was progression from negative to evidence of simple pneumoconiosis in 2 of 479 workers who had been reported negative in 1953. Two workers originally diagnosed as having simple pneumoconiosis had developed coalescent lesions. The latest survey [46] covered the 16-year period after the original study, however, only 1 of the 5 plants initially surveyed was available for the followup. Among employees diagnosed as not having pneumoconiosis at the time of the 1953 survey, only two subsequently developed evidence of simple pneumoconiosis. Of 441 individuals who joined the work force between 1953 and 1967, none had roentgenographic evidence of pneumoconiosis as of 1969. The 2 workers who had simple pneumoconiosis in 1953 progressed to complicated pneumoconiosis, and several individuals with doubtful or definite coalescent lesions showed progression.

All atmospheric dust concentrations were reported, with one exception, to be below 8 mppcf. In this area of the plant respiratory protection was mandatory.

It has been suggested by some investigators [43,47,48] in addition to those who conducted the PHS diatomite industry surveys [44] that natural, uncalcined diatomaceous earth promotes a form of pneumoconiosis in workers exposed for long periods of time to this material. According to Smart and Anderson, [48] a benign type of linear pulmonary fibrosis develops, leading to few if any symptoms and no demonstrable disability.

Animal Toxicity

Silicosis similar to that seen in man has been produced in a number of animal species: rats, [49] guinea pigs, [50] rabbits, [50] dogs, [51] and monkeys. [52]

Animal studies with various forms of free silica have demonstrated a capacity for the minerals to induce a fibrogenic response in organs other than the lungs. [53]

King et al, [49] in studies on the relative fibrogenicity in rat lung tissue of tridymite, cristobalite, quartz, and cryptocrystalline fused quartz injected intratracheally, demonstrated that the most fibrogenic form was tridymite followed in descending order by cristobalite and quartz.

Brieger and Gross, [54] also employing intratracheal injections, produced a typical silicotic tissue reaction in rat lungs and lymphatic tissues following injection of 30 mg of quartz and coesite dusts.

Goldstein and Webster [55] studied the relative pathogenicity of an approximately 90% pure quartz dust of different size ranges of particles (less than 1, 1-3, 2-5 μm) but having equal surface area. They found that fibrosis was least severe in the rats intratracheally injected with the quartz suspension having the smallest particle size. In addition, the degree of fibrosis varied with the quantity (by weight) or size of particles but not with the surface area. King et al [56] in an earlier study found maximal fibrogenicity produced in rats by 50 mg intratracheal injections of a quartz-cristobalite dust suspension in the particle size range of 0.5-2 μm.

Chronic inhalation studies by Gardner [50] with guinea pigs and rabbits produced a cellular proliferation and laying-down of fibrous tissue in tracheobronchial lymph nodes and lungs after a few months of quartz dust exposure. After a period of about two years, the lesions presented almost all the essential characteristics of the silicotic nodules seen in human cases of silicosis. Dust exposure concentrations for the 2-year, 8-hour/day, 6-day/week study were approximately 4,400 mppcf with respirable dust of 91% quartz.

Gardner's [53] summary of experiments in which rabbits were injected intravenously with various free silica dusts showed that responses to cristobalite and tridymite were more severe than those from quartz, and the fibrosis that followed was of a diffuse form, rather than nodular.

Chronic animal inhalation studies by Wagner et al [51] with flux-calcined diatomaceous earth of 61% cristobalite content and at dust exposure concentrations of 2 mppcf produced fibrotic nodules in hilar lymph nodes in dogs exposed for periods up to 2.5 years. No fibrosis of pulmonary parenchyma was observed in dogs, guinea pigs, or rats chronically exposed at 2 or 5 mppcf.

Neymann's [52] studies on experimental silicosis in monkeys showed varying degrees of dust-laden macrophages, fibrocytes, hyalinized collagen fibers, and interstitial fibrosis in animals exposed to 3 μm quartz dust particles for up to 27 months.

The cytotoxic effects of free silica on alveolar, lymph node, and peritoneal macrophages have been demonstrated in vivo and in vitro by a number of investigators, including Marks, [57] Vigliani et al, [58]

Heppleston and Styles, [59] Heppleston, [60] Vigliani, [61] Pervis and Ghislandi, [62] Allison et al, [63] Burrell and Anderson, [64] and Zaidi. [65]

These studies suggested that the cytotoxic and fibrogenic activity was due to the rupture of the macrophage lysosomal membrane and the release of a factor, probably lytic enzymes, which produce cytoplasmic damage as they diffuse into the surrounding medium. Following lysis of the macrophages, the phagocytized free silica particles are liberated and thus are free to cause further damage to fresh macrophages. Further tissue changes, ie, perivascular aggregation of lymphoid tissue and fibrosis, may follow but it is uncertain what chain of events leads from the damaged macrophage to the fibrosis. Heppleston [60] discussed a "factor" or "factors" released from the free silica damaged macrophages which was thought responsible for stimulating collagen formation. The studies by Vigliani [61] and by Pervis and Ghislandi [62] demonstrated a similar reaction. The macrophages after ingestion of free silica particles undergo degeneration resulting in the liberation of certain toxic substances as well as the ingested particles. The ingested particles are again taken up by fresh macrophages to repeat the continuous cycle. The toxic substances initiate the cellular reaction which consists of new macrophages, mast cells, fibroblasts, and plasma cells. Phospholipids are also released from dying macrophages and cause stimulation of fibroblasts which leads to collagen formation.

Although there is general agreement that deposited free silica particles are engulfed by phagocytic cells, which are rapidly destroyed,

the fibrogenic effects are not yet fully explained. Why this substance of simple chemical composition and low chemical reactivity has such a selective toxicity for one cell type, the macrophage, whereas other particles of comparable size and surface area (such as carbon particles or diamond dust) are ingested by cells without harmful effects is a question of importance in relation to the pathogenesis of silicosis that merits further investigation. [63]

In 1932 Kettle [66] reported that coating silica particles with iron inhibited its ability to cause silicosis in animals. Gross et al [67] later pointed out that this inhibition was short-lived, and that the limited duration of the inhibition was related to the disappearance of the iron from the dust and the tissue after a few months.

Denny et al [68] in 1937 demonstrated that metallic aluminum powder completely inhibited the development of silicosis in animals. Because of this successful inhibition of silicosis and the apparent lack of toxicity of the metallic aluminum dust, [69] experiments involving workers exposed to free silica in plants and mines were initiated to determine the effectiveness of aluminum dusts to prevent or arrest development of silicosis in workers. [70-72] Although some subjective improvement was noted among some of the workers given daily inhalation treatment of aerosolized aluminum dust over periods of 2-3 years, no improvement was observed in the chest roentgenograms or in lung functions. [72]

Studies by Schlipkoter and Brockhaus [73] and Schlipkoter et al [74] showed that polyvinylpyridine (PVP) and polyvinylpyridine-N-oxide (PVPNO) inhibited silicosis in rats exposed by intraperitoneal and intratracheal

injections of quartz dust and PVP or quartz-containing coal mine dust and PVPNO. The authors suggested that the adsorption of the PVP compound on the dusts was responsible for the modified silicosis. Other mechanisms, however, cannot be excluded. Because this phenomenon has important implications for the treatment of human disease, there is obvious need for further exploration of this finding.

Correlation of Exposure and Effect

The epidemiologic studies and data presented earlier attempt to relate the prevalence of silicosis in industrial workers to the degree and duration of exposure to free silica. It is evident that the higher the dust concentration of free silica, the more rapid the development of silicosis and its prevalence. Conversely, as dust is controlled, the frequency of occurrence of silicosis decreases, the severity of the disease lessens, and the length of time for the disease to become manifest increases. As a consequence of this extended time for the development of the disease, it becomes more difficult to establish a relationship between lifetime dust exposure and disease incidence. In addition, environmental data obtained from the early epidemiologic studies do not permit judgment of the adequacy of present standards since information on working conditions prior to initiation of the studies is often lacking or poorly defined. Table XI-1 presents data from epidemiologic studies made prior to 1940 in industries in which silicosis was known to occur to a significant

degree. [33] Free silica exposures in excess of the listed "permissible maximum safe dust concentration" were considered responsible for the production of silicosis in workers for a given industry. It is noteworthy that for the majority (4 or 6) of the industries represented the maximum dust limits considered appropriate, even in these relatively early studies, were significantly below the 10 mppcf limit presently accepted in the US for exposure to quartz dust. (See Table XI-1) Subsequent reports by other investigators [25-27,29,30,32,33,37-39,44] are considered more accurate in relating the environmental conditions to the medical studies and the prevalence of silicosis. From these reports, data also became available for the first time on workers employed after dust-control measures were instituted in some industries. In all cases the prevalence of silicosis was substantially higher in workers employed prior to application of dust controls. The metal mine study of 1958-1961 [26] showed an overall prevalence rate of silicosis of 3.4%. Full shift, weighted average mine dust exposure of underground mine employees was 6.8 mppcf. In contrast, earlier studies of silicosis among metal mine workers [16,24] found more than 60% of the workers with roentgenologic evidence of silicosis. Exposure concentrations were not reported but were undoubtedly substantially higher than 6.8 mppcf. Dreessen et al [25] reported that 86% of the miners working during a 1939 survey were exposed to dust concentrations between 6 and 30 mppcf.

Environmental and medical studies have not been made to reevaluate the prevalence of silicosis in ferrous foundry workers studied in 1950 by Renes et al. [27] However, increased incidence of silicosis beyond the

9.2% reported in 1950 would be anticipated in the workers with 20 or more years of exposure. This assumption is based on longer exposure of the workers to the 13-20% free silica dust at concentrations of 3-75 mppcf or higher.

The study of West Virginia potteries by Flinn et al [29] suggests that significant consideration should be given to the relationship of low dust concentrations (below 10 mppcf), duration of exposure (years of work), and the prevalence of silicosis. It was suggested that exposure to more than 4 mppcf of dust for prolonged periods of time in pottery factories may result in silicosis, the hazard increasing with increasing dust concentration. From a total of 189 cases of silicosis in 2,516 workers examined, 96 cases were found in those exposed to daily concentrations of dust below 7 mppcf. Seventy-eight cases were found at concentrations between 6-7 mppcf; 7 cases between 5-6 mppcf; 9 cases between 4-5 mppcf, and 2 cases at below 4 mppcf. Previous exposures at higher dust concentrations were probably responsible for the last 2 cases. An increasing percentage of workers with silicosis was identified with increasing length of employment. At dust concentrations between 4-8 mppcf, workers with 30 or more years of work exhibited a prevalence of silicosis in excess of 50%. With this incidence of disease at relatively low dust concentrations, the possibility exists that the prevalence of silicosis in workers exposed below 4 mppcf would increase given a long enough period of exposure.

The study of the silica brick industry by Fulton et al [32] is another report in which relatively low dust concentrations, duration of

employment, and incidence of silicosis suggest that reconsideration be given to the long established "safe" dust exposure of approximately 10 mppcf. Forty-two percent (210 of 499) of workers exposed to average dust concentrations ranging from 0-9.9 mppcf were diagnosed as having silicosis. An additional 218 employees exposed to 10-19.9 mppcf average dust concentrations were also silicotic. The disease was not found where the average exposure was less than 2 mppcf. Free silica content of the brick dust ranged from 75-90%.

Studies of environmental data and prevalence of silicosis among workers in the Vermont granite industry provide a major basis for the correlation of effects from exposure to granite dust. [17,33,34,37-39]

Russell et al [17] first showed (1924-25) the results of exposure of workers to a 25-35% quartz content granite dust at concentrations averaging 27-59 mppcf. Among these workers the first case of early silicosis appeared after approximately 2 years of exposure to the highest (59 mppcf) average concentration. Within 4 years a 100% prevalence of the disease was found. These findings prompted Russell to conclude that a safe level of dust exposure in the Vermont granite sheds was somewhere between 9-20 mppcf. A restudy [33] of the industry approximately 12 years later (1937) further established the relationship of granite dust concentration to progression of silicosis, ie, the higher the dust exposure, the greater the prevalence of the disease and the more rapid the progression. The data suggested that at an average dust exposure of 6 mppcf there were no unfavorable health effects on the worker and led Russell [33] to suggest a tentative threshold of 10 mppcf.

A study of the industry in 1955-56 by Hosey et al [34] showed that engineering controls initiated in 1937 had reduced essentially all granite shed dust concentrations to about 5 mppcf. Airborne dust samples were found to contain approximately 25% quartz. Only one new, but doubtful, case of silicosis was found in the group of men whose exposure had started in 1937. Workers who had been employed prior to 1937 and who were positive for silicosis had an average of 32.4 years of exposure to granite dust while those without the disease had an average of 26.3 years. Of all the workers studied, 50% had started work before and 50% after dust controls were initiated. Thus it was recognized at the time of this study, [34] 18 years after the controls were started, that a final judgment as to the efficacy of the control measures and reduced dust exposures, in relation to the prevalence and progression of silicosis, could not be made until a greater time span had elapsed. Ashe and Bergstrom's data [35] published in 1964 contributed to further evaluation of the dust controls instituted some 26 years earlier. No cases of silicosis were found in workers whose exposure started after 1937. Dust concentrations averaged 3 mppcf (range 0.5-8.3 mppcf). The authors [35] concluded that "careful surveillance of the working environment and annual X-rays of the exposed workmen must be continued to determine the ultimate efficacy of dust control in the Vermont granite industry."

The most recent studies (1969-70) of the Vermont granite industry, approximately 32 years after initiation of the 1937 control measures, are those by Theriault et al. [37-39] Their findings were based on environmental data determined by personal respirable mass samples, and

pulmonary function and roentgenographic evaluations. These studies included both prospective and retrospective data and merit special consideration for developing a correlation of exposure and effect at the relatively low average granite shed dust concentrations reported, 523 μg/cu m for granite dust and 50 μg/cu m for quartz dust.

A lifetime estimate of exposure to granite dust and quartz dust was calculated for each of 792 workers from the dust concentration data and a complete occupational history. These exposure concentrations reportedly caused a significant decrease in pulmonary functions for FVC, FEV 1, and TLC. This decrease, in excess of that calculated for three control groups, was estimated at 2 ml/dust-year. A total of 32.5 dust-years of exposure was required to affect the ventilatory function of 50% of the workers. Chest roentgenograms of 30% of the workers examined were classified as abnormal (opacities compatible with pneumoconiosis) according to the UICC/Cincinnati classification. [42] Increase in dust-years exposure correlated with the increase in size of rounded, dust-induced lung opacities and their profusion. Forced vital capacity was lower for people with abnormal roentgenograms and it decreased as profusion of opacities increased. Forty-six dust-years of exposure were necessary to produce opacities on 50% of the roentgenograms classified as abnormal. This represented a delay of about 13.5 years between the effects of dust exposure on ventilatory capacity and the appearance of opacities on chest roentgenograms. It was suggested that early detection of dust effects in groups of workers is better accomplished by pulmonary function tests than by roentgenographic evaluations. The change reported, a 2-ml decrement of

FVC/year in excess of normal, would not appear to be clinically significant from a functional standpoint; however, the presence of p- and q-type lung opacities probably reflects permanent changes in pulmonary tissue that could impair the health of the worker. In any case, effects were recorded at concentrations below the dust exposure standard of 10 mppcf used since 1937.

Data from environmental and medical studies of the California diatomite industry by Cooper and Cralley [44] showed roentgenographic changes interpreted as consistent with a diagnosis of pneumoconiosis in 9% of workers exposed to diatomaceous earth dusts at concentrations ranging from 1-66 mppcf. Doubtful changes were found in an additional 9%. The incidence of disease was associated with exposures to the airborne dusts containing cristobalite and a trace of tridymite as the forms of free silica. Cristobalite content ranged from 15-61%. The extent of severity of pneumoconiosis, as evidenced by the roentgenographic changes, appeared to correlate with the cristobalite content of the dust and the length of exposure. Workers employed in mill operations for 5 or more years and exposed to the highest (61%) cristobalite content dusts at an average concentration of 11 mppcf (21-99 mppcf) showed a 47% incidence of pneumoconiosis.

The observations of Cooper and Cralley [44] are supported by those reported by Vigliani and Mottura [43] of workers manufacturing ceramic filter candles exposed to an 80% cristobalite content dust. A rapidly developing form of silicosis was identified in the majority of exposed workers who had worked 4 or more years in the industry. Their dust

exposures were estimated at 11-14 mppcf. The silica brick industry study by Fulton and his co-workers [32] also associated cristobalite and tridymite exposures with an accelerated progression of silicosis, ie, the same average duration of exposure was required to produce stage 3 silicosis among workers exposed to chiefly cristobalite and tridymite dusts (62% and 17%) as produced a stage 2 silicosis in uncalcined brick workers whose exposure was mainly to quartz dust (87%). Roentgenographic surveys covering 16 years [45,46] of the California diatomite industry following reduction of more than 84% of the cristobalite dust levels to below 5 mppcf revealed no new cases of pneumoconiosis in workers who had joined the work force since control measures were introduced in 1953.

Accelerated and frequently fatal silicosis among sandblasters has been documented in several published reports. Merewether [75] in 1936 reported that sandblasters in Great Britain had an average employment duration of 10.3 years prior to death from silicosis. The length of employment of all other fatal silicosis cases, irrespective of occupational cause, was 40.1 years. Ziskind et al [76] in a 1973 report of shipyard silica sandblasting operations discussed the fate of 22 silicotic sandblasters. Of these, 11 died at an average age of 48.5 years, with an average silica exposure of 11 years. Eight showed massive disease on chest roentgenograms; 1 had silicoproteinosis; and 6 had complicated pulmonary tuberculosis.

In the 11 survivors, the average age was 44 years with an average exposure of 12.5 years. Seven had extensive disease as seen on chest X-rays. Pulmonary function studies showed depressions of all functional

parameters which were more marked in these cases which were ultimately fatal. The rate of deterioration was very rapid, was related to change of chest roentgenographic category, and greatly exceeded standard predicted regressions.

It should be noted that fatal, accelerated silicosis as seen in American shipyard sandblasters follows approximately the same average length of exposure to silica dust as reported in the earlier study by Merewether. [75]

Environmental data from the report of Ziskind et al, [76] using personal gravimetric samplers, indicated an average 37.3 mg/cu m concentration of respirable dust outside the sandblasters' protective hood; the percentage of free silica in the dust was 83.6%; the majority of particles were below 3μm in diameter.

The use of silica sand for blast cleaning operations was prohibited in Great Britain 25 years ago under enactment by the Ministry of Labour and National Service. [77]

IV. ENVIRONMENTAL DATA

Dust Measurement

To evaluate either the hazard to health from exposure to dust or the effectiveness of dust control measures, one must have a method or methods for the evaluation of the dustiness. Ideally the methods employed should be as closely related to the health hazard as possible. When determining exposure to dusts containing free silica, in addition to determining the percentage of free silica the method should measure that portion of the dust causing silicosis, ie, that dust which penetrates and is retained in the pulmonary, nonciliated regions of the lungs.

Through the years, many collection methods have been used in the determination of dustiness, and these methods have been reviewed by a number of authors. [78-83] Because these reviews are comprehensive, only the basic principle of the major methods will be briefly discussed here.

(a) Count procedures: The concern of industrial hygienists over the years has been to measure that fraction of a dust that can cause pneumoconiosis. Since it has been recognized that only dust particles smaller than approximately 5 μm in aerodynamic diameter are deposited and retained in the lung, methods were sought to measure concentrations of this dust. [83,84] Microscopic counting of dust collected by impingement has long been used for this purpose. Dust counting as an index of dust concentration and consequently of workers' exposure has been used in South Africa by Kitto [85] using the konimeter and in Australia by Owens [86] with a jet dust sampler. In the United Kingdom, thermal precipitation has been frequently used for dust collection [84] while in the United States

the Greenburg-Smith and midget impingers have been commonly employed. [87-89] In these investigations, the lower limit of dust size included in counts was determined by procedure or was implicit in the counting procedure employed. Where a 10X (16 mm) objective lens was used with light-field counting, as in impinger counts, the usual lower limit of diameter of particles seen was approximately 1.0 μm. Others have used dark-field illumination with which it is possible to see particles as small as 0.1 μm diameter. [80]

Because of differences in sampling techniques and instruments used, comparisons of dust concentration with silicosis prevalence in different parts of the world is difficult. This points to the fact that if dust concentrations are measured by count procedure, the procedure employed should follow a standardized method to minimize differences. Such standard methods for impinger sampling and dust counting have been published. [89-91]

Because the prevalence of silicosis in the 1920's and earlier was severe, more effort was devoted to improving dust conditions than to refining and developing methods of dust sampling and measurement. [17,24-29] Although counting methods are inefficient and give variable results, they clearly showed the effectiveness of dust control measures. [25-27,33,44] Later, with efforts to further reduce silicosis, researchers also turned to improving dust measurement methods. [37,79,82,83]

(b) "Total" mass concentration methods: The simplest method of measuring dust concentrations is to determine the total weight of dust collected in a given volume of air. The "total" mass, however, is determined to a considerable extent by the large dust particles, which

cannot penetrate to the pulmonary spaces to cause silicosis. The proportion of dust small enough to penetrate to the pulmonary spaces ("respirable" dust) is extremely variable, ranging in industrial dust clouds from as little as 5% to more than 50% by weight. [80,84]

Thus, the "total" dust concentration by weight is not a reliable index of "respirable" dust concentrations or an index of a silicosis hazard.

(c) Respirable mass size-selective measurement (personal sampling): For evaluation of a silicosis hazard, the method now generally preferred is personal (breathing zone) respirable mass sampling. [78] Dust collection devices now available for this method of sampling also provide a means for a size-frequency analysis of the collected dust. A traditional method for such an analysis has been to collect a sample on a membrane filter and examine it by high-powered optical microscopy (about 1000X), supplemented, perhaps, by electron microscopy, as described by McKee and Fulwiler. [92] Present-day instrumentation permits collection of a dust in such a manner that the sample is size-separated by the design and flow characteristics of the sampling device. Such equipment includes impactors, centrifugal and gravitational separators, and a range of miniature cyclones. [84,92-94] In addition to particle-size separation, these instruments are also capable of collecting a quantity of dust sufficient for an analysis for free silica content of the dust as recommended in Appendix II.

Respirable mass samples are preferably taken over a full 10-hour shift. However, multiple, shorter period (2-4 hour) samples may be collected over an individual's full-shift exposure period, the samples

pooled for analytical purposes, and the average respirable mass concentration of free silica calculated on a full-shift basis. The recommended equipment and the method for collection of dust containing free silica are presented in Appendix I.

Technical Feasibility of Attainment of Standards

(a) Metal mines: Although there is a lack of published information on current dust levels in metal mines, data from Flinn et al [26] substantiate the existence and capability of engineering controls and technology for reducing metal mine dust levels to comply with the recommended standard.

(b) Foundries: In a study to compare impinger counts (mppcf) with results obtained by respirable mass (mg/cu m) sampling for dust containing quartz, Ayer et al [78] compared respirable mass and impinger measurements in a number of Michigan foundries. They found that, in general, foundries that could meet the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLV) [95] for free silica by impinger count could meet the ACGIH TLV for free silica by respirable-mass measurement. If reduction of respirable free silica levels in foundries is necessary, the technical means for reducing the dust concentrations to meet the required limit are available.

(c) Ceramics industries: Recent data on free silica concentrations in American ceramic factories are not available. The British Ceramic Research Association [96] has in recent years taken a great number of samples, most of which were personal respirable mass dust samples. As a trade association, this group does not ordinarily publish

results of dust measurements. They seem confident, however, that dust standards somewhat stricter than present ACGIH TLVs could be maintained.

(d) Crushing, grinding, and mixing of minerals containing free silica: Although few workers are employed in individual crushing, grinding, and mixing operations in a given plant, overexposure of workers is common. [44] Existing techniques of enclosure, local and general exhaust ventilation, wetting, and the use of respirators are adequate to reduce to acceptable levels the workers' exposure to dust.

Engineering Control

(a) Foundries: Ventilation designs to control free silica exposures in specific foundry operations are given in the ACGIH Industrial Ventilation manual. [97]

(b) Ceramic Industries: Research and successful application of ventilation, blowing and exhausting, and dilution ventilation have been carried out by the British Ceramic Research Association [98] and have undoubtedly reduced dust levels in this industry in the British Isles. The application of these methods as well as the control procedures outlined in the Industrial Ventilation manual [97] appear to be sufficient to control dust levels in American ceramic plants to the recommended level.

(c) Crushing, grinding, screening, etc: Principles for control of dust from crushers and similar devices and for material handling are given in the Industrial Ventilation manual. [97]

(d) Abrasive blasting: Because of the severe silicosis hazard associated with abrasive blasting with silica sand and the extreme difficulty in controlling the hazards associated with its use in abrasive

blasting, it is recommended that silica sand or other substances containing more than 1% free silica be prohibited as abrasive blasting materials. [99,100]

V. DEVELOPMENT OF THE STANDARD

Basis for Previous Standards

In the United States, as elsewhere in the world, there are so many dusty trades in which the extent and nature of dust exposure is so varied that the results in one industry are not always comparable to those in another. Table XI-1 emphasizes this variability and shows the concentrations of free silica-bearing dusts that had been accepted as permissible prior to 1940 for the particular industries in the localities indicated. [33]

Hygienic exposure values for dust containing free silica have been based on the quantitative concept that the magnitude of the toxicity is proportional to the concentration of free silica in the dust. When this magnitude of toxicity is represented by an exposure limit, then the limit is inversely proportional to the percentage of free silica in the dust and can be expressed in mppcf as derived from a particle count of the dust-laden environment and a general particle count formula of:

$$\text{Threshold limit} = \frac{K}{\%SiO2} \text{ mppcf}$$

One of the first recommended "upper limits" for quartz-bearing industrial dusts was that suggested by Russell [33] for the Vermont granite industry based upon studies in that industry. A limit of 10 mppcf for dust containing 25-35% quartz was recommended.

Threshold Limit Values (TLVs) for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes is a guide adopted by the American Conference of Governmental Industrial Hygienists (ACGIH) for use in the control of occupational health hazards. The value for quartz first published in 1946 [101,102] was originally called a maximum allowable concentration (MAC) value and followed the pattern suggested by the particle count formula given above. However, only three ranges of free silica (quartz) content were considered as indicated below.

Range of SiO2, %	MAC - mppcf
Silica--High (above 50% free SiO2)	5
Silica--Medium (5-50% free SiO2)	20
Silica--Low (below 5% free SiO2)	50

Review of the early studies of the Public Health Service [17,25,26,29,34] and others [18,32] suggested that the results of the engineering and medical studies were reasonably consistent with values calculated from the count formula using a factor, designated K, equal to 250, and by adding a constant 5 to the percentage of free silica in the denominator. This formula was published by ACGIH in 1962. [103]

$$TLV = \frac{250}{\%SiO2 + 5} \text{ mppcf}$$

To make the TLV consistent with a 1970 revision of the TLV for nuisance dusts, the numerator K was raised to 300 and the constant 5 raised to 10 in the denominator. [104]

Prior to the 1970 revision of the count formula, a respirable dust concentration formula utilizing respirable mass measurements of dust was introduced [95]:

$$\text{TLV} = \frac{10}{\%\ \text{respirable free silica} + 2}\ \text{mg/cu m}$$

The formula was based upon the collection of dust by size-selective sampling devices. [82] Such instruments collect a fraction of dust which is capable of penetrating to the gas-exchange portion of the lung where long-term retention of dusts occurs. The concentration of airborne free silica in this size fraction should relate more closely to the degree of health hazard. As with the count formula, a constant was added to the denominator to prevent excessively high respirable dust concentrations when the fraction of free silica in the dust is low. The constant of "2" limits the concentration of respirable dust with less than 1% free silica to 5 mg/cu m.

In addition to quartz, other forms of free silica have been assigned a specific TLV based on experimental or human industrial experience data that indicated a need for individual identification.

Cristobalite (above 5%) was originally listed in 1960 [105] with a TLV of 5 mppcf based on studies in the diatomite industry by Cooper and Cralley [44], Smart and Anderson [48], analogy with the TLV for "silica",

[102] and experimental studies in animals by Wagner et al. [51] In 1968 [95] the TLV was reduced to one-half the value obtained from either the count or mass formula for quartz based upon a review of existing documentation and information to the TLV committee [106] in a personal communication by Smart. This information suggested that the limit of 5 mppcf for cristobalite did not possess a sufficient safety factor for the prevention of pneumoconiosis. Tridymite was likewise assigned one-half the quartz value based upon animal toxicity data developed by King et al [49] in which tridymite was found to be the more active form of free silica studied when its dust was administered by intratracheal injection into the lungs of rats. Analogy was also made with cristobalite.

Although insufficient industrial experience was available to indicate the degree of hazard presented by fused silica dust, the same limit as that required by the quartz formulae was adopted in 1969. [107] Intratracheal injection studies with rats by King et al [49] found fused silica considerably less active than quartz.

Tripoli and silica flour were added to the TLV list in 1972 [108] with the recommendation that the standard for these materials be derived using the respirable mass formula for quartz. Documentation for inclusion of tripoli on the list came from the study of McCord et al [109] who induced tissue proliferation by direct intraperitoneal implantation of tripoli dust in rats and guinea pigs similar to that produced by quartz. Silica flour was included on the list based upon data of King and co-workers [49] and Hatch and Kindsvatter [110] who considered silica flour, because of its fine particle size, to have a significant fibrogenic potential.

The 1968 ACGIH recommended TLVs for quartz have been adopted by the US Department of Labor under the Walsh-Healey Public Contracts Act regulations (41 CFR 50. 204). The TLVs have also been adopted by the US Department of Interior under the Metal and Nonmetallic Health and Safety Act (Sec 6, 80 stat 774; 30 USC 725).

The Federal Coal Mine Health and Safety Act of 1969 (PL 91-173) provides that the Secretary of Health, Education, and Welfare prescribe a formula for determining the applicable standard for coal mines where quartz amounts to more than 5%. Such a formula has been published using 0.1 mg/cu m of respirable quartz as a basis. [30 CFR Part 70.101 published in Federal Register, vol 36, page 4941, dated March 16,1971 and 30 CFR Part 71.100 published in Federal Register, vol 37, page 6368, dated March 28, 1972] The limit thus becomes: mg/cu m respirable dust = 10/% quartz. The 1968 TLV for free silica and the statutory limit for quartz at 5% and 2 mg/cu m of respirable dust are the principal bases for the quartz limit for coal mines. Thus the allowable level of airborne quartz in coal mines is 100 μg/cu m, twice the limit recommended in this criteria document.

Because methods employed by different countries for assessment and reporting of dust concentrations in the workplace vary considerably, comparison of standards recommended by various countries for exposure to dust containing free silica cannot be made with certainty. Examples of standards for free silica adopted by several countries other than the US follow.

The Federal Republic of Germany has adopted a MAK value of 0.15 mg/cu m for quartz, including cristobalite and tridymite. According to Schutz, [111] this standard is based upon a comparison of different local

and foreign standards, results of silicosis statistics, mean dust levels in several industries, and calculations based upon the amount of dust which could be retained in the lungs of exposed workers.

The Swedish values for quartz use a gravimetric determination of the total amount of dust per cubic meter of air. [112] This quantity is related to the free silica content if this mineral exceeds 2.5%. By a formula based on dust quantity and percentage of free silica, a dust index is derived which relates to a given exposure limit. Values exceeding 1.0 represent a silicosis risk. If the free silica is less than 2.5%, a standard of 15 mg/cu m is used. If dust contains large amounts of cristobalite or an unusually great proportion of respirable particles, a lower value is applied.

DeGueldre, [81] in a review of methods adopted by different authorities for assessing the hazard relating to exposures to dusts in mines, included the following standards for France and the USSR on a list of criteria without identifying the basis for their adoption.

France has required a dust index for each workplace since 1956. The index is derived from a formula utilizing the number of dust particles per cu cm below 5 μm, the percentage content by weight of free silica as determined by X-ray diffraction of dust below 5 μm, and a constant dependent upon the sampling and examination methods adopted. The dust index, related to the silicosis risk, determines the frequency of medical examinations. Dust concentrations with an index of 5 or less are considered satisfactory; between 5 and 6, doubtful; and those above 6, dangerous.

The USSR expresses a standard in which "maximum permissible concentrations are as weights of fine dust, probably under 5 microns."

Based upon the percent free silica, the following concentrations are permitted. [81]

Mineral and Organic Dust	Maximum Permissible Concentration mg/cu m
Over 70% crystalline silica	1
10-70% free silica	2
Silicate dust with 10% free silica	4
Other mineral dust with 10% free silica	5
Minerals and mixtures with no silica	6
Coals with more than 10% free silica	2
Coals with less than 10% free silica	4
Coals with no silica	10

The present federal standard for free silica exposure is an 8 hour time-weighted average based upon the 1968 ACGIH TLV formulas of 250/%SiO2 + 5 = mppcf or 10 mg/cu m/%SiO2 + 2 for respirable quartz. One-half this amount has been established as the limit for cristobalite and tridymite. [29 CFR Part 1910.93 published in the Federal Register, volume 39, page 23543, dated June 27, 1974]

Basis for Recommended Environmental Standard

The literature contains many publications on exposures to dusts containing free silica. Unfortunately, data necessary for development and recommendation of a standard to protect the health of workers against the harmful effects of exposure to such a potent pneumoconiosis-producing material are seldom contained in the published reports. [18,47] Epidemiologic studies too frequently have not included environmental data

or such data, if available, have related only to the then present conditions with no correlation with past exposures. In addition, reevaluation studies of a given industry on the state of the worker's health resulting from continued exposure to free silica-bearing dusts have not generally been made. Refinements in the technology of sampling and analysis of dusts and of methods for monitoring biological response will make possible a more precise and valid evaluation of the effects of exposure to dusts which may cause silicosis.

A review of the data from epidemiologic studies of workers in metal mines [25,26] and in foundries [27] reveal that the medical data are reasonably consistent with impinger-count dust concentration data. In these studies the prevalence of silicosis was reduced significantly in the work environments where dust levels were controlled at or below 10 mppcf. Additional data on the effects of exposure of workers at dust levels below 10 mppcf can be found from the studies in the pottery industry, [29] the silica brick industry, [30-32] and the granite industry. [17,33,36-39]

In pottery workers Flinn et al [29] found that at dust levels of less than 8 mppcf there was an increasing prevalence of silicosis with increasing length of exposure at dust levels between 4.0-7.9 mppcf. The prevalence of silicosis ranged from 0.3% among workers exposed for less than 10 years to 85% among workers with over 40 years of exposure at this level (see Table III-2). Two cases of early silicosis were observed in 798 workers exposed for 10-29 years at less than 4 mppcf. However, these cases could have received higher exposure at some previous work period and thus one cannot say with certainty that they occurred as a result of the lower exposure.

While the study of Fulton and co-workers [32] of the silica brick industry showed a significant prevalence of silicosis of 52% in 1,035 employees examined, interpretation of these data must take into account the absence of free silica dust exposure data for several years preceding the survey. Such information is considered essential for an accurate evaluation of the reported health effects due to the inhaled dust. This is particularly true in light of the 42% prevalence of silicosis found in workers exposed at an average concentration of 0-9.9 mppcf as determined at the time of the 1939 study. In all probability dust exposures significantly higher than 10 mppcf were experienced in previous years by the workers in these same operations and could have been responsible in part for the recognized cases of silicosis. The reported 22% (14 of 65) incidence of silicosis in workers examined where average dust exposures were 2-3.9 mppcf would tend to support the conclusion that higher dust exposures contributed to the prevalence of silicosis. Review of the literature on silicosis revealed no other report with unequivocal diagnosis of silicosis based on dust exposures at levels between 2-4 mppcf.

The study of Fulton et al suggests that cristobalite and tridymite have a capacity greater than that of quartz to induce silicosis. An average length of exposure of 17.9 years was required to produce stage 2 silicosis among the green-brick workers whose exposure was restricted to that form of dust containing 88% quartz. Men in the burned-brick department were found to have stage 3 silicosis after an exposure of like duration to burned-brick dust of 80% cristobalite and tridymite. Average dust concentrations were essentially the same, 15.9 and 16.9 mppcf, respectively. Animal studies confirm the greater activity of cristobalite

and tridymite. [49,53] Interpretation of available data from the silica brick study [32] suggests an exposure limit for free silica dust below 10 mppcf. However, the enhanced biological response resulting from exposure to the mixed cristobalite-tridymite dust would suggest a limit closer to 5 mppcf as being more appropriate for an exposure level at which silicosis should not occur. However, a greater toxicity of cristobalite and tridymite than of quartz, if expressed as respirable mass rather than as particle count, does not necessarily follow from the epidemiologic studies based on impinger count.

Surveys of the diatomite industry in California [44-46] associate a 9% incidence of silicosis in workers exposed to dusts from calcined diatomite containing up to 61% cristobalite in the parent material and up to 32% in airborne dust. Reduction of dust concentrations to a point where more than 84% of the samples counted were routinely below 5 mppcf reduced the incidence of silicosis to zero in workers whose employment began after dust control measures were instituted. The 5 mppcf level was suggested as the maximum exposure concentration for the industry. [44] Subsequent studies, [45,46] the most recent being conducted 16 years after the initial survey, appear to confirm the validity of the 5 mppcf level for dusts containing cristobalite. Again, no evidence of silicosis was reported for individuals employed since initiation of the dust control program.

It is from the Vermont granite industry that the most extensive and complete environmental and medical data are available for establishing a recommended environmental limit for exposure to free silica. These data have been accumulated over approximately a 50-year period extending from the 1924 study of Russell et al [17] to that of Theriault and co-workers

[37-39] in 1969-72. With the exception of the reports by Theriault et al, all occupational environment dust exposures were determined by microscopic counts of impinger-collected dust samples. The derived air dust concentrations in mppcf and the associated health effects provide the major portion of the material used as the bases for the present exposure limits (TLV's) for quartz and other free silica polymorphs [95,104,107,108] in addition to the 10 mppcf granite dust exposure limit in use in that industry since 1937. [33-35]

The studies of Theriault and co-workers [37-39] of Vermont granite workers are important from several standpoints, among them their use of size-selective respirable mass sampling, coupled with gravimetric determinations of dust concentrations, instead of impinger counts, as in past studies.

These investigators [38-39] also based interpretations of granite dust toxicity on pulmonary function tests as well as on X-ray evidence. While the observed average decrement in function may not have clinical significance, it appears to presage radiographically evident changes, and thus could be a more sensitive index of effects in a group of workers, whether or not it would be sufficiently sensitive for diagnosis of disease in an individual. Yet, these studies do not demonstrate a safe concentration of silica. The authors found that 50% of the workers had radiographic evidence of silicosis at 46 dust-years (ie 46 years of exposure at a dust level equivalent to about 50 μg/cu m of free silica) and functional evidence at over 32 dust-years. But the curves drawn to fit their data suggest a significant incidence of silicosis at 0 dust-years. [39] Based on a plot of radiographically evident silicosis against dust-

years, 30% of the working population had silicosis with no exposure. Of course, this undoubtedly represents imperfections in their data or in available methods of analysis of their data, but it is not evident how to use inferences from these analyses in deriving an environmental limit. One can speculate that their environmental samples were unrepresentative of the years of exposure preceding the sampling, which surely contributed to the development of silicosis; the authors attempted to make some estimate of the extent to which past exposures might have been higher than indicated by their more recent atmospheric sampling and, though their estimates seem reasonable, they could have been in error. Another curious observation was that the silica content of the dust sampled (9%) was much lower than previous analyses had demonstrated; they suggested that this was due to process changes which caused a dilution of the silica content of the total dust. It is believed important that these studies be confirmed in the granite sheds and be extended to other operations producing airborne silica, as a likely prerequisite to further refinement of an occupational health standard for free silica.

Russell et al [17] studied 972 granite shed workers, dividing them into 4 exposure groups according to average dustiness: 37-60, 27-44, 20, and 3-9 mppcf. The group with the highest dust exposure showed the unmistakable indication of the seriousness of the hazard of exposure to granite dust by development of early silicosis in 40% of workers after two years and 100% after 4 years of exposure. The development of silicosis in the remaining groups appeared to be proportional to the dust exposure. An experience similar to the highest exposure occurred at the second highest

exposure (27-44 mppcf) where early stages of silicosis appeared after 4 years of exposure and more advanced stages developed by the 7th year.

In the group exposed at an average of 20 mppcf there was little indication of severe effects upon the health of the workers. However, the authors concluded that one would hesitate to state positively that no harm would come to persons exposed for many years to a concentration under 20 mppcf. In the case of the lowest exposure group where the average dust concentration was 6 mppcf (range 3-9 mppcf), there was no indication of any untoward effects of dust exposure on workers.

From the above, the authors [17] interpreted that average dust exposure for the 2 highest exposure groups was clearly harmful to workers. They concluded that, even though harmful effects were found, a safe limit of dust exposure apparently lies somewhere between 10 and 20 mppcf.

In a restudy of the granite workers [33] Russell revised his original estimate [17] of 10-20 mppcf as the desirable limit for granite dust exposures. Rather than basing his new recommendation upon data from the 20 mppcf average granite dust exposure group, which still carried "some question as to the harmful effect" of the dust exposures at that average concentration, Russell apparently used the progression of silicosis from the milder forms to the more severe forms; he also used the further complication of tuberculosis in the highest average dust exposure group (27-44 mppcf) as the basis for his new limit of about 10 mppcf.

Following this study and enforcement of the 10-mppcf limit by the Vermont Department of Health, dust control progressed in the granite sheds so that by the time of the study by Hosey et al [34] few exposures in the granite sheds studied exceeded 5 mppcf. The effectiveness of the control

measures was evidenced by the absence of new cases of silicosis, with the exception of one doubtful case, in men starting work in the granite sheds after 1937. Furthermore, chest roentgenographic surveys of granite workers showed a reduction in the prevalence of silicosis from 45% in 1937 to 15% in 1956.

Confirmation of the safety of the limit of 5 mppcf was reported by Ashe and Bergstrom [35] in 1964. Their study, 26 years after dust control began, likewise found no cases of silicosis in workers employed after the start of dust control. Environmental data also indicated a probable greater margin of safety for dust exposures at the time of the study; average concentrations were 3 mppcf.

Based on the impinger-count dust concentration data and the reported absence of identifiable dose-response effects, the granite shed studies [33-35] indicate that a limit of 5 mppcf has, up to this time, been an effective control for the prevention of silicosis in men exposed to granite dust of 25-35% quartz.

As had most of the investigators before them, Hosey et al [34] and Ashe and Bergstrom [35] concluded that careful surveillance of the work environment and of the worker's health was needed to determine the ultimate efficacy of dust control in the granite sheds industry.

From the above studies in Vermont granite sheds, a safe level for silica can be interpreted as 5 mppcf. Because of variations in types, size, and density of particles in other industries, it is not clear that the same limit, in terms of number of particles, will properly describe safe exposures in these other industries producing airborne free silica. But on the basis that 5 mppcf is equivalent, in Vermont granite sheds, to

50 μg/cu m, (see discussion below) it seems appropriate to apply this limit, in terms of respirable mass, to other operations producing dusts containing free silica. Thus, an environmental limit of 50 μg of respirable free silica/cu m is recommended.

Reno et al [113] and Sutton and Reno [114] compared impinger-count measurements with size-selective mass concentrations from granite shed worker environments in an attempt to establish a relationship between these methods of sampling free silica. They concluded that 10 mppcf of granite dust (containing approximately 25-35% free silica) was equivalent to 100 μg/cu m of free silica. Their work has been reviewed and evaluated by Ayer and his associates. [78,79] On the basis of their review, Ayer et al supported the conclusion of Reno and Sutton and their collaborators, ie, that 10 mppcf of total granite dust is approximately equivalent to 100 μg/cu m of respirable free silica. Theriault and associates [37] came to a slightly different conclusion, viz, that 10 mppcf is equivalent to about 80 μg/cu m, but they presented no data or argument supporting the conclusion. Thus, a safe level of silica of 5 mppcf for the granite workers indicates a level of 50 μg/cu m in terms of respirable free silica.

A review of data from other industries [26,27,29,32,44] does not reveal any significant difference in the degree of toxicity of free silica in the form of quartz to which workers are exposed as compared with that in the granite industry. They do reveal, however, that the 10 mppcf standard has not been entirely adequate for protection of workers in those industries, a condition which has been suggested by the data from the granite industry. Consequently, the recommended standard of 50 μg/cu m is considered applicable to all work environments where exposure to the quartz

form of free silica may occur. It is recommended that the studies in the granite industry be confirmed and that similar studies be undertaken in other industries to determine more precisely the significance of exposure to free silica in those industries so that alternate recommendations can be made should they be indicated.

The epidemiologic studies of Fulton et al [32] and Cooper and Cralley [44] have suggested that cristobalite and tridymite are more active than quartz in producing fibrotic change in lung tissue. King et al [49] and Gardner [53] have confirmed this in animal studies. In addition, experimental evidence indicates that microcrystalline free silica, because of its extremely fine particle size, may have a greater potential for inducing fibrotic change. [56,110] Because of these factors, it has been recommended [95] that a standard for these forms of free silica be one-half that recommended for quartz. Regrettably, there are no studies which relate mass respirable quantities of cristobalite, tridymite, or microcrystalline free silica to a prevalence of silicosis in an exposed population. However, the epidemiologic studies cited [32,44] above and follow-up studies in the diatomite processing industry [45,46] have indicated that if exposure levels of cristobalite and tridymite are kept below 5 mppcf no cases of silicosis are likely to develop in an exposed worker population. Similar data for microcrystalline free silica are lacking.

While the respirable mass concentration of 50 μg/cu m cannot be shown at this time to be equivalent to the 5-mppcf particle count concentration in operations other than granite work, it is believed that a free silica concentration of 50 μg/cu m in air is sufficiently low to

protect workers exposed to cristobalite, tridymite, or microcrystalline free silica against the development of silicosis, thus no separate standard for these forms of free silica is recommended at this time. Further research is needed to validate inferences about the safety of the 50 μg/cu m limit for other forms of free silica; meanwhile, it is recommended that the limit of 50 μg/cu m should apply to any form of free silica.

Despite the questions raised above about the studies of Theriault and associates, [37-39] their approach seems clearly superior to that of past studies. Respirable mass sampling of worker populations should give a lower variance in results and should show more clearly whether other dusts may potentiate or antagonize silica toxicity (Theriault et al suggested that other components of granite dust slightly increased the toxicity of silica). Perhaps more importantly, correlation of effects judged by X-ray evidence and by pulmonary function tests would be expected to demonstrate the superiority of pulmonary function tests as an early indicator of silicosis (and for this reason, tests of pulmonary function are recommended for routine medical monitoring of silica workers in this recommended standard), and such testing should be included in the design of further research.

It is recognized that many workers are exposed to small amounts of free silica or are working in situations where, regardless of amounts used, there is only negligible contact with the material. Under these conditions it would not be necessary to comply with many of the provisions of this recommended standard, which has been prepared primarily to protect workers' health under more hazardous circumstances. Concern for workers' health requires that protective measures be instituted below the enforceable limit

to ensure that exposures stay below that limit. For these reasons "exposure to free silica" has been defined as exposure above half of the environmental limit, thereby delineating those work situations which do not require the expenditure of health resources for environmental and medical monitoring and associated recordkeeping. This level has been chosen on the basis of professional judgment rather than on quantitative data that delineate nonhazardous areas from areas in which a hazard may exist.

The length of time necessary for silicosis to develop when workers are exposed to relatively low levels of free silica makes it necessary to retain medical and environmental records for extended periods of time for effective evaluation of control measures. The time of retention of these records as they relate to workers exposed to free silica should be at least 30 years following termination of employment.

Subsequent to the completion, review, and approval of this document, a summary of new information was furnished NIOSH (personal communication, H. Ohman, Vasteras, Sweden, September 1974). At exposure levels, expressed in increments of 10 μg/cu m, from 10 to 280 μg/cu m of respirable free silica, none of the groups exposed at concentrations up to 50 μg/cu m had radiographic evidence of silicosis, but at all higher levels there was at least one case of silicosis, the percentage affected increasing with concentration level. Exposures were calculated on the basis of 40-year exposures as constituting a working lifetime. If review and analysis of the data and methods, information not now available, supports the inferences based on the summary, the study would offer additional evidence for the environmental limit of 50 μg/cu m, in this case from foundry operations.

Basis for Recommended Sampling Method

"Impinger sampling combined with its microscopic counting method has served well in the past as a tool in reducing exposures to dusts which give rise to pneumoconiosis". [115] However, in spite of its success as a monitoring method, the impinger is deficient in most of the factors which are desirable for evaluating a dust standard. [79] Results obtained with the impinger are not closely related to the health hazard when dust is in the form of agglomerates, as is the case of most redispersed dust. Many dust particles are sufficiently large so that when they are inhaled they are removed by the upper respiratory tract. Thus they never reach the pulmonary spaces where tissue change can occur, yet they are collected and counted by the impinger method and are considered in the total count. Where virtually all dust is in the form of discrete, respirable-size particles, the counts may be very much lower, even though the hazard is far greater than for a dust of greater size. [79] In addition, careful training of dust counters is required before their counts approach the average value of experienced counters. The cost of the determination of an average exposure is high. Any one impinger sample usually measures only 10-30 minutes of exposure and at least 5 samples are required to determine an exposure with any degree of confidence. [82] It is evident that the impinger method falls short of the ideal with regard to relevance to health hazard, simplicity, reproducibility, and unit cost. [79]

The Johannesburg Conference on Pneumoconiosis of 1959 [116] recommended that "measurements of dust in pneumoconioses studies should relate to the 'respirable fraction' of the dust cloud...". During its 1968

annual meeting, the ACGIH accepted a report of the TLV Committee which recommended adoption of a quartz TLV for respirable dust in mg/cu m. [106]

The use of size-selective sampling of respirable dusts as a means of evaluating inhalation hazards has been reviewed by Hatch and Gross, [84] by Morrow, [83] the AIHA-ACGIH Aerosol Technology Committee, [82,117] and Ayer et al, [78] and all have well stated the advantages to be gained by using this method of sampling. These include: the ability to sample over a full shift or a major fraction thereof; automatic compensation by the size-selective device for shape, density and degree of agglomeration of dust; the ability to use personal samplers and obtain truer "breathing zone" exposure; the greater possibility for standarizing analytical determinations (as contrasted with optical count); and a much lower cost per determination of weighted exposure to dust. The samples can also be used for determination of free silica content, weight concentration, and particle size distribution.

The size-selective, respirable mass collection of dust provides a method and data that can be more closely related to the health hazard associated with the inhalation of free silica particles. The method is simple, reproducible, and relatively inexpensive. The size-selective mass method separates out the large dust particles by an inertial or gravitational method, allowing only those sizes of dust to pass which are capable of penetrating to the pulmonary, nonciliated portion of the lung. The method is ideally suited for collection of the essentially insoluble free silica dusts which exert their damaging effect in the pulmonary area [79] and is the method recommended for collection of dust samples for

evaluation of a silicosis hazard. Detailed procedures for application of the method are given in Appendix I.

Basis for Recommended Analytical Method

Three principal methods are currently used for the qualitative and/or quantitative determination of free silica in workplace dusts. These analytical methods are: the colorimetric chemical procedure, infrared spectrophotometry, and X-ray diffraction.

At present the colorimetric procedure [118-121] is the method most universally used. However, there are two serious drawbacks to this wet chemical method which prevent its recommendation as the method of choice: (1) the analytical results are highly operator-dependent, requiring extreme adherence to a timed, precise protocol due to color instability; (2) the method does not distinguish between the free silica polymorphs--quartz, cristobalite, and tridymite--which at present have different Federal standards for permissible airborne concentrations. [29 CFR Part 1910.93 published in the Federal Register, volume 37, page 22139, dated October 18, 1972]

The infrared procedure [40,122-125] is a relatively new analytical method for free silica which has the potential for the qualitative identification of the free silica polymorphs. [126] The method has been routinely applied for the determination of quartz only. [122,127] Another drawback is the dependence of the analytical results on particle size. [123,125,128] Samples having an average particle size greater than 2 μm have a reduced absorbance at the analytical bands of 12.5 and 12.8 μm. [129]

The X-ray diffraction procedure, [130-132] on the other hand, is specific for the various forms of free silica, [133] including the microcrystalline variants. [134] The method is sensitive, detecting as little as 25 μg of quartz on a silver membrane filter. [135] Moreover, this procedure requires less sample preparation than either the infrared or the colorimetric procedures. [136]

Comparative studies by NIOSH of the three analytical methods utilizing field samples of respirable Georgia granite dust indicate that all three give equivalent percentages of free silica on field samples. The results of analyses of 45 side-by-side granite shed dust samples, [137] collected on three different days, are presented in Table V-1.

TABLE V-1

PERCENTAGE OF FREE SILICA RECOVERED FROM GEORGIA GRANITE DUST BY THREE DIFFERENT ANALYTICAL METHODS

Analytical Method	Number of samples	Mean % free silica	% Deviation from overall mean
Colorimetric	18	23.6	+0.9
Infrared (512 cm^{-1})	12	24.5	+5.0
X-ray Diffraction	15	22.2	-5.0
	45	23.4 (overall mean)	

None of the methods described above are ideal for analysis of dust samples for free silica under all conditions [137]; but, because of its sensitivity, speed, minimum sample preparation time, ability to identify the polymorphs of free silica, and capabilities for automation, the X-ray diffraction method is recommended as the method of choice for the quantitative and qualitative analysis of dust containing free crystalline silica. Detailed procedures for application of the method are given in Appendix II. When conditions warrant, the colorimetric or infrared spectrophotometry methods may be used. Such conditions may occur: (1) when interfering materials in the sample will decrease the sensitivity of the X-ray diffraction method by blocking the primary diffraction peaks; (2) when more than 1 polymorph of free silica is present which would interfere with the accuracy of the results obtained; (3) when the quantity of the total sample is small or when cristobalite, tridymite, or other polymorphs of free silica are a significant fraction of the sample. The experience of the laboratory performing the analysis and their knowledge of conditions under which the samples being analyzed are collected will, in a large measure, determine which alternate method should be used.

When infrared or colorimetric analytical methods are used, the procedure for these methods [138,139] as given in the NIOSH Manual of Analytical Methods should be followed.

These methods will provide as accurate a means for qualitative and quantitative analysis of free silica in collected respirable dust samples as is presently available.

VI. WORK PRACTICES

(a) Substitution: The most certain and direct method of eliminating the silicosis hazard is to substitute other less toxic material for free silica. [140] In abrasive blasting, where silica sand can be replaced with an abrasive containing less than 1% free silica, such substitution should be considered. A number of such materials are available. These include slag products, metallic shot and grit, garnet, nut shells, cereal husks, and sawdust. [99,100]

(b) Dust suppression: The use of water to allay or prevent formation of dust is as old as the history of industry. Where sand can be used wet, little or no dust is generated. In many sand and mineral handling operations, a moisture content can be determined that will substantially reduce dust while not interfering with the process. The use of water sprays may improve dust conditions considerably [26,140,141]; however, sprays may have a limited use in reducing respirable dust to acceptable levels. [141] Similarly, wetting down piles of dust is helpful as long as the moisture content remains high, but water often does not penetrate far enough into the pile. [141] Wet drilling controls the greatest part of the dust, but enough fine, free silica particulate may be generated so that supplementary control is required. [26,140-142] Thus, although the use of water is encouraged and may be sufficient to solve certain dust problems, it does not necessarily provide a complete solution.

(c) Ventilation: In spite of the wide distribution and acceptance of the Industrial Ventilation manual [97] and the holding of many conferences and training programs, many ventilation systems are still

designed without reference to accepted principles and procedures. This may stem in part from the fact that engineering principles of contamination control are not generally a part of engineering curriculum. Furthermore, even when systems are properly designed, the installation, adjustment, or maintenance may be unsatisfactory.

Visual inspection to ensure that exhaust hoods are in place, intact, open, and connected to the ventilation systems would identify many problems. Plant managers should schedule visual inspection for physical integrity and also mechanical checks to ensure that needed repairs are performed promptly. [97]

In many operations the source of dust is not fixed but depends upon the work being performed. For such dust sources, a movable dust hood often provides the best solution. Such hoods usually require proper placement to make them effective, thus the workers can control their effectiveness. Consequently, they should be so designed to ensure that proper use is not difficult. Improper use of movable hoods generally increases the hazard to the worker and to those around him, therefore, conscientious use of such hoods is required.

(d) Wet drilling and dust collection: Drilling of free silica-bearing rock is a common source of dust which frequently causes silicosis. [16,24-26] Both wet drills and drills with attached dust collectors are available. Even with wet drills and dust collectors, dust concentrations should be monitored and ventilation may be needed. [140] Wet drilling with surface-active agents has improved this method of dust control. Dry drilling without controls should be prohibited.

(e) Labeling and apprisal of hazard: In many dust exposures involving a potential for silicosis, labeling may not be appropriate. The free silica-containing dust is generated from rock, sand, gravel, clay, or other minerals. Shipment of hazardous materials, however, may require labeling. Carloads and bags of silica flour are shipped but users may not be aware of the hazard involved with this material. Likewise, flux-calcined diatomaceous earth contains substantial percentages of cristobalite and tridymite and can generate very hazardous dust. Labels for such materials should be provided.

(f) Protective equipment and clothing: Certain operations, such as abrasive blasting in confined spaces, can be performed safely only with respiratory protection. [99,100] Other dusty operations, in maintenance and repair for instance, are carried out intermittently where exhaust ventilation is not feasible. These operations may also require protective respirators.

For extreme hazards, such as in abrasive blasting, the employer must not only supply suitable respirators but must require their conscientious use. For respirators to provide effective protection, the employee must be trained in their use; further, they must fit properly and be properly maintained. [99,100]

Protective clothing has not been widely used for health protection against free silica dust. Experiments in British potteries have demonstrated, however, that dirty work clothes can be a significant source of dust in that industry. [98] Investigators found that cotton and wool clothing tend to retain dust, and that these materials are less satisfactory than others for health protection. They cited also the

importance of design of work clothing; for example, pleats and other design features can cause significant dust retention. Test procedures for evaluating dust retention of clothing have been developed by the British Ceramic Research Association. It is suggested these procedures be used until better ones have been demonstrated. [98]

(g) Dust monitoring: In order to determine the extent of the potential silicosis problem, the need for periodic monitoring of the operations where exposure to free silica is suspected is required. Schedules for the monitoring of respirable free silica dust concentrations are given in Chapter I, Section 7, of this document.

Construction, agriculture, and service occupations have not been regularly investigated for free silica dust hazards. Although results of the few investigations to date have shown that environmental levels are in excess of current standards, they tend to be negative for silicosis. [143,144] Great changes that have occurred in these industries warrant exploratory and epidemiologic studies to more clearly define the silicosis hazard and the development of methods for control of dust exposure.

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VIII. APPENDIX I

AIR SAMPLING METHOD FOR FREE SILICA

General Requirements

To evaluate the worker's exposure, environmental sampling for free silica dust requires the application of respirable dust sampling techniques such as those presented in the ACGIH Air Sampling Instruments [145] and those recommended by the AIHA-ACGIH Aerosol Technology Committee. [82] The dust is collected with a size-selective personal sampler positioned in the worker's breathing zone. Dust penetrating the precollector is collected on a low ashing polyvinyl chloride (PVC) filter and the free silica content is determined by X-ray diffraction (Appendix II) after redeposition of the dust on a silver membrane filter.

(a) Samples collected shall be representative of the individual worker's exposure.

(b) Sampling data sheets shall include:

(1) The date and time of sample collection.

(2) Sampling duration.

(3) Volumetric flowrate of sampling.

(4) The name of the worker being sampled or the description of the sampling location.

(5) Sampler serial number.

(6) Name of person taking sample.

Personal Sampling

(a) Size-selective device

Personal samples shall be collected using a two-stage, 10-mm nylon cyclone size-selective sampler.

(b) Filters and filter holders

The size-selective sampler shall be connected to a 2-piece, 37-mm cassette containing as the collecting medium a 37-mm, low-ashing polyvinyl chloride (PVC) filter with a 5.0-μm pore size. (Gelman VM -1 filters are unacceptable for use in the analytical method recommended because of the high background reading produced by the filter upon ashing.) Duplicate filters shall be subjected to identical handling, but without air being drawn through them, so that they may serve as blanks or controls.

(c) Personal sampling pumps

A portable battery operated pump, conforming to the requirements of 30 CFR-47, which is equipped with a pulsation dampener and which will draw 1.7 liters of air per minute (liters/minute) for at least 8 hours shall be used. Present technology of personal sampling pump batteries limits the sample collection time to approximately 8 hours.

(d) Sampling time

The sampling time shall be for a full work shift and the exposure calculated for up to a continuous daily 10-hour exposure. Multiple, shorter (2-4 hour) samples may be collected over a full shift and pooled for determination of an individual's average exposure to free silica during the total work shift.

Workroom (General) Air Sampling

(a) Size-selective sampling devices

When high volume respirable dust samples are required from the area of the worker's environment, a 1/2-inch metal cyclone or its equivalent shall be used to obtain samples.

(b) Filters and filter holders

The size-selective sampler is connected to the filter holder cassette containing the appropriate filter. Samples collected for chemical analysis must be collected on a 37-mm low-ashing polyvinyl chloride (PVC) filter with a 5.0-μm pore size.

(c) Sampling pumps

Any nonpulsating flow pump with a capacity of at least 10-15 in. Hg at 9 liters/minute will be adequate if the 1/2-inch cyclone is used. For larger samplers a pump of greater capacity will be required.

(d) Sampling time

The general air samples should be collected for a period of 4 consecutive hours taken during collection of the personal samples.

Collection and Shipping of Samples

(a) The quantity of dust collected in the filter holder cassette assembly should not exceed 5 mg. The filter is kept in the cassette for shipment.

(b) Whenever an air sample or series of air samples is collected, a bulk sample of the suspected parent material of the atmospheric contaminant should be obtained and shipped back to the laboratory with the air samples.

(c) After sampling, remove the filter cassette from the sampling train. Stopper the end of the cassette. Ship the cassette to the analytical laboratory in a suitable container to prevent damage in transit.

Sampling Methods for Quantitative and Qualitative Analysis of Free Silica

There are several difficulties, especially from small sample size, in analyzing for the percent and/or form of free silica on each personal sample, so the following methods of sampling are recommended to provide an appropriate sample size for such determinations: higher volume respirable dust samples as collected with a 1/2-inch cyclone; groups of pooled personal samples; any other method shown to be equivalent in collection of a suitable size sample.

A minimum total sample of 500 μg of dust or a minimum free silica dust sample of 25 μg is needed for accurate analysis of the collected dust.

Calibration of Sampling Trains

Since the accuracy of an analysis is no greater than the accuracy of the volume of air which is measured, accurate calibration of a sampling pump is essential for correct interpretation of the pump's indicated volume. The frequency of calibration is dependent on the use, care, and handling to which the pump is subjected. In addition, pumps should be recalibrated if they have been misused or if they have just been repaired or received from a manufacturer. If the pump receives hard usage, more frequent calibration may be necessary.

Ordinarily, pumps should be calibrated in the laboratory both before they are used in the field and after they have been used to collect a large number of field samples. The accuracy of calibration is dependent on the

type of instrument used as a reference. The choice of calibration instrument will depend largely on where the calibration is to be performed. For laboratory testing, primary standards such as a spirometer or a soapbubble meter are recommended, although other standard calibrating instruments such as a wet-test or a dry-gas meter can be used. The actual setup will be the same for these instruments.

Instructions for calibration with the soapbubble meter follow. If another calibration device is used, equivalent procedures should be followed.

The calibration setup for a personal sampling pump with a filter cassette is shown in Figure XI-1. The procedure is described below.

(a) Check the pump battery with a voltmeter to assure adequate voltage for calibration. Charge the battery if necessary.

(b) Place the PVC filter in the filter cassette.

(c) Assemble the sampling train as shown in Figure XI-1.

(d) Turn the pump on and moisten the inside of the soapbubble meter by immersing the buret in the soap solution and draw bubbles up the inside of the buret until they are able to travel the entire length of the buret without bursting.

(e) Adjust the pump rotameter to provide a flowrate of 1 liter/minute.

(f) Check the water manometer to insure that the pressure drop across the sampling train does not exceed 13 inches of water (1 in. of Hg).

(g) Start a soapbubble up the buret and, with a stopwatch, measure the time it takes the bubble to move from one calibration mark to another. For a 1000-ml buret, a convenient calibration volume is 500 ml.

(h) Repeat the procedure in (g) above at least twice, average the results, and calculate the flowrate by dividing the volume between the preselected marks by the time required for the soapbubble to traverse the distance.

(i) Data for the calibration include the volume measured, elapsed time, pressure drop, air temperature, atmospheric pressure, serial number of the pump, pump number, date of the calibration, and name of the operator.

IX. APPENDIX II

ANALYTICAL METHOD FOR DETERMINATION OF FREE SILICA IN ATMOSPHERIC DUST

The following method for determination of the polymorphs and/or free silica content in airborne dusts employs X-ray diffraction as recommended in the NIOSH Manual of Analytical Methods. [146] This method is based upon the work of Bumsted, [135] Leroux and Powers, [130] Talvitie and Brewer, [147] Bradley, [148] the Handbook of X-rays, [149] and Leroux et al. [150]

(a) Principle of the Method

(1) Atmospheric dust samples are collected on low-ashing PVC membrane filters.

(2) The filters are ashed and the residues along with an internal calibration standard are redistributed onto silver-membrane filters.

(3) Each sample is scanned by X-ray diffraction to determine the polymorphs of free silica that may be present.

(4) If present, the mass of each polymorph is determined by measuring the ratio of the diffraction peak intensities of the free silica polymorph and the internal standard, and comparing this ratio to a calibration curve.

(b) Range and Sensitivity

(1) The analytical range extends from 5 μg/sq cm to 200 μg/sq cm for each free silica polymorph; the total atmospheric dust loading on the filter must not exceed 1 mg/sq cm.

(2) The sensitivity is 5 μg for each free silica polymorph.

(3) A minimum total dust sample of 500 μg or a minimum free silica dust sample of 25 μg is needed for accurate analysis of collected dusts.

(c) Interferences

(1) Several minerals have diffraction peaks that correspond in position to the major peak for quartz; these include micas (biotite, muskovite, potash, feldspars [microcline], plagioclase), sillimanite, graphite, iron carbide, and zirconium silicate. The presence of these interferences is usually encountered in specific, recognizable situations and can be seen in the X-ray diffraction pattern. Analytical measurements can be carried out at a secondary quartz peak with a commensurate decrease in sensitivity.

(2) Comparable interference may occur from other free silica polymorphs such as cristobalite and tridymite. This can be determined by X-ray diffraction analysis. If interference occurs, secondary peaks must be used.

(3) Diffraction peak interference may also occur for the fluorite (CaF2) internal calibration standard. Compensation must be made for increased peak intensity from the interfering minerals, or an alternate standard must be employed.

(4) In certain cases, some elements (iron and iron compounds) present in the sample may give rise to appreciable X-ray fluorescence leading to high background intensity. This situation may be circumvented by using a diffracted beam monochromator or by utilizing an alternate X-ray tube target material.

(d) Precision and Accuracy

(1) Leroux et al [150] stated that dust deposits may be analyzed with a precision of better than ±3%.

(2) Bumsted [135] reported 21 replicate measurements of quartz in 2 mg of coal dust as having an average of 0.63% (12.6 μg), a range of 0.51-0.91% (10.2-18.2μg) and a standard deviation of 0.114 (± 1.4 μg); he reported an accuracy of ± 30 % (± 3.8 μg) of the quartz present in these samples. No information is available regarding the accuracy for cristobalite or tridymite.

(e) Advantages and Disadvantages

(1) The X-ray diffraction method offers sensitivity equivalent to or greater than other methods (infrared or colorimetric), is nondestructive to the sample and is rapidly performed.

(2) The X-ray diffraction method is limited to a sample size of a few milligrams. Application of the method requires a rather high degree of technical skill and expensive equipment.

(f) Apparatus

(1) X-ray diffraction equipment, including copper and/or molybdenum taget X-ray tubes. (Gelman VM-1 filters are unacceptable for use with X-ray diffraction because of the high background reading produced by the filter upon ashing.)

(2) Low temperature radiofrequency asher or muffle furnace.

(3) Ultrasonic bath.

(4) Filtration apparatus.

(5) 25-mm diameter silver membrane filters having a 0.45 μm pore size.

(6) Aluminum weighing pans.

(7) Porcelain crucibles with covers.

(8) 100-ml Pyrex beakers.

(9) Glass microscope slides.

(10) Nonserrated, nonmagnetic forceps.

(11) Metal spatula.

(g) Reagents

(1) Quartz, cristobalite, and tridymite powders and other free silica polymorphs as needed: Acid washed and wet-sieved through 10 μm sieves.

(2) Fluorite (CaF2): 400 mesh powder, analytical grade.

(3) Wetting agent.

(4) Petroleum jelly.

(h) Procedure-Cleaning of Equipment

It is important that all equipment be kept as free of contaminant dust as possible.

(1) The spatula, forceps, etc may be satisfactorily cleaned by using ethyl alcohol and disposable nonlinting tissues.

(2) The aluminum weighing pans are cleaned by rinsing them twice with distilled water and twice with ethyl alcohol, and allowing them to dry in a dust-free environment.

(i) Analysis of samples

(1) Either of the following described methods may be used to ash the sample:

(A) With forceps and spatula, place the filter sample in an aluminum weighing pan and situate within the sample compartment of the low-temperature asher so that the sample exposure to the radiofrequency-excited oxygen plasma is optimized. The sample is ashed for

1 hour at 100 watts RF power and at an oxygen flow rate of 75 cc/min, using the techniques recommended in the instrument manual.

(B) Using forceps and spatula, put the filter sample in a porcelain crucible, cover loosely and place in muffle furnace. Maintain for 2 hours at 600 C (800 C if graphite is present in the sample).

(2) Carefully scrape the ash residue into a 100-ml beaker. Rinse the weighing pan (or crucible) several times with about 5 ml of water and pour the rinse water into the beaker. Add 1 ml of the fluorite standard solution, a few drops of wetting agent, and distilled water to bring volume up to 50 ml.

(3) Ultrasonically agitate the beaker and its contents for 30 minutes at maximum setting.

(4) Filter solution, under suction, through a 25-mm diameter, 0.45-μm pore size silver-membrane filter. Thoroughly wash down the filter holder with distilled water to ensure that all dust particles have been transferred to the filter.

(5) Remove the filter with forceps, place in a Petri dish, and dry at 105 C for 15 minutes.

(6) The silver filter is then attached to a glass microscope slide with petroleum jelly and inserted into the X-ray diffractometer. A portion of the filter should be inserted beneath the clamping surface of the diffractometer.

(7) The diffractometer is then scanned over the 2 Theta-range corresponding to d = 4.5 to 2.8 Angstroms (for a copper tube, 2 Theta = 18-32 degrees and for a molybdenum tube, 2 Theta = 9-15 degrees). The

presence of crystalline forms of silica are determined by the occurrence of diffraction peaks as follows:

Mineral	d (Most Intense)	d (Second Most Intense)
Quartz	3.34 Angstroms	4.26 Angstroms
Cristobalite	4.05 "	2.49 "
Tridymite	4.07 "	3.80 "
Fluorite Standard	3.15 "	----

The presence of interfering compounds can be determined by the presence and identification of other X-ray diffraction peaks.

(8) The intensity of the most intense diffraction peak for quartz, cristobalite, and tridymite, or other free silica polymorphs is determined by measuring peak height or peak area from the diffraction scan or by scaler (fixed time or fixed count) measurement at peak position. All measurements must be corrected for background. Comparable measurements are made for the fluorite standard at d = 3.15 Angstroms. If diffraction peaks from other compounds interfere with the most intense peak for quartz, cristobalite, tridymite, or other free silica polymorphs, the second most intense peak for these free silicas must be employed.

(9) The free silica to calcite intensity ratios are determined, and the mass of quartz, cristobalite, tridymite, and/or other polymorphs of free silica is determined from the appropriate calibration curve.

(j) Calibration and Standard

(1) Standards

(A) A standard solution of fluorite is prepared by adding 20 mg of fluorite to 100 ml of distilled water containing a few drops of wetting agent and then agitating.

(B) Known amounts of quartz, cristobalite, tridymite, and/or other polymorphs of free silica are weighed to the nearest 0.1 mg and are added to 100 ml distilled water containing a few drops of wetting agent to provide five standard solutions of each mineral covering a concentration range of 0.01 - 0.3 mg/ml.

(2) Standard curve

(A) One ml of the standard fluorite solution and one of the free silica solutions are added to 50 ml distilled water and are analyzed according to steps (i) (4) through (i) (8). Similar data are collected for each of the free silica solutions.

(B) Standard curves are prepared for quartz, cristobalite, tridymite, or other forms of free silica in which the intensity ratio of the free silica standard to fluorite is plotted against free silica mass in micrograms. This plot should give a nearly straight line that passes through the origin.

(k) Calculations

The concentration of free silica in air can be expressed as micrograms of free silica per cubic meter of air sampled (μg/cu m).

$$\mu g\ SiO2/cu\ m = \frac{\mu g\ Q + \mu g\ C + \mu g\ T + \mu g\ P}{Vs}$$

where:

$\mu g\ SiO2/cu\ m$ = total micrograms of free silica per cubic meter of air sampled.

$\mu g\ Q$, $\mu g\ C$, $\mu g\ T$, $\mu g\ P$ = quantity of free silica as determined from the appropriate calibration curve.

Vs = volume of air sampled in cubic meters.

Q,C,T,P = quartz, cristobalite, tridymite, polymorph of free silica.

X. APPENDIX III

MATERIAL SAFETY DATA SHEET

The following items of information applicable to any product or material containing free silica shall be provided in the appropriate section of the Material Safety Data Sheet or other approved form. If a specific item of information is not applicable (eg, flash point) the initials "na" should be inserted.

(a) Section I. Source and Nomenclature.

(1) The name, address, and telephone number of the manufacturer or supplier of the product.

(2) The trade name and synonyms for a mixture of chemicals, a basic structural material, or for a process material; the trade name and synonyms, chemical name and synonyms, chemical family, and formula for a single chemical.

(b) Section II. Hazardous Ingredients.

(1) Chemical or widely recognized common name of all hazardous ingredients.

(2) The approximate percentage by weight or volume (indicate basis) which each hazardous ingredient of the mixture bears to the whole mixture. This may be indicated as a range or maximum amount, eg 10-20% by volume; 10% maximum by weight.

(3) Basis for toxicity for each hazardous material such as an established standard in appropriate units.

(c) Section III. Physical Data.

Physical properties of the total product including boiling point and melting point in degrees Fahrenheit; vapor pressure in millimeters of

mercury; vapor density of gas or vapor (air = 1); solubility in water, in parts/hundred parts of water by weight; specific gravity (water = 1); percent volatile, indicate if by weight or volume, at 70 degrees Fahrenheit; evaporation rate for liquids (indicate whether butyl acetate or ether = 1); and appearance and odor.

(d) Section IV. Fire and Explosion Hazard Data.

Fire and explosion hazard data about a single chemical or a mixture of chemicals, including flash point, in degrees Fahrenheit; flammable limits in percentage by volume in air; suitable extinguishing media or agents; special firefighting procedures; and unusual fire and explosion hazard information.

(e) Section V. Health Hazard Data.

Toxic level for total compound or mixture. Effects of exposure, and emergency and first-aid procedures.

(f) Section VI. Reactivity Data.

Chemical stability, incompatibility, hazardous decomposition products, and hazardous polymerization.

(g) Section VII. Spill or Leak Procedures.

Detailed procedures to be followed with emphasis on precautions to be taken in cleaning up and safe disposal of materials leaked or spilled. This includes proper labeling and disposal of containers with residues, contaminated absorbants, etc.

(h) Section VIII. Special Protection Information.

Requirements for personal protective equipment, such as respirators, eye protection, protective clothing, and ventilation, such as local exhaust (at site of product formulation, use or application), general, or other special types.

(i) Section IX. Special Precautions.

Any other precautionary information.

U.S. DEPARTMENT OF LABOR
Occupational Safety and Health Administration

Form Approved
OMB No. 44-R1387

MATERIAL SAFETY DATA SHEET

Required under USDL Safety and Health Regulations for Ship Repairing, Shipbuilding, and Shipbreaking (29 CFR 1915, 1916, 1917)

SECTION I

MANUFACTURER'S NAME	EMERGENCY TELEPHONE NO.
ADDRESS *(Number, Street, City, State, and ZIP Code)*	
CHEMICAL NAME AND SYNONYMS	TRADE NAME AND SYNONYMS
CHEMICAL FAMILY	FORMULA

SECTION II - HAZARDOUS INGREDIENTS

PAINTS, PRESERVATIVES, & SOLVENTS	%	TLV (Units)	ALLOYS AND METALLIC COATINGS	%	TLV (Units)
PIGMENTS			BASE METAL		
CATALYST			ALLOYS		
VEHICLE			METALLIC COATINGS		
SOLVENTS			FILLER METAL PLUS COATING OR CORE FLUX		
ADDITIVES			OTHERS		
OTHERS					

HAZARDOUS MIXTURES OF OTHER LIQUIDS, SOLIDS, OR GASES	%	TLV (Units)

SECTION III - PHYSICAL DATA

BOILING POINT (°F.)		SPECIFIC GRAVITY (H_2O=1)	
VAPOR PRESSURE (mm Hg.)		PERCENT, VOLATILE BY VOLUME (%)	
VAPOR DENSITY (AIR=1)		EVAPORATION RATE (_____ =1)	
SOLUBILITY IN WATER			
APPEARANCE AND ODOR			

SECTION IV - FIRE AND EXPLOSION HAZARD DATA

FLASH POINT (Method used)	FLAMMABLE LIMITS	Lel	Uel
EXTINGUISHING MEDIA			
SPECIAL FIRE FIGHTING PROCEDURES			
UNUSUAL FIRE AND EXPLOSION HAZARDS			

SECTION V - HEALTH HAZARD DATA

THRESHOLD LIMIT VALUE

EFFECTS OF OVEREXPOSURE

EMERGENCY AND FIRST AID PROCEDURES

SECTION VI - REACTIVITY DATA

STABILITY	UNSTABLE		CONDITIONS TO AVOID
	STABLE		

INCOMPATIBILITY *(Materials to avoid)*

HAZARDOUS DECOMPOSITION PRODUCTS

HAZARDOUS POLYMERIZATION	MAY OCCUR		CONDITIONS TO AVOID
	WILL NOT OCCUR		

SECTION VII - SPILL OR LEAK PROCEDURES

STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED

WASTE DISPOSAL METHOD

SECTION VIII - SPECIAL PROTECTION INFORMATION

RESPIRATORY PROTECTION *(Specify type)*

VENTILATION	LOCAL EXHAUST	SPECIAL
	MECHANICAL *(General)*	OTHER

PROTECTIVE GLOVES	EYE PROTECTION

OTHER PROTECTIVE EQUIPMENT

SECTION IX - SPECIAL PRECAUTIONS

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORING

OTHER PRECAUTIONS

XI. TABLE AND FIGURE

TABLE XI-1

PERMISSIBLE CONCENTRATIONS OF DUST CONTAINING FREE SILICA FOR THE PARTICULAR INDUSTRIES IN THE LOCALITIES INDICATED*

Industry	Percentage Silica in the Dust	Permissible Maximum Safe Dust Concentration Million Particles/cu ft
South Africa gold mines**	80	4.5
Ontario gold mines**	About 35 (in the rock)	8.5
Australia sandstone**	90 (in the rock)	6
Barre granite***	31 to 38	10-20
Pennsylvania anthracite coal	35	5-10
Broken Hill, Australia**	10 to 17	14

*National Silicosis Conference, Bulletin No. 13, Division of Labor Standards, United States Department of Labor.
**Based upon engineering practice.
***Based upon clinical studies.

From Reference 33

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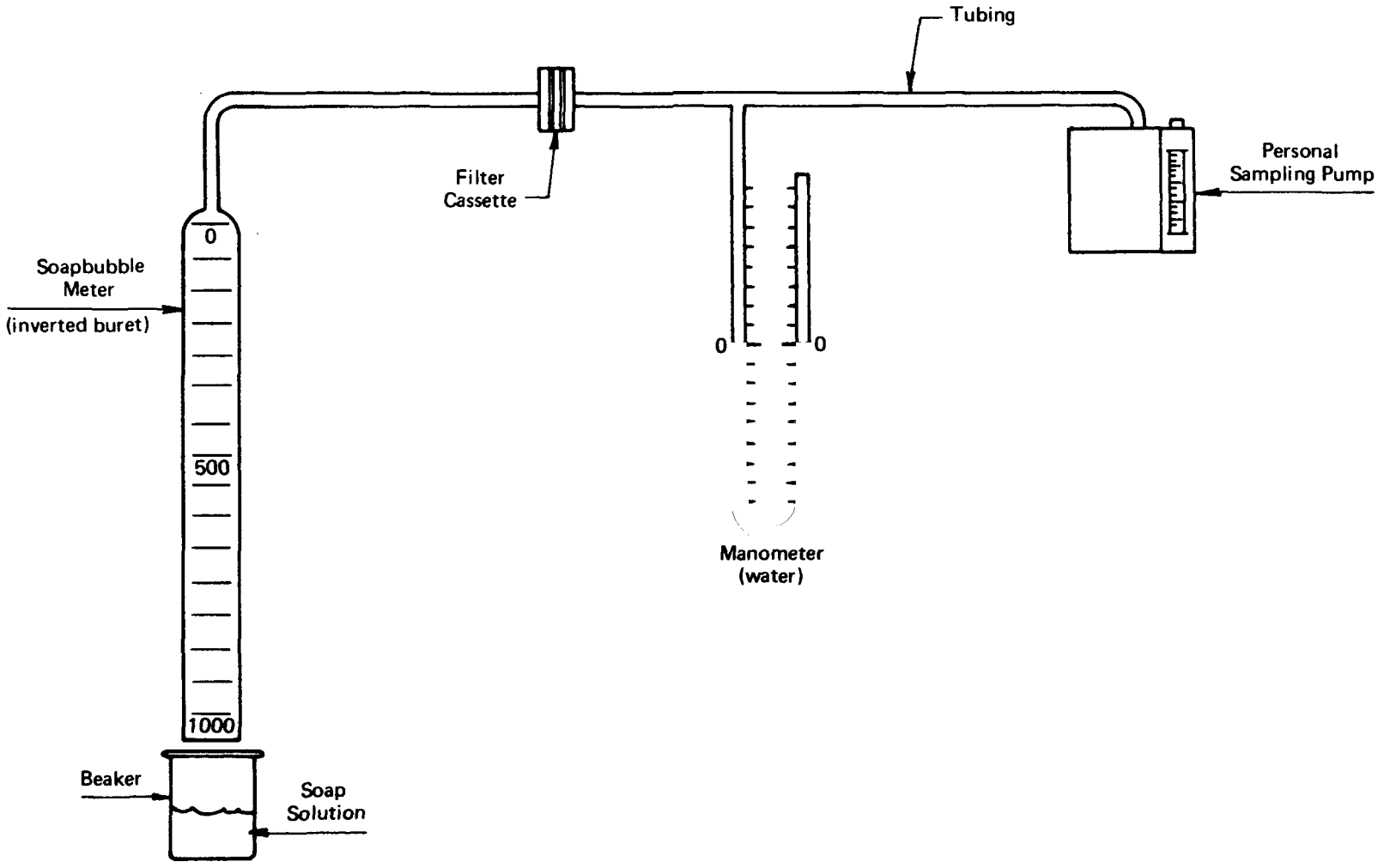

FIGURE XI - 1. CALIBRATION SETUP FOR PERSONAL SAMPLING PUMP WITH FILTER CASSETTE