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]