REVIEW OF THE LITERATURE ON CRYSTALLINE SILICA

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ABSTRACT: A review and update of technical papers associated with crystalline silica exposure hazards has been prepared for the purpose of augmenting information presented in the 1975 document, "Criteria for a Recommended Standard....Occupational Exposure to Crystalline Silica." No changes in the recommended standard are proposed, although there have been many assessments of the silica hazards in foundries, abrasive-blasting operations, silica flour and abrasive cleaner manufacturing, and the firebrick and ceramic industries.

A series of epidemiological studies which address the risk of respiratory malignancy among workers exposed to crystalline silica are referenced. Also noted are several animal studies that report effects of silica exposure. Among these are studies aimed at a better understanding of the mechanism of silicosis induction as well as studies reporting the development of malignancies after silica exposure by inhalation, intratracheal inspection, and intraperitoneal instillation.

Two studies, one in the United States and one in Sweden, reporting the extent of silica exposure in the foundry industry, and recent advancements and recommendations in the area of engineering controls to reduce silica exposure are described.

The most recent developments in techniques for workplace sampling and analysis of crystalline silica are discussed, including a recently completed collaborative test of both x-ray diffraction and infrared spectrometric methods for silica analysis.

Also included in the review is a summary of OSHA inspections (by SIC codes) and a listing of NIOSH Health Hazard Evaluation Reports on crystalline silica.

KEY TERMS: crystalline-silica, respiratory-disorders, epidemiology,toxicology, foundries,control-technology,x-ray-diffraction, spectrometry, silicosis, cancer

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DISCLAIMER

This document represents a review of recent papers and research efforts of many investigators, and is not intended as a statement of policy position for the National Institute for Occupational Safety and Health (NIOSH).

PREFACE

Potential exposure to crystalline silica is unavoidable in many industries, and is widely dispersed in the general and industrial environment. Unfortunately, the continued presence of silicosis, a preventable disease in the workforce, indicates that all industries are not providing sufficient control measures to limit worker exposure. The National Institute for Occupational Safety and Health (NIOSH) seeks to highlight the occupational situations in which evidence of silica-related diseases are reported so that appropriate corrective measures may be taken by an informed public.

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SYNOPSIS

When the criteria document on crystalline silica [1] was published in 1975, there was still a great deal of uncertainty in the industrial hygiene community regarding this common mineral. Sampling and analytical methods had been changed to a totally different concept and were still undergoing development and refinement; the standard had been redefined from a measure of total silica-containing dust to a measure of silica mass; and the uncertainty about the validity of conversions from one measurement system to the other cast doubt on the relevance of historical studies on exposure effects. These factors, together with a rapid advance in instrumental technology, contributed to a proliferation of studies on the multiple aspects of crystalline silica exposure control and effects. No single source was available where these studies could be found. This report has brought this information together as an update of that found in the criteria document [1].

Among these recent publications are studies in a Swedish foundry [2], in the U.S. diatomite industry [3], and in the Vermont granite sheds [4,5,6,7]. Other reports indicate additional cases of silicosis in the manufacture of firebricks [8,9], in foundries [10,11], in gold mines [3,9,12], in the Welsh slate industry [13], and in the Swedish diatomite industry [14].

Of particular interest are the reports of silicosis in the abrasive cleaner industry [15,16], silica flour mining and milling [17,18,19,20], slate pencil manufacturing [21], wood filler use [22], and in the pottery industry [23]. These industries are known to use silica, and almost no information exists on the prevalence of silicosis in these industries. The state of a worker's health in a number of industries that use or manufacture products containing silica does not appear to have been examined. For example, almost no information concerning the building trades was found, even though a Swedish study [24] suggested that some construction workers may be at risk of developing silicosis. A few dry-wall tapers were examined, and they showed evidence of lung damage; however, affected workers had simultaneous exposure to asbestos [25].

Several reports [9,11,12,26,27,28,29,30,31,32] provide evidence of cancer, particularly of the respiratory tract, in worker populations with exposure to free silica. Retrospective studies of miners [12,26], and workers in fire-brick and fire-clay manufacturing [9], several foundries [11,27,28,29,30,31,32], and pottery manufacturing [23] reported varying degrees of excess risk of respiratory cancer mortality. Two studies of groups of diagnosed silicotics, one in Sweden [33] and one in Ontario, Canada [34] reported a similar elevation of cancer mortality. Although these studies report statistically significant excesses of cancer, no causative relationship has been demonstrated. In many instances, especially in the foundry industry, silica is only one of many toxic substances encountered by the worker. The effect of mixed exposures is still unclear.

A number of cases of acute silicosis, often in association with proteinosis, have been described, particularly in sandblasters [35,36,37,38]. It would appear that particular attention should be given to the working conditions and medical examinations of sandblasters and others who work in especially dusty conditions. However, as was pointed out by Jones et al. [37], it is often difficult to identify workers who have been exposed to crystalline silica in sandblasting operations.

Reports describing several medical tests designed to detect lung damage in persons with silicosis were found [40,41,42]. However, in all cases the patients had changes already evident in chest radiographs and the methods did not appear appropriate for routine examination of employees. In one case [42], the procedure required the use of radioactive material.

In several reports, kidney damage [12,43,44,45] or hepatosplenic silicosis [45] was mentioned following exposure to silica. These reports are inconclusive, and suggest a need for further research in this area.

Evidence from animal studies [46,47,48,49,50], indicated a relationship between silica and carcinogenic response. Syrian hamsters and several species of rats have been exposed to silica by inhalation, intratracheal and intrapleural instillation, and even by direct dusting on internal organs. Neoplastic growths resulted in the respiratory and lymphatic systems of certain animal groups. Mixed exposure studies were also conducted. The mechanisms of several responses have not been fully explained, and their significance is still in doubt. However, these studies, as a group, must be considered in the ultimate assessment of the role of crystalline silica in carcinogenesis.

Since the publication of the NIOSH criteria document, "Criteria for a Recommended Standard....Occupational Exposure to Crystalline Silica" [1], most analytical methods research has been directed to the improvement and optimization of the x-ray diffraction method. Detection limits have been lowered, standardization altered, and analytical precision improved. Methods improvement is a continual effort, and the NIOSH Manual of Analytical Methods [51] reflects this fact. The most recent development of this process, as it relates to crystalline silica, is the collaborative test recently conducted on the x-ray diffraction (XRD) and infrared spectrometry (IR) methods. This test provides a definitive evaluation of the capability of these methods individually as well as the relationship of each to the other. The final report on this test will be available early in 1983, and its findings will be reflected in the next revision of the NIOSH Manual of Analytical Methods.

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CONTENTS

I. INTRODUCTION

Potential exposure to crystalline silica is widespread in the United States workforce because numerous industries [1,84,85,86] use or produce materials that contain a significant percentage of silica (see Appendix I). The National Occupational Hazard Survey of 5,000 industries was conducted by NIOSH in 1972-74 [84], from which NIOSH estimated that approximately 3,200,000 workers in 238,000 plants were potentially exposed to free silica. About 260,000 were full-time workers, potentially exposed at least 4 hours/day. Another 200,000 worked in areas where quartz was present. In 1972, silica was one of five substances selected by OSHA for its Target Health Hazards Program [85]. By the time the program was terminated in 1976, 1,197 inspections for silica had been conducted. Of the 8,423 samples analyzed, 37% exceeded OSHA's permissible exposure limit for respirable silica.

On November 11, 1974, NIOSH recommended an occupational health standard for crystalline silica exposure to the Department of Labor [1]. This standard was designed to help prevent the development of chronic lung disease in persons occupationally exposed to crystalline silica. Medical monitoring, including chest radiographs and pulmonary function tests, and a permissible exposure limit of 50 micrograms of respirable free silica per cubic meter of air (50 $\mu g/m^3$) as a time-weighted average (TWA) for up to a 10-hour workday, 40-hour workweek, were recommended in the standard.

This report does not alter the basic recommendations made in the criteria document [1], but presents information developed since its publication. Additional studies on sampling and analytical methods, work practices and engineering controls, human mortality patterns, and human and animal toxic effects as they relate to crystalline silica exposure, are included.

Crystalline silica is defined as silicon dioxide with molecules arranged in fixed regular patterns. It occurs in nature in several polymorphic forms which are chemically identical, and differ from one another only in their crystalline arrangement. Primary among these are quartz, cristobalite, and tridymite. Quartz is by far the most abundant form of silica. In addition, there are high temperature forms of each of the three common polymorphs which occur only at specific elevated temperatures (e.g. quartz inversion occurs at 573 °C), are unstable, and are designated alpha and beta for low and high temperature forms, respectively. In general usage and in this report, crystalline silica, free silica, silica, and quartz are used interchangeably and in all cases refer to alpha-quartz. All three forms have been associated with the development of silicosis in workers. A long-standing issue that has been receiving increasing attention and study is that of the relationship between silica exposure and the development of respiratory cancer. An increased incidence of lung cancer has been observed in workers employed in industrial settings where crystalline silica exposure and resultant silicosis occurred. However, no dose-response relationship

has been established. Likewise, smoking histories of these populations are not available.

Most of the studies cited describe situations where mixed exposures occurred. This was especially true in the foundry industry, which has been the subject of many retrospective studies. A series of experiments with rats have produced a body of evidence that show neoplasms developing after crystalline silica administration. The disease mechanism has not been determined, however, and the limitations of the human epidemiology studies leave many unanswered questions. Further research is needed.

Extensive exposure information on crystalline silica has been amassed through the compliance inspection program of the Occupational Safety and Health Administration (OSHA). This information, which covers the period July 1972 to June 1982, is summarized.

II. HUMAN EFFECTS

In 1974, NIOSH made its recommendation on crystalline silica to help prevent the development of silicosis in workers [1]. That recommendation was based, primarily, on numerous case histories and epidemiological studies that described silicosis in workers exposed to crystalline silica. More recent reports of adverse effects of exposure to free silica have stressed several different factors, such as the results of medical tests or the incidence of silicosis in a particular industry. In addition, complications or associated diseases and diagnostic procedures, some not routine, have been described.

Silicosis - Group Results of Medical Tests

In 1975, Navratil et al. [52] examined the medical records of 200 patients. Most had been miners and all had been diagnosed as having silicosis 3-17 years earlier; nearly all were smokers. The degree of severity of the silicosis was determined by evaluating available chest x-rays, classified by the protocol of the International Classification of Pneumoconiosis (ICP) established by the International Labor Organization in 1971.

The results of lung tomographies and occupational histories were also available to confirm the diagnosis of silicosis. In 65 cases, the disease had progressed from what was described as simple silicosis into a more complicated form. In another 65 cases, severe silicosis had already been diagnosed. Generally, there was a time-related tendency toward increased size and profusion of nodular opacities. Statistically significant increases of pulmonary function impairment were slow to develop. Even in the most severely affected group, these changes did not develop for 6-10 years. However, the authors noted that the mortality rate for those patients with severe functional disorders was high. Persons who also had chronic bronchitis developed more significant functional disorders during their disease than other patients. Changes in blood gases and acid-base balance were examined for 3-10 years in 58 persons, but significant changes were rarely found, even in individuals with severe ventilatory impairment.

In 1978, Cardesi and Provana [53] examined the records of 204 patients who had been admitted to a hospital with a diagnosis of silicosis or lung cancer, to determine if any correlation existed between the two diseases. One hundred seventy-nine were men and 25 were women. There were 135 cases of lung cancer which were classified into one of five histological groups. Thirty-two persons, 29 men and 3 women, had silicosis without lung cancer. Twelve cases were massive, 9 were nodular, and 11 were peribronchiolar-vascular. Thirty-seven individuals had both silicosis and lung cancer. In 7 cases, silicosis was massive, in 11 cases it was nodular, and in 19 cases it was peribronchiolar-vascular. While 53.6% of the persons

with silicosis also had lung cancer, no correlation with occupation could be made because occupational histories were incomplete.

In 1975, Teculescu et al. [40] studied static lung volumes and intrapulmonary mixing by closed circuit helium dilution in 233 individuals with silicosis and 41 others suspected of having pneumoconiosis. Forty-four persons served as controls. Test results were abnormal in about 63% of the patients. Average mixing time and ventilation for the 274 patients were approximately twice the values found in controls. These changes were more pronounced in persons who also had chronic bronchitis or an impairment of ventilatory function. Approximately 25% of the patients had no evidence of restrictive or obstructive ventilatory defect; however, one-third of these persons had lung changes observed by the closed-circuit mixing tests.

In 1973, Cocarla et al. [54] examined the serum of 105 persons with silicosis and silico-tuberculosis, using the Middlebrook-Dubos test for tubercle bacillus antibodies. However, the authors found the test to be of little diagnostic value.

An editorial in the American Review of Respiratory Disease in 1978 drew several conclusions from the available literature on silicosis and tuberculosis [55]. These were: (1) tuberculosis has been a major cause of morbidity and mortality in silicotics, (2) the silicotic has an increased risk of developing tuberculosis, (3) this risk is higher in cases of pure silicosis than in mixed dust pneumoconiosis, and (4) the percentage with tuberculosis is highest among patients with advanced forms of silicosis. Possible relevant factors mentioned were impaired function of macrophages and effects on the immune system.

The results of studies on preventive therapy were also examined. The reviewer concluded that the evidence is equivocal and that isoniazid therapy is effective in preventing tuberculosis among persons with pure silicosis, and that the therapy should be considered for persons with silicosis if there is reason to believe they are infected with Mycobacterium tuberculosis. It should be noted that the risk associated with tuberculosis should be weighed against the possibility that isoniazid might be metabolized to a carcinogen.

In 1978, an editorial in Lancet [56] cautioned that it appeared necessary to consider inhaled silica dust as a rare cause of renal disease. This statement was based on two case reports. In one case, a person who had worked with refractory bricks developed proteinuria. A renal biopsy demonstrated focal glomerulonephritis and degenerative changes in the proximal tubules, although no change in tubular function was observed [43]. A second case involved a sandblaster with acute pulmonary silicoproteinosis who died of acute renal failure [44].

In another case report in 1972, silicotic nodules in the liver and spleen together with calcification in abdominal lymph nodes were discovered in a person who developed silicotuberculosis after nearly 15 years as a

miner [45]. The authors felt that hepatosplenic silicosis is probably not uncommon in patients with severe silicosis. Their patient had died because of severe respiratory failure.

Retrospective Studies Concerning Cancer and Exposure to Silica

In 1979, Katsnelson and Mokronosova [9] examined the results of several epidemiological studies of industries in Russia to determine if exposure to nonfibrous mineral dusts was associated with the development of malignant tumors. Industries examined where there was exposure to silica included a plant producing silica firebricks, two plants producing aluminosilicate fireclay, and a gold mine. The firebricks contained over 90% quartz, and the fireclay dusts contained 15-30% quartz although total airborne dusts were 3-4 times greater than in the firebrick industry. The amount of airborne silica in the gold mines was similar to that in the firebrick industry. Recent exposures were in the 1-2 mg/m^3 range; however, historical exposures were probably higher. The numbers of male and female workers were about equal in these industries except for all underground mining activities, where the women were not employed. In the firebrick plant, 75% of the men and 1.5% of the women admitted smoking, and in a fireclay plant, the respective percentages were 66% and none.

All of the epidemiological studies were conducted in a similar manner [9]. Death records were examined to determine the number of deaths from malignant tumors over a 20- to 26-year period. These records were then compared with company records to identify who had been employed by the company for at least 3 years. The proportion of the general population and the corresponding proportions for industrial workers dying from tumors at all sites, or at specific sites, were calculated. From age-adjusted proportions, a relative risk was determined. There was a significant increase ($P < 0.05$) in death from lung and gastric cancer and tumors at all sites for underground workers, but not surface workers of a gold mine. The relative risk increased in persons with silicosis. In the firebrick plant, men, but not women, had an increased risk of death from lung cancer and tumors at all sites. All male workers in the fireclay plants had an increased risk of death from lung and gastric cancer and tumors at all sites. No significant excess of deaths from any disease was found in women except for one fireclay plant in which the relative risk of tumors at all sites was elevated. The authors concluded that even though the death rate from cancer was higher in occupational groups exposed to silica, the results tended to support the conclusion that silica-containing dust was probably a cocarcinogen, and not a direct carcinogen. This conclusion was based on the differences in the risk for men and women and the finding that increased incidence of deaths from tumors of all sites is correlated with high total dust load rather than the silica content of the dust.

The causes of death for silicotics exposed in copper mines, gold mines, or firebrick factories were also discussed [9]. The main causes of death were tuberculosis, heart-lung insufficiency, and malignant tumors. These

three causes accounted for 54-72% of the deaths for stage I silicotics, 60-69% for stage II, and 83-100% for stage III. As the stage increased, the percentage of tuberculosis deaths increased and the percentage of malignant tumor deaths decreased. The authors noted that the large effect of tuberculosis as a competing cause of death had to be taken into account, and that the calculated relative risk of death from cancer appeared less.

In 1978, McDonald and coworkers [12] conducted a retrospective mortality study of 1,321 men who had worked at least 21 years in a gold mine. Only 9 were lost to follow-up, and the cause of death was ascertained for 657 of the 660 who had died. The ratio of observed to expected deaths was computed from South Dakota rates for various causes of death. The cohort had 81 excess deaths from 1937 to 1973, an excess that could be attributed primarily to pneumoconiosis or respiratory tuberculosis. The cohort was further divided into five dust exposure categories: very high, high (greater than 6 million particles per cubic foot, mppcf), moderate, low, and very low (1 mppcf maximum). The relative risk of death from pneumoconiosis for moderate exposure was 2.9 times that in the combined low and very low categories; it was 19.9 times greater for combined high to very high exposure. Similar trends, 3 and 16 times greater, were seen for respiratory tuberculosis. Because of seven deaths from these two causes in the low and very low exposure groups which were used as the basis for calculating relative risks, the risks in the groups with greater exposure could have been underestimated. The higher exposure categories also had a 1.6 times greater risk of developing malignant neoplasms, but trends relating to increased exposure were not evident.

The authors concluded that past dust exposure had produced substantial excess mortality. However, there was no evidence of a separate and specific increase in respiratory cancer. The study was specifically concerned with the risk of cancer from exposure to crystalline cummingtonite-grunerite, because of its chemical similarity to amosite asbestos (fibrous grunerite).

Mortality patterns of workers in the same South Dakota gold mine had been studied earlier (in 1976) by Gillam et al. [120]. A retrospective cohort study was conducted, restricted to 440 workers who had been employed underground at that facility for at least 60 months and who had never mined underground elsewhere. Over the thirteen years studied (1960-1973), 71 deaths occurred among these miners compared to 52.9 expected an excess significant at $P < 0.05$. Ten deaths attributable to respiratory malignancy, as compared to 2.7 expected, represented a significant excess ($P < 0.01$).

When the analysis addressed the time interval from onset of underground employment in the mine to death from respiratory disease, it was found that no excess of nonmalignant disease risk was noted during the first nineteen years of underground exposure. Respiratory cancer showed a significant excess of exposure intervals of nineteen years or less (3 observed vs. 0.56 expected, $P < 0.05$) and of twenty or more years (7 observed vs. 2.18 expected, $P < 0.01$).

Environmental sampling in the mine showed that crystalline silica levels were well below the applicable OSHA standard. Arsenic, chromium and nickel were present only at trace levels and radon daughters were not detected. Samples were also analyzed for asbestos by scanning electron microscopy, phase-contrast optical microscopy, transmission electron microscopy, and energy dispersive x-ray analysis. Airborne fibrous material was identified as composed primarily of fibrous grunerite (amosite).

The observed excess of malignant respiratory disease among the cohort was attributed by the authors to asbestos, singly or in combination with cigarette smoke. They further stated that the excess of nonmalignant respiratory disease could be ascribed to asbestos, with a possible additive role from low exposures to free silica dust.

In 1982, Costello [26] studied a cohort of 12,258 white metal miners who worked in 50 different mines (11 types) extracting mostly copper, lead-zinc and iron ores. Of the 12,258 miners studied, 1,987 (16.2%) were deceased. A mortality study showed increased SMR's ($P < 0.01$) for respiratory cancer, including cancer of the trachea, bronchus and lungs, and nonmalignant respiratory diseases including pneumoconiosis and accidents. In an attempt to evaluate the effect of other potential causative factors, a case control study was designed to examine the type of ore mined, radon daughter exposure, radiographic evidence of silicosis, presence of airway obstruction, diesel exhaust exposure, and smoking habits. Respirable silica exposure was not studied. Respiratory cancer incidence decreased significantly in ex-smokers and nonsmokers and increased in cigarette smokers.

Gibson and colleagues [11] examined the incidence of lung cancer mortality in a steel foundry in Canada. Persons who had worked at the facility prior to 1967 for 5 or more years and who were at least 45 years old were included in the study. There were 439 foundry workers and 1,103 nonfoundry workers. This cohort of 1,542 workers was followed until January 1977, and only 6 were lost to follow-up. There were 21 deaths from lung cancer in foundry workers, and only 11 in the nonfoundry group. A foundry worker over 45 years of age was about 5 times more likely to die of lung cancer than a nonfoundry worker. This statistically significant increase was similar to that found by comparing the cohort with mortality patterns in an adjacent urban population (Toronto in 1971). Crane operators were at the highest risk, followed by finishers, molders, coremakers, and furnace workers. Exposures to respirable free quartz in these five areas of the foundry averaged 0.38-0.75 times the 1976 TLV. Previous exposure data were not available. The authors suggested three explanations for their results: higher exposures to quartz and other substances in the past, a cocarcinogenic effect of the respirable dust, or the presence of unidentified carcinogens.

In 1976, Koshela et al. [27] studied the mortality of iron, steel and nonferrous foundry workers in Finland who died between 1952-1970. The foundry workers were compared with the general male population of the

country, and SMR's were calculated. An excess of lung cancer deaths was reported with 13.9 expected and 21 observed.

A further study of lung cancer mortality among Finnish foundry workers was conducted by Tola et al. [28] in 1979 in 13 iron foundries. The workers were classified by jobs according to degree of exposure to polycyclic aromatic hydrocarbons. The job classifications were as follows: (1) heavy exposures (casters, knock-out men, molders); (2) some exposure (coremakers); (3) low exposure (fettlers); and (4) miscellaneous or undefined. It was felt that smoking habits did not vary significantly between the exposed and the nonexposed groups in the study, and, therefore, did not introduce a confounding effect into the study. The authors concluded that iron foundry workers may have an increased risk of developing lung cancer.

In an extensive report of mortality patterns within the United States steel industry, Redmond [29] reported increased risk of cancer of the respiratory systems for workers in the mason and blacksmith shops with the steel manufacturing industry in the United States.

In 1979, Egan et al. [30] reviewed the death records maintained by the International Molders and Allied Workers Union when conducting a proportional mortality study of this group of foundry workers. White and black workers were analyzed separately, but mortality patterns did not differ between the two groups. A significant excess of deaths attributable to cancer of the trachea, bronchus and lung (PMR = 147, $P < 0.01$), and nonmalignant respiratory disease including pneumoconiosis (PMR = 610, $P < 0.01$) was found. Several foundry contaminants were discussed as possible causative agents, among which were aromatic hydrocarbons, polynuclear aromatic hydrocarbons, BCME, silica, and talc. Smoking histories were not available. Based on a survey conducted by the National Center for Health Statistics, the authors indicated that unless the smoking patterns of foundry operative and kindred workers were significantly different from those of non-foundry operative and kindred workers in general, the lung cancer excess could not be explained by smoking.

In 1981, a follow-up of the study [31] showed that the increased risk of lung cancer among a group of younger foundry workers may indicate that modern foundry technology has not reduced the carcinogenic risk of the foundry environment.

The specific mortality experience of men employed in a captive gray iron foundry was studied by Decoufle and Wood [32] in 1979. Two thousand eight-hundred sixty-one employees were identified based on the criterion of at least one year of employment in "blue collar" foundry jobs prior to 1968. Excess mortality due to respiratory cancer was observed in all groups, but was statistically significant only for black workers employed more than 5 years prior to 1938.

The five studies cited above [25,29,30,31,32] all deal with foundry exposures where airborne respirable silica is a major problem. Foundry

workers have been reported in studies such as these to have an increased incidence of lung cancer, and some investigators have attributed this prevalence to crystalline silica exposure. It should be noted, however, that there are many toxic (possibly carcinogenic) substances in the foundry environment. At this point, there is insufficient data to name a single cause for this increased incidence of respiratory malignancy.

In 1982, workers in the pottery industry were studied by Thomas [23] who used the death claim records of the International Brotherhood of Pottery and Allied Workers as his cohort. Workers were classified according to the product line of their work experience: (1) chinaware, (2) general ware and (3) sanitary ware (plumbing fixtures). Only workers in the third product line showed a statistically significant increase in deaths from lung cancer (PMR = 1.80, $P < 0.01$). It should be noted that in the sanitary ware product line, talc is used to dust plaster-of-paris molds. Many of the industrial grades of talc have been found to contain asbestos, so the cancer incidence cannot be unequivocally ascribed to silica exposure.

In 1980, Westerholm [33] reviewed the Swedish national pneumoconiosis register, which contains all silicosis cases notified since 1931. His study addressed eight comprehensive questions, one of which was, "Is silicosis associated with an increased mortality from lung cancer?" Occupational groups considered in the assessment of the lung cancer risk of silicotics were mining, quarrying and tunneling, steel and iron industries, and the ceramic industry. The lung cancer mortality data indicated an association between silicosis and lung cancer for those workers who were employed in mining and iron and steel industries. However, Westerholm noted that these data did not imply a causal relationship, only that the rate of lung cancer deaths is increased in silicotics when compared to the general population.

A similar study was conducted by Finkelstein et al. [34] in Ontario, Canada in 1982. They reviewed the mortality data for 1,190 Ontario miners who had been awarded compensation for silicosis from 1940 through 1975. Four cohorts were established based on the initial year of formal disability: 1940-49, 1950-59, 1960-69, 1970-75. SMR's for deaths due to lung cancer were elevated in the first and second decade cohorts (303 and 195) but not in the latter two. For all nonmalignant respiratory diseases (excluding tuberculosis) the SMR's were elevated in all four cohorts.

A number of the studies cited above have been reviewed by Goldsmith et al. [63] in a 1982 report that discusses the human epidemiological and animal study evidence as it related to silica exposure and increased incidence of lung cancer. Three candidate hypotheses were proposed to explain the excesses of lung cancer among silica-exposed workers. These hypotheses were: (1) silica directly induces lung cancer; (2) silica causes silicosis, which may be an intermediate pathologic state leading to lung cancer; (3) silica, linked with PAH and smoking or from the ambient working environment, impairs lung clearance. Thus, the effective dose and/or duration of exposure is increased inducing neoplasia in the adjacent pulmonary tissue.

Puntoni et al. [37] examined the mortality rates for 20 classifications of shipyard workers in Genoa, Italy in 1979. Company records were supplied in three groups for active or retired workers who were at least 35 years of age on January 1, 1960 and 1970, and June 30, 1975. No indication of the completeness of these records was given. The number of expected deaths was calculated from information obtained on the male population in Genoa. The relative risk of death was significantly elevated in shipyard workers compared with both control groups in the following categories: total deaths; cancer at all sites; cancer of the colon (excluding rectum); cancer of the lung, bronchus, and trachea; cancer of the kidney and bladder; respiratory disease; ictus and senility; cirrhosis of the liver; and all other causes. One of the groups, stakers, performed sandblasting and were also exposed to solvents, naphtha, and mineral oil. This group had an excessive incidence of total deaths; cancer at all sites; cancer of the larynx; cancer of the lung, bronchus, and trachea; deaths from respiratory disease; and deaths from cirrhosis of the liver. Information from other groups also exposed to solvents suggested this exposure as a factor in the deaths from cirrhosis of the liver.

The authors believed that laryngeal cancer was related to silica exposure, based on similar findings in workers in a refractory material factory. The latter study has not been published and, therefore, is not available for review.

Silicosis and Sandblasting Practices

In 1975, Jones et al. [64] described the results of pulmonary function tests of 55 sandblasters in Louisiana who had developed silicosis. Vital capacity, FEV_1, residual volume, and pulmonary diffusing capacity were measured, and total lung capacity (TLC) was calculated. The patients were divided into three groups. Group I (n=14), with simple nodular silicosis, had normal pulmonary function. Group II (n=10), with large opacities, had moderate obstruction, normal lung volumes, and mild impairment of alveolar gas transfer. Group III (n=31), with major distortions of lung architecture, had restriction of lung volume, more marked obstruction, and further reduction in gas transfer. Results from sequential pulmonary function tests for 26 persons were available so that annual rates of change in ventilatory function could be calculated. In Group III, large declines in ventilatory function were observed; FEV_1 decreased by 120 ml a year and TLC diminished by 187 ml a year. The authors noted that they knew of at least 135 sandblasters with silicosis, 30 of whom had died. They also noted the difficulties in identifying individuals who might be at risk, because persons exposed to silica from sandblasting often worked in small facilities on a contract basis and considered themselves to be painters and not sandblasters.

In another report in 1976 from the same laboratory, Ziskind et al. [65] studied 22 sandblasters with silicosis who worked in shipyards. Eleven of them died during the study; their average age at death was 48.5 years and

they had been exposed to silica for an average of 9.7 years. Nine of the sandblasters also had developed mycobacterial infection and one had developed silico-proteinosis. Only 5 of the dead men had worked with air-supplied hoods; 4 had worked in enclosed spaces. The average age of the 11 survivors was 43.6 years, and they had been exposed for an average of 12.4 years; 4 had bacterial infections. Seven of the survivors had worn air-supplied hoods. In 15 of the 22 cases, 8 of which were fatal, massive lesions were observed on chest radiographs. The authors commented that their results supported the contention that sandblasting could be associated with the onset of silicosis.

Ziskind et al. [65] also measured the amount of airborne silica in sandblasting areas of open steel fabrication yards and determined protection factors for both air supplied and non-air supplied hoods being used to limit worker exposure to the dust. Continuous samples were collected using gravimetric respirable dust samplers both inside and outside the protective hoods worn by the sandblasters. Samples were analyzed either by x-ray diffraction or colorimetry; the results are given in Table II-1. It is notable that the hoods in many cases did not reduce worker exposure to silica below the 50-$\mu g/m^3$ limit recommended by NIOSH [1].

TABLE II-1

RESPIRABLE DUST AND FREE SILICA CONCENTRATIONS DURING SANDBLASTING

Position of Sampler	Levels of Sandblasting Activity					
	Slow Operation (2-2.5 Hour Blasting)		Moderate Operation (2.5 Hour Blasting)		"Busy" Operation	
	Mean Respirable Dust (mg/m^3)	Silica (%)	Mean Respirable Dust (mg/m^3)	Silica (%)	Mean Respirable Dust (mg/m^3)	Silica (%)
Outside hood	2.0 (6)*	67.4	6.9 (6)	54.5	37.2 (7)	83.6
Inside non air-supplied hoods	1.3** (12)	30.8	2.8 (7)	42.2	11.0 (4)	70.3
Inside air supplied hoods	0.6 (11)	39.3	2.6 (2)	77.2	5.9 (5)	55.1

Adapted from Reference [65]

*Number of samples is given in parentheses.

**35% of the sandblasters did not wear respirators under hoods.

To examine possible immunologic effects induced by silica exposure, Schuyler et al. [66] studied several aspects of cell-mediated immune response in 1977. Sixteen Louisiana sandblasters and 13 control subjects, patients at a pulmonary clinic, were examined. The silica-exposed group responded to low concentrations of concanavalin A with depressed lymphocyte stimulation. However, results of the following evaluations were similar in both groups: (1) 48-hour skin test responses to four recall antigens, (2) lymphocyte number, (3) T and B cell percentages, and (4) lymphocyte stimulation in response to phytohemagglutinin, pokeweed mitogen, and antigens. The authors noted that their findings were in contrast to the marked effects of silica on cell-mediated immunity and macrophage function that have been observed in laboratory animals. Possible explanations given for the differences were species differences, lack of recent silica exposure in their subjects, or measurement of functions that did not accurately reflect effects of silica. In a follow-up study, Schuyler et al. [67] observed that the depressed lymphocytic proliferation in subjects with silicosis was not due to factors in the serum and was not correctable by supplementation with allogenic lymphocytes.

In 1978, Schuyler et al. [38], in a brief abstract, described evidence of lymphocyte, but not macrophage, dysfunction in samples collected by bronchoalveolar lavage from persons with silicosis. The number and type of cells recovered and the percentage viable were similar in silicotics and controls. However, only 1 of 5 silicotics produced cells that induced macrophage migration inhibition when mixed with guinea pig peritoneal macrophages and exposed to streptokinase-streptodornase; cells of 7 of 11 control subjects possessed this capability.

In a related article in 1979, Reiser and Last [41] examined the information available on the immunological aspects of silicosis. They concluded that the information was unclear about the extent to which immunological mechanisms are directly involved in the pathogenesis of silicosis. The authors noted that the two major complications of silicosis in humans that might be associated with alterations in immune function would be tuberculosis and Caplan's syndrome. While tuberculosis was at one time a major cause of death in persons with silicosis, there did not appear to be sufficient information on the incidence of rheumatoid arthritis in silicotics to evaluate the significance of Caplan's syndrome.

In a 1977 study, Suratt et al. [42] reported the development of silicosis in 4 men first employed as sandblasters an average of 35 months before onset of symptoms; 3 died approximately 59 months after their first exposure to crystalline silica. All 4 patients were smokers and had sandblasted granite and marble tombstones in enclosed but vented rooms. The sand was reused until it was too fine to cut stone. When working conditions were first investigated, sandblasters were wearing mine respirator masks with disposable filters. The blasting room operator was exposed to 15 mppcf of sand containing 98% free silica. During a second investigation, the operator was exposed to 3.4 mg silica/m^3 as a time-weighted average (TWA),

but the workers were wearing masks and hoods with continuous external air flow.

Potential Techniques for Evaluating Silicotics

In 1974, Tada et al. [68] used bronchial arteriography to examine alterations of blood supply in the lungs of six patients with silicosis. Chest x-rays had revealed large opacities in 4 subjects and small opacities in 2. All four persons with large opacities had enlargement of the bronchial arterial trunk, considerable neovascularization, and peripheral bronchopulmonary arterial shunts. The authors observed that bronchopulmonary shunts were present in lesions, possibly representing areas of obstructive pneumonitis; all findings were compatible with chronic inflammation of the lungs. No such changes were observed in the individuals with small opacities.

In 1974, Siemsen et al. [69] examined 30 patients with radiographic evidence of pneumoconiosis (17 with silicosis, 7 with silicotuberculosis, and 6 with asbestosis) by performing gallium-67 scans of the lungs and comparing them with nondiseased controls. Gallium-67 concentrations in the lungs were elevated in all persons with pneumoconiosis. In approximately one-fourth of the patients, the results of the gallium scans suggested a greater area of the lung was involved than that observed by chest radiographs. The authors observed that this technique cannot differentiate specific diseases, such as silicosis, but suggested that gallium scans might have potential use in the early detection of lung changes, and in the assessment of progression of the disease.

In 1979, D'Andrea and coworkers [70] examined whether activities of angiotensin enzyme in serum could indicate silicosis. Increased levels of this enzyme were found, but the rise was only moderate.

III. ANIMAL STUDIES

Cell and tissue culture tests have been used extensively as a screening device for studying fibrogenic dusts. A number of these techniques were reviewed by Kaw [71] in 1977. Another in vitro test for determining the potential fibrogenicity of dusts is based on their ability to induce hemolysis in erythrocytes. This test is based on a presumed correlation between the ability of a material to rupture the erythrocyte membrane, and the lysosomal membrane by a similar mechanism. In 1978, Potts et al. [72] found that Min-U-Sil®, a material composed primarily of crystalline silica with a mean diameter of 1 μm, induced extensive hemolysis of rat erythrocytes. In 1977, Summerton and coworkers obtained similar results with human red cells [73]. In 1976, Chvapil et al. [74] also found rapid hemolysis of red blood cells of dogs in the presence of silica, with lipid peroxidation occurring in the presence of silica only in viable erythrocytes. Hemolysis and induction of lipid peroxidation appeared to be independent processes. The authors suggested that lipid peroxidation is a possible mechanism contributing to the development of fibrosis.

The possibility that inhalation of silica could affect susceptibility to infection was examined in female mice [75] in 1979. One group of mice was exposed to silica with a mean diameter of 1.95 μm at a concentration of about 4.9 mg/m^3. Statistically significant decreases in the ability of splenic lymphocytes to respond to T and B cell mitogens occurred after 21 weeks of exposure, but no differences were observed at 15, 33, and 36 weeks. A second group of mice was exposed to silica at 2.2 mg/m^3 for 3 weeks, and a third group was exposed at 5.1 mg/m^3 for 20 weeks. Each group was then exposed to influenza virus. These mice did not show increased susceptibility to influenza infection when compared to control animals.

In 1975, De Mesquita and Kerr [76] examined the effect of silica on the subcutaneous implantation of fibrosarcomas in female rats. Over 42 days, both tumor size and weight were less when the transplanted tumor was coated with silica dust before implantation in the recipient. However, by the end of 42 days, all tumors, including those coated with silica, were invasive, and silica apparently did not interfere with successful transplantation of the tumors.

In 1974, Shanker et al. [77] examined the regional lymph nodes of rats 1-8 days after the intraperitoneal injection of 50 mg of quartz in physiological saline. Twelve of the 25 animals had been exposed to a total body irradiation of 500 roentgens (0.13 C/kg) 7 days earlier. In nonirradiated animals, macrophages were primarily responsible for phagocytosis and subsequent transportation of quartz to the lymph nodes. Areas of the lymph nodes containing quartz particles increased from 3.3% on the 1st day to 33% on the 8th day. However, the reticulum cells of the lymph nodes contained quartz particles, and the affected area, approximately 14%, did not change substantially from the 1st to the 4th days. The authors

proposed that the delay they observed in the fibrogenic effect of quartz on the lymph nodes of irradiated rats was due to the suppression of peripheral monocytes caused by the radiation.

In 1977, Singh and coworkers [78] injected 25 male rats intratracheally with 50 mg of quartz dust (particle size less than 5 μm). The animals, and an equal number of controls, were killed for examination at 30, 90, and 150 days. By 30 days, lesions in the lungs were composed primarily of macrophages and a few lymphocytes. At 90 days, some collagen was evident, and the deposition of collagen increased substantially by 150 days. There was a near linear increase in both wet and dry weight of the lung over the study period, although collagen content increased significantly only at 150 days. The concentration of amino acids in the lung, liver, and serum was determined, and significant increases were noted in concentration of most of the amino acids, especially hydroxyproline. By 150 days, total amino acid content in the lung increased, while the concentration of free amino acids decreased in the liver and increased in the serum. The authors concluded that some collagen forms in the lung from available precursors there, while other amino acids were contributed by organs such as the liver.

In 1979, Chvapil and colleagues [79] examined the sequence of alterations in rat lungs shortly after administration of silica by intratracheal injection. One group of rats received 75 mg of silica, and the lungs were examined 6-144 hours afterwards. Wet weight of the lung and DNA content increased in 24 hours, followed by an increase in noncollagenous protein and total lipids at 72 hours. Finally, collagen content significantly increased in 6 days. A second group of rats received instillations of 10, 30, 50 and 75 mg of silica dust. Their lungs were examined 6 days after exposure. At the lowest dose, all of the changes occurred except for increases in collagen. Collagen was increased for the 30 mg exposure by 70%, a net deposition of 13 mg. The authors found this rapid increase in collagen content quite surprising, and they felt that in later stages of fibroproliferative inflammation the excess of deposited collagen was partially resorbed.

In 1976, Horvath [80] postulated that the negative surface charge of quartz dust could be related to its ability to cause silicosis. She administered 50 mg of three types of dust, two containing 93% quartz (particle size 0.07-1.11 μm and 0.03-3.50 μm) and one containing 21% quartz (of comparable size distribution), intratracheally to three groups of 10 rats. Six other groups received similar suspensions, but two basic dyes were first reacted with the silica to alter the surface charge. After 8 to 12 months, the lungs of 28 of the 30 rats given quartz alone had nodular fibrosis, and all of these rats had alveolar septum hypertrophy and emphysema. While exposure to the dye-coated quartz resulted in hypertrophy and emphysema in all of the animals by 12 months, only 7 of the 60 had evidence of nodular fibrosis. This experiment does not demonstrate conclusively that surface charge is responsible for silicosis, since other theories involving surface factors were not disproven by the results.

In a 1972 study of asbestos effects, Wagner and Wagner [46] made intrapleural inoculations into Wistar rats. Quartz was used as a nonfibrous control substance. Those rats treated with quartz developed silicotic growths in the mediastinum, pericardium, liver, and spleen. Histologically, the observed tumors were designated as malignant lymphomas of the histiocytic type (MLHT).

In a 1976 study, Wagner [47] designed experiments to answer questions raised in her previous work. Two hundred-forty specific pathogen free (SPF) Wistar rats, approximately 39 days old, were used in the study. Eighty were used as controls, and 160 were inoculated intrapleurally with 20 mg of quartz (> 5 μm diameter) suspended in sterile saline. Two males and two females were killed at 5-week intervals, up to 120 weeks. In an additional experiment, different dusts were administered to selected animals: Silica II (cristobalite), Silica III (Min-U-Sil®, 99% quartz), coal dust, and carbon black. Silica III was administered by three routes: intraperitoneal inoculation, intravenous inoculation, and direct application to the thymus. All other dusts and saline controls were administered intraperitoneally. Malignant histiocytic lymphomas were found in rats inoculated intrapleurally with silica, confirming the earlier study. Carbon black and coal dust produced no MLHT. Other routes of administration of silica (intraperitoneal, intravenous, direct dusting) failed to produce tumor growth.

An additional study in 1980 was designed by Wagner et al. [48] to assess the reactions of different silica polymorphs and different rat strains. The silica dusts used were: Tridymite, Min-U-Sil® (93% quartz), D&D® (a pure quartz), Snowit® (commercially prepared quartz), DQ12® (0.1 μm quartz), and cristobalite. A 20 mg saline suspension of each was used for intrapleural injection.

Aldersly Park, Agus and PVG rats were injected with the 6 silica varieties and saline controls.

Tridymite proved to be the most active form of silica dust in terms of lymphoma incidence, with cristobalite the least. The number of lymphomas produced per 32 animals inoculated was tridymite, 16; Min-U-Sil®, 11; D & D®, 8; Snowit®, 8; DQ12®, 5; cristobalite, 4.

Aldersly Park rats had a tumor incidence of 35%, Agus rats, 5%, PVG strain 8%.

Syrian Golden hamsters were injected intratracheally with polycyclic aromatic hydrocarbon combined with SiO_2, MgO_2, and agar gel by Stenback and Rowland [49] in 1979. Ten groups of 24 male and 24 female hamsters received intratracheal instillation of single and combined agents. Respiratory tract tumors developed in 21 of 48 animals treated with BaP + SiO_2. In group 6, which received BaP alone, only 5 of 48 developed respiratory tumors. None of the hamsters treated with SiO_2 alone developed tumors.

Holland et al. [50] reported in 1982 on a study of the effect of oil shale dusts on Syrian Golden hamsters and Sprague-Dawley rats. Both raw and spent shale dusts were ground to a median aerodynamic diameter of 2.45 μm. Min-U-Sil® (quartz) of comparable particle size was used as a vehicle to compare fibrogenic potential of the shale dust. Doses were matched for quartz content.

Both species were exposed intratracheally and by inhalation to the dusts under study. The hamsters showed only minimal fibrogenic response. The response of the rats is summarized in Table IV-1. Of the 49 tumors observed, 9 were adenomas and 40 were carcinomas. The authors noted that exposure levels were high, nearing 10 to 20 times that which might be expected in an industrial setting.

TABLE IV-1

Response of Sprague-Dawley Rats to Oil Shale Dust and Quartz Dust

	No. of Test Animals	No. With Pulmonary Fibrosis	% With Fibrosis	No. With Malignant Tumors	% With Malignant Tumors
Intratracheal Instillation					
Shale Dust	220	19	8.6	1	.46
Quartz	36	15	42	6	17
Inhalation					
Shale Dust	107	35	33	25	23
Quartz	57	44	77	17	30

Adapted from Reference [49]

In 1979, Rylander and colleagues [81] examined the free lung cell response of guinea pigs exposed to silica for 8 hours (74% of the particles had an aerodynamic diameter below 3 μm). Ten animals were exposed to silica alone, and 10 also were exposed to cigarette smoke. Twenty-four hours after exposure, free lung cells were recovered in a saline wash. The number of macrophages recovered was significantly less when compared with controls for exposure to silica alone ($P < 0.01$), but significantly greater ($P < 0.01$) for exposure to silica and smoke. The number of leukocytes increased only in the combined exposure group.

IV. STUDIES PROVIDING INFORMATION ON EXPOSURE HISTORIES

Data received from the Vermont granite industry was the most extensive and complete environmental and medical information available for evaluation in the criteria document on crystalline silica [1]; studies of that industry were an important basis for a recommended exposure limit. Several reports between 1977 and 1981 [4,5,6] described changes in workers exposed under conditions typical of current practices.

In 1977, Musk et al. [4] described the results of a 4-year follow-up study of pulmonary function in 974 granite shed workers. Annual decreases in FVC and FEV_1 were 0.07-0.08 and 0.05-0.06 liter/year for granite shed workers compared with 0.03-0.04 liter/year for the general population. It was concluded that cigarette smoking did not account for the differences. However, no exact correlation between dust exposure level and decrement was obtained, possibly because only a single dust exposure concentration was used to characterize each person's 4-year exposure.

In 1981, Graham et al. [5] presented results of another evaluation of the pulmonary function losses in Vermont granite workers. The investigation represented a long-term follow-up of workers who had been studied earlier [4]. Average increases of 0.006 liter/year in FEV_1 and of 0.108 liter/year in FVC were observed among 487 current workers between 1974 and 1979. The authors thought that this unexpected trend could be explained by differences in techniques of producing and evaluating tracings between themselves and Musk et al. [4], as well as the detection of a leak in a spirometer that was possibly used in both studies. The authors [5] concluded that the issue of pulmonary function loss was not settled. However, the data presented do show annual FEV_1 decrements of 0.019 liter/year among workers of 0.007 liter/year, among workers with 20 or more years of exposure, and of 0.038 liter/year among retired granite workers, all based on measurements taken in 1970, 1974, and 1979. Although the corresponding average FVC measurements showed increases that may have been explained in part by the authors, the decreses in FEV_1 over 9 years among workers with low dust exposures should not be overlooked.

In a 1980 study of Vermont granite shed workers, Craighead and Vallyathan [6] examined the lungs of 15 deceased workers. Medical histories, including chest radiographs and complete autopsy records, were available. Examinations were performed by light and polarized microscopy, scanning electron microscopy, x-ray energy spectrometry, and backscatter electron imaging. All individuals had begun employment after 1937, when improvements in dust control had been introduced. None had radiographic evidence of silicosis; however, all had either localized or diffused fibrosis, and crystalline silica was found in the lung tissues.

In 1978, Ohman [2] calculated the inhaled dose of respirable quartz dust for 498 workers at a Swedish foundry. From company records, which included some dust measurements dating from the late 1940's, and individual

interviews, Ohman developed a complete occupational history for each worker. Chest radiographs made over several years were also available. From the occupational histories, the amount of silica inhaled over a working lifetime was estimated, and these figures were then compared to the workers' health. There were no cases of silicosis found for inhaled doses estimated to be below 5 g. For doses of 5-7.5 g, about 58% of the workers developed silicosis. According to the author's calculations, a 5-gram accumulation of inhaled dust would correspond to an exposure of 50 $\mu g/m^3$ over a 50-year working life.

In 1978, Nelson et al. [16] identified 16 cases of silicosis in Ontario plants where workers were exposed to silica flour, which is produced by the crushing and milling of quartz or quartzite rock. Types of industries studied were abrasive cleansers, foundry supply parting compounds, and silica flours. The workers had been exposed an average of 10.3 years (range, 4-27), and the mean dust concentration (14 cases) was estimated to be 3.7 mppcf.

At 4 plants with 9 cases of silicosis, records of dust sampling by the Ontario Ministry of Health over 37 years were available. To obtain a conversion factor for the respirable mass, the investigators generated airborne silica flour in a dust chamber and analyzed samples by the dust count and respirable mass methods. A conversion factor of 1 mppcf equivalent to 0.11 mg/m^3 was calculated. Average dust counts from representative locations were then used to estimate the airborne concentrations at which silicosis had been observed. For the 9 cases, the average dust concentration was 0.21-0.55 mg/m^3. Based on a 30-year working life, the authors estimated that these exposures would correspond to 0.04-0.16 mg/m^3. It was concluded that the TLV for respirable silica should be 0.05 mg/m^3 for silica flour.

In January, 1972, OSHA initiated the Target Health Hazard Program (THHP), wherein inspection efforts were focused on five major occupational hazards: carbon monoxide, lead, silica, asbestos, and cotton dust. This effort was continued until March 1976, at which time a second program was initiated, titled the National Emphasis Program (NEP).

Recognizing that these OSHA efforts had produced a massive data base for industrial exposures to crystalline silica, NIOSH requested this data. The transmitted information included raw data for all inspections monitoring silica concentrations between July of 1972 and July of 1982, and summarized data grouped by SIC codes. All samples were collected by respirable mass sampling techniques, and Permissible Exposure Levels (PEL) were determined by use of the formula:

$$mg/m^3 \text{ respirable dust} = \frac{10}{\% \text{ quartz} + 2}$$

The total data base included 23,864 individual respirable silica samples drawn from worksites representing 306 separate Standard Industrial

Classifications (SIC's). All but 5.5% of the samples were drawn from the manufacturing industries, with the majority (13,985 or 58.6%) from the primary metals industries. The largest single industrial source in which silica inspections were made was ferrous foundries. There were 10,850 samples collected in this industrial group alone. Severity of exposure was indexed according to a ratio between the actual concentration of respirable free silica (in mg/m^3) and the PEL. Thus, a severity index of 1.0 indicated SiO_2 concentration at the PEL, while a severity index of 2.0 represented a silica concentration of twice the allowed exposure for a worker.

The number of compliance samples was tallied, and 4,835 or 20.3% of the total exceeded the OSHA PEL at least by a factor of 2. This figure, which does not include all violations of acceptable exposure limits, is certainly a significant level of overexposure to respirable silica.

A summary of this data, by SIC code, is presented in Appendix III.

A congressionally mandated function of NIOSH (under the Occupational Safety and Health Act) is the performance of Health Hazard Evaluations (HHE's). HHE's are initiated in response to requests from either management or worker representatives. Eighty-four HHE's involving crystalline free silica exposure have been completed. Of these, 42 (50%) have produced data indicating exposures exceeding the NIOSH recommendation for SiO_2.

In each case, the study entailed an industrial hygiene evaluation and a medical evaluation of employees who were exposed to hazardous agents. All data has been reported in detail along with recommendations for abatement of hazardous or unhealthful conditions. A list of these HHE's are shown in Appendix II.

These reports, coupled with the OSHA inspection data, offer exposure information with all analytical data on free silica concentrations derived via mass sampling techniques.

An example of long-range planning geared to protecting the health of workers is a program developed by the National Industrial Sand Association (NISA). NISA has developed a manual [82] outlining operations within the sand industry, physiological effects of exposure to crystalline silica, sampling and analytical techniques, a medical surveillance program for workers, and an epidemiological recordkeeping program designed to provide the necessary information for future evaluations of hazards to workers.

Epidemiological studies are often hampered by the lack of specific exposure data that can be related to biological effects of that exposure. This program, if followed as recommended, will provide the tools for future evaluations.

Two unusual case histories of workers who developed silicosis, apparently related to workplace exposure, were found [39,57]. These cases

are particularly interesting because silica exposure was generally not thought to be a hazard in their occupations. In addition, evidence of silicosis was found in several studies of industrial operations that were either not extensively examined in the past or not widely recognized as hazardous.

In 1974, Levy and Margolis [39] reported a case of siderosilicosis and atypical epithelial hyperplasia in a 34-year-old man. He had been employed as an oxyacetylene torch cutter in a steel foundry for 5 years using only a welder's helmet for eye protection. This individual worked 12-15 feet from sandblasting operations. The authors reported that OSHA had investigated this foundry and found respirable silica at 6.82 mg/m^3 and iron oxide at 19.4 mg/m^3 in this person's work area. However, the authors did not mention whether other potentially fibrogenic metals were present that may have contributed to the worker's condition.

In 1975, Barnes [57] reported an unusual case of silicosis in a man who worked 40 years as a cinema projectionist. Further inquiry into his industrial and family history revealed that his brother, who died from silicosis, and his father, who died from silicotuberculosis, had both been film projectionists for many years. The worker mentioned that the projection room was filled with a fine white powder released from copper-carbon rods of film projector arc lamps that burned during operation. Analysis of this powder revealed that it contained 6% free silica.

Because modern projectors still use copper-carbon rods, the author [57] felt that silicosis remains a potential hazard to projectionists, and that they should have a chest x-ray taken periodically. The period of exposure in the three cases was 20 years for the father, 19 years for the son who died in 1954, and 40 years for the other son. Exposure times before obvious radiographic changes were observed were 11 and 25 years, respectively, for the two sons.

Occasionally, silicosis can develop and progress extremely rapidly. A 30-year-old Australian man developed a progressive form of silicosis 2 years after first exposure; 30 months later he died [35]. Examination of the lungs revealed extensive fibrous tissue containing rounded nodules. Nodules were also present in the lymph nodes. Some alveoli contained Periodic Acid Schiff (PAS) positive material, indicating silicoproteinosis. The subject had been employed in the manufacture of metal-polishing compounds. On about 60 occasions, over 38 months, he had opened 24 bags of finely ground silica powder and then poured the powder into a vat of molten material. A dust mask was available, but its use was not enforced; the man reportedly smoked on occasion while doing this operation.

In 1972, Xipell et al. [36] described a similar case, that of a 58-year-old Australian man who had worked as a quartz miller for 7 years. He had previously worked as a dairy farmer for 20 years and in a brick factory for 6 years. He first complained of a persistent dry cough and then

of exertional dyspnea; he died 3 years after the onset of symptoms. Light icroscopy of biopsy and necropsy material revealed acute silicosis with associated alveolar lipoproteinosis.

The silica flour industry has been the subject of two NIOSH Health Hazard Evaluations undertaken in 1977 at the request of the Mine Safety and Health Administration (MSHA), and a Current Intelligence Bulletin [18,19,20]. Silica flour is produced by grinding and milling crystalline silica. The silica flour from the mills surveyed was determined to be 99% free silica, and the airborne samples collected showed mean particle diameters ranging from 2.3 to 5.2 μm. X-ray diffraction analysis of the 91 respirable samples showed that 67% exceeded the MSHA standard for crystalline silica (100 $\mu g/m^3$) while 85% exceeded the NIOSH recommended standard (50 $\mu g/m^3$).

NIOSH investigators found a high incidence of silicosis among workers employed at two mining and milling operations that were producing "silica flour" for uses such as industrial and toothpaste abrasives, paint extenders, and fillers for cosmetics [15,17]. Exposures were very high; 77 of 91 (85%) dust samples exceeded the NIOSH-recommended standard of 50 $\mu g/m^3$ silica. Eighty-six workers participated in the survey. While none of the 25 workers employed less than a year had evidence of silicosis, twenty-three of 61 (37%) present or former workers with more than a year of exposure had developed silicosis, and seven of these suffered from progressive massive fibrosis (PMF). The NIOSH Current Intelligence Bulletin #36 issued the warning that "workers engaged in the manufacture and use of silica flour, are at serious risk of developing silicosis," and recommended the control of exposure and the labeling of products containing silica flour.

An unpublished progress report of a 1977 study of an abrasive soap factory identified many cases of silicosis [58]. In abrasive soaps, fine silica particles are mixed with alkali or oxalic acid. In the plant examined, 204 workers had been employed since it began operation in 1954. The progress report described the first 62 workers examined and 14 death certificates. On four of the certificates, silicosis had been listed as the cause of death. Twenty-two of the 62 workers had abnormalities in chest x-rays characteristic of silicosis, and six cases had progressed to an advanced stage of silicosis.

A woman developed silicosis and died in 1979, 25 years after she had intentionally inhaled an abrasive scouring powder containing 90% silica [59]. She remained asymptomatic for 5 years, at which time she progressively became short of breath. Diffuse nodular densities were evident in chest radiographs taken 11 years after exposure. Her condition continued to deteriorate, and pulmonary function tests revealed severe obstruction with moderate restrictive ventilation. A biopsy of the liver revealed prominent silicotic nodules in the portal regions of the parenchyma. The glomeruli of the kidney were also affected. The patient was considered to have a nonspecific vasculitic syndrome associated with

severe silicosis. Apparently, six months later, she died of acute respiratory failure.

In 1978, Irwig and Rocks [3] described the results of a survey of 1,973 white men, aged 45-54, who had been employed underground in South African gold mines for at least 10 years. There were 134 individuals with radiographic evidence of silicosis. However, results from a respiratory disease questionnaire revealed no differences between persons with or without silicosis, except an incidence of chest illness. The mean forced vital capacity (FVC) did not differ between the groups. The mean forced expiratory volume in 1 second (FEV_1) was 5% lower for persons with silicosis, and the mean forced expiratory flow (FEF) of the FVC (FEF 25-75%) was 14% lower. The authors then took into account the possibility that differences in dust exposure, height, age, and smoking history could be responsible for the differences in lung function tests. The authors concluded that miners with silicosis did not have more symptoms of chronic bronchitis and that silicosis had little effect on lung function. They suggested that chronic obstructive lung disease may be a more important cause of morbidity in gold miners than silicosis.

A 1953-54 study done by the Public Health Service (PHS) in five diatomite plants in western states was described in the NIOSH criteria document on crystalline silica [1]. Of 869 workers examined, 78 (9%) had radiographic evidence of pneumoconiosis, which appeared to correlate with the cristobalite content of the dust and length of exposure. Cooper and Jacobson [60] have since reported on a 21-year follow-up of workers at one plant that had contributed 68% of the members in the original study group. A review of chest radiographs of 428 current workers, 129 of whom had been employed at least 20 years, revealed changes in 20 (4.5%). However, 8 of these changes had been present in preemployment films or were not interpreted as indicative of pneumoconiosis. Only 12 (2.8%) were regarded as evidence of pneumoconiosis probably related to workplace exposure. All but 1 of the 12 affected workers had been employed before 1953. Of the other 11 with positive films in the second survey, 6 had already developed definite pneumoconiosis at the time of the study, which was conducted 21 years earlier. Only four individuals had complicated or coalescent lesions, and they had been employed 27-46 years. The authors attributed the reduction of diatomite pneumoconiosis to a strict program of dust control, supplemented by the mandatory wearing of respirators.

In 1978, Beskow [14] described the case histories of six diatomite workers who developed silicosis. The workers had been exposed to kieselguhr, a powder made by heating lake ooze containing diatom walls to a temperature of about 1000°C. The kieselguhr contained quartz, cristobalite, and tridymite. Duration of exposure to kieselguhr was 2-20 years, and age at time of diagnosis was 38-49 years. All six cases progressed to stage III (advanced) silicosis. One worker also had possible tuberculosis, two had tuberculosis and spontaneous pneumothorax, and one had advanced amyloidosis in the kidneys. These six cases constituted all instances of diatomaceous

silicosis that had been registered (since 1931) with the Workers Protection Board (WPBS) of Sweden.

NIOSH [21] conducted a medical and industrial hygiene survey in the North Carolina brick industry in 1980. Medical screening methods included spirometry, chest radiography, sputum cytology, and a questionnaire for 541 brick makers, 253 clay pipe makers, and 785 controls. The ranges of personal exposures to respirable free silica were 0.4-692 $\mu g/m^3$ in brick plants and 8-200 $\mu g/m^3$ in pipe plants. Mixing operations in brick plants resulted in the highest geometric mean concentration of all job categories, 113 $\mu g/m^3$. For 4 of the 10 job categories surveyed in brick plants, geometric mean exposures were between 50 and 100 $\mu g/m^3$.

Based on all of the medical tests, compelling statistical evidence did not exist to support the hypothesis of increased respiratory disease among brick makers in relation to the control group. However, a statistically significant decrement in the mean FEV_1/FVC for black brick makers was observed in comparison to controls. Also, white brick makers had lower mean values for FVC and FEV_1/FVC than controls. The report cautioned that the apparent absence of marked respiratory disease in the groups studied did not necessarily mean that the same results would be found in workers exposed for longer periods, in retired workers, or in workers who left the industry and who may have been more sensitive to the dust. Recommendations were made to implement a combined medical/environmental monitoring program and to reduce the higher dust levels.

In 1978, Lesser et al. [8] examined 9 patients, 54-73 years old, with disabling pneumoconiosis. All had occupational histories of kaolin exposure in the manufacture of firebricks. All had radiographic evidence of pneumoconiosis, 6 had chronic bronchitis, 1 had emphysema, 1 had lung cancer, and 2 had tuberculosis. All but 1 had reduced arterial blood oxygen, and 1 had increased carbon dioxide retention. All had various degrees of airway obstruction. Most had worked with kaolin for many years, but 2 had only 2 to 4 years of exposure. The amount of free silica in respirable dust samples was measured at 1 firebrick factory. Results showed free silica concentrations as high as 4.5%, and workers working in dusty areas were frequently exposed to silica at concentrations in excess of the 1973 Threshold Limit Value (TLV) set by the American Conference of Governmental Industrial Hygienists (ACGIH). Around the crusher mill, as much as 8.9% of the airborne dust was cristobalite; in settled dust it was as high as 15%.

In 1977, Jain et al. [22] examined 151 workers in small-scale slate pencil factories in India. The shale used in making the pencils had a silica content of 69%, and the authors believed that the working conditions were poor. Of the 151 workers examined, 144 (95%) were men. One hundred twenty-four of the workers (82%) were 14-30 years old. Seventy-eight percent had at least one of the following complaints: productive cough (75%), exertional dyspnea (46%), chest pain (28%), and blood-stained sputum (4%). The overall incidence of radiographic evidence of silicosis was 57%

(82 workers). The incidence and severity of silicosis appeared to be directly related to the length of exposure. Silicosis was observable in some workers after only 2 years of exposure.

In 1980, Glover et al. [13] performed a cross-sectional survey of respiratory disease in Welsh slate workers. Employed slate workers, persons identified through electoral registers as previous slate workers, and a control group not exposed to any dusts were examined. There were 725 men qualifying as slate workers without other dust exposure, and 530 nonexposed men. Ninety-four percent of the total population sample attended a special clinic for medical examinations. The following information was collected for each man: age, height, tobacco use, respiratory disease symptoms, chest radiographs, lung function tests, and occupational history. The prevalence of pneumoconiosis was high in slate workers; one-third had some radiographic evidence of silicosis. Ten percent of the slate workers had at least grade II pneumoconiosis. However, there was little evidence of progressive massive fibrosis (PMF). PMF diagnosis was difficult because similar radiographic shadows were observed in persons who had recovered from tuberculosis. Many of the older men in both groups, including controls, had chest radiographs indicative of healed tuberculosis. About 33% of the slate workers complained of cough and phlegm, and about 17% had at least grade II dyspnea. These rates were much higher than observed for the control group, even though both groups contained similar percentages of smokers. These symptoms showed a strong correlation with a degree of fibrosis. FEV_1 and FVC were similarly affected, with the greatest decreases in persons with the most severe pneumoconiosis.

The authors [13] estimated (based on their data) that all of the Welsh underground miners who had opened new extraction areas and the finishers (grinding and polishing) employed in very dusty areas would develop at least grade II pneumoconiosis after 20-30 years of employment. They also estimated that 50-60% of the underground rockmen and slate makers (splitters and dressers) would be similarly affected if exposed throughout a working lifetime. They felt that 15-20% of the quarry rockmen (open pit), sawyers, and underground assistants would also be affected. However, Glover et al. noted limitations of their study. These included the lack of reliable dust measurements, the failure of 6% of the population identified to submit to testing, and the use of a survivor population. The last factor is the most significant, because severely affected workers may have died and, thus, would not have been counted in the study.

In 1972, Kawakami and colleagues [25] described three cases of pneumoconiosis among five workers who had long-term exposure to a fine mineral powder used in the Japanese furniture industry for filling the grains of surfaces of wooden products. About 50% of this material was composed of quartz, 39.4% of which was less than 5 μm in diameter. The three workers, one woman and two men aged 53, 45, and 58, had been exposed 12, 26, and 40 years, respectively. All were moderate smokers, and the woman also had active pulmonary tuberculosis.

Even though there is widespread potential for exposure to silica in the building trades industry, almost no information exists concerning silicosis in this industry. One group, drywall tapers, was examined, but they were also exposed to asbestos in most cases [61,62]. In a 1978 evaluation of 15 consumer spackling and patching compounds and 10 industrial drywall taping compounds, 15 of the compositions contained 5-70% quartz [61]. Workers can be exposed while mixing the spackle material and during sanding. In a 1978 clinical field survey of 114 taping workers, 25 (22%) had chronic bronchitis, 10 (9%) had dyspnea upon exertion, 10 (9%) had dry rales, 15 (13%) had bronchitis, and 9 (8.3%) had abnormal chest radiographs [62]. Abnormal chest radiographs occurred only in workers employed at least 10 years as drywall tapers. Most (69%) of the persons with adverse changes smoked. These workers were also exposed to asbestos, and it is not clearly indicated in the report that the toxic signs found had any relationship to silicosis.

A survey of the prevalence of silicosis in ferrous foundries in Denmark was conducted from 1967 to 1969 [10]. A total of 146 cases, 127 of which were stage I, were found among 4,061 persons. In 896 fettling shop workers, the percentage with silicosis progressed approximately linearly from none observed at 6 years of exposure to 50% with 30 years of employment. Molders showed a low incidence of silicosis, only 5% after 35 years of exposure, but then a rapid increase to 35% after 40 years. In 1977, Bonnevie [10] obtained occupational histories for 1,103 of the workers, 37 of whom had silicosis. Eleven of the 38 foundries initially examined were represented. In all cases, the workers with silicosis tended to have been exposed to silica longer than their coworkers who had no radiographic evidence of silicosis. The author concluded that conditions in the working environment were primarily responsible for the development of silicosis, and that individual susceptibility played only a minor role.

V. EXTENT OF EXPOSURE AND CONTROL MEASURES

Sources of Exposure

In a 1977 report completed by the Swedish National Board of Occupational Safety and Health [24], industries were classified by dust exposure levels into four groups. Group 1 was considered to have very high dust exposures. Group 2 dust exposures were in a median range, but still exceeded the Swedish standard for silica (0.1 mg/m^3). Group 3 industries were rated acceptable, with medium exposure levels. Group 4 was rated good with low dust levels. The four groups are listed in Table V-1.

TABLE V-1

SWEDISH INDUSTRIES CLASSIFIED BY DUST EXPOSURE LEVEL

Group 1 Very High Dust Exposure	Group 2 Medium Level Dust Exp. ≥ 0.1 mg/m3	Group 3 Acceptable Dust Exp. Levels	Group 4 Low Dust Exp. Levels
Blast-cleaning	Asphalt works	Manufacturers of:	Brick sawing
Bulk materials handling	Brick works	concrete products	Manufacture of:
Dry Mortar mixing	Building construction	concrete roofing tiles	bricks
Iron foundries	Diatomaceous earth	glue	ceramic products
Manufacture of:	handling and	honeycomb clinker	grinding wheels
roofing felt	extraction	plastic products	ready-mix concrete
scouring powder	Ferrous-alloy plants	rubber products	Underground mining
Mechanical street	Glass industry	sealing compounds	
sweeping	Lime and Dalomite plants	Surface mining	
Rock crushing	Manufacture of:		
Stone-working	abrasive paper		
	cement		
	cleaning and polishing materials		
	emery cloth		
	paint		
	prefabricated building materials		
	welding electrodes		
	Non-ferrous foundries		
	Primary crushing		
	Potato handling		
	Quartz industry		
	Steel foundries using quartz sand		
	Steelworks		

Adapted from Reference [24]

Silicon dioxide can be an undesired constituent present when a desired substance is obtained. Examples include coal and hard rock mining, and refining of metals such as copper, nickel, or gold. Cristobalite or tridymite can be introduced in high temperature processes such as calcining of diatomaceous earth or manufacture of silica brick [1]. Certain industrial processes use materials containing silica as part of the plant equipment. Examples include the use of sand in abrasive blasting, sand casting in foundries, and certain pyrometallurgical processes.

Silicon dioxide may also be present in natural materials that have commercial value. One example includes certain types of quartz, such as opals, that are of great value as jewelry. More common examples include sand, granite, and sandstone used in building materials, asphalt, and tombstones. This category could also include clays used in ceramics and firebrick manufacture. Silica is also used in a relatively pure form as an ingredient in a mixture to form a final product. This category would include the manufacture of glass and asbestos cement pipe and the use of silica sand in cement or mortar. Powdered silica is used in paints, porcelain, scouring soaps, dental fillings, and wood filler; pure quartz is used in electronic equipment.

The degree to which workers are exposed to free silica can be quite variable even within similar industrial categories. For example, in mining (open pit or underground), the technique used for removal of the mineral deposit and the percentage of silica contained in the surrounding rock all affect worker exposure. Work that tends to scatter finely divided silica into the air, often in poorly ventilated areas in restricted spaces, is especially hazardous. These conditions are frequently encountered in abrasive blasting, and Great Britain has banned the use of silica sand in blasting since 1949 [1]. Other factors that affect worker exposure involve the physical form of the material being handled. Operations such as quarrying of sandstone or granite can be particularly hazardous, because the associated cutting and grinding operations can disperse considerable quantities of free silica into the air. In contrast, many uses of materials containing silica in the building trades do not involve extensive cutting or milling of materials containing silica, and work in these trades has not been traditionally associated with a high incidence of silicosis.

In pyrometallurgic techniques for the refining of copper and nickel, silica must be present to separate iron as a silicate in slag from the other metals [87,88]. If the ore does not contain sufficient silica, a relatively pure form of silica may be added to certain furnace melts. This can cause the release of free silica into the workplace atmosphere. In such situations it may be harder to control silica exposures than exposures to metals, as is evident in the results of a NIOSH survey at a ferronickel refinery [87]. The average nickel concentration in 78 personal samples was 30 $\mu g/m^3$. None exceeded the current Federal standard, and only 18 were in excess of 15 $\mu g/m^3$. In contrast, 3 of 30 personal samples exceeded the Federal standard for free silica. Only 3 samples were below the exposure limit of 50 $\mu g/m^3$ recommended by NIOSH.

In 1978, data from the National Occupational Hazard Survey (NOHS) indicated that about half of all workers potentially exposed to free silica are employed in manufacturing [84]. Five Standard Industrial Classifications (SIC's) within the manufacturing industry account for 73% of the total employment; these include stone, clay, and glass products (32); primary metal industries (33); fabricated metal products (34); machinery, except electrical (35); and transportation (40-44). Among full-time workers potentially exposed to silica, 86% are employed in industries that account for 91% of the total workers.

The percentages of exposed workers who had access to medical examinations are given in Table V-II. A number of workers receiving periodic medical examinations, also work in areas where measures have been taken to control exposure. In Table V-III, the percentage who receive chest x-rays and who work in controlled or uncontrolled areas is also listed.

TABLE V-II

PERCENTAGE OF WORKERS EXPOSED TO FREE SILICA
WHERE
MEDICAL EXAMINATIONS AND CONTROLS WERE AVAILABLE

Industry	SIC	Periodic Chest X-rays	Pulmonary Function Tests	X-rays and Personal Protection	X-rays and Other Controls	X-rays but No Controls
All		33	20	1	17	27
Contract Construction	15	60	9	0	1	4
Manufacturing		46	30	2	28	35
Stone, Clay, Glass	32	50	37	2	31	47
Primary metals	33	61	41	2	36	52
Fabricated metals	34	24	7	0	19	15
Machinery, not electrical	35	41	23	6	28	29
Transportation	40-44	56	46	1	42	41

Adapted from Reference [84]

Control Methods

In 1977, the Swedish National Board of Occupational Safety and Health [24] examined a number of industries using or producing silica-containing materials. It also made specific recommendations for dust control in particularly hazardous operations in those industries. The following synopsis of its recommendations emphasizes applications in the stone-working industry, abrasive blasting, sand casting operations, and the relining of furnaces.

Several types of stone, including granite, gneiss, marble, limestone, slate, sandstone, and soapstone, are handled in the stone-working industry [24]. The percentage of quartz in these stones varies considerably. Several operations were considered especially hazardous. Table V-III lists these operations and measures recommended for dust control [24]. In addition to these controls, specific recommendations were given for free-jet blasting. These included the use of a general ventilation system with a downward airflow speed of 0.3–0.5 meter per second admitted as diffusely as possible. In addition, the use of a gas-tight room with smooth walls was recommended, and the operator was to wear an air-supplied breathing apparatus with a hood or helmet.

TABLE V-III

STONE-WORKING CONTROL MEASURES

Operation	Average Respirable Dust Exposure (mg/m^3)	Control Measure	Industry
Jet-Piercing	21*	No specific control device available	Granite quarrying
Dry-drilling	25	Portable or central dust exhaust units	Quarrying large stones
Wedge-hole drilling blocks	17	Exhaust hoods	Splitting large
Edge chiseling, hammer dressing, and smoothing	24	Local exhaust	Finishing edges on building stones
Sandblasting	32	Use of enclosed blasting machine or sand substitutes	Finishing surfaces
Dry grinding	18-42	Local exhaust on grinding machine or exhaust hood	Tombstones, windowsills
Finish lettering	10	Removable device with exhaust capacity of 800 m^3/hour	Tombstones
Assembly	10-80	Local exhaust for drilling and grinding hoods for facade stones; no recommendations for sawing	Drilling, sawing, grinding, and facade stones in building construction

*Varies considerably with wind conditions
Adapted from Reference [84]

Blasting is a process used for cleaning rusted metallic surfaces. Blasting materials can be composed of silicon carbide, aluminum oxide, or steel shot, as well as quartz sand [24]. In 1977, the Swedish National Board of Occupational Safety and Health [24] recommended the following measures to reduce exposure to quartz dust during blasting:

(1) Substitution of quartz-free blasting materials.

(2) Placement of objects to be blasted in a closed cabinet or chamber so that the operator can perform his/her work while outside the chamber.

(3) A down-draft ventilation system for rapid removal of dust after blasting, with air exhausted through the floor grating and make-up air diffusely admitted through a perforated ceiling.

(4) Blasting material automatically returns to the blast bell to avoid manual handling.

(5) A hood-type supplied air respirator for blasters who must work inside the blasting chamber.

(6) Blasting material deposited in the blasting area after blasting has been completed, is removed by a powerful vacuum cleaner.

Sand casting in steel, iron, and nonferrous foundries has been associated with an increased risk of silicosis [24]. Sand molds are composed of quartz sand, a binder, and an additive. In some, but not all cases, sand free of quartz is a suitable substitute. Operations in which there is potential worker exposure to free silica include sand preparation, molding, coremaking, casting, knock-out, decoring, fettling, preliminary cleaning, dressing, and clearing of furnace and ladle linings. In 74 Swedish nonferrous foundries examined in 1969 [24], dust exposure was greater in mechanized sand preparation areas than in areas where the sand was prepared manually.

The Swedish National Board of Occupational Safety and Health [24] recommended enclosure of bins, feeders, mixers, sand screens, sand beaters, magnetic separators, and conveyers to lower exposure in sand preparation. It noted that attention must be given to condensation problems in enclosed sand preparation equipment, and recommended the use of special openings to admit dilution air.

The Swedish studies [24] found dust exposure in molding to be less than in most other foundry operations. To control the dust, they recommended transfer of the material by enclosed belt or pneumatic conveyers, placement of the material into closed containers at the molding sites, use of concrete flooring and regular vacuum cleaning to prevent dispersion of dust from the

floor, installation of a conveyer below each molding site to carry away molding material, cleaning of rims of molding boxes with a nozzle connected to a central vacuum, and a moisture content in molding material of at least 2.5%.

The coremaking area was the least dusty part of the Swedish foundries examined [24]. Nevertheless, recommendations for further dust reduction were made; these included the use of enclosed conveyers and closed hoppers for carrying dry sand to the coremaking machines, regular vacuum cleaning of floors and machines, and local exhaust ventilation. The casting areas were considered very dusty, and the Swedish Board recommended that casting be carried out close to a specially designed hood. In the foundries, the average dust concentration in the knock-out operation exceeded the Swedish exposure limits. In automated plants, enclosure and sound isolation were recommended for knocking out. Exhaust hoods tailored to the process were recommended in areas requiring some manual handling; the use of exhaust ventilation below the shake-out grate in the roller-table mechanization was considered ineffective.

Fettling was considered the most difficult dust control problem in foundries [24]. The average dust concentration for all the foundries examined exceeded the Swedish limit. A particularly dusty process was free-jet blasting with quartz dust. Dust control measures similar to those in preliminary cleaning and dressing were recommended. It was also recommended that rotary tumbling barrels be rubber-lined for noise control, and exhaust ventilation equivalent to 2,000-2,500 m^3/hour (1177-1471 cfm) for a 1-meter diameter barrel through one of the trunnions, be provided. Additional suggestions included minimizing the need for dressing by changing the prior steps, automating the process, and installing local exhaust.

Furnaces and ladles used to hold molten metal are often lined with material containing quartz, and excessive exposure to crystalline silica was found in furnace departments of Swedish iron foundries [24]. Recommendations for reducing exposure during clearing and relining of furnaces and ladles were made.

In steelmaking, dust problems were encountered in areas such as the preparation of refractory mortar and clearing and relining of furnaces and ladles [24]. Not all bricks used for refractory linings contained quartz, and it was not possible to use low-silica bricks in all processes. The Swedish Board recommended that ladles and furnaces, if possible, be cleared mechanically in a separate area, possibly outdoors and away from the operable furnaces. Ladles can be cleared by a pneumatic breakout tool with a sound-isolated and pressurized cab for worker protection. Continuous flushing with water aids in dust abatement. Certain furnaces, such as converters, are generally too large to remove from the work area. The Swedish Board recommended the use of an exhaust hose with a 10,000 m^3/hour (5,886 cfm) capacity placed close to the converter bottom to lower the dust levels. It also recommended a similar system operating at 2,000-5,000 m^3/hour (1177-2542 cfm), depending on the diameter, for

ladles, and that a local exhaust ventilation on the pneumatic tools be used for chiseling out the lining. Relining was found to be a less dusty operation than clearing. Nevertheless, downward-directed airflow was also recommended for relining. In addition, vacuuming of the new bricks on the pallet and local exhaust and well-designed hoods for brick sawing were recommended.

The Swedish Board [24] also examined other industries using silica and made recommendations for control of dust. In rock and gravel crushing plants, dusty operations included drilling, loading, transporting, crushing and screening. In building and civil engineering, exposure occurred in preparation of the building site during rock drilling and loading and transport of blasted rock. During building construction, workers were exposed to free silica while chiseling, plugging, grinding, sawing, rough casting of building materials, and constructing floating inner floors. In asphalt batching plants, the crushed stone added to the asphalt emulsion was a source of exposure throughout the process from transportation through stockpiling.

In the ceramics industry, preparation and surface treatment of bricks and clay preparation and grinding, scouring, and finishing operations for earthenware and porcelain were especially dusty. In the glass industry, quartz exposure from the sand could occur during the unloading of raw materials, charging of furnaces, manufacture of crucibles, and in batch processes (in comparison with automated processes). In the manufacture of asphalted roofing felt, a mineral coating that often contained quartz and a bottom coating of sand or soapstone were added to asphalt impregnated felt. Potentially dusty operations involved handling of crushed materials in delivery, batch mixing, and sprinkling in the coating machine.

A number of recommendations were made by the Swedish Board for dust control [24]. These included:

(1) Automation, process enclosure, and ventilation for open processes.

(2) Enclosure of storage bins and conveyer belts.

(3) Use of paved roads and ventilated cabs for transport of materials.

(4) Use of stationary equipment such as saws instead of portable units so that the procedure could be carried out in a ventilated hood.

(5) Use of special exhaust devices such as drills with local exhaust devices and chiseling hammers with exhaust bores in the center.

(6) Wet grinding or local exhaust on the wheel.

(7) Washing or wetting of materials such as rocks, gravel, or sand that contain fine dusts.

(8) Paving of roadways in work areas.

(9) Spraying of stockpiles with a synthetic resin or asphalt emulsion.

(10) Regular vacuum cleaning of floors, machinery, and dust-covered materials within process areas.

An alternate to engineering controls would be the substitution of less hazardous materials. In 1979, Davis [89] described a small foundry operation that substituted olivine for silica-based sand in selected processes. This change was an attempt to provide a low-cost alternative to extensive controls. Free silica concentrations were lowered, but, as would be expected, overall air quality was unimproved.

A 1979 NIOSH-funded study [121] of foundry engineering control technology involved an in-plant review of 24 ferrous and nonferrous foundries. The participating plants had been selected to represent a wide area in the foundry industry and to represent the widest range of engineering process controls possible. The final conclusions of the study listed four areas of concern: air contamination, heat stress, nonionizing radiation, and noise reduction.

The findings on air contamination addressed the following areas: (1) cleaning and finishing of ferrous castings, (2) green sand systems, (3) melting and pouring, and (4) molding and coremaking. Area 1 was considered the most difficult area for controlling dust levels, and current ventilation practices are inadequate. Electrostatic fog, a new commercially available method for supplemental dust control, is promising. Substitution of nonsilica molding materials (olivine, zircon, chromite) has the capability of reducing silica content of foundry dust by 80%. However, this substitution will not eliminate silica from a foundry, and little is known about the inhalation toxicology of these alternate materials. Respirable silica exposure in melting and pouring areas varies with the type of furnace, charging procedures, and control methods used to protect operating personnel.

Recommendations to improve foundry respirable silica levels include reduction of burn-on caused by metal penetration into mold and core materials, use of air-supplied respirators on hoods in cleaning rooms, use of high-velocity, low-volume (HVLV) control methods, continued exposure and background sampling, and continuing study of ventilation and control methods to improve overall workplace air quality.

These conclusions and recommendations have been incorporated into a new criteria document, "Criteria for Assessment and Control of Health Hazards in Foundries" (unpublished) [122].

NIOSH conducted a Control Technology Assessment in three silica flour milling plants. This assessment had as its major emphasis, the reduction of exposure hazards in the industry. In a 1982 report by Caplan et al. [90], three dust control measures were recommended: (1) injection of agglomerating agents into the products; (2) water spraying of surfaces of silica flour-filled bags during conveyance; and (3) bulk loading into hopper trucks and railroad hopper cars under exhaust ventilation control. Dust levels in the subject plants have been significantly reduced by these measures and generally improved housekeeping practices.

VI. CRYSTALLINE SILICA SAMPLING AND ANALYSIS

Introduction

The NIOSH criteria document in 1974 on crystalline silica recommended respirable mass size-selective measurement (referred to as the respirable mass method) to collect and measure the respirable portion of airborne dust and x-ray diffraction to measure the crystalline silica content [1]. Both the sampling and analytical methods were relatively new at the time, but in the eight years intervening, both techniques have been refined and are in widespread use today. The x-ray diffraction method for analysis has been collaboratively tested (by Stanford Research Institute).

Sampling

(a) Dust Count and Respirable Mass Methods

Historically, airborne dust levels have been determined by a particle count method in which particles are collected by impingement and counted by using an optical microscope. The respirable size range is determined by the visibility limitations of the optical microscope at the lower limit, and by a subjective judgment at the upper limit. This collection method was in use during the period in which many epidemiological assessments of the hazards from crystalline silica exposure were made, and there has been a resistance to abandoning this technique.

The more recent size-selective respirable mass sampling technique has been in use, on a regular basis, for several years now, and is in fact, the method recommended in the 1974 criteria document on crystalline silica [1]. Dust particles are collected in a two-stage sampler. The first stage is a 10-mm nylon cyclone operated at a sampling rate of 1.7 liters/minute. At this sampling rate, the cyclone selects the dust fraction from 2 to 10 μm (aerodynamic diameter). This respirable fraction is collected on the second stage, which is a 37-mm, 5 μm pore size polyvinyl chloride membrane filter. The dust mass is measured and analyzed for the crystalline silica content. This method has been most applicable in estimating silica exposure during a workshift.

In addition to the usual factors such as adequacy of the pump and calibration procedures, Williams and Hawley [91] took into consideration several other factors when obtaining samples for analysis of respirable free silica. These included the need to operate sampling devices using cyclones at the recommended flowrate of 1.7 liters/minute, so that actual pulmonary deposition could be closely approximated. In addition, it was recommended that workers not remove the sampling equipment (and inadvertently contaminate the filter by inversion of the cyclone). The authors preferred to use a 37-mm polyvinyl chloride membrane filter with a nominal pore size of 5 μm, because such filters have a low moisture absorption capacity.

However, they cautioned that static charges should be removed from the filter before weighing to obtain accurate results. They also commented that the NIOSH recommendation of 50 μg free silica/m^3 was more stringent than the OSHA standard of 10 mg/m^3/% quartz + 2; when the silica content exceeded 2%, they recommended the use of a weighing balance in which the smallest weight change detectable would be 1 μg.

Knight et al. [92] compared the results of different sampling strategies in hardrock mines in Canada. Short-term sampling of 5 ml of air with a konimeter was compared with full-shift size-selecting gravimetric sampling in mines with high (above 30%), medium (10-30%), and low (below 10%) quartz content. There was a poor relationship between the daily mean number of dust particles/m^3 and respirable quartz dust concentrations. The relationship was worse when dust counts were compared with total respirable dust concentrations.

Another factor was the variability of the amount of combustible dusts (i.e., diesel fuel emissions) in the gravimetric samples. Large variations were found in the composition of the respirable portion of dusts, even for mines considered to have a high quartz content. In these mines, 22% of the dust (range, 0-70%) was composed of quartz and 57% (range, 8-96%) was combustible dust. The remaining material was not analyzed. For 23 mines, quartz exposure varied two- to four-fold over 3 to 6 shifts, and shift-to-shift variations were somewhat greater for each working station. The amount of quartz measured for different operations was extremely variable for drilling, slushing, and pipe and track operations, and the results were also variable for mucking, drilling, and haulage, and for shift bosses. These results clearly demonstrated the difficulties that could be encountered in attempting to accurately assess exposure in hardrock mines. Thus, it would appear especially difficult to establish reasonable dose-response relationships for silicosis in studies of such workers using particle counting techniques.

As in a study by Ayer et al. (who found a correlation between sampling methods of 10 mppcf and 0.1 mg/m^3) [7], two other studies have compared the results of paired samples measured by the respirable mass and dust count methods [93] (Johns-Manville Corporation, written communication, August 1976). Both of the latter studies concluded that the respirable mass method yielded results indicative of a greater hazard than the dust count method, which would pose difficulties if both methods were allowed by a standard.

Blehm and Buchan [93] collected 30 pairs of 20-minute breathing zone samples during and after clay mixing operations in a small pottery factory operating shop. The quartz used in making the clay was 99.8% pure, and this figure appeared to have been used in determining the permissible exposure limits. When the results of the 20-minute sample collections were converted to TWA's, only samples collected during mixing exceeded OSHA's permissible limit of 2.72 mppcf, and the mean values for all sampling periods exceeded its permissible limit of 0.1 mg/m^3. Furthermore, the estimated decrease in concentration after completion of mixing depended on the method used for

the measurement (from 3.4 to 1.3 mppcf compared with 1.17 to 0.22 mg/m^3). Because particle size also decreased from a mean of 1.39 μm to 0.8 μm, these results tended to support the contention of the authors that the presence of a few large particles greatly affected the results obtained by the respirable mass method. However, the implication that the number of particles present was a better estimate of exposure than mass, which appeared less demonstrable, because particle size selectors took into account the percentage of larger particles that could become deposited in the lung [91,94]. The presumption that all airborne dust found was from quartz can also be questioned.

Chase examined 191 pairs of samples collected at an asbestos-pipe manufacturing facility (Johns-Manville Corporation, written communication, August 1976). Each result was described as being below 50%, 50-100%, or above 100% of the current OSHA standards for silica. Of the 191 paired samples, 127 yielded equivalent results by both respirable mass and impingement measurements. Only 30 samples gave values above the standard by one method: five were above the dust count standard but not the respirable mass standard, while 25 exceeded the respirable mass standard but not the dust count standard. However, results of samples measured by the respirable mass method were more often in the 50-100% range than paired samples collected by impingement. Chase also performed a statistical analysis on the results of a previous study of exposure levels in the Vermont granite sheds, and he concluded that hazards evaluated by impingement and respirable mass were significantly different. When the two measurements were not in agreement, the respirable mass measurement resulted in "more hazardous" readings twice as often (32 to 16).

NIOSH has calculated the percentages of free silica in each of the Johns-Manville samples and compared conversion factors for those containing 1-10% and 1-15% silica with the conversion factors from the granite shed studies [95]. There were 95 and 123 data pairs in the two respective ranges. From average dust counts, total respirable mass, and percentages of silica for each of the 11 locations examined by Johns-Manville, NIOSH calculated overall TWA's. For the 1-10% silica range, TWA's were 3.3 mppcf, 0.44 mg/m^3, and 5.8% silica. For the 1-15% range, corresponding values were 3.3 mppcf, 0.43 mg/m^3, and 7.5% silica. The conversion factors calculated from these results were 10 mppcf = 0.076 mg SiO_2/m^3 (1-10%) and 10 mppcf = 0.101 mg SiO_2/m^3 (1-15%). Complete equivalency between these calculations and those of the granite sheds would not be expected, because the granite shed data averaged 4.4% silica and the two dust samples would not be expected to have identical physical characteristics. Considering these points, the results are in extremely close agreement.

(b) An Alternate Method

In 1975, Tomb and Treaftis [96] reported the design and testing of a disposable, two-stage respirable dust sampler. The first stage was an integral flat plate impactor that removed particles with an aerodynamic diameter greater than 10 μm. The second stage collected particles on a

37-mm membrane filter (5-μm pore size). The unit was not commercially available, and the impaction head might require some redesign for mass production. The impactor was designed to operate at a flowrate between 2 and 3 liters/minute. Tomb and Treaftis [96] suggested the following advantages of the impactor unit over the cyclone: (1) reduced analytical error; (2) elimination of cyclone maintenance and replacement; and (3) improved technical integrity of the sampling head.

In view of the fact that the size-selective respirable mass method for crystalline silica sampling has now been in use for over ten years, and that during that time OSHA has collected more than 23,000 samples for crystalline silica analysis in addition to NIOSH surveys, and studies by industrial hygiene groups in the private sector, a significant data base has been accumulated for further evaluation of this technique. The European Industrial Hygiene Community has also adopted the mass sampling approach.

It is now possible not only to sample from a selected range of particle sizes, but to characterize the particle size distribution of a given sample. These advancements in sampling technology coupled with a rapidly and steadily expanding body of exposure data offer a strong argument for the turn to the mass standard and retirement of the particle count approach.

Analysis

(a) Introduction

The NIOSH Manual of Analytical Methods [51] includes procedures for the analysis of free silica by x-ray diffraction [93,94], infrared spectrometry [93], and by a wet chemical spectrophotometric method [93]. NIOSH's criteria document recommended the x-ray diffraction method, but allowed for the use of either of the other two methods where warranted. Since that time, emphasis has been placed on x-ray diffraction methodology, and there have been many refinements.

The method of analysis given in the crystalline free silica document [1] was essentially P&CAM 109 of the NIOSH Manual of Analytical Methods [51]. This method, which used an internal standard, was evaluated under contract [103], and the conclusion reached was that an external standard method was consistently more precise. Such a method, including an absorption correction procedure [40,53], was written up as P&CAM 259 [51].

(b) X-Ray Diffraction

In x-ray diffraction analysis, dust samples are dispersed onto a suitable filter medium and irradiated with either Cu K-Alpha (0.1542 nm radiation from a copper target tube) or Mo K-Alpha (0.07107 nm radiation from a molybdenum target tube). Each free silica polymorph is quantitatively measured by comparing the intensity of a diffracted line specific for the polymorph with an appropriate standard curve. The x-ray

diffraction measurement is subject to several interferences and experimental error. Potential errors arise from sample preparation, particle size distributions, matrix effects, filter background effects, and the presence of interfering minerals. Various methods of compensation have been proposed. The crystalline silica criteria document [1] discussed methods for correcting for interfering minerals and background intensity caused by x-ray fluorescence.

Anderson [97] considered direct interferences as the main source of error, but Altree-Williams et al. [98] did not experience significant difficulties in analyzing samples from a variety of industries. Altree-Williams and coworkers [98] collected samples and standards on polycarbonate filters and analyzed them without redeposition. The polycarbonate was backed by a silver filter for analysis so that absorption corrections could be made if necessary. Samples were analyzed from among the following: cement, ceramics, clay, coal, granite, gypsum, limestone, sand, sandstone, brickworks, and ferrous and nonferrous foundries. No problems due to interference were found, and the authors concluded that interferences were not a major limitation of x-ray diffraction analysis of quartz.

Particle size distribution has an effect on x-ray diffraction line intensity, and one article discussed the potential magnitude of error and the need for size matching between samples and calibration standards [99].

Anderson [97] considered the thin layer of amorphous silica, which normally coats the surface of free-silica particles, and he suggested that this layer may be responsible for the decrease in the diffraction intensity observed in x-ray analysis of quartz particles less than 2 μm in diameter. Knight [100] in response to Anderson's article, noted that errors from particle size distribution and the amorphous layer should cancel out when calibration standards and dust samples are equivalent in particle size distribution.

Several groups have evaluated the x-ray diffraction method for the measurement of quartz, but no consensus has been reached concerning several points, including methods to correct for matrix effects [51,101,102,103, 104,105,106] and the choice of an acceptable filter [99,102,103,105,107, 108,109,110,111]. An important matrix effect is the absorption of radiation by the dust in heavily loaded samples. How much the intensity of the diffracted radiation is reduced depends on the composition of the dust, the dust layer thickness, and the angle of diffraction. This matrix effect can be corrected by either an internal standard method [51,102] or by a direct approach [51,101,103,104]. For the internal method, a known amount of a standard mineral is mixed with the calibration standards and with the sample. The ratio of the intensity of the standard phase to that of the sample phase is then used as a quantitative measure of the amount of sample material present. The standard mineral must not be a component of the sample dust or yield a diffraction line which overlaps with a line from a

component of the dust sample. Ideally, it should have a strong diffraction line near the free silica diffraction line of interest.

The direct method corrects the matrix effects by measuring the attenuation of a diffraction line from the filter medium, or a standard mineral attached to the sample holder beneath the filter [51,101,103,104]. Its diffraction line must also be free from overlap with diffracted radiation from the sample. Different filter types produce different levels of background upon which the sample peak is superimposed. This background intensity should always be subtracted from the peak intensities. High background levels have an adverse effect on detection limits; several researchers have chosen the silver membrane filter based on its low background [51,102,103,104].

Since completing the criteria document, NIOSH has issued two revised methods (S315 [101], P&CAM 259 [51]) for x-ray diffraction analysis of free silica. In the first method (S315), the dust is collected on a 25-mm diameter, 1.2-μm pore size silver membrane, and the silica is measured directly using the silver diffraction peak to correct for matrix effects. In the second method (P&CAM 259), the dust is collected on a polyvinyl chloride filter (25-mm diameter, 5-μm pore size). This filter is subsequently ashed, and the residue is redeposited on a 0.45-μm pore size silver membrane, at which point the methodology becomes essentially the same as that in S315. The second method (P&CAM 259) advocates ashing and redeposition in order to ensure that the deposition will be homogeneous and of uniform thickness, a rigid requirement for the direct method.

Both methods specify the use of a copper target x-ray tube and instrument calibration by a mica or silica standard (2-theta equals 26.9 for Cu K-Alpha irradiation). Measurement of the free silica mass requires calculating a calibration constant for each crystalline polymorph. While S315 recommends preparing free silica standards either in a dust dispersion chamber or from dispersals in isopropyl alcohol, P&CAM 259 recommends only the dispersal technique. Method S315, is based primarily on work by Peters [103], which eliminates several pretreatment steps, including filter ashing, addition of the internal standard, and redeposition, but it does not guarantee depositions of uniform thickness.

Peters [103] screened the following internal standards: alumina (Al_2O_3), calcite ($CaCO_3$), zirconia (ZrO_2), silicon (Si), and reagent grade fluorite (CaF_2). He reported that reagent grade fluorite produced a poor diffraction line intensity, and he recommended the use of silicon. Bumsted [102] described reagent grade fluorite as a inferior standard to "native" fluorite (written communication, August, 1976). Bumsted's studies, upon which the criteria document method was partially developed, used only native fluorite as an internal standard.

Peters [103] also compared the analytical precision and accuracy of the internal standard and direct methods. The quartz diffraction line intensity at 26.7 2-theta (Cu K-Alpha) was measured and matrix effects were corrected

by both the internal standard and direct methods. Overall, a similar accuracy was found for both methods, but the direct method was significantly more precise. Linear regression analyses yielded coefficients of variation of 42% for the internal standard method and 7% for direct analysis. Although the bulk of Peters' data demonstrated equivalent quartz values by both methods, some data cast doubt on the accuracy of the direct method. In a series of tests evaluating sources of error in the preparation of calibration standards containing 50 µg quartz (discussed later in this section), the direct method yielded an average of only 33.1 µg of quartz. Peters did not explain the cause for this low value.

The effects of several commercially available filters on the results of x-ray diffraction analysis have been examined by several groups. Peters [103], Knight et al. [92,109], Allen et al. [110], Pollack [111], and Edmonds et al. [99] analyzed the dust directly on the collection filter. Bumsted [102], Freedman et al. [108], and Mangia [107] redeposited the dust onto a fresh filter for analysis. Bumsted [102] and Peters [103] both observed a broad diffuse background for all organic filters evaluated at 26-28 2-theta (Cu K-Alpha). Table VI-1 summarizes the relative x-ray intensities reported by Peters. Bumsted [102] evaluated several other membrane filters including Whatman 41® (cellulose), Sartorius® (cellulose nitrate), and G.E. Nuclepore® (polycarbonate). These three filters exhibited relative background intensities comparable to that of the Millipore® filter. Both authors concluded that the background from the organic filters was unacceptable for measurement, and they concurred that the silver membrane filter should be used. Also, both suggested rotation of the filter to correct the uneven distribution of dust on the filter surface, although no data were presented to prove that sample spinning is necessary.

TABLE VI-1

PROPERTIES OF RESPIRABLE DUST COLLECTION FILTERS

Type	Material	Manufacturer	Relative Intensity* (2 theta = 26)
Silver	Silver	Selas Flotronics®	1.0
VM-1	PVC	Gelman®	4.54
FWS-B	PVC	MSA®	4.65
DM-800	Acrylonitrile	Gelman®	8.00
AA	Cellulose esters	Millipore®	9.18

Adapted from Reference [103]
*Relative x-ray intensity measured on a clean filter with Cu K-Alpha radiation

Several groups [99,105,106,107,108] reported success with x-ray diffraction measurement of dust dispersed on organic membrane filters. According to Freedman and coworkers [108], the Gelman® DM 450 membrane filter exhibits a flat x-ray background in the region of interest (26.6 for Cu K-Alpha). A 100-μg sample of quartz deposited on a DM 450 filter yielded peaks of 300 counts per second (cps) while the background count was measured at 80 cps. Although Bumsted reported that the Sartorius® cellulose nitrate membrane filter has a background intensity more than nine times that of the silver membrane filter [102], Mangia found agreement between the results of x-ray diffraction and IR analysis of quartz using these filters [107].

Several investigative groups have directly analyzed quartz dust on the organic membrane filters used for collection [99,110,111]. Allen and coworkers [110] collected samples of sandblasting dust on 37-mm Millipore® filters. The dust samples were analyzed colorimetrically and by x-ray diffraction. In the x-ray diffraction measurement, the sample was rotated and the integrated intensity of the diffraction line at 26.6°(Cu K-Alpha) was measured by planimetry. Allen et al. [110] corrected for sample matrix effects using the broad diffraction peaks from the Millipore® filter. An intensity attenuation factor was calculated by comparing the background from the sample dust filters with the background from standards of pure quartz on filters. This factor was divided into the quartz integrated intensity to obtain a corrected intensity. The quartz mass was assessed by comparing the corrected intensity with standard curves. Allen and coworkers claimed an overall precision of ±20% (approximately two standard deviations) in the 10-μg to 8-mg range for the respirable quartz calibration curve data. Accuracy was ±35% for 10 field samples relative to the colorimetric method.

Pollack [111] evaluated the direct measurement of quartz on 37-mm polyvinyl chloride membrane filters with the sample restricted to a 16-mm diameter. He had seven such filters (a blank, and standards of 20, 60, and 100 µg) step-scanned on an instrument equipped with a rotating copper anode tube running at 11 kilowatts. He concluded that with this instrument it was possible to measure quartz directly on PVC filters with a sensitivity comparable to that published for silver filter work.

Edmonds et al. [99] directly measured silica particles collected on silver (0.45-µm pore size) and polyvinyl chloride (0.6-µm Millipore® and 5-µm MSA®) membranes. Restricting the collection area to a 12-mm diameter on 25- or 37-mm filters yielded greater sensitivity than using the whole surface. Thin deposits of quartz led to preferred orientation on smooth-surfaced polyvinyl chloride membranes and an increased x-ray diffraction intensity per unit weight compared with results using silver membranes. The x-ray diffraction response for the quartz standard, Min-U-sil 15®, was as much as three times that of an equal weight of Min-U-sil 5®. However, Min-U-sil 5®, with the smaller particle size, was preferred as a standard by the authors because it would more closely match a filter sample preceded by a cyclone.

Knight et al. [92,109], Leroux et al. [104], Peters [103], and NIOSH [51] examined several important considerations for use of silver membrane filters in the x-ray diffraction analysis of silica. Three methods are direct analysis procedures, and all compensate for sample self-absorption by comparison of the intensity of the silver diffraction line transmitted through the dust layer with the intensity from a clean silver filter [51,103,104]. Peters' [103] calculation of the quartz weight included a term for the intensity of the instrument calibration standard. He chose a mica chip for instrument calibration, while NIOSH [51] used a quartz stone and Leroux and coworkers [104] used pure quartz deposited on a silver membrane filter.

Peters [103] evaluated silver filters for field collection of samples. He compared the flow resistance and the retention of 0.8-µm particles on a series of 25-mm silver membrane filters with pore sizes of 0.8, 1.2, 3.0, and 5.0 µm. He found greater than 40% particle penetration for the 5.0-µm pore size and high flow resistance for the 0.8-µm pore size filter. He concluded that the 1.2-µm pore filter was the best compromise of flow rate and particle retention. Knight and coworkers [109] collected and analyzed dust samples on 37-mm diameter, 5-µm pore size silver membrane filters. They noted lower diffraction line intensities at low dust loadings which they attributed to two factors: high penetration of the dust into the filter pores and shadowing by the rough filter membrane surface.

Leroux et al. [104] and Knight et al. [92,109] compared the analytical efficiency of molybdenum and copper target tubes. The advantages of irradiation with the copper tube were better peak resolution, higher peak intensity, higher signal to noise ratio, and a shorter counting time for the

same statistical accuracy. The advantages of irradiation with the molybdenum tube were lower sample self-absorption and a longer linear operating range of quartz concentrations. Knight and coworkers preferred the copper tube; Leroux and coworkers preferred the molybdenum target tube.

Peters [103] evaluated sources of error in the preparation of standards by the liquid suspension method. Using a ruggedness test, he sought possible differences in precision and accuracy when the following factors were varied: solvent, time of powder dispersion by ultrasonic agitation, method to agitate the powder in the solvent, storage time of the stock suspensions, method for taking a suspension aliquot, silver filter pore size, filter cleanup, and method for drying the filter. Each sample contained 1 mg quartz, 15 mg alumina, and 4 mg silicon powder (internal standard) in 1,000 ml solvent. Quartz was measured by both the internal standard and direct methods. The only factor considered by the author to affect accuracy was pipetting of the sample aliquots, although other results presented in this same report contradict this conclusion. Peters preferred isopropyl alcohol to water as the solvent for standard preparation due to faster filtration speed, faster sample drying, and relatively low solubilities for some internal standards (calcite and fluorite).

The infrared spectrometric [112] and spectrophotometric [113] methods are still in use in the industrial hygiene community, but have been used with decreasing frequency as x-ray diffraction technology has advanced and the necessary equipment has become more widely available. NIOSH efforts have been expended almost exclusively in refining x-ray techniques. One natural phenomenon led NIOSH analytical chemists to use traditional chemical methods in solving a specific problem.

In 1980, when Mt. St. Helens erupted in Washington State and released tons of volcanic ash into the atmosphere, Public Health Service (PHS) personnel were called in to assess the potential health hazards. When the NIOSH laboratories made preliminary x-ray diffraction analytical runs, the resultant recorder tracings were impossible to interpret [114]. The plagioclase silicates, of which the ash was primarily composed, created strong interference masking possible quartz, cristobalite and tridymite peaks. This problem also resulted when infrared spectrometric analysis was attempted.

The wet chemical method [113] employs a hot phosphoric acid digestion step, which serves to dissolve silicates and retain the free silica in particulate form. This technique was used on the respirable fraction of the volcanic ash samples as a preliminary "clean-up" step prior to x-ray diffraction analysis. As a result, a relatively silicate-free sample was found and the presence of both quartz and cristobalite was confirmed. These findings were substantiated by evaluation of primary, secondary and tertiary diffraction peaks for quartz and cristobalite, and also by infrared scans of the "clean" samples.

The results of this study indicated the possibility of interfacing two distinctly different methods to solve inherent interference problems in complex samples. The work of Dollberg et al. [114] has documented one application of this principle; it is possible that this approach may be utilized in many instances where silicates and other minerals obscure essential diffraction peaks.

(c) Other Analytical Methods

Weiss et al. [115] reported improvements for quantitative differential thermal analysis (DTA) of quartz in dust. In their procedure, 40-mg samples containing as little as 1% quartz were packed between layers of pure silicon carbide in a 300-mg sample holder. A specially prepared microholder that required only 2 mg of material was also tested. Since DTA for quartz is subject to error from sample packing in the holder, Weiss and coworkers relied upon careful hand tamping to achieve reproducible packing. Particle size could affect the response in DTA of quartz [1,97,115]; therefore, Weiss and coworkers compensated for differences in particle size by grinding samples and measuring the average particle sizes of samples and standards with a combination of air elutriating classification and microscopic analysis. Peak height for an average particle size of 2 μm was less than half that for 5 μm particles. The quartz content in foundry dust was measured by micro DTA and x-ray diffraction (internal standard method), and the two were in agreement (Table VI-2).

TABLE VI-2

FOUNDRY DUST ANALYSIS FOR QUARTZ BY DTA MICROANALYSIS AND X-RAY DIFFRACTION

Sample	Micro DTA Results (%)	X-Ray Diffraction Analysis (%)
A	25.8	23.5
B	12.1	14.5
C	8.1	11.5
D	12.5	15.5
E	55.2	60.0

Adapted from Reference [115]

Hurley and White [116] developed an x-ray fluorescence method for the analysis of quartz in coal mine dust. The measurements were performed on a Siemens SRS-1 Sequential X-Ray Spectrometer equipped with a chromium target tube. The x-ray analyzing crystal was ammonium dihydrogen phosphate (ADP) that was oriented along the (200) diffraction plane. The analytical line used was Si K-Alpha at 143.67 2-theta. Samples were ground until they passed through a 325-mesh screen; then 0.5 g was mixed with 0.1 g of methyl cellulose and pressed into a 1 1/4-inch diameter pellet. The exact position of the Si K-Alpha peak was related to the composition of all material containing silicon. Silicates caused a slight peak shift of approximately 0.0125 2-theta for Si K-Alpha; quartz did not cause a peak shift. The spectrometer detector was positioned for a fixed time count on the low intensity side of the Si peak, at the angle expected for the peak half height. The measurement was repeated on the high intensity side. The peak intensity was equal to the added intensities of the two measurements and was, therefore, a measurement of the total silicon content (assumed to be silicates and crystalline silica in the coal dust). The peak shift was approximately equal to the difference between the intensities, and the ratio of the difference to the sum was a function of the amount of crystalline silica present for a given percentage of total silicon.

(d) Most Recent Efforts in Methods Research

The Inorganic Methods Development Section, Methods Research Branch, Division of Physical Sciences and Engineering, NIOSH, has recently completed, under contract, a collaborative test of P&CAM 259. Included in the collaborative test contract was an infrared spectrometry method for silica in coal dust, which was being tested in a joint effort with the Bureau of Mines.

The goal of this work was to verify the reliable performance of these analytical methods in a number of laboratories, and document their applicability and limitations. Both methods were being tested using samples from the same statistical procedures so that a valid comparison of the precision and accuracy of the two methods was possible.

Before a method can be collaboratively tested in a number of laboratories, it must be ruggedness tested to determine that minor procedural changes allowed by the method write-up do not affect the quality of the results. Several changes were made in the write-ups of these two silica methods as a result of ruggedness test. The final form of these methods will appear in a future revision of the NIOSH Manual of Analytical Methods, and in the final contract report [117]. The primary change in the methods has been the addition of procedures for working with: (1) calcite (both methods), and (2) Kaolinite (IR method).

Although the ultimate goal of a collaborative test (by NIOSH) is a "Class A-Recommended" method, neither the XRD or the IR method met requirements for this designation.

In the course of the test, both methods were optimized and ruggedized by the contractor and then tested by 15 representative laboratories.

In general, conclusions drawn from the collaborative test program were:

(1) Accurary: The two methods have low bias. On the average, the XRD method, the IR method, and a reference method produced approximately the same analytical results.

(2) Precision: In general, precision of analytical data is divided into three aspects: intralaboratory analytical precision, interlaboratory (multiple laboratories) analytical precision, and interlaboratory sampling and analytical precision.

In general, intralaboratory analytical precision was in the range of 7-8% relative standard deviation (RSD). When this data was combined with interlaboratory results, the RSD increased to about 17%. The total error increased markedly when interlaboratory results were calculated to include variability due to personal sampling techniques. For samples with loadings of 200 μg SiO_2, the total RSD was approximately 18%. This figure increased to about 32% for lesser sample loadings.

These error rates were considerably higher than expected. A NIOSH Class A method should yield an RSD of no more than 12.7% (results within 25% of the tone value 95% of the time). By this criterion, therefore, both the XRD and the IR methods fall short of Class A standards in that both interlaboratory analytical precision and combined sampling and analytical precision demonstrate excessive error. Both methods will be "Class B-accepted."

A full report of the collaborative test will be published late in 1983.

VII. INFORMATION GAPS

(a) Additional laboratory and epidemiological studies are needed to determine the relationship between silica dose, as measured by the respirable mass method, and the risk of developing silicosis. Potential effects on the kidney and the effects of silica on the incidence of lung cancer should also be assessed.

(b) The toxic effects of cristobalite and tridymite need further study.

(c) The exposure data from the OSHA inspection program and the NIOSH Health Hazard Evaluation Program should be studied in depth. Worksites, where hazardous exposures were noted, should be further studied to ascertain the degree of improvement in environmental conditions and the subsequent effect on the health of workers.

(d) Although completion of the collaborative test on XRD and IR methods for silica analysis has answered questions about accuracy and precision of these methods, there is still a need for advancement of the "state of the art." Further study directed toward the reduction of sampling error is needed.

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APPENDIX I

OCCUPATIONS WITH POTENTIAL EXPOSURE TO CRYSTALLINE SILICA

Abrasive blasters
Abrasive makers
Auto garage workers
Bisque-kiln workers
Brick layers
Buffers
Buhrstone workers
Casting cleaners (foundry)
Carborundum makers
Cement makers
Cement mixers
Ceramic workers
Chemical glass makers
Chippers coal miners
Construction workers
Cosmetics makers
Cutlery makers
Diatomaceous earth calciners
Electronic equipment maker
Enamellers
Fertilizer makers
Flint workers
Foundry workers
Furnace liners
Fused quartz workers
Glass makers
Glaze mixers (pottery)
Granite workers
Grinding wheel makers
Grindstone workers
Hard rock miners
Insecticide makers
Insulators
Jewelers
Jute workers
Kiln liners
Masons
Metal workers
Miners
Mortarmen
Oil purifiers
Oilstone workers
Optical equipment makers
Paint mixers
Polishing soap makers
Porcelain workers
Pottery workers
Pouncers (felt hat)
Pulpstone workers
Quarry workers
Quartz workers
Refractory makers
Road constructors
Rock workers
Rubber compound mixers
Sand workers
Sandpaper makers
Sandstone grinders
Sawyers
Scouring soap workers
Silica brick workers
Silicon alloy makers
Silver polishers
Slate workers
Smelters
Sodium silicate makers
Spacecraft workers
Stone workers
Street sweepers
Subway construction workers
Tile makers
Toothpaste makers
Tube mill liners
Tumbling barrel workers
Tunnel construction workers
Whetstone workers
Wood filler workers

Adapted from Reference [86]

APPENDIX II
OSHA INSPECTION DATA BY SIC CODES
Crystalline SiO Samples
2

		No. of Samples Collected	Samples with conc. > 2x PEL	% of Samples Exceeding 2x PEL
Agriculture, Forestry and Fishing				
01	Agricultural Products - Crops	4	0	0
02	Agricultural Products - Livestock Services	4	0	0
07	Agricultural Services	35	27	77
Mining				
12	Bituminous Coal and Lignite Mining	4	0	0
13	Oil and Gas Extraction	8	7	88
14	Mining and Quarrying of Nonminerals except Fuels	31	19	61
Construction				
15	Building Construction - General Contractors	45	13	29
16	Construction other than Building Construction - General Contractors	424	127	30
17	Construction - Special Trade Contractors	289	29	10
Manufacturing				
20	Food and Kindred Products	187	97	52
22	Textile Mill Products	52	14	27
23	Apparel and Other Finished Products made from Fabrics and Similar Material	16	0	0
24	Lumber and Wood Products, except Furniture	13	1	8
25	Furniture and Fixtures	31	0	0
26	Paper and Allied Products	82	11	13
27	Printing, Publishing and Allied Industries	31	0	0
28	Chemicals and Allied Products	640	86	13
29	Petroleum Refining and Related Industries	214	23	11
30	Rubber and Miscellaneous Plastics Products	269	25	9
31	Leather and Leather Products	14	0	0
32	Stone, Clay, Glass and Concrete Products			
	321 Flat Glass	82	7	9
	322 Glass and Glassware, Pressed or Blown	229	25	11
	323 Glass Products Made of Purchased Glass	37	4	11

APPENDIX II (Continued)
OSHA INSPECTION DATA BY SIC CODES
Crystalline SiO Samples
2

		No. of Samples Collected	Samples with conc. > 2x PEL	% of Samples Exceeding 2x PEL
324	Cement, Hydraulic	65	0	0
325	Structural Clay Products	635	168	26
326	Pottery and Related Products	945	217	23
327	Concrete, Gypsum and Plaster Products	347	41	12
328	Cut Stone and Stone Products	270	72	27
329	Abrasive, Asbestos and Miscellaneous Nonmetallic Mineral Products	558	89	16
33	Primary Metal Industries			
331	Blast Furnace, Steel Works, Rolling and Finishing Mills	639	205	32
332	Iron and Steel Foundries	10,850	2,483	23
333	Primary Smelting and Refining of Nonferrous Metals	146	13	9
334	Secondary Smelting and Refining of Nonferrous Metals	39	0	0
335	Rolling, Drawing and Extruding of Nonferrous Metals	23	5	22
336	Nonferrous Foundries (Casting)	2,170	189	9
339	Miscellaneous Primary Metal Products	68	31	46
34	Fabricated Metal Products, except Machinery and Transportation Equipment	1,265	272	22
35	Machinery, except Electrical	1,377	184	13
36	Electrical and Electronic Machinery, Equipment and Supplies	474	107	23
37	Transportation Equipment	600	117	20
38	Measuring, Analyzing and Controlling Instruments, Photographic, Medical	137	36	26
39	Miscellaneous Manufacturing Industries	211	20	9
	Transportation, Communication, Electric, Gas and Sanitary Services			
40	Railroad Transportation	11	0	0
41	Local and Suburban Transit and Interurban Highway Passenger Transportation	2	0	0
42	Motor Freight, Transportation and Warehousing	45	13	29

APPENDIX II (Continued)
OSHA INSPECTION DATA BY SIC CODES
Crystalline SiO$_2$ Samples

		No. of Samples Collected	Samples with conc. ≥ 2x PEL	% of Samples Exceeding 2x PEL
44	Water Transportation	12	0	0
47	Transportation Services	7	0	0
49	Electric, Gas and Sanitary Services	46	9	20
Wholesale Trade				
50	Wholesale Trade - Durable Goods	55	3	5
51	Wholesale Trade - Nondurable Goods	77	2	3
Retail Trade				
52	Building Material, Hardware, Garden Supplies and Mobile Home Dealers	24	24	100
55	Automotive Dealers and Gasoline Service Stations	8	6	75
57	Furniture, Home Furnishings and Equipment Stores	1	0	0
59	Miscellaneous Retail	46	0	0
Finance, Insurance, Real Estate				
Services				
72	Personal Services	3	0	0
73	Business Services	36	4	11
75	Automotive Repair Services and Garages	29	2	7
76	Miscellaneous Repair Services	25	8	32
80	Health Services	2	0	0
82	Educational Services	10	0	0
84	Museum, Art Galleries, Botanical and Zoological Gardens	13	0	0
87	Miscellaneous Services	8	0	0
Public Administration				
Nonclassifiable Elsewhere				
99		1	0	0
Total		23,864	1,035	20

APPENDIX III

NIOSH Health Hazard Evaluations Involving SiO_2 Exposure

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Health Hazard Evaluation Report No. 73-073-143, Inland Manufacturing Company, General Motors Corp., Dayton, OH, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1973, 6 pp.

Health Hazard Evaluation Report No. 73-095-113, Banner Iron Works, St. Louis, MO, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1973, 14 pp.

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Health Hazard Evaluation Report No. 73-135-138, Kaiser Permanente Cement Company, Lucerne Valley, CA, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1973, 19 pp.

Health Hazard Evaluation Report No. 73-184-176, Philadelphia Quartz Company, Chester, PA, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1973, 7 pp.

Health Hazard Evaluation Report No. 74-009-174, Wheeling-Pittsburg Steel Corp., Steubenville, OH, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1974, 10 pp.

Health Hazard Evaluation Report No. 74-049-191, Owens-Illinois Glass Company, East Point, GA, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1974, 10 pp.

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Health Hazard Evaluation Report No. 74-110-306, Morris Bean and Company, Yellow Springs, OH, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1974, 28 pp.

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Health Hazard Evaluation Report No. 77-000-002, Artanis Offset, Inc., New York, NY, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1977, 11 pp.

Health Hazard Evaluation Report No. 77-000-066, Platte Chemical Company, Fremont, NE, US Dept of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1977, 1 pg.

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