HETA 91–0047–2672
Golden Valley Electric Association
Healy, Alaska

Margaret S. Filios, RN, ScM
Joseph E. Burkhart, CIH

PREFACE

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ACKNOWLEDGMENTS AND AVAILABILITY OF REPORT

This report was prepared by Margaret Filios, RN, ScM, Joseph E. Burkhart, CIH, and Ronnie J. Cornwell (retired) of the Respiratory Disease Hazard Evaluations and Technical Assistance Program, Clinical Investigations Branch, Division of Respiratory Disease Studies (DRDS). Assistance was provided by Kathleen Fedan, BS, Clinical Investigations Branch. Desktop publishing was performed by Terry Stewart.

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February 1998

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SUMMARY

In November 1990, the National Institute for Occupational Safety and Health (NIOSH) received a request for a Health Hazard Evaluation (HHE) from the plant manager, Golden Valley Electric Association, Healy Power Plant, Healy, Alaska. The request stated that the employees had complained about irritating odors in the plant, smoke from the coal pulverizers, and exposure to morpholine and amine, and that two employees were recently diagnosed with asthma.

As a first step in this investigation, all employees were asked to complete a screening questionnaire which was mailed to them. After reviewing the questionnaire responses, NIOSH investigators conducted an initial environmental and medical evaluation at the plant May 30 – June 9, 1991. An interim report containing the results of the first industrial hygiene survey was issued by NIOSH in December 1991. Concern was subsequently expressed by several employees that the evaluation was conducted during a time period when the plant's doors and windows were open and therefore would not reflect the worst–case conditions of the winter months, when the plant is closed off to the outside environment. In addition, union representatives indicated that more employees had developed asthma and requested a second site visit and evaluation during the winter months. A second environmental and medical evaluation was conducted February 4–15, 1993.

The environmental surveys consisted of collecting personal and area environmental measurements to determine the extent of workplace exposures. The medical surveys consisted of a respiratory and work history questionnaire, pre– and post–shift pulmonary function testing, and serial peak flow monitoring of select employees based on symptoms reported on the screening questionnaire or a reported diagnosis of asthma. Each medical survey was conducted to document acute and chronic respiratory symptoms and disease (primarily asthma) temporally related to workplace exposures, and to determine if a difference in symptom and disease prevalence existed between the May 1991 and February 1993 evaluation periods.

During the spring 1991 survey, three personal breathing zone (PBZ) and seven area air samples were obtained and analyzed for respirable dust and crystalline silica. The respirable dust concentrations measured on the three personal samples were 0.04, 0.1, and 0.5 milligrams per cubic meter of air (mg/m^3). Respirable dust concentrations measured on the area samplers ranged from 0.01 to 0.7 mg/m^3. Crystalline silica was detected on one PBZ sample collected on a utility man; however, the amount detected was not sufficient to quantify. Quartz was also detected on two of the area samples (5th floor near DA tank and bottom floor baghouse). Only the bottom floor baghouse sample had a quantifiable amount (0.06 mg/m^3) of quartz. For

approximately 2 hours during sampling, on the bottom floor of the baghouse, a workman used a broom to sweep settled fly ash off the floor. Qualitative silica analysis of the settled dust indicated the percent quartz by weight ranged from 6.9 percent to 8.8 percent with an average of 7.8 percent.

Sampling for respirable dust and crystalline silica was a primary focus during the 1993 winter survey. During that survey, 13 PBZ and 35 work area (WA) samples were obtained and analyzed for respirable dust and crystalline silica. Respirable dust concentrations for the 13 PBZ samples ranged from none detected (ND) to 1.17 mg/m^3. The mean respirable dust concentration of these 13 samples was 0.24 mg/m^3, with a standard deviation of 0.38. The highest exposure (1.17 mg/m^3) was collected on a maintenance mechanic. Crystalline silica was not detected on any of the PBZ samples collected during the February 1993 survey.

Respirable dust concentrations for all 35 area samples ranged from ND to 3.84 mg/m^3, with a mean and standard deviation of 0.32 mg/m^3 and 0.83, respectively. The highest potential for a respirable dust exposure (3.84 mg/m^3) was measured in the middle of the coal tunnel, where an employee sometimes sits to observe the coal flow on the conveyor belt.

Crystalline silica was detected on five (14%) of the 35 WA samples collected. Exposure concentrations for those five samples ranged from 0.02 – 0.18 mg/m^3. Of those five samples, four were collected in the coal tunnel and one was collected in the basement of the baghouse. All four samples collected in the coal tunnel exceeded the NIOSH recommend exposure limit (REL) of 0.05 mg/m^3 for crystalline silica.

Results of samples collected for elemental metals indicated only trace quantities present on the samples. The primary metals observed on the samples were iron (Fe), aluminum (Al), chromium (Cr), calcium (Ca), magnesium (Mg), titanium (Ti), and Zirconium (Zr). All sampling results for the elements identified were far below any existing exposure criteria.

Results from PBZ samples indicated that sulfur dioxide exposures ranged from a low of 1.0 parts per million (ppm) to a high of 2.5 ppm. Of the samples collected, six had sulfur dioxide concentrations that exceeded both the NIOSH REL and the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV) of 2.0 ppm for an 8–hour time–weighted average (TWA) exposure. None of the PBZ concentrations collected exceeded the OSHA PEL of 5 ppm for an 8–hour TWA exposure.

Sampling for the amines morpholine and hydroquinone was conducted during the February 1993 survey. No morpholine or hydroquinone was detected on any of the samples.

Twenty–two (73%) of 30 employees participated in the May 1991 medical survey, and 24 (83%) of 29 employees participated in the February 1993 medical survey. Seventeen employees, representing 77% of the participants of the May survey, were present for both surveys. A total of four employees were confirmed to have physician–diagnosed asthma; two were documented during the May 1991 evaluation and two were documented during the February 1993 evaluation. Of these four cases, one was classified as definite occupational asthma, two were classified as “possible” cases of occupational asthma, and the remaining one appeared to represent increased airway irritability of a post–infectious origin. There was no statistically significant difference in the proportion of participants with asthma between May 1991 and February 1993. During both surveys, acute symptoms of highest prevalence were “chest tightness” and “chest wheezes or whistles,” and chronic symptoms of highest prevalence were “cough” and “phlegm”; respiratory symptom prevalences did not increase significantly over the 20–month period between the two surveys.

A total of four participants had baseline spirometry results that fell below the normal range. All four exhibited an obstructed lung pattern. Two were current cigarette smokers and two were former smokers. None of the 17 participants present at both surveys exhibited a decrease in their FEV_1 greater than that expected over a 20–month period.

Peak flow monitoring results revealed two participants with variability within the range considered normal at the time of the May 1991 survey, but greater than normal variability (>20%) at the time of the February 1993 survey. However, only one of these participants had a pattern of variability which was suggestive of a temporal association with work.

Analysis of the environmental data collected during both the May 1991 and February 1993 surveys indicated that a potential health hazard existed from exposure to sulfur dioxide and crystalline silica. Results from both surveys showed a potential for exposure to high concentrations of sulfur dioxide in the baghouse area. In addition, crystalline silica (quartz) was also detected in air samples collected in the baghouse and coal tunnel.

The chest symptoms and asthma appear to be consistent with the effects of exposure to respiratory tract irritants. Although the proportion of employees with chest symptoms and asthma did not differ significantly between the 2 surveys (different seasons), the findings are clinically important. Each case of occupational asthma represents a sentinel health event indicating a potentially hazardous worksite environment. Exposure to sulfur dioxide, other respiratory irritants, and mineral dust may explain these findings and may have both induced and exacerbated the respiratory symptoms and asthma.

Keywords: SIC Code 4911 (Electric Services), electric power generation, baghouse, fly ash, respirable dust, crystalline silica, sulfur dioxide, spirometry, peak flow, asthma.

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INTRODUCTION

In November 1990, the National Institute for Occupational Safety and Health (NIOSH) received a request for a Health Hazard Evaluation (HHE) from the plant manager, Golden Valley Electric Association, Healy Power Plant, Healy, Alaska. The request stated that the employees had complained about irritating odors in the plant, smoke from the coal pulverizers, exposure to morpholine and amine, and that two employees were recently diagnosed with asthma.

In January 1991, a short questionnaire was mailed to all 31 current employees to learn more about the nature and frequency of their concerns. Responses were received from 74% (23/31) of the workers. The three most commonly voiced complaints were: (1) the practice of reversing the exhaust fans during the winter in the boiler room and baghouse; (2) the use of fly ash as a cleaning agent during blow–down (plant cleaning); and (3) the use of compressed air supplied by air hoses during blow–down. More than 70% of the respondents reported at least one respiratory symptom (i.e., cough, phlegm, breathlessness, chest tightness, and/or wheeze) on their questionnaires; wheeze was reported by more than 50%, and 40% reported chest tightness. Additionally, of the 14 non–smokers who replied, 86% reported having at least one respiratory symptom. A majority of the workers reporting symptoms worked in areas with reportedly high potential for exposure to coal dust and fly ash. The data from this questionnaire indicated that environmental and medical evaluations were warranted.

A survey involving both environmental and medical components was conducted May 30–June 9, 1991. During the survey, several employees expressed concern that because this survey was conducted during the summer season when the plant's doors and windows were open, the results would not reflect the worst–case conditions present during the winter months, when the plant is closed off to the outside environment. Also, union representatives indicated that additional employees had developed asthma since the May 1991 survey and requested a second evaluation during the winter months. A second environmental and medical survey was therefore conducted February 4–15, 1993. Personal breathing zone (PBZ) and work area (WA) environmental measurements were collected during both surveys to determine potential exposures of workers. Both medical evaluations consisted of a respiratory and work history questionnaire, pre– and post–shift pulmonary function testing, and serial peak flow monitoring of select employees based on symptoms reported on the screening questionnaire or a reported diagnosis of asthma. The medical data were used to document acute and chronic respiratory symptoms and disease (primarily asthma) related to workplace exposures and determine whether symptom and disease prevalence differed between summer and winter.

NIOSH issued an interim report in December 1991 containing the results of the May 1991 environmental evaluation. Participants in both medical evaluations were provided their individual spirometry and peak flow monitoring results after each survey. This report presents the findings of both the May 1991 and February 1993 environmental and medical surveys.

BACKGROUND

Process Description

The Healy power plant is a pulverized coal–fired, steam–driven, electric generating facility. The unit went on line in November 1967 as a positive draft unit. In 1980, a baghouse with induced draft fan was added for particulate removal. This changed the boiler to a negative draft of 0.5 inches of water. Low sulfur (0.2%) coal is trucked to the site directly from a coal mine 5 miles away. Coal is pushed by bulldozer, from a stockpile, into a primary crusher. The crushed coal is then transported by an enclosed conveyor into two 250–ton storage bunkers. Coal is gravity fed from each bunker to Foster Wheeler MB–19 pulverizers. The pulverized coal is then blown into the boiler through four burners, two from each pulverizer. The boiler is a Foster Wheeler water tube negative–draft unit. Coal use per day is 480–500 tons.

Fly ash from the baghouse is conveyed pneumatically to a point where it is mixed with water and discharged to the settling pond. After dredging the ash pond, the wet ash is taken back to the coal pits.

Boiler makeup water comes from a 200–ft. well and is passed through a zeolite softener. It is fed to an evaporator where turbine extraction steam is used to evaporate the water, leaving behind the impurities. Water treatment in the boiler cycle consists of adding caustic soda, sodium phosphate, a polymer dispersant, a feedwater pH buffer, and a corrosion inhibitor. Some of these are rarely added, others are added on a daily basis, both automatically and manually.

A total of 30 individuals were employed during the May 1991 survey. At the time of the February 1993 survey, Golden Valley Electric employed 29 individuals, 25 of whom were operators, production, or maintenance personnel, and four of whom were involved in clerical or administrative work. A description of job duties follows:

Operators: Various job titles under this category include Control Room Engineer, Assistant Control Room Engineer, Relief Control Room Engineer, Auxiliary Operator, Relief Assistant Control Room Engineer, and Coal Equipment Operator. Control Room Engineers are responsible for operating the boiler and electrical controls from the control room. Assistant Control Room Engineers work in the control room and also take readings throughout the entire plant except the basement. Relief Control Room Engineers are responsible for general cleaning and assisting maintenance mechanics (Mechanical Repairmen) when not providing relief for other operators. Auxiliary Operators are responsible for monitoring equipment in the basement of the plant and operating the automatic ash removal equipment. Other duties include clean–up of the first three floors and helping the Assistant Control Room Engineer during abnormal operating conditions. The Relief Assistant's duties are the same as the Relief Control Room Engineer, except he relieves the Assistant and Auxiliary positions only. Coal Equipment Operators use a bulldozer and loader to move coal into the bunkers. Other duties include cleaning the coal–handling area, blow–down and general clean–up of the plant, and assisting maintenance. During the period of our surveys, Control Room Engineers and Operators worked 12–hour rotating shifts, with three employees to a shift. Two Coal Equipment Operators worked 4 days a week, 10 hours per day.

Maintenance (Repairmen): This category includes Mechanical/Welding Repairmen, Electrical Repairmen, Instrument Repairmen, Assistants, and the Utilityman. Mechanical/Welding Repairmen are responsible for the repair or installation of all mechanical equipment, welding, machining, and general building maintenance. Electrical Repairmen's primary duties are to maintain and install electrical equipment. The Instrument Repairman maintains, repairs, and installs instruments. Additional duties include monitoring and adding chemicals to the boiler water cycle. The Utilityman is responsible for general housekeeping duties and assisting where needed. During the period of our surveys, two maintenance crews worked 4 days a week, 10 hours per day.

METHODS

Industrial Hygiene

The industrial hygiene portion of this investigation was designed to characterize the physical and chemical make–up of the air throughout the plant and to determine potential worker exposures. During both the summer and winter surveys, full–shift WA and PBZ monitoring was conducted. Area sampling stations were located on each of the seven floors of the plant. At each station, samples for respirable dust, crystalline silica, elemental metals, aldehydes, amines, hydrocarbons, carbon monoxide, nitric oxide, nitrogen dioxide, and sulfur dioxide were collected. In addition to the full–shift WA air samples, short–term WA air samples were collected for sulfur dioxide. Settled dust samples were collected and analyzed for asbestos and silica content.

Respirable Dust/Crystalline Silica

PBZ and WA samples, for the estimation of respirable dusts and respirable silica dust exposure, were collected on pre–weighed, 37–millimeter (mm) diameter, 5–micron (µm) pore size, polyvinyl chloride (PVC) membrane filters, mounted in series with 10–mm Dorr–Oliver nylon cyclones. Air was drawn through the filter at a flow rate of 1.7 liters per minute (lpm) using a battery–powered sampling pump.

All air samples were analyzed for respirable dusts and total respirable crystalline silica (alpha quartz, tridymite, and cristobalite). Respirable dust content was analyzed gravimetrically according to NIOSH Method 0600. Respirable crystalline silica dust content was analyzed by NIOSH Method 7500, using X–ray diffraction.[1]

Elemental Metals

WA and PBZ samples for the estimation of exposure to elemental metals were collected on 37–mm diameter, 0.8–µm pore size cellulose ester membrane filters, mounted in closed–face cassettes. Air was drawn through the filters at a flow rate of 1.7 lpm using a battery–powered sampling pump for a full shift. All air samples collected for elemental analysis were digested according to NIOSH Method 7300 and analyzed using a scanning inductively coupled plasma emission spectrometer.[1]

Aldehydes

Full–shift PBZ and WA samples for formaldehyde and acrolein were collected on solid sorbent tubes (ORBO–23) using a constant flow sampling rate of 100 cubic centimeters per minute (cc/min). The collected samples were analyzed for each analyte utilizing a gas chromatograph equipped with a nitrogen–phosphorus detector according to NIOSH Analytical Methods 2501 and 2541.[1]

Total Hydrocarbons

Full–shift WA samples for the estimation of total hydrocarbon exposure were collected on charcoal sorbent tubes at constant air flow sampling rates of 50 and 200 cc/min. Sampling times varied between 7–8 hours. Samples were qualitatively and quantitatively analyzed according to NIOSH Analytical Method 1501 using gas chromatography / mass spectrometry (GC/MS). Bulk air samples were collected at the higher flow rate to saturate the sampling tube. These bulks are qualitatively screened using a gas chromatograph equipped with a flame ionization detector (GC/FID). If no hydrocarbon peaks are detected on the screening samples, then it is doubtful if peaks would be detected on the lower flow (50 cc/min) samples. If hydrocarbon peaks are identified from the screening, then the lower flow sample would be analyzed and quantitated by the GC/MS for the specific compounds detected during the screening. Identification is made by matching the sample spectra to reference spectra. Once identified, the peaks are converted to actual concentration for each hydrocarbon identified.[1]

Amines

The two major amines used in the boiler feed water at this facility were hydroquinone and morpholine. General WA samples for hydroquinone were collected on 0.8 micrometer pore size, cellulose ester membrane filters. Full–shift air samples were collected at a flow rate of 1.5 lpm using battery–powered sampling pumps. After sampling, the filters were immediately transferred into a vial containing 10 milliliters (ml) of a 1% acetic acid solution. Hydroquinone samples were analyzed using high performance liquid chromatography according to NIOSH Analytical Method 5004.[1]

General WA samples for morpholine were collected on silica gel tubes. Full–shift air samples were collected at a flow rate of 200 cubic centimeters per minute (cc/min) using a battery powered sampling pump. Samples were analyzed using gas chromatography according to NIOSH Analytical Method S–150.[1]

Carbon Monoxide

PBZ air samples for the estimation of carbon monoxide (CO) exposures were collected using Dräger diffusion detector tubes (Catalog No. 67 33191, National Dräger, Pittsburgh, Pennsylvania). These tubes operate on the diffusion properties of gases. The detection range of this sampling method is 6 to 75 ppm for an 8-hour sampling duration. The accuracy for this method, as reported by the manufacturer, is ± 25%.

In addition, WA samples for CO were collected using a direct reading Interscan, Series 4000 CO monitor. This monitor was connected to a Metrosonic, Model 714 data logger for signal storage and subsequent analysis. Each monitor was calibrated before, and rechecked after the survey using 25 ppm certified span gas. The limit of detection for this meter is 1% of a full scale reading, which corresponds to 1 ppm for CO.

Oxides of Nitrogen

Full–shift WA and PBZ exposure estimates for oxides of nitrogen (NO_x + NO_2) were determined using Palmes® passive dosimeters (NIOSH Method 6700).[1]

In addition, area samples for the estimation of nitrogen dioxide and nitric oxide were also collected using an Interscan, Series 4000 NO_2 direct reading monitor for nitrogen dioxide and Interscan, Series 4000 NO direct reading monitor for nitric oxide. These monitors were also connected to Metrosonic, Model 714 data loggers for signal storage and subsequent analysis.

Sulfur Dioxide

PBZ samples for the estimation of sulfur dioxide (SO_2) were collected using Dräger 5/a–D long–term diffusion indicator tubes (Cat.#81–01091). The limit of detection for this method is 0.7 ppm SO_2 for an 8–hour exposure.

General WA samples for the estimation of potential exposure to SO_2 were collected by drawing air through filters in two 2–piece cassettes connected with tygon tubing using constant flow air sampling pumps, calibrated at 1.5 lpm. The sampler configuration consisted of a mixed cellulose ester membrane filter with a 0.8 micrometer pore size housed in a 2–piece polystyrene cassette preceded by a cellulose filter impregnated with potassium hydroxide glycerine solution housed in a 2–piece cassette. Sulfates, sulfites and other particulate matter are collected on the first filter, while sulfur dioxide passes through the initial filter and is collected on the second filter. Each sample was analyzed by ion chromatography according to modified NIOSH Analytical Method 6004.[1]

Medical

All employees were mailed a screening questionnaire prior to the May 1991 survey. This questionnaire was used to determine the nature and prevalence of respiratory complaints among employees at the Healy plant (to assist NIOSH in determining the appropriate level of response to the HHE request) and later, to identify a sub–group of employees for serial peak flow monitoring.

The purpose of the May 1991 medical evaluation was to identify and document acute and chronic respiratory symptoms and disease (primarily asthma) temporally related to workplace exposures. The purpose of the second evaluation in February 1993 was the same as the first, and also to determine if symptom and disease prevalence differed given the changed exposure circumstances (spring vs. winter–time exposures).

Identical methods were utilized during the May 1991 and February 1993 evaluations. All employees were invited to participate. A questionnaire, cross–shift spirometry, and serial peak flow monitoring of a sub–group of employees were used to evaluate employees for possible respiratory health effects.

Data from each respective survey were examined cross–sectionally (at two separate points in time) using descriptive statistics to evaluate symptom and disease prevalence. To determine if a seasonal difference existed, data from participants who were present at both surveys were evaluated longitudinally (across time). All descriptive statistics were calculated using the Statistical Analysis System (SAS) version 6.04.[2] McNemar's test for paired–sample nominal scale data was used to determine if the proportion of participants reporting symptoms or disease differed between the two surveys .[3,4] Longitudinal changes in pulmonary function were evaluated as recommended by Hankinson and Wagner,[5] with a decrement of $\geq$15% in the forced expiratory volume in one second (FEV_1) over one year considered an abnormal decline.

What follows is a single description of the methods that were used during both evaluations.

Questionnaire

A modified version of the Medical Research Council (MRC)[6] questionnaire on respiratory symptoms, supplemented with questions concerning asthma and acute respiratory symptoms, smoking habits, demographic information, and work history, was administered by trained NIOSH personnel at the time of the pulmonary function testing.

The following definitions were established for the purpose of questionnaire analysis:

ACUTE RESPIRATORY SYMPTOMS	
Wheeze	Wheezing or whistling in the chest other than that associated with a cold.
Attacks of dyspnea with wheezing or whistling*	Any previous attack.
Attacks of dyspnea or cough*	Unprovoked attack of shortness of breath or cough in the last year.
Chest tightness	Any tightness of the chest occurring for longer than 1 minute within the past 12 months.
CHRONIC RESPIRATORY SYMPTOMS	
Chronic cough	A cough on most days for as much as 3 months during the year.
Chronic phlegm	The production of phlegm on most days for as much as 3 months during the year.
Chronic bronchitis	Cough and phlegm on most days for as much as 3 months for 2 or more years.
*Chronic dyspnea**	Shortness of breath walking with similar age individuals on level ground.

* Dyspnea is defined as shortness–of–breath, or difficult breathing.

Acute symptoms were determined to be temporally related to work if a participant reported that symptoms began after starting work at Golden Valley Electric, were brought on or exacerbated by exposures at work, and improved when the participant was away from work.

Asthma is a clinical syndrome characterized by airway inflammation with resultant increased responsiveness of the tracheal–bronchial tree to a variety of stimuli with variable or reversible airflow obstruction.[7,8,9] Airflow changes can occur spontaneously, with treatment, with a precipitating exposure, or with diagnostic maneuvers such as nonspecific inhalation challenge. Symptoms of asthma include episodic wheezing, chest tightness, and dyspnea, or recurrent attacks of cough and sputum production, often accompanied by rhinitis (runny nose).[10] Occupational asthma (OA) is characterized by variable airflow obstruction related to exposure in the workplace environment to airborne contaminants.[11] Variable airflow obstruction can be documented by cross–shift spirometry or serial peak expiratory flow rate (PEFR) measurement.

"Asthma" was first defined on our questionnaire as an indication that the condition existed at the time of the evaluation. The NIOSH surveillance case definition for occupational asthma was then applied to identify participants with asthma related to workplace exposures (see Appendix I). For classification purposes, the criterion "Exposure to an agent or process previously associated with occupational asthma" was interpreted to mean a specific, known agent or process previously associated with OA – typically those that produce an immunologic response, or asthma that develops after a single intense irritant exposure.

Spirometry

Spirometry was performed using a dry rolling–seal spirometer interfaced to a dedicated computer. At least five maximal expiratory maneuvers were recorded for each person each time spirometry was performed. All values were corrected to BTPS (body temperature, ambient pressure, saturated with water vapor). The largest forced vital capacity (FVC), and forced expiratory volume in one second (FEV_1) were the parameters selected for analysis, regardless of the curves on which they occurred. Testing procedures conformed to the American Thoracic Society's[12] recommendations for spirometry. Predicted values were calculated using the Knudson[13] reference equations. Test results were compared to the 95th percentile lower limit of normal (LLN) values obtained from Knudson's reference equations to identify participants with abnormal spirometry patterns of obstruction and restriction.[13] Five percent of the population will have predicted values that fall below the normal range, or LLN, while 95% will have predicted values above the lower limit.

Using this comparison, obstructive and restrictive patterns are defined as:

Obstruction: Observed ratio of FEV_1/FVC% below the LLN.
Restriction: Observed FVC below the LLN; and FEV_1/FVC% above the LLN.

The criteria for interpretation of the level of severity for obstruction and restriction, as assessed by spirometry, is based on the NIOSH classification scheme (available upon request from the Division of Respiratory Disease Studies). For those persons with values below the LLN, the criteria are:

Classification	*Obstruction (FEV_1/FVC x 100)*	*Restriction (% Predicted FVC)*
Mild	**> 60**	**> 65**
Moderate	**≥ 45 to ≤ 60**	**≥ 51 to ≤ 65**
Severe	**< 45**	**< 51**

Cross–shift spirometry was used to document acute airway response and was performed pre– and post–shift on the last day of the participant's work week and again on the first day of the following work week. A decrement of 10% or greater in FEV_1 across a work shift on either the last day or the first day of the work week, or an improvement of 10% or greater over a weekend was considered an acute response and suggestive of a relationship with workplace exposures.

Peak Flow Monitoring

Peak flow meters were used to document the variability of acute airway responses. Selected participants were given log sheets and instructed in a standardized manner in the use of the Mini–Wright Peak Flow Meter. Participants were asked to record flow results from three blows every 2 hours while awake, for 8 consecutive days. An attempt was made to obtain data from the 2 days at the end of the work week, at least 2 days off, and the first several days of the next work week. In addition, participants were asked to record the presence of symptoms and use of medication during the 2 hours prior to the recording of their peak flow measurements.

Peak flow logs from each worker were reviewed for completeness. An individual worker's record from a 24–hour survey day was considered valid (interpretable) if it contained peak flow results from at least three recording times that spanned at least 8 hours that day. A worker's entire record was included in the analysis if valid records from a minimum of 4 of the 8 survey days were present, including at least 1 day off work. Logs which failed to meet these minimum criteria were excluded from analysis.

At each peak flow recording time, only the best value (largest of the three recorded values) was used for calculations and subsequent interpretation. For each worker, an *overall mean* peak flow was calculated, using the best value from all available recording times. In addition, for each survey day with valid results, a *daily mean* was calculated from the best values on that day. Diurnal variation in peak flow was calculated as the difference between the daily maximum and minimum best values for the survey day divided by the daily mean. Overall variation in peak flow was calculated as the difference between the maximum and minimum best values for the entire survey, divided by the overall mean. Both the overall mean and daily mean are expressed as a percentage. Overall variation of ≥ 20% is suggestive of increased airway responsiveness. If daily mean peak flow values are lower on work days than days away from work, or variation ≥ 20% is seen on work days and absent on days off work, a relationship between airflow changes and workplace exposures is suggested.

EVALUATION CRITERIA

To assess the hazards posed by workplace exposures, NIOSH investigators use a variety of environmental evaluation criteria. These criteria suggest exposure levels to which most workers may be exposed for a working lifetime without experiencing adverse health effects. However, because of wide variation in individual susceptibility, some workers may experience occupational illness even if exposures are maintained below these limits. The evaluation criteria do not take into account individual hypersensitivity, pre–existing medical conditions, or possible interactions with other work place agents, medications being taken by the worker, or environmental conditions.

The primary sources of evaluation criteria for the workplace are: NIOSH Criteria Documents and Recommended Exposure Limits (RELs),[14] the Occupational Safety and Health Administration (OSHA) Permissible Exposure Limits (PELs),[15] and the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs®).[16] The objective of these criteria for chemical agents is to establish levels of inhalation exposure to which the vast majority of workers may be exposed without experiencing adverse health effects.

Occupational health criteria are established based on the available scientific information provided by industrial experience, animal or human experimental data, or epidemiologic studies. Differences between the NIOSH RELs, OSHA PELs, and ACGIH TLVs® may exist because of different philosophies and interpretations of technical information. It should be noted that RELs and TLVs® are guidelines, whereas PELs are standards which are legally enforceable. OSHA PELs are required to take into account the technical and economical feasibility of controlling exposures in various industries where the agents are present. The NIOSH RELs are primarily based upon the prevention of occupational disease without assessing the economic feasibility of the affected industries and as such tend to be conservative. A Court of Appeals decision vacated the OSHA 1989 Air Contaminants Standard in *AFL–CIO v OSHA*, 965F.2d 962 (11th cir., 1992); and OSHA is now enforcing the previous 1971 standards (listed in 29 CFR 1910.1000, Table Z–1–A). However, some states which have OSHA–approved State Plans continue to enforce the more protective 1989 limits. For exposures with evaluation criteria, NIOSH encourages employers to use the 1989 OSHA PEL or the RELs, whichever are lower.

Evaluation criteria for chemical substances are usually based on the average PBZ exposure to the airborne substance over an entire 8– to 10–hour workday, expressed as a time–weighted average (TWA). Personal exposures are usually expressed in parts per million (ppm), milligrams per cubic meter (mg/m^3), or micrograms per cubic meter ($\mu g/m^3$). To supplement the 8–hour TWA where there are recognized adverse effects from short–term exposures, some substances have a short–term exposure limit (STEL) for 15–minute peak periods; or a ceiling limit, which is not to be exceeded at any time. Additionally, some chemicals have a "skin" notation to indicate that the substance may be absorbed through direct contact of the material with the skin and mucous membranes.

It is important to note that not all workers will be protected from adverse health effects if their exposures are maintained below these occupational health exposure criteria. A small percentage may experience adverse health effects because of individual susceptibility, a pre–existing medical condition, previous exposures, and/or a hypersensitivity (allergy). In addition, some hazardous substances may act in combination with other work place exposures, or with medications or personal habits of the worker (such as smoking, etc.) to produce health effects even if the occupational exposures are controlled to the limit set by the evaluation criterion. These combined effects are often not considered by the chemical specific evaluation criteria. Furthermore, many substances are appreciably absorbed by direct contact with the skin and thus potentially increase the overall exposure and biologic response beyond that expected from inhalation alone. Finally, evaluation criteria may change over time as new information on the toxic effects of an agent become available. Because of these reasons, it is prudent for an employer to maintain worker exposures well below established occupational health criteria.

Respirable Silica

Crystalline silica, referred to as free silica, is defined as silicon dioxide (SiO_2) in the form of quartz, tridymite, and cristobalite. The chief concern of excessive free silica exposure is the development of silicosis. This form of pneumoconiosis is characterized by a nodular pulmonary fibrosis caused by the deposition of fine particles of crystalline silica in the lungs. In silicosis, as in many other pneumoconioses, the various stages of progression of silicotic lesions are related to the degree of exposure to free silica, the duration of exposure, and the length of time the dust has to react with the lung tissue. Symptoms usually develop insidiously, with cough, shortness of breath, chest pain, weakness, wheezing, and nonspecific chest illnesses. Silicosis usually appears after years of exposure, but may appear in a shorter time if exposure concentrations are very high. This latter form is referred to as rapidly–developing or acute silicosis, and its etiology and pathology are not as well understood. Silicosis is usually diagnosed through chest x–ray and occupational history of exposure to silica–containing dust. Among the different crystalline structures and surface properties of quartz particles, some forms may have a greater capacity to produce silicosis.[17,18,19]

Epidemiological studies have shown an association between silicosis and lung cancer. Recently, a committee of the International Agency for Research on Cancer (IARC) reclassified crystalline silica (quartz or cristobalite) from occupational sources as a substance "carcinogenic to humans" and evidence suggests that individuals with silicosis are at increased risk for lung cancer.[20] NIOSH recommends that crystalline silica be considered a potential occupational carcinogen.[14,19,21]

The NIOSH REL for respirable crystalline silica is 0.05 mg/m^3 as a TWA for up to 10 hours per day during a 40–hour week. This REL is intended to prevent silicosis. The ACGIH TLV® for respirable crystalline silica, as quartz, is 0.05 mg/m^3 of air.

The OSHA PEL for crystalline silica is somewhat different in that the allowable PEL is calculated for respirable dust (not crystalline silica) exposures if the dust contains ≥ 1% free crystalline silica. That calculation is:

$$PEL_{(mg/m^3)} = \frac{10}{\%Silica + 2}$$

For example, the allowable respirable dust exposure criteria using the OSHA equation for a dust containing 15% crystalline silica is 0.59 mg/m^3 for a 8–hour TWA exposure. In essence, the lower the silica content of the dust, the higher the allowable respirable dust exposure criteria; and the higher the silica content, the lower the respirable dust exposure criteria.

Elemental Nickel

Metallic nickel compounds cause sensitization dermatitis.[22] NIOSH[23] considers nickel a potential occupational carcinogen, as nickel refining has been associated with an increased risk of nasal and lung cancer.

The ACGIH TLV® for soluble compounds of nickel is 100 µg/m^3 for a TWA exposure. The OSHA PEL for nickel is 1000 µg/m^3. The NIOSH

REL for nickel is 15 µg/m^3 as a TWA for up to 10 hours per day during a 40–hour week.

Aldehydes

Formaldehyde

Formaldehyde is a colorless gas with a strong odor. Exposure can occur through inhalation and skin absorption. The acute effects associated with formaldehyde are irritation of the eyes and respiratory tract and sensitization of the skin. The first symptoms associated with formaldehyde exposure, at concentrations ranging from 0.1 to 5 parts per million (ppm), are burning of the eyes, tearing, and general irritation of the upper respiratory tract. There is variation among individuals in terms of their tolerance and susceptibility to acute exposures of the compound.[24] In two separate studies, formaldehyde has induced a rare form of nasal cancer in rodents. Formaldehyde exposure has been identified as a possible causative factor in cancer of the upper respiratory tract in a proportionate mortality study of workers in the garment industry.[25]

NIOSH, OSHA, and ACGIH have classified formaldehyde as a potential occupational carcinogen. NIOSH recommends that exposures to formaldehyde be controlled to the "lowest feasible limit" through the use of engineering controls. The lowest feasible limit typically refers to the limit of detection (LOD) of the analytical method, which for formaldehyde is generally around 0.016 ppm. On May 27, 1992, OSHA amended its existing regulation (29 CFR 1910.1048) for occupational exposure to formaldehyde to take effect on June 26, 1992. The final amendments lowered the 8-hour PEL for formaldehyde from 1 ppm to an 8-hour TWA of 0.75 ppm. The amendments also added medical removal protection provisions to supplement the existing medical surveillance requirements for those employees suffering significant eye, nose, or throat irritation, and for those suffering from dermal irritation or sensitization from occupational exposure to formaldehyde. Additional hazard labeling, including a warning that formaldehyde presents a potential cancer hazard, is required where formaldehyde air concentrations, under reasonably foreseeable conditions of use, may potentially exceed 0.5 ppm. The final amendment also provides for annual training of all employees exposed to airborne formaldehyde concentrations of 0.1 ppm or higher. The ACGIH has recommended that occupational exposure to formaldehyde be controlled so that no exposure exceed 0.3 ppm for any period of the work shift.

Acrolein

Acrolein, another acutely toxic aldehyde, produces intense irritation to the eyes and mucous membranes of the respiratory tract. Because of acrolein's pungent, offensive odor and the intense irritation of the conjunctiva and upper respiratory tract, severe toxic effects from acute exposure are rare as workers will not tolerate the vapor even in minimal concentrations. Acute exposure to acrolein may cause bronchial inflammation, resulting in bronchitis or pulmonary edema. The carcinogenic potential of acrolein has not been adequately determined, but one of its potential metabolites, glycidaldehyde, is considered to be carcinogenic.[26, 27] Acrolein has been found to be mutagenic to bacteria and to induce sister chromatid exchange *in vitro.*[28] The NIOSH REL, ACGIH TLV®, and the OSHA PEL for occupational exposure to acrolein is 0.1 ppm for an 8–hour, time–weighted exposure.

Hydrocarbons

Hydrocarbons describe a large class of chemicals which are organic (i.e., containing carbon) and have sufficiently high vapor pressure to allow some of the compound to exist in the gaseous state at room temperature. Not all hydrocarbons exhibit the same toxicological effects; therefore, exposure criteria are dependent on the particular hydrocarbon and toxic effect. Generally, overexposure to these substances may cause irritation of the eyes, respiratory tract, and skin. Since they are central nervous system depressants, overexposure may also cause fatigue, weakness, confusion, headache, dizziness, and drowsiness.[22,23]

Morpholine

Morpholine vapor is an irritant of the eyes, nose, and throat. In industry, some instances of skin and respiratory tract irritation have been observed, but no chronic effects have been reported. Workers exposed for several hours to low vapor concentrations complained of foggy vision with rings around lights, the result of corneal edema, which cleared within 3–4 hours after exposure.[22] The NIOSH REL, OSHA PEL and ACGIH TLV® for a full–shift TWA exposure to morpholine is 20 ppm.

Hydroquinone

Short–term exposure to hydroquinone can cause headaches, dizziness, nausea, vomiting, increased respirations, breathing difficulty, sensation of suffocation, ringing noise in ears, paleness, bluish discoloration of skin, green or brownish discoloration of urine, and irritation of the skin and eyes. Long–term (chronic) exposures can cause depigmentation of the skin, brownish discoloration of the cornea, and blurred vision.[22]

The current OSHA PEL and ACGIH TLV® for hydroquinone, expressed as a TWA exposure, is 2 mg/m^3. The NIOSH REL differs in that the exposure limit is based on a 15–minute ceiling concentration of 2 mg/m^3.

Carbon Monoxide

Carbon monoxide (CO) is a colorless, odorless gas, slightly lighter than air. It is produced whenever incomplete combustion of carbon–containing compounds occurs. Typical environmental sources of carbon monoxide exposure, to name a few, are poorly vented heating systems, automobile exhaust, and cigarette smoke. The combination of incomplete combustion and inadequate venting often results in overexposure. The danger of this gas derives from its affinity for the hemoglobin of red blood cells, which is 300 times that of oxygen. The hazard of exposure to CO is compounded by the insidiousness with which high concentrations of carboxyhemoglobin (CO–Hb) can develop without marked symptoms.[22] Intermittent exposures are not cumulative in effect and, in general, symptoms occur more acutely with higher concentrations of CO. The NIOSH REL for a TWA exposure to CO is 35 ppm. The OSHA PEL for CO is 50 ppm for a TWA exposure. The ACGIH TLV® for a TWA exposure to carbon monoxide is 25 ppm.

Nitrogen Dioxide

Nitrogen dioxide may cause severe breathing difficulties which may be delayed in onset. Nitrogen dioxide gas is a respiratory irritant; it causes pulmonary edema and rarely, among survivors, widespread inflamination and fibrotic obstruction of small airways.[29] Brief exposure of humans to concentrations of about 250 ppm caused cough, production of mucoid or frothy sputum, and increasing dyspnea. The effects expected in humans from exposure to nitrogen dioxide for 60 minutes are: 25 ppm, respiratory irritation and chest pain; 50 ppm, pulmonary edema with possible subacute or chronic lesions in the lungs; 100 ppm, pulmonary edema and death.[22]

The NIOSH REL for nitrogen dioxide is a 15–minute STEL of 1 ppm. The OSHA PEL for nitrogen dioxide is 5 ppm for a ceiling exposure.

The ACGIH TLV® for a TWA exposure to nitrogen dioxide is 3 ppm with a 5 ppm STEL.

Sulfur Dioxide

Sulfur dioxide gas is one of the combustion by–products produced in coal–fired power plants, and is a severe irritant of the eyes, mucous membranes, and skin. Its irritant properties are due to the rapidity with which it forms sulfurous acid on contact with moist membranes. In combination with certain particulate matter and/or oxidants, the effects may be markedly increased. Approximately 90% of all sulfur dioxide inhaled through the nose is absorbed in the upper respiratory passages, where most effects occur. High concentrations of sulfur dioxide may produce respiratory paralysis and pulmonary edema. Exposure to concentrations of 10 to 50 ppm can cause irritation to the eyes and nose, rhinorrhea ("runny nose"), choking, cough, nosebleeds, and in some instances, reflex bronchoconstriction ("wheezing") with increased pulmonary resistances.[22] The NIOSH REL for sulfur dioxide is 2 ppm for an 8–hour TWA exposure and 5 ppm for a 15–minute STEL exposure. The ACGIH TLV® is identical to the NIOSH REL. The OSHA PEL for sulfur dioxide is 5 ppm for a 8–hour TWA exposure.

RESULTS

Industrial Hygiene

Samples, for which TWAs were computed, were collected over the length of a normal work period. If the sampling period was less than 8 hours, a zero value was not assigned to the unsampled portion of the workshift in computing the TWAs because it was assumed that there was similar exposure throughout the workshift.

Respirable Dust/Crystalline Silica

During the May 1991 survey, several bulk samples of settled dust (fly ash) were collected throughout the facility. Qualitative silica analysis of these samples indicated that quartz was the only form of crystalline silica present. The percent quartz by weight ranged from 6.9 percent to 8.8 percent with an average of 7.8 percent.

Three PBZ and seven WA air samples obtained during the May 1991 survey were analyzed for respirable dust and crystalline silica. The respirable dust concentrations measured on the 3 PBZ samples were 0.04, 0.1, and 0.5 mg/m^3.

Respirable dust concentrations measured on the WA samplers ranged from 0.01 to 0.7 mg/m^3.

Crystalline silica was detected on one PBZ sample collected on a utility man; however, the amount detected was not sufficient to quantify. Quartz was also detected on two of the area samples (5th floor near DA tank and bottom floor baghouse). Only the bottom floor baghouse sample had a quantifiable amount (0.06 mg/m^3) of quartz. For approximately 2 hours during sampling, on the bottom floor of the baghouse, a workman used a broom to sweep settled fly ash off the floor.

The silica concentrations measured on these samples were all below the OSHA PEL. Only the sample collected in the bottom floor of the baghouse exceeded the NIOSH REL, and this sample was collected during a time when the settled dust was being disturbed.

Sampling for respirable dust and crystalline silica was the primary focus during the February 1993 survey. During that survey, 13 PBZ and 35 WA samples were obtained and analyzed for respirable dust and crystalline silica. Results of the PBZ samples collected during the 1993 survey are presented in Table 1. Respirable dust concentrations for the 13 PBZ samples ranged from none detected (ND) to 1.17 mg/m^3. The mean respirable dust concentration of those 13 samples was 0.24 mg/m^3, with a standard deviation of 0.38. The highest exposure (1.17 mg/m^3) was collected on a maintenance mechanic. Crystalline silica was not detected on any of the PBZ samples collected during the winter survey.

Results of the 35 WA samples are presented in Table 2. In summary, respirable dust concentrations for all 35 samples ranged from ND – 3.84 mg/m^3, with a mean and standard deviation of 0.32 mg/m^3 and 0.83, respectively. The highest potential for a respirable dust exposure (3.84 mg/m^3) was measured in the middle of the coal tunnel where an employee sometimes sits to observe the coal flow on the conveyor belt.

Crystalline silica was detected on 5 of the 35 (14%) WA samples collected. Exposure concentrations for those 5 samples ranged from 0.02 – 0.18 mg/m^3. Of those five samples having quantifiable silica, four were collected in the coal tunnel and one was collected in the basement of the baghouse. All four samples collected in the coal tunnel exceeded the NIOSH REL of 0.05 mg/m^3 for crystalline silica.

Elemental Metals

During the May 1991 survey, two settled dust and 15 (12 WA and 3 PBZ) air samples were collected and analyzed for elemental metals. Nineteen different elements were detected in the bulk samples and 13 detected in the air samples. Seven elements detected in the settled dust but not in the air samples were beryllium, cobalt, lithium, phosphorus, vanadium, yttrium, and zirconium. The concentration of elemental metals measured on the air samples was well below the evaluation criteria. However, one sample collected on a welder indicated an exposure to nickel of 0.02 mg/m^3. NIOSH considers nickel to be potential occupational carcinogens and exposure should be reduced to the lowest feasible level.

During the February 1993 survey, 30 WA full–shift samples were collected throughout the plant and subsequently analyzed for elemental compounds. Results of that analysis indicated only trace quantities of metals present on the samples. The primary metals observed on the samples were iron (Fe), aluminum (Al), chromium (Cr), calcium (Ca), magnesium (Mg), titanium (Ti), and zirconium (Zr). All sampling results for the elements identified were far below any existing exposure criteria; and in many cases, the amounts observed on the samples were at or slightly above the minimum detectable concentration.

Aldehydes

Nine WA samples were collected and analyzed for aldehydes during the May 1991 survey. No aldehydes were detected on any of the samples. The minimum detectable concentration (MDC) was 0.06 ppm.

Since no aldehydes were detected during the May 1991 survey, aldehyde sampling was not performed during the February 1993 survey.

Amines

During the May 1991 survey, eight WA samples were collected on silica gel sampling tubes and analyzed for morpholine. No morpholine was detected on any of the samples collected. The MDC was 0.11 ppm.

Sampling for morpholine and hydroquinone was conducted during the February 1993 survey. In all, 30 WA samples were collected throughout the plant for both morpholine and hydroquinone. No morpholine or hydroquinone was detected on any of the samples. The MDC for morpholine and hydroquinone was 0.02 ppm and 0.003 ppm, respectively.

Hydrocarbons

Seven charcoal tube samples were taken for quantitative analysis and 2 bulk samples were collected for qualitative analysis in May 1991. The representative bulk charcoal tube samples had a much larger sample volume than the charcoal tube samples taken for quantitative analysis. Analysis of the bulk samples indicated the presence of C_5–alkanes, acetone, 1,1,1–trichloroethane, toluene, C_3–alkylbenzenes, C_{10}–alkanes, and limonene. These substances were present in the bulk samples in such minute quantities there was no need to analyze the remaining samples.

Because the samples collected during the May 1991 survey were essentially negative for hydrocarbons, no additional hydrocarbon samples were collected during the February 1993 survey.

Carbon Monoxide

During the May 1991 survey, carbon monoxide samples were collected throughout the plant using long–term carbon monoxide detector tubes. No carbon monoxide was detected in any of the sampling. The minimum quantifiable concentration for these long–term detector tubes for an 8–hour sample was 6 ppm.

During the February 1993 survey, 13 PBZ and 20 WA samples for carbon monoxide were collected using passive detector tubes. Only trace quantities of carbon monoxide were detected on the samples, all at levels that were not quantifiable.

Nitrogen Dioxide

Ten measurements (5 PBZ and 5 WA) for nitrogen dioxide were collected during the May 1991 survey. Nitrogen dioxide concentrations determined from those samples ranged from ND to a high of 0.04 ppm. The sampling results were far below existing exposure criteria.

During the February 1993 survey, 44 (14 PBZ and 30 WA) samples were collected and analyzed for nitrogen dioxide. The results of that analysis showed that 24 (55%) of those samples had no detectable nitrogen dioxide. Of the 20 samples that had quantifiable nitrogen dioxide, the detected range was 0.05 to 0.26 ppm. These sampling results were similar to the nitrogen dioxide data collected during the previous survey. All nitrogen dioxide sampling results were far below existing exposure criteria and do not indicate that a health hazard existed from nitrogen dioxide exposure. The MDC of nitrogen dioxide for the sampling method was 0.05 ppm.

Sulfur Dioxide

The full–shift WA samples collected during the May 1991 survey indicated sulfur dioxide TWA concentrations on the 7th floor of 0.02 ppm, at the top floor of the baghouse of 0.65 ppm, and at bottom floor of the baghouse of 0.32 ppm. These results do not indicate hazardous levels when compared to the criteria. However, direct reading measurements using sulfur dioxide detector tubes indicated higher short–term measurements from 1 to 2 ppm at the top and bottom floors of the baghouse. These short–term results do indicate a potential sulfur dioxide health hazard to workers if they were to be in these areas for an extended period.

Three different methods were used to determine sulfur dioxide exposures during the February 1993 survey: direct reading meters, passive detector tubes, and treated filters. The passive detector tubes were used to collect PBZ samples, whereas the direct reading meters and filters were used to determine potential WA exposures. Results from the 13 PBZ samples collected on workers (Table 3) indicated that sulfur dioxide exposures ranged from a low of 1.0 ppm to a high of 2.5 ppm. Of the 13 PBZ samples collected, 6 exceeded both the NIOSH REL and the ACGIH TLV® of 2.0 ppm for an 8–hour TWA exposure. one of the

PBZ samples collected exceeded the OSHA PEL of 5 ppm for a TWA exposure.

No data were available from either the direct reading meters or the treated filters used to measure sulfur dioxide concentrations within the plant. The direct reading meters were set up at two locations: the top floor and basement of the baghouse. Mid–way through sampling on the first day of the survey, the meters were observed to be very unstable and were providing erratic results.

Briefly, the meters detect sulfur dioxide as a result of a chemical reaction which occurs within a wet cell sensor of the meter. We believe that the failure of these meters was a result of the high temperatures (90–114^{o} F) and extremely low relative humidity (2–12%) within the baghouse. Due to these conditions, the wet cell sensors in the meters became desiccated. This condition was later confirmed by weighing the sensors and comparing that post–weight with the manufacturers specifications.

We also believe that the chemically treated filter samples used for measuring sulfur dioxide suffered desiccation problems. Although the samples were analyzed, the results were very questionable and did not compare with any of the detector tube samples collected during either this survey, or the previous survey. Therefore, those results were also voided.

Asbestos

Asbestos was used as a thermal insulating material in several areas of the power plant. Over the years, the insulating materials have been damaged or deteriorated thereby creating potential exposure to workers. The company has conducted asbestos abatement projects in some areas of the plant and has identified several other areas for abatement. Areas identified by the company for abatement include: bottom half of turbine and piping, evaporator (east end), DA storage tank (east end), IB boiler feed pump outlet check valve and piping, steam line to pulverizer, boiler sight glass piping, boiler low water trip piping, steam sample line off back of steam drum, and air heater steam for preheat coils outside lunch room.

Only during the May 1991 survey were samples collected of representative vacuum settled dust from each level of the plant. The samples were examined by polarized light microscopy to determine fiber content. The materials found in all the samples were essentially the same. The predominant feature was an abundance of rounded, isotropic glass balls of various sizes (fly ash). Non–fibrous mineral components were quartz, feldspar, and carbonate particulate. Very little fibrous material was present in any sample, less than 1 percent of the total matrix, and consisted of cellulose, polyester, and mineral (glass) fibers. No fibrous material meeting either the mineralogical or morphological characteristics of asbestos was seen in any sample.

Medical

May 1991 Survey

Twenty–two (73%) of 30 employees participated in the survey, completing the questionnaire, baseline spirometry, and cross–shift spirometry on at least one day. Ten employees were asked to perform peak flow monitoring based on a review of the screening questionnaire. These employees had indicated experiencing chest tightness or wheezing within 12 months prior to filling out the questionnaire. Seven of these 10 employees agreed to use the peak flow meters.

Ninety–five percent (21/22) of the participants were Caucasian males. Participants' ages ranged from 23–52 years, with an average of 38 years (standard deviation [sd] = 8). Fifty–nine percent (13/22) of the participants were "ever smokers" (that is, current smokers and ex–smokers combined). Of these 13, six were current smokers and seven were ex–smokers. Overall, "ever" smokers had a median of 10 pack–years (a pack–year is equivalent to smoking an average of one pack per day for a year) of cigarette smoking. Current smokers had a median of 13 pack–years, versus a median of 6 pack–years for ex–smokers. Forty–one percent (9/22) of the participants had never smoked cigarettes.

At the time of the survey, the duration of employment at Golden Valley Electric ranged from one to 24 years, with a median duration of seven years. Sixty–four percent (14/22) reported working as a Control Room Engineer or Operator, and 27% (6/22) reported working as Repairmen. The distribution of these job classifications were a reflection of the distribution of jobs within the plant as a whole at the time of this survey.

Respiratory Symptoms

Table 4 presents the prevalence of each symptom by cigarette smoking habit and the prevalence of symptoms among survey participants as a whole from the May 1991 survey. Overall, acute symptoms of highest prevalence were chest tightness (6/22, 27%) and chest wheezes or whistles (15/22, 23%). Chronic symptoms of highest prevalence were cough (7/22, 32%) and phlegm (5/22, 23%). Six (27%) participants reported a history of hayfever or other allergies. Of the participants with chest tightness, three (50%) also reported a history of hayfever or other allergies.

Among "ever" smokers, the most frequently reported acute symptom was chest tightness (4/13, 31%). Among "never" smokers, the most frequently reported symptoms were chest wheezes or whistles (3/9, 33%) and attacks of dyspnea with cough (3/9, 33%). Cough was the most frequently reported chronic symptom for both ever smokers (3/13, 23%), and never smokers (4/9, 44%).

Three (14%) participants met the criteria for work–related acute chest symptoms. Two were current cigarette smokers and one was a non–smoker. None of the three reported a prior history of hayfever or other allergies, or pre–existing asthma.

Spirometry

All 22 participants performed spirometry at least once during the survey. Twenty–one (95%) participants performed cross–shift spirometry on the last day of their work week, while 20 (91%), performed spirometry on the first day of the following week. Nineteen (86%) performed cross–shift spirometry on both days. Baseline spirometry results revealed one (5%) participant with results that fell below the normal range, a mild obstructive pattern and a cross–shift decrement in FEV_1 of 10% on the last day of the work week. Although none of the participants had a cross–shift decrement greater than 10% on either day, a total of three participants had an observed FEV_1 decline between 5–10% on the last day of their work week. Of the 21 participants for whom we had the appropriate measurements, none exhibited a weekend improvement.

Peak Flow Monitoring

Interpretable peak flow data were available for the seven participants who used a meter. Peak flow variability was < 20% over the recording period for all seven.

Asthma

Two (9%) of 22 participants reported a physician diagnosis of asthma. A review of their medical records confirmed the diagnosis. One of the two met the NIOSH surveillance case definition for occupational asthma (OA) by having a physician diagnosis of asthma, work–related acute chest symptoms, and a work–related change in FEV_1 of 10%. The second participant was categorized as a "possible" case because although he had a physician diagnosis and work–related chest symptoms, he did not have any of the diagnostic test criteria needed to fulfill the case definition.

Both of these individuals had a positive methacholine challenge test, although the criteria for defining the results as positive were not provided by the diagnosing physician, nor was the timing of the testing (whether following exposure at work or after a period away from work) recorded. Asthma was attributed by the physician to the workplace, but not to a specific agent within the workplace environment.

February 1993 Survey

Twenty–four (83%) of 29 employees participated in the February 1993 survey. Five employees who had not participated in the previous survey chose to participate in this survey, as well as two employees who had started work in the interim. Three participants from the May 1991 survey who were still employed at the time of the February 1993 survey did not participate, either by choice or because they were unavailable. Another employee had left employment at the plant, and a fifth participant was dropped from analysis because of insufficient data. All 24 participants completed the questionnaire and baseline spirometry. To determine if peak flow variability had changed since the preceding survey, the same seven participants who had used a peak flow meter in May 1991 were again approached and three agreed to use a meter. In addition, one participant who had reported chest symptoms on the screening questionnaire but who had been previously unavailable, and a participant who had been diagnosed with asthma in the interim, were approached and both agreed to use a meter. A total of five participants used a peak flow meter during this survey.

Because we re–visited essentially the same employee population, demographic and employment characteristics were similar, reflecting usual changes over time. Ninety–six percent (23/24) of the participants were male and 88% were Caucasian. The median age was 40 years, and the average age was 41 years (sd=7). Participants' ages ranged from 28–54 years. Tenure ranged from one to 25 years, with a median duration of 9 years.

Sixty–two percent (15/24) of the participants in this survey were "ever" smokers: five were current smokers and 10 were ex–smokers. Overall, "ever" smokers had a median of 16 pack–years of cigarette smoking. Current smokers had a median of 26 pack–years, versus 11 pack–years for ex–smokers. Thirty–eight percent (9/24) of the participants had never smoked cigarettes.

The distribution of employees among the various job classifications remained basically unchanged from the previous survey. However, two employees reported changing their job category or duties, and one reported reducing work hours in response to a respiratory health problem since the May 1991 survey.

Respiratory Symptoms

Symptoms of highest overall prevalence were chest tightness (10/24, 42%), chest wheezes or whistles (9/24, 38%), cough (9/23, 39%), and phlegm (8/24, 33%) (Table 5). Table 5 also reveals that chest tightness and chronic cough were the most frequently reported symptoms among both "ever" smokers and "never" smokers alike. Forty percent (6/15) of "ever" smokers and 44% (4/9) of "never" smokers reported that chest tightness occurred, while the prevalence of chronic cough was 33% (5/15) and 50% (4/8) for "ever" and "never" smokers, respectively.

A history of hayfever or other allergies was reported by thirteen (54%) of the participants. Two (8%) participants met the criteria for work–related acute chest symptoms. One was a smoker and one was a non–smoker; both reported a prior history of hayfever or other allergies. Neither of these two workers reported asthma.

Spirometry

All 24 participants performed spirometry at least once during the survey. Cross–shift spirometry data were collected on at least one work day for 23 participants, and 16 performed cross–shift spirometry on both the last day of their work week and the first day of the following week. Because of their work schedules, three participants were evaluated initially at the beginning of their work week (i.e., the order of testing was reversed). Baseline spirometry results revealed four participants with results that fell below the normal range: three exhibited a mild obstructive pattern, and one had a moderate obstructive pattern. Two were current smokers and two were ex– smokers. Two of these participants reported experiencing chest tightness, though the temporal pattern did not meet the criteria for being work–related.

None of the 23 participants who performed cross–shift spirometry had a decrement of ≥ 10% on either the last day, or the first day, of their work week. One participant had a cross–shift decrement in FEV_1 between 5–10% on the last day of the work week, and one participant had an FEV_1 decline between 5–10% on both the last day of the work week and on the first day of the following week. This same individual had a weekend improvement between 5–10%. Of the three individuals who were tested in the reverse order (over the work week), all had an observed decline in FEV_1 of less than 5%.

Peak Flow Monitoring

Interpretable peak flow data were available for all five participants who used a meter. Two participants exhibited a ≥ 20% variability in peak flow over the recording period, and one of the two had a pattern of variability that was suggestive of a temporal association with work. Unfortunately, the peak flow data from the second worker were not sufficient to allow for the evaluation of possible work–related effects. The maximum variability in peak flow for these two participants ranged from 21% – 29%, and both recorded the presence of symptoms and the use of inhalers, such as bronchodilators and steroids, to control their symptoms.

Asthma

Four (17%) participants reported currently having physician–diagnosed asthma. Two of the four had asthma documented during the May 1991 survey, and two reported being diagnosed over the 20–month period between the May 1991 survey and the February 1993 survey. A review of the medical records for these two additional participants confirmed the physician diagnosis, and both had a positive methacholine challenge test. As with the two participants initially diagnosed with asthma, the criteria for defining the results of the methacholine challenge test as positive were not provided, nor was the timing of the testing recorded. Neither of these two additional participants met the NIOSH case definition for OA. Both lacked the objective evidence of work–related spirometry or peak flow changes, and neither met the criteria for work–related acute chest symptoms. However, one of the two participants was categorized as a "possible" case since he reported using medication to control symptoms which he related to work. The second participant was diagnosed with asthma following a severe respiratory infection, reported a cough as his only symptom, and was not on any medication.

Longitudinal Evaluation

Seventeen employees, representing 77% of the participants of the May 1991 survey, participated in both surveys. Selected characteristics of the five employees who participated only in the May 1991 survey and the 17 who participated in both surveys are presented in Table 6. While these two groups were of similar age, they differed with respect to all other characteristics examined. Employees who participated in both surveys had worked at Golden Valley Electric, on average, three times as long as the May–only participants;

53% (9/17) were "ever" smokers versus 80% (4/5) of the May–only participants. None of the May–only participants reported asthma, and a greater proportion reported a history of allergies and symptoms, except for chronic dyspnea and attack of dyspnea with cough, than participants in both surveys.

Table 7 presents the prevalence of respiratory symptoms and conditions of the 17 individuals who participated in both surveys. The prevalence of all symptoms, except for attacks of dyspnea with cough, were the same or higher during the February 1993 survey. Application of a statistical test (McNemar's) revealed no significant difference in the prevalence of any symptom between the May 1991 survey and the February 1993 survey. Among other changes that occurred, the number of participants reporting a history of allergies increased from four to seven, and the proportion of participants with work–related chest symptoms decreased. Again, these differences were not statistically significant. Two participants had given up cigarette smoking between the first and second surveys.

A review of the data also revealed that the number of participants with a confirmed physician diagnosis of asthma increased from two to four over the 20–month period between the two surveys. There was no statistically significant difference in the proportion of participants with asthma from May 1991 to February 1993. All four workers with confirmed asthma had been employed at Golden Valley Electric for over 10 years. Two were Control Room Operators and two were Repairmen. One of the Operators had changed jobs between the first and the second survey, originally working as a Repairman. Only one of the physician–diagnosed asthmatics exhibited an abnormal lung function pattern; the three others had spirometry results within the normal range. Two of the four had never smoked, one was an ex–smoker, and one was a current smoker. Only one of the four reported a history of allergies (a childhood food allergy).

None of the 17 participants present at both surveys exhibited a decrease in their FEV_1 that was greater than expected for a 20–month time interval. Peak flow monitoring results revealed two participants with variability within the range considered normal at the time of the May 1991 survey who subsequently exhibited a greater than normal variability (>20%) at the time of the February 1993 survey. However, only one of these participants had a variability pattern that was temporally work–related.

DISCUSSION

In this investigation, a total of four employees at Golden Valley Electric were confirmed to have physician–diagnosed asthma; two were documented during the May 1991 evaluation and two were documented during the February 1993 evaluation. Of these four, one was classified as having definite occupational asthma, two were classified as "possible" cases of occupational asthma, and the remaining employee appeared to have increased airway irritability of a post–infectious origin. Both of the employees with "possible" occupational asthma had worked in occupations or industries that are associated with exposure to both fibrogenic dusts and/or agents that may cause allergy–induced asthma. The type of work reported included seasonal work in the timber industry, short–term employment (less than one month) in seafood processing, farming, highway construction, and work in a quarry and a barite mine. Only one of these two participants reported working for an extended period of time at such jobs (approximately three to five years). Both were employed at Golden Valley Electric for over 10 years.

During both surveys, the prevalence of both acute and chronic chest symptoms were relatively high, with "chest tightness" the acute symptom of highest prevalence and "cough" the chronic symptom of highest prevalence. Although the increases in prevalence of chest symptoms and asthma between seasons were not statistical significance, the small size of the study population severely limits the power of the study to confirm or rule out all but the largest differences. Additionally, no definitive conclusions can be drawn regarding the difference in symptom prevalence between those who chose to participate in both surveys and the small number (5) who participated in only one survey, among whom only one had left employment.

The NIOSH surveillance case definition for OA is fairly broad and clinical in its orientation, and is not explicit as to whether the exposure to a substance should be acute or chronic in order to classify a case as occupational asthma. Given our restriction of the case definition to specific agents known to produce sensitization or asthma that develops after a single intense irritant exposure, only one of the four employees with asthma was classified as having definite OA. It is possible that the two employees classified as "possible" cases may indeed have OA. The fact that both reported symptoms associated with work, and medication utilization that correlated with exposure at work, suggests a relationship to the workplace. These individuals may not have experienced an acute decrement over the course of either the work shift or work week because of their medical treatment for their asthma. Additionally, respiratory symptoms may not have improved away from work because of increased irritability of the airways to non–specific triggers outside of the workplace. Non–specific triggers in the general environment include cigarette smoke, cold dry air, household cleaning agents, exercise, and inert dust.[9, 30, 31, 32]

Over 15% of all newly–diagnosed adult–onset asthma may be related to workplace exposures.[33,34] Causes of occupational asthma include an immunologic response of the body to an antigen (i.e., allergy–induced asthma) or irritant–induced asthma (also referred to as RADS or reactive airways dysfunction syndrome) due to exposures to extremely high levels — such as those associated with spills or unintended releases — of irritant gases, fumes, or chemicals on one or multiple occasions.[34, 35, 36, 37, 38] Asthma that may be attributable to irritant exposures has received less study than allergy–induced asthma, and its prevalence is unknown, with discussions in the scientific literature focusing primarily on cases meeting the criteria for RADS.

Irritant gases associated with coal combustion, particularly sulfur dioxide, are an important class of chemical agents that have been found to directly induce inflammation, reflex bronchoconstriction, and non–specific bronchial hyperreactivity (NSBH).[30, 39, 40, 41] Long–term, low level ($\leq$1.5 ppm) occupational exposure to sulfur dioxide has been associated with an increase in respiratory symptoms, such as chronic phlegm, wheeze, and mild exertional dyspnea,[42] and various experimental studies have indicated that respiratory effects of sulfur dioxide may be greater in asthmatics, particularly in the presence of exercise and low humidity.[43] Kipen[44] described 10 cases of adult–onset asthma that did not meet the criteria for being either allergen–induced or irritant–induced asthma (i.e., requiring an acute high exposure). "Low–Dose Reactive Airways Dysfunction Syndrome" was the term the authors used to describe this condition, and suggested that the development of adult–onset asthma was associated with recurrent low–dose irritant exposure. During the course of these investigations, we received no reports of required hospitalization due to an acute irritant exposure. However, the practice of reversing exhaust fans during the winter, and description of the plant filling with boiler gas on occasion, may have resulted in peak exposures that, in combination with chronic low–level exposures, may have induced or exacerbated acute chest symptoms and asthma in this worker population.

Additionally, exposures to particulates associated with coal–fired electric generation may also have contributed towards the development of non–specific respiratory symptoms and respiratory disease seen at this facility. Occupational exposures to mineral dust, such as coal dust and its by–product, pulverized fuel ash (which consists primarily of silica and silicates), have been associated with the development of chronic bronchitis, emphysema, asthma, and pulmonary fibrosis (caused by retention of dust in the lung).[45, 46, 47] Davison[48] reported a case of work–related asthma from pulverized fuel ash in an atopic power plant attendant.

CONCLUSIONS

Analysis of the environmental data collected during both surveys indicated that a potential health hazard existed from exposure to sulfur dioxide and crystalline silica. Environmental sampling results from both surveys showed a potential for exposure to high concentrations of sulfur dioxide in the baghouse area. In addition, crystalline silica (quartz) was detected in the bulk and air samples collected in the baghouse and coal tunnel.

One definite case of occupational asthma was documented, and there were two additional possible cases. Each case of occupational asthma represents a sentinel health event indicating a potentially hazardous worksite environment. The high prevalences of acute and chronic chest symptoms and asthma experienced by the participants during both surveys appear to be consistent with the effects of exposure to respiratory tract irritants. Although sulfur dioxide and crystalline silica dust were the only agents that posed a potential health hazard, it is impossible to say with certainty that these are the agents responsible, given the numerous respiratory irritants that workers may be potentially exposed to at this worksite.

An important consideration in evaluating crystalline silica exposure is the percentage of these materials in the dust. In particular, the silica content in the fly ash at this facility seemed consistent at approximately 7%. Exposure to silica is likely to occur during "blow down" when the employees use air hoses and brooms to clean the settled fly ash from the plant surfaces. During the cleaning operations not only would the individuals doing the cleaning be overexposed, but other personnel throughout the plant would be as well. And, not only are the plant personnel potentially exposed to the contaminants in the fly ash, but so are the workers' families. When the workers clean the settled dust from the plant or enter the baghouse to remove and/or clean filters, their clothing becomes very soiled. These work clothes are then taken home to be laundered which results in potential exposure for the family.

Another major concern on the part of the workers was exposure to asbestos. The facts are: (1) asbestos containing materials (ACM) were used throughout the facility, (2) some of the ACM has been damaged and/or deteriorated and is friable, (3) asbestos abatement projects to remove or repair ACM have been conducted in certain areas of the plant, and (4) additional areas have been identified for additional abatement projects. Even though we did not find any asbestos in the settled dust samples collected during our visit, this does not mean that the potential for airborne asbestos exposure does not exist. Friable asbestos was found in several areas workers are required to work in or on without adequate protection.

The respiratory protective devices observed being used during the spring 1991 survey were certified for protection against the particulates, gases, and vapors that may be present in the work environment. However, based on observations of workers with full beards wearing a respirator, and discussions with the workers and management, problems within the company’s respiratory protection program existed. First, the workers and plant management were not adequately informed or trained in the proper use of the respirators provided. Secondly, there was no established, written respiratory protection program at the plant.

RECOMMENDATIONS

Environmental

- The procedure of using compressed air to clean the settled fly ash from the plant surfaces should be eliminated. Company management and employees should work together to arrive at a procedure which will be adequate to clean the facility without creating dusty conditions.

- Personnel required to work in the coal tunnel and bottom floor of the bag house should be provided with proper respiratory protection for both respirable dusts and crystalline silica. Where accepted engineering control measures have not been developed or when necessary due to the nature of the work involved (for example, while establishing controls or occasional entry into hazardous areas to perform maintenance), employees may work for reasonable periods of time in concentration of airborne contaminants exceeding permissible levels if they are protected by appropriate respiratory protective equipment. When respiratory protective equipment is used, a written program for selection, maintenance, training, fitting, supervision, cleaning, and use shall be established. It is recommended that a respirator program consistent with the requirements of ANSI Z88.2–1980, OSHA standard 29 CFR 1910.134, and the NIOSH Respirator Decision Logic be established at the plant. OSHA (29 CFR 1910.132) requires that protective equipment, including personal protective equipment, be provided at no cost to the employee if the potential exists for exposure to a substance capable of causing injury or impairment. The CFR further requires that if employees provide their own protective equipment, the employer shall be responsible to assure its adequacy, including proper maintenance and sanitation of the equipment. NIOSH further recommends that: (1) the employer shall assure that protective clothing that has become contaminated is cleaned or laundered before its reuse, (2) the employer shall assure that no employee take home equipment or clothing that is contaminated, (3) the employer shall inform any person who launders such clothing of the potentially harmful effects and of procedures to safely handle the clothing, and (4) the employer shall provide change rooms for employees who are required to change into protective clothing.

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

WARNING!
FREE SILICA WORK AREA
Avoid Breathing Dust
Use Appropriate Respiratory Protection
May Cause Delayed Lung Injury (Silicosis)

  The posting should be printed both in English and in the predominant language of non-English-speaking workers, unless they are otherwise trained and informed of the hazardous areas. Illiterate workers shall receive such training. Enforcement of respiratory protection needs to be stressed to all workers.

- Exposures to sulfur dioxide were above the NIOSH REL, but below the OSHA PEL. It is recommended that Golden Valley adopt a monitoring program to assess the sulfur dioxide concentrations within the plant, in particular the upper floors of the baghouse. Such monitoring should assess exposures during procedures which are likely to result in increased sulfur dioxide concentrations. If personal exposures are found to be in excess of environmental criteria, appropriate control measures should be taken.

! If asbestos–containing materials are removed, hazardous exposures will likely result if (1) proper removal techniques, (2) exposure controls, or (3) proper disposal methods are not used. Considerable attention has been paid to these three subject areas in asbestos abatement over the last decade in the United States. A fourth area, which may require more effort, is education and training. The ability to identify potential asbestos–containing material, to understand the health hazards associated with exposure to asbestos, and to undertake appropriate control techniques are equally important. All asbestos abatement policies and procedures should meet the OSHA asbestos regulations as specified in 29 Code of Federal Regulations (CFR), Section 1910.1001 (General Industry Safety and Health Standards) and 29 CFR 1926.58 (Construction Standards for Asbestos).

! Golden Valley Electric should conduct periodic industrial hygiene sampling of all plant operations. This will be particularly important in the event of a process or chemical ingredient change. It is not necessary to sample the entire plant each time, but rather target particular processes. All sampling records should be maintained in accordance with OSHA regulations, and results posted for employee inspection. A list of industrial hygiene consultants is available in a number of different professional journals.

Medical

A. Golden Valley Electric Association should institute a medical monitoring program. The following recommendations for medical monitoring are based on NIOSH guidelines for workers exposed to crystalline silica [49] and a compilation of NIOSH recommendations concerning occupational asthma. A medical examination and screening tests should be available to all workers prior to job placement to provide a baseline for future evaluations, and at least once every three years thereafter.

Examinations should include at a minimum:

! A medical and occupational history to collect data on current and prior exposure to coal dust, fly ash, and other dusts which can cause pneumoconiosis; previous exposures to agents known to cause asthma; signs and symptoms of respiratory disease; and smoking. Special attention should be paid to pre–existing lung symptoms or history of asthma.

! A chest x–ray (posterior–anterior 14" x 17"), preferably obtained using a high kilo–voltage technique, and classified by a B reader according to the 1980 International Labour Organization (ILO) International Classification of Radiographs of Pneumoconioses. Film quality and technique should conform to specifications outlined by Wagner et al.[50]

! Pulmonary function testing (spirometry) including forced vital capacity (FVC) and forced expiratory volume at one second (FEV_1), using equipment and methods consistent with ATS recommendations.[51]

! Skin testing for tuberculosis (TB). The association of TB with silicosis and silica exposure is well–known.[20] Skin testing procedures should be in accordance with the 1990 ATS/CDC guidelines.[52]

The examination should also include the eyes, because of the potential for over–exposure to sulfur dioxide.

It must be emphasized that under the Americans with Disabilities Act (Public Law 1–1–336 [S.993]; July 26, 1990) employment–related medical evaluations cannot be used to make decisions concerning employability; the employer cannot make any inquiries concerning whether an applicant has any unusual medical conditions prior to making a job offer.[53] Thus, a history of pre–existing asthma or silicosis (or other pneumoconiosis) would not be grounds for refusing an individual employment. Prior to placement within the facility (i.e., once hired), however, medical examination results can be used in decisions regarding placement or the need for accommodations.

B. Workers with abnormal spirometry or symptoms such as persistent cough, cough at night, wheezing, or difficulty breathing; a positive finding on the chest x–ray; or all of the previously mentioned, should be referred for a more thorough medical evaluation by a physician qualified to advise the employee and company whether the employee has a condition that is likely to be aggravated by continuing workplace exposure(s) and associated with increased risk of impairment of respiratory health. Workers who develop any otherwise unexplained symptoms of work–related asthma in the period between examinations should also receive such an evaluation.

C. Each employee should receive a written copy of his/her medical examination results in full detail, with the results reviewed with a health care professional at the time the employee receives them.

D. All cases of silicosis or suspected work–related asthma should be reported to the State health department, and recorded as required by OSHA. To enhance the uniformity of reporting, NIOSH has developed reporting guidelines and case definition for both occupational asthma (Appendix I)[7] and silicosis (Appendix II).[54] These definitions and guidelines are recommended for surveillance of work–related illnesses by State health departments and regulatory agencies receiving reports of cases from physicians and other health care providers.

E. A plant–wide no–smoking policy and smoking cessation program should be implemented if not already in place.[55] Cigarette smoking may predispose workers to the development of irritant–induced asthma [44,35], and changes in lung function may be potentiated by occupational exposure to respirable mineral dust and cigarette smoking.[56]

F. Medical records should be maintained separately and in a confidential manner, and access should be limited to health care personnel. Records of environmental exposures applicable to an employee should be included in the employee's medical records. These records should be kept for at least 30 years following an employee's termination of employment. Maintaining records for this length of time is necessary because of the long latency associated with silicosis and other chronic lung conditions.

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Table 1
Personal Sampling Results
for Respirable Dust and Crystalline Silica
February 1993

HETA 91–0047–2672
Golden Valley Electric, Healy Power Plant
Healy, Alaska

Sample Number	Volume (liters)	Respirable Dust Concentration (mg/m^3)	Silica Dust Concentration (mg/m^3)	Sampling Location	Date
2097	928.2	1.02	ND	Mechanical Repairman	Feb 4
2104	924.8	0.05	ND	Instrument Repairman	Feb 4
2109	941.8	0.05	ND	Instrument Repairman	Feb 4
2122	746.3	0.09	ND	Assistant Operator	Feb 4
2128	805.8	1.17	ND	Maintenance Mechanic	Feb 4
2145	950.3	0.05	ND	Auxiliary Operator	Feb 4
2156	970.7	0.05	ND	Utilityman	Feb 4
2095	938.4	0.04	ND	Electrician	Feb 5
2103	817.7	0.00	ND	Control Room Operator	Feb 5
2140	958.8	0.06	ND	Auxilary Operator	Feb 5
2146	946.9	0.00	ND	Storeroom	Feb 5
2150	974.1	0.10	ND	Utilityman	Feb 5
2082	576.3	0.49	ND	Utilityman	Feb 6

ND = Not Detected

Table 2
Work Area Sampling Results
for Respirable Dust and Crystalline Silica
February 1993

HETA 91–0047–2672
Golden Valley Electric, Healy Power Plant
Healy, Alaska

Sample Number	Volume (liters)	Respirable Dust Concentration (mg/m³)	Silica Dust Concentration (mg/m³)	Sampling Location	Date
2089	758.2	0.00	ND	Control Room	Feb 4
2090	863.6	0.08	ND	Basement (near feed chemicals)	Feb 4
2091	846.6	0.01	ND	2nd Floor (by coal pulveriser)	Feb 4
2092	844.9	0.01	ND	Burner Deck	Feb 4
2094	826.2	0.02	ND	5th Floor Baghouse	Feb 4
2096	878.9	0.06	ND	Turbine Deck	Feb 4
2098	827.9	0.05	0.02	Baghouse (basement)	Feb 4
2099	844.9	0.02	ND	6th Floor (by air intakes)	Feb 4
2102	846.6	0.06	ND	5th Floor (near DA tank)	Feb 4
2107	620.5	0.15	ND	Baghouse (top floor)	Feb 4
2087	435.2	2.80	0.06	Coal Tunnel (bottom)	Feb 5
2100	727.6	0.00	ND	Baghouse (basement)	Feb 5
2101	785.4	0.05	ND	Control Room	Feb 5
2106	440.3	0.00	ND	5th Floor (near DA tank)	Feb 5
2108	435.2	3.84	0.18	Coal Tunnel (middle)	Feb 5
2110	566.1	0.23	ND	Coal Tunnel (top)	Feb 5
2116	790.5	0.04	ND	Turbine Deck	Feb 5
2137	727.6	0.10	ND	Baghouse (middle floor)	Feb 5
2138	742.9	0.07	ND	Burner Deck	Feb 5
2143	787.1	0.08	ND	7th Floor (near air intakes)	Feb 5
2144	633	0.03	ND	Boiler (basement)	Feb 5
2151	528.7	0.00	ND	Baghouse (top floor)	Feb 5
2158	765	0.04	ND	2nd Floor (by coal pulveriser)	Feb 5
2063	664.7	0.05	ND	Baghouse (top floor)	Feb 6
2064	596.7	0.12	ND	Baghouse (basement)	Feb 6
2065	746.3	0.00	ND	Control Room	Feb 6
2066	649.4	0.02	ND	Turbine Deck	Feb 6
2067	647.7	0.00	ND	Boiler (basement)	Feb 6
2068	627.3	0.00	ND	5th Floor (near DA tank)	Feb 6
2072	610.3	0.03	ND	2nd Floor (by coal pulveriser)	Feb 6
2075	595	0.10	ND	7th Floor (intakes)	Feb 6
2088	642.6	0.03	ND	Burner Deck	Feb 6
2093	538.9	1.41	0.05	Coal Tunnel (bottom)	Feb 6
2105	612	0.08	ND	5th Floor Boiler (by Baghouse)	Feb 6
2152	538.9	1.74	0.07	Coal Tunnel (middle by heater)	Feb 6

ND = Not Detected

Table 3
Personal Sampling Results
for Sulfur Dioxide
February 1993

HETA 91–0047–2672
Golden Valley Electric, Healy Power Plant
Healy, Alaska

Date	Sample Number	Occupation	SO_2 Concentration (ppm)
Feb 4	2097	Mechanical Repairman	1.1
Feb 4	2104	Instrument Repairman	void (tube broken)
Feb 4	2109	Instrument Repairman	1.6
Feb 4	2122	Assistant Operator	1.2
Feb 4	2128	Maintenance Mechanic	2.5
Feb 4	2145	Auxiliary Operator	1.1
Feb 4	2156	Utilityman	1.0
Feb 5	2095	Electrician	2.2
Feb 5	2103	Control Room Operator	2.5
Feb 5	2140	Auxiliary Operator	2.1
Feb 5	2146	Storeroom	2.1
Feb 5	2150	Utilityman	2.1
Feb 6	2082	Utilityman	1.5

Table 4
Prevalence of Respiratory Symptoms by Cigarette Smoking Habit
May 1991

HETA 91–0047–2672
Golden Valley Electric, Healy Power Plant
Healy, Alaska

Respiratory Symptom*	**Cigarette Smoking Habit**				**Survey Total**	
	Ever		***Never***			
	(Number = 13)		(Number = 9)		(Number = 22)	
	Yes	**%**	**Yes**	**%**	**Yes**	**%**
Acute:						
Chest Tightness	4	31	2	22	6	27
Chest Wheezes/Whistles	2	15	3	33	5	23
Attacks of Dyspnea with Wheeze	1	8	1	11	2	9
Attacks of Dyspnea with Cough	1	8	3	33	4	18
Chronic:						
Cough	3	23	4	44	7	32
Phlegm	2	15	3	33	5	23
Bronchitis	1	8	2	22	3	14
Dyspnea	1	8	1	11	2	9

*(*See text for definitions)*

Table 5
Prevalence of Respiratory Symptoms by Cigarette Smoking Habit
February 1993

HETA 91–0047–2672
Golden Valley Electric, Healy Power Plant
Healy, Alaska

Respiratory Symptom*	**Cigarette Smoking Habit**				**Survey Total**	
	Ever		***Never***			
	(Number = 15)		(Number = 9)		(Number = 24)	
	Yes	**%**	**Yes**	**%**	**Yes**	**%**
Acute:						
Chest Tightness	6	40	4	44	10	42
Chest Wheezes/Whistles	6	40	3	33	9	38
Attacks of Dyspnea with Wheeze	4	27	1	11	5	21
Attacks of Dyspnea with Cough	3	20	1	11	4	17
Chronic:						
Cough	5	33	4**	50	9**	39
Phlegm	4	27	4	44	8	33
Bronchitis	4	27	3	33	7	29
Dyspnea	1	7	1	11	2	8

*See text for definitions
**One participant did not complete the questions on cough

Table 6
Selected Characteristics of Participants by Survey Participation

HETA 91–0047–2672
Golden Valley Electric, Healy Power Plant
Healy, Alaska

Characteristics	*Survey Participation*			
	May 1991 Only		**Both Surveys**	
Number of Participants	5		17	
Age (median)	32		38	
Tenure (median)	4		12	
Smoking Status	**Number**	**%**	**Number**	**%**
Ever	4	80	9	53
Never	1	20	8	47
Respiratory Symptom*	**Number**	**%**	**Number**	**%**
Chest Tightness	3	60	3	18
Chest Wheezes/Whistles	2	40	3	18
Attacks of Dyspnea with Wheeze	1	20	1	6
Attacks of Dyspnea with Cough	—	—	4	24
Chronic Cough	3	60	4	24
Chronic Phlegm	3	60	2	12
Chronic Dyspnea	—	—	2	12
Medical History	**Number**	%	**Number**	**%**
Asthma	—	—	2	12
Allergies	2	40	4	24

*See text for definitions

Table 7
Comparison of Respiratory Symptoms and Conditions Reported by 17 Employees Present for Both Surveys

HETA 91–0047–2672
Golden Valley Electric, Healy Power Plant
Healy, Alaska

Condition	Survey*			
	May 1991		February 1993	
	Yes	%	Yes	%
Respiratory Symptom				
***Acute*:**				
Chest Tightness	3	18	7	41
Chest Wheezes/Whistles	3	18	5	29
Attacks of Dyspnea with Wheeze	1	6	4	24
Attacks of Dyspnea with Cough	4	24	2	12
Chronic:				
Cough	4	24	7**	44
Phlegm	2	12	6	35
Dyspnea	2	12	2	12
Work–related Symptoms	2	12	1	6
Medical Condition				
Asthma (physician–diagnosed)	2	12	4	24
Allergies	4	24	7	41

*Differences not statistically significant at the p = 0.05 level
**One participant did not complete the questions on cough

APPENDIX I

APPENDIX I

NIOSH Surveillance Case Definition for Occupational Asthma [7]

A. A physician diagnosis of asthma

AND

B. An association between symptoms of asthma and work and any one of the following:

1. Workplace exposure to an agent or process previously associated with occupational asthma.

OR

2. Significant work–related spirometry changes in forced expiratory volume in one second (FEV_1) or peak expiratory flow rate (PEFR).

OR

3. Significant work–related changes in airways responsiveness as measured by nonspecific inhalation challenge.

OR

4. Positive response to inhalation provocation testing with an agent to which the patient is exposed at work. Inhalation provocation testing with workplace substances is potentially dangerous and should be performed by experienced personnel in a hospital setting where resuscitation facilities are available and where frequent observations can be made over sufficient time to monitor for delayed reactions.

Patterns of work–related disease association can vary. The following examples are patterns that may suggest an occupational etiology: symptoms of asthma develop after a worker starts a new job or after new materials are introduced on a job (a substantial period of time may elapse between initial exposure and development of symptoms); symptoms develop within minutes of specific activities or exposures at work; delayed symptoms occur several hours after exposure, during the evenings of the workdays; symptoms occur less frequently or not at all on days away from work and on vacations; symptoms occur more frequently on returning to work. Work–related changes in medication requirements may have similar patterns, also suggesting an occupational etiology.

Many agents and processes have been associated with occupational asthma,[10,54] and others continue to be recognized. Changes in nonspecific bronchial hyperactivity can be measured by serial inhalation challenge testing with methacholine challenge or histamine. Increased bronchial reactivity (manifested by reaction to lower concentrations of methacholine or histamine) following exposure and decreased bronchial reactivity after a period away from work are evidence of work–relatedness.

APPENDIX II

NIOSH SURVEILLANCE GUIDELINES: SILICOSIS [54]

Reporting Guidelines

State health departments and regulatory agencies should encourage physicians (including radiologists, pathologists, and other health care providers) to report all diagnosed or suspected cases of silicosis. These reports should include persons with

— a physician's provisional or working diagnosis of silicosis, **OR**

— a chest x–ray interpreted as consistent with silicosis, **OR**

— pathologic findings consistent with silicosis

To set priorities for workplace investigations, State health departments and regulatory agencies should collect appropriate clinical, epidemiologic, and workplace information about persons reported to have silicosis.

Surveillance Case Definition

A. 1. History of occupational exposure to airborne silica dust

AND

2. Chest x–ray or other imaging technique interpreted as consistent with silicosis

OR

B. Pathologic findings characteristic of silicosis

Delivering on the Nation's promise:
Safety and health at work
For all people
Through research and prevention

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Table 1
Personal Sampling Results
for Respirable Dust and Crystalline Silica
February 1993

HETA 91–047
Golden Valley Electric, Healy Power Plant
Healy, AK

Sample Number	Volume (liters)	Respirable Dust Concentration (mg/m^3)	Silica Dust Concentration (mg/m^3)	Sampling Location	Date
2097	928.2	1.02	ND	Mechanical Repairman	Feb 4
2104	924.8	0.05	ND	Instrument Repairman	Feb 4
2109	941.8	0.05	ND	Instrument Repairman	Feb 4
2122	746.3	0.09	ND	Assistant Operator	Feb 4
2128	805.8	1.17	ND	Maintenance Mechanic	Feb 4
2145	950.3	0.05	ND	Auxiliary Operator	Feb 4
2156	970.7	0.05	ND	Utilityman	Feb 4
2095	938.4	0.04	ND	Electrician	Feb 5
2103	817.7	0.00	ND	Control Room Operator	Feb 5
2140	958.8	0.06	ND	Auxilary Operator	Feb 5
2146	946.9	0.00	ND	Storeroom	Feb 5
2150	974.1	0.10	ND	Utilityman	Feb 5
2082	576.3	0.49	ND	Utilityman	Feb 6

ND = Not Detected

Table 2
Work Area Sampling Results
for Respirable Dust and Crystalline Silica
February 1993

HETA 91–047
Golden Valley Electric, Healy Power Plant
Healy, AK

Sample Number	Volume (liters)	Respirable Dust Concentration (mg/m^3)	Silica Dust Concentration (mg/m^3)	Sampling Location	Date
2089	758.2	0.00	ND	Control Room	Feb 4
2090	863.6	0.08	ND	Basement (near feed chemicals)	Feb 4
2091	846.6	0.01	ND	2nd Floor (by coal pulveriser)	Feb 4
2092	844.9	0.01	ND	Burner Deck	Feb 4
2094	826.2	0.02	ND	5th Floor Baghouse	Feb 4
2096	878.9	0.06	ND	Turbine Deck	Feb 4
2098	827.9	0.05	0.02	Baghouse (basement)	Feb 4
2099	844.9	0.02	ND	6th Floor (by air intakes)	Feb 4
2102	846.6	0.06	ND	5th Floor (near DA tank)	Feb 4
2107	620.5	0.15	ND	Baghouse (top floor)	Feb 4
2087	435.2	2.80	0.06	Coal Tunnel (bottom)	Feb 5
2100	727.6	0.00	ND	Baghouse (basement)	Feb 5
2101	785.4	0.05	ND	Control Room	Feb 5
2106	440.3	0.00	ND	5th Floor (near DA tank)	Feb 5
2108	435.2	3.84	0.18	Coal Tunnel (middle)	Feb 5
2110	566.1	0.23	ND	Coal Tunnel (top)	Feb 5
2116	790.5	0.04	ND	Turbine Deck	Feb 5
2137	727.6	0.10	ND	Baghouse (middle floor)	Feb 5
2138	742.9	0.07	ND	Burner Deck	Feb 5
2143	787.1	0.08	ND	7th Floor (near air intakes)	Feb 5
2144	633	0.03	ND	Boiler (basement)	Feb 5
2151	528.7	0.00	ND	Baghouse (top floor)	Feb 5
2158	765	0.04	ND	2nd Floor (by coal pulveriser)	Feb 5
2063	664.7	0.05	ND	Baghouse (top floor)	Feb 6
2064	596.7	0.12	ND	Baghouse (basement)	Feb 6
2065	746.3	0.00	ND	Control Room	Feb 6
2066	649.4	0.02	ND	Turbine Deck	Feb 6
2067	647.7	0.00	ND	Boiler (basement)	Feb 6
2068	627.3	0.00	ND	5th Floor (near DA tank)	Feb 6
2072	610.3	0.03	ND	2nd Floor (by coal pulveriser)	Feb 6
2075	595	0.10	ND	7th Floor (intakes)	Feb 6
2088	642.6	0.03	ND	Burner Deck	Feb 6
2093	538.9	1.41	0.05	Coal Tunnel (bottom)	Feb 6
2105	612	0.08	ND	5th Floor Boiler (by Baghouse)	Feb 6
2152	538.9	1.74	0.07	Coal Tunnel (middle by heater)	Feb 6

ND = Not Detected

Table 3
Personal Sampling Results
for Sulfur Dioxide
February 1993

HETA 91–047
Golden Valley Electric, Healy Power Plant
Healy, AK

Date	Sample Number	Occupation	SO_2 Concentration (ppm)
Feb 4	2097	Mechanical Repairman	1.1
Feb 4	2104	Instrument Repairman	void (tube broken)
Feb 4	2109	Instrument Repairman	1.6
Feb 4	2122	Assistant Operator	1.2
Feb 4	2128	Maintenance Mechanic	2.5
Feb 4	2145	Auxiliary Operator	1.1
Feb 4	2156	Utilityman	1.0
Feb 5	2095	Electrician	2.2
Feb 5	2103	Control Room Operator	2.5
Feb 5	2140	Auxiliary Operator	2.1
Feb 5	2146	Storeroom	2.1
Feb 5	2150	Utilityman	2.1
Feb 6	2082	Utilityman	1.5

Table 4
Prevalence of Respiratory Symptoms by Cigarette Smoking Habit
May 1991

HETA 91–047
Golden Valley Electric, Healy Power Plant
Healy, AK

Respiratory Symptom*	Cigarette Smoking Habit				Survey Total	
	Ever		Never			
	(Number = 13)		(Number = 9)		(Number = 22)	
	Yes	%	Yes	%	Yes	%
Acute:						
Chest Tightness	4	31	2	22	6	27
Chest Wheezes/Whistles	2	15	3	33	5	23
Attacks of Dyspnea with Wheeze	1	8	1	11	2	9
Attacks of Dyspnea with Cough	1	8	3	33	4	18
Chronic:						
Cough	3	23	4	44	7	32
Phlegm	2	15	3	33	5	23
Bronchitis	1	8	2	22	3	14
Dyspnea	1	8	1	11	2	9

*(*See text for definitions)*

Table 5
Prevalence of Respiratory Symptoms by Cigarette Smoking Habit
February 1993

HETA 91–047
Golden Valley Electric, Healy Power Plant
Healy, AK

Respiratory Symptom*	**Cigarette Smoking Habit**				**Survey Total**	
	Ever		***Never***			
	(Number = 15)		(Number = 9)		(Number = 24)	
	Yes	**%**	**Yes**	**%**	**Yes**	**%**
Acute:						
Chest Tightness	6	40	4	44	10	42
Chest Wheezes/Whistles	6	40	3	33	9	38
Attacks of Dyspnea with Wheeze	4	27	1	11	5	21
Attacks of Dyspnea with Cough	3	20	1	11	4	17
Chronic:						
Cough	5	33	4**	50	9**	39
Phlegm	4	27	4	44	8	33
Bronchitis	4	27	3	33	7	29
Dyspnea	1	7	1	11	2	8

*See text for definitions
**One participant did not complete the questions on cough

Table 6
Selected Characteristics of Participants by Survey Participation

HETA 91–047
Golden Valley Electric, Healy Power Plant
Healy, AK

Characteristics	***Survey Participation***			
	May 1991 Only		**Both Surveys**	
Number of Participants	5		17	
Age (median)	32		38	
Tenure (median)	4		12	
Smoking Status	**Number**	**%**	**Number**	**%**
Ever	4	80	9	53
Never	1	20	8	47
Respiratory Symptom*	**Number**	**%**	**Number**	**%**
Chest Tightness	3	60	3	18
Chest Wheezes/Whistles	2	40	3	18
Attacks of Dyspnea with Wheeze	1	20	1	6
Attacks of Dyspnea with Cough	—	—	4	24
Chronic Cough	3	60	4	24
Chronic Phlegm	3	60	2	12
Chronic Dyspnea	—	—	2	12
Medical History	**Number**	%	**Number**	**%**
Asthma	—	—	2	12
Allergies	2	40	4	24

*See text for definitions

Table 7
Comparison of Respiratory Symptoms and Conditions
Reported by 17 Employees Present for Both Surveys

HETA 91–047
Golden Valley Electric, Healy Power Plant
Healy, AK

Condition	Survey*			
	May 1991		February 1993	
	Yes	%	Yes	%
Respiratory Symptom				
***Acute*:**				
Chest Tightness	3	18	7	41
Chest Wheezes/Whistles	3	18	5	29
Attacks of Dyspnea with Wheeze	1	6	4	24
Attacks of Dyspnea with Cough	4	24	2	12
Chronic:				
Cough	4	24	7**	44
Phlegm	2	12	6	35
Dyspnea	2	12	2	12
Work–related Symptoms	2	12	1	6
Medical Condition				
Asthma (physician–diagnosed)	2	12	4	24
Allergies	4	24	7	41

*Differences not statistically significant at the p = 0.05 level
**One participant did not complete the questions on cough

APPENDIX I

NIOSH Surveillance Case Definition for Occupational Asthma [7]

A. A physician diagnosis of asthma

AND

B. An association between symptoms of asthma and work and any one of the following:

Workplace exposure to an agent or process previously associated with occupational asthma.

OR

2. Significant work–related spirometry changes in forced expiratory volume in one second (FEV_1) or peak expiratory flow rate (PEFR).

OR

Significant work–related changes in airways responsiveness as measured by nonspecific inhalation challenge.

OR

Positive response to inhalation provocation testing with an agent to which the patient is exposed at work. Inhalation provocation testing with workplace substances is potentially dangerous and should be performed by experienced personnel in a hospital setting where resuscitation facilities are available and where frequent observations can be made over sufficient time to monitor for delayed reactions.

Patterns of work–related disease association can vary. The following examples are patterns that may suggest an occupational etiology: symptoms of asthma develop after a worker starts a new job or after new materials are introduced on a job (a substantial period of time may elapse between initial exposure and development of symptoms); symptoms develop within minutes of specific activities or exposures at work; delayed symptoms occur several hours after exposure, during the evenings of the workdays; symptoms occur less frequently or not at all on days away from work and on vacations; symptoms occur more frequently on returning to work. Work–related changes in medication requirements may have similar patters, also suggesting an occupational etiology.

Many agents and processes have been associated with occupational asthma [Chan–Yeung 1990], and others continue to be recognized. Changes in nonspecific bronchial hyperactivity can be measured by serial inhalation challenge testing with methacholine challenge or histamine. Increased bronchial reactivity (manifested by reaction to lower concentrations of methacholine or histamine) following exposure and decreased bronchial reactivity after a period away from work are evidence of work–relatedness.

APPENDIX II

NIOSH SURVEILLANCE GUIDELINES:SILICOSIS [54]

Reporting Guidelines

State health departments and regulatory agencies should encourage physicians (including radiologists, pathologists, and other health care providers) to report all diagnosed or suspected cases of silicosis. These reports should include persons with

— a physician's provisional or working diagnosis of silicosis, **OR**

— a chest x–ray interpreted as consistent with silicosis, **OR**

— pathologic findings consistent with silicosis

To set priorities for workplace investigations, State health departments and regulatory agencies should collect appropriate clinical, epidemiologic, and workplace information about persons reported to have silicosis.

Surveillance Case Definition

A. 1. History of occupational exposure to airborne silica dust

AND

2. Chest x–ray or other imaging technique interpreted as consistent with silicosis

OR

B. Pathologic findings characteristic of silicosis