Silica
The Deadly Dust

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AOEC

NIOSH

Silica (Silicon Dioxide - SiO_2)

- Crystalline silica occurs naturally in the earth's crust
- Earth's most abundant mineral
- Three most common forms
  - Cristobalite
  - Tridymite
  - Quartz – most abundant component of soil & rock

Silica (Silicon Dioxide - SiO_2)

- Respirable particles of silica (<5 μ in diameter) produced when crystalline silica-containing rock and sand is used or processed
  - Mining, milling, and stone work
  - Quarrying and tunnel operations
  - Foundry and boiler work
  - Sandblasting and drilling
  - Pottery and glass making

Photo: NIOSH Safety and Health Topic: Silica

Silica (Silicon Dioxide - SiO_2)

- Occupational exposure associated with respiratory diseases
  - Silicosis (chronic, accelerated, acute)
  - Progressive pulmonary fibrosis
  - Chronic obstructive pulmonary diseases
  - Lung cancer
  - Increased risk for TB

Silica (Silicon Dioxide - SiO_2)

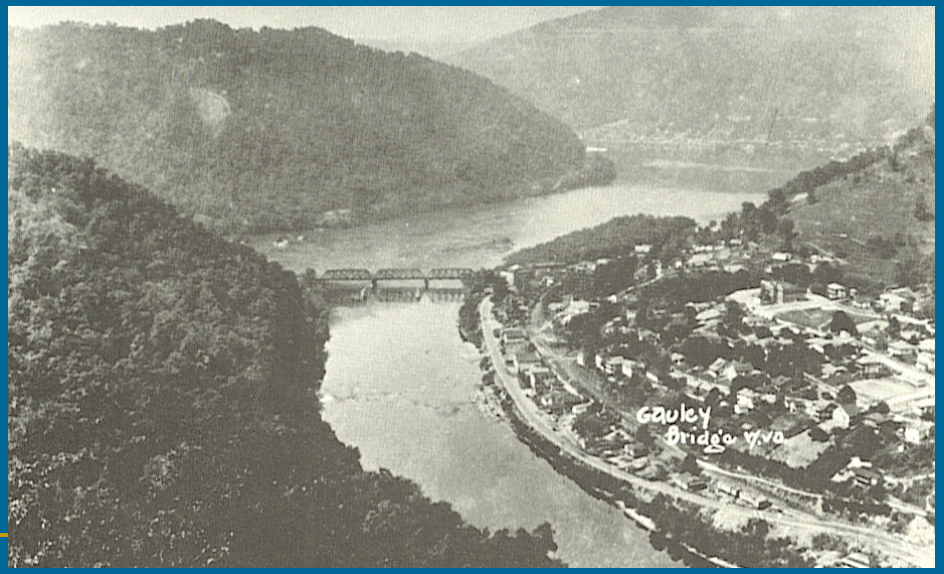
- Occupational exposure associated with other diseases
  - Systemic autoimmune diseases
    - Rheumatoid arthritis, SLE, scleroderma, small vessel vasculitides
  - Renal Disease
    - Glomerulonephritis, nephrotic syndrome, end-stage renal disease

Silica – Historical Overview

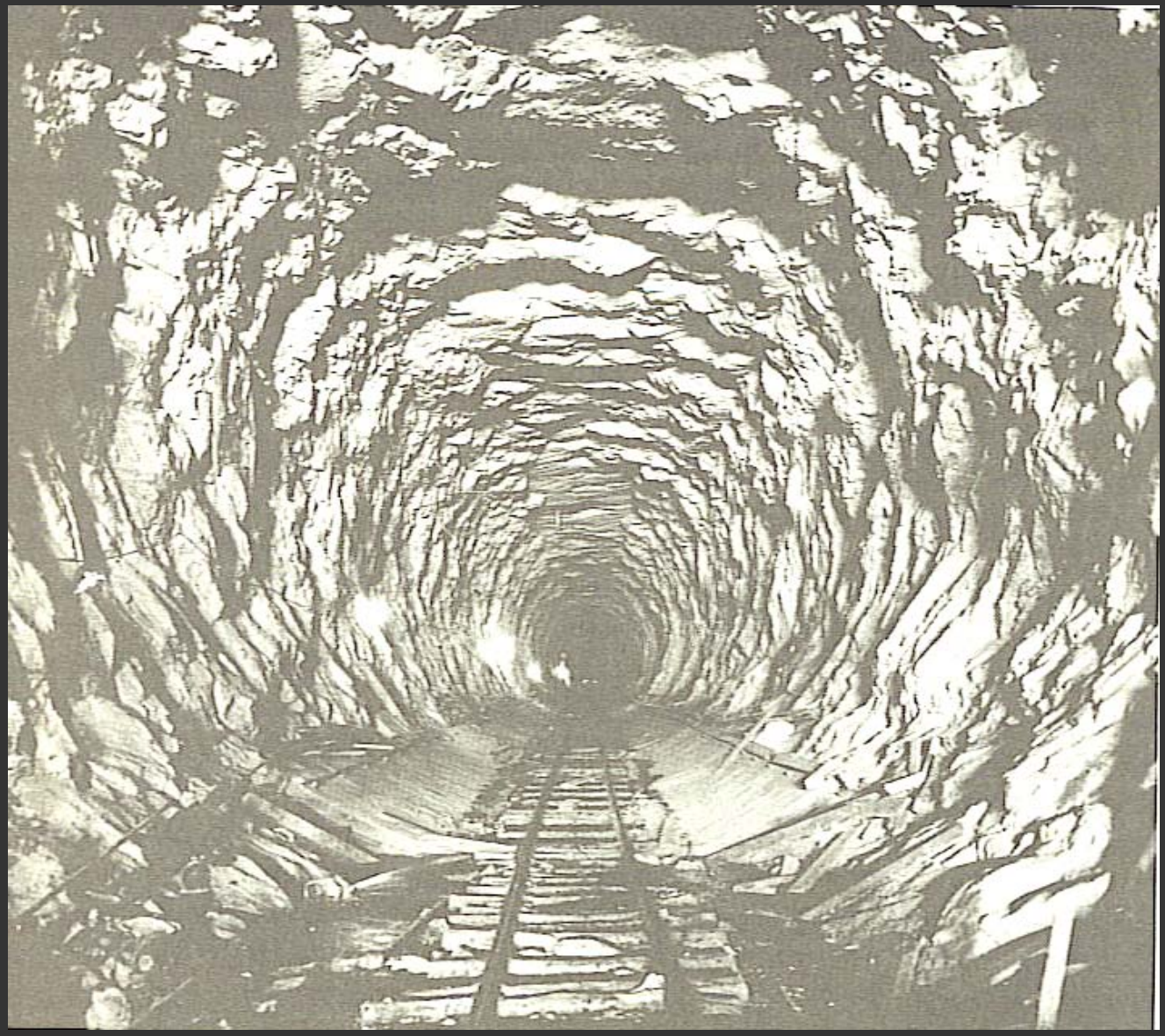
- Silicosis
  - First reported by ancient Greeks
- Prevalence
  - Peaked in the industrial countries in the last half of the 19th century
  - Disease still prevalent in the developing world and not eliminated from the developed world

Silica – Historical Overview

- Largest industrial disaster in US history
  - Gauley Bridge, WV – 1930-1932
- Tunnel construction
  - Silica content of the rock - >90%
- >475 workers died
- 1,500 were disabled from chronic silicosis

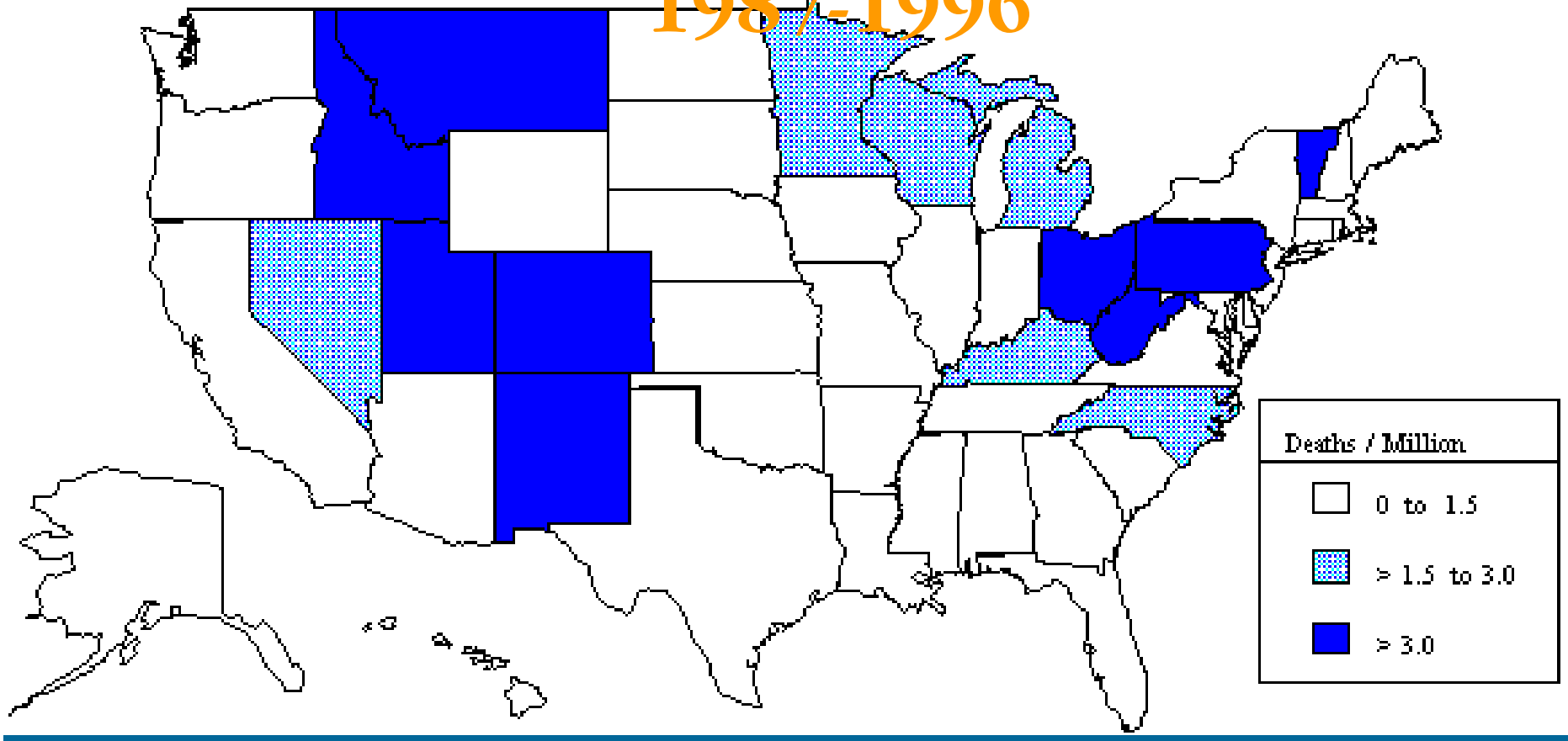


View of Gauley Bridge, WV circa 1930
Source: The Hawk's Nest Incident, Cherniak M, 1986



View of Hawk's Nest tunnel interior, March 13, 1932
Source: The Hawk's Nest Incident, Cherniak M, 1986

Silicosis: Crude mortality rates by state, U.S. residents age 15 and over, 1987-1996



NIOSH

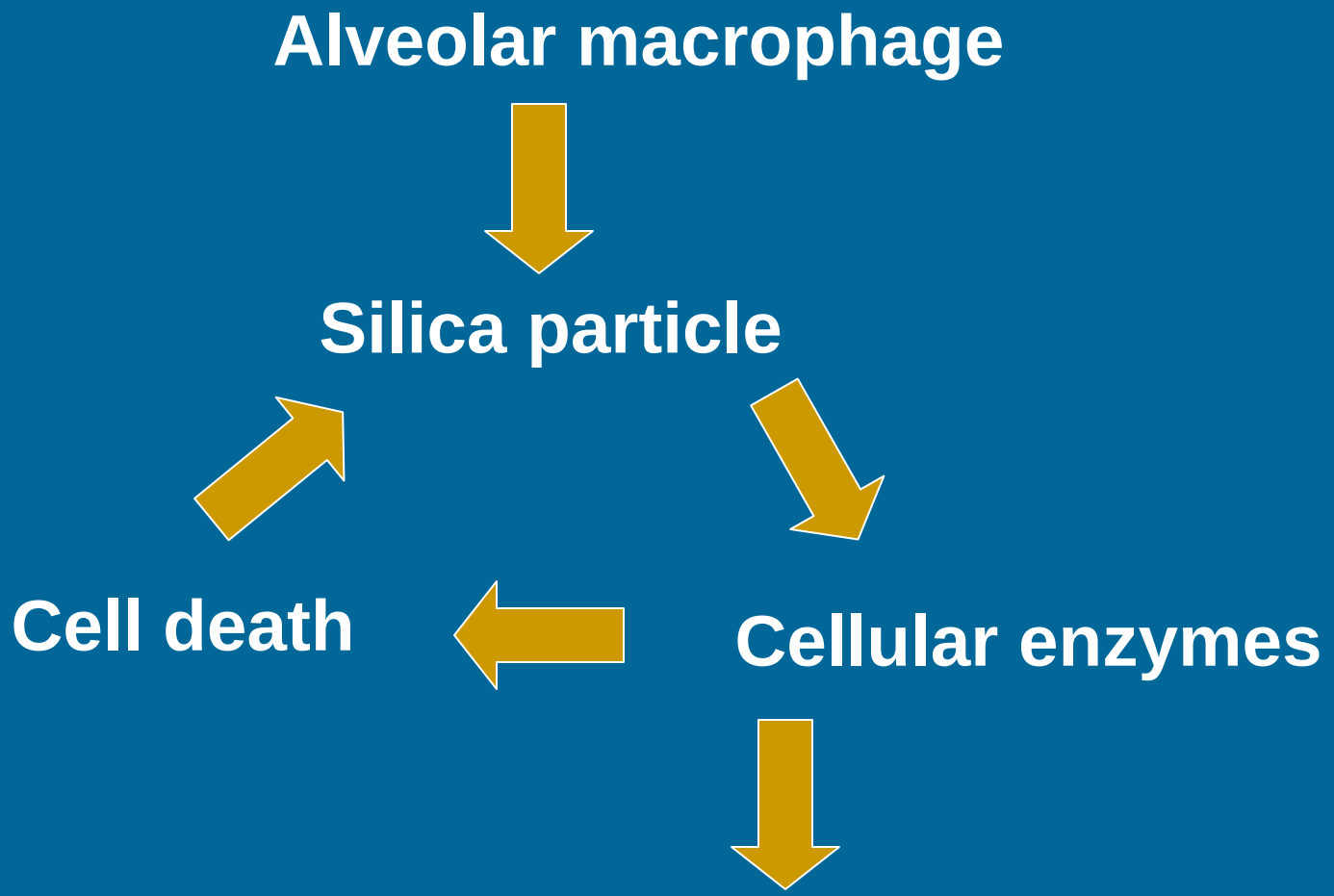
Silicosis

- A pulmonary disease caused by inhalation of dust particles of respirable size
- Three presentations and severity of the disease
  - Classic silicosis
  - Accelerated silicosis
  - Acute silicosis

Silicosis

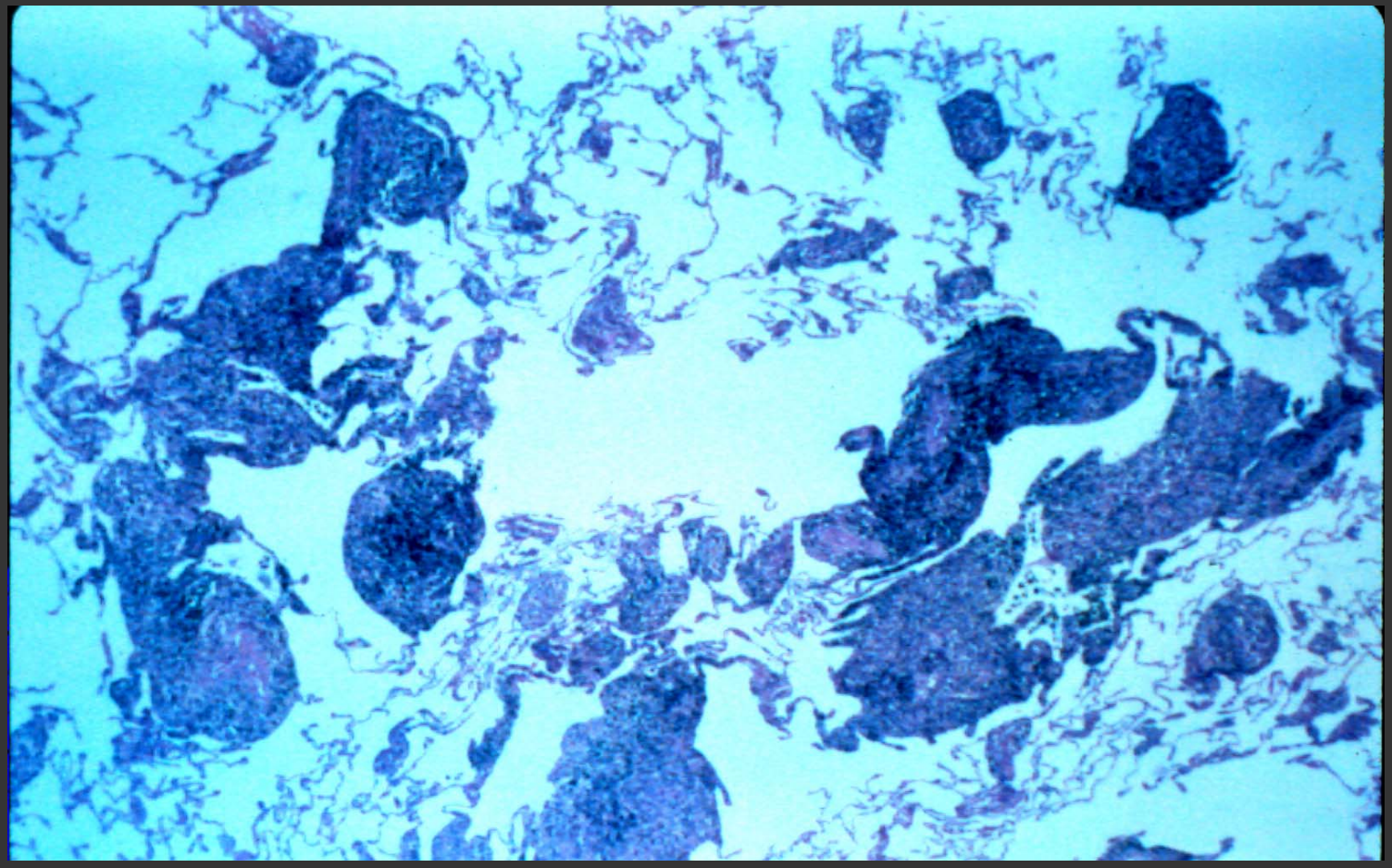
- Chronic (classic) silicosis
  - 20+ years of exposure to low-medium dust levels
- Accelerated silicosis
  - 5-10 years of higher dust exposure
- Acute silicosis
  - <1-3 years exposure to extremely high levels of free crystalline silica

Pathogenesis



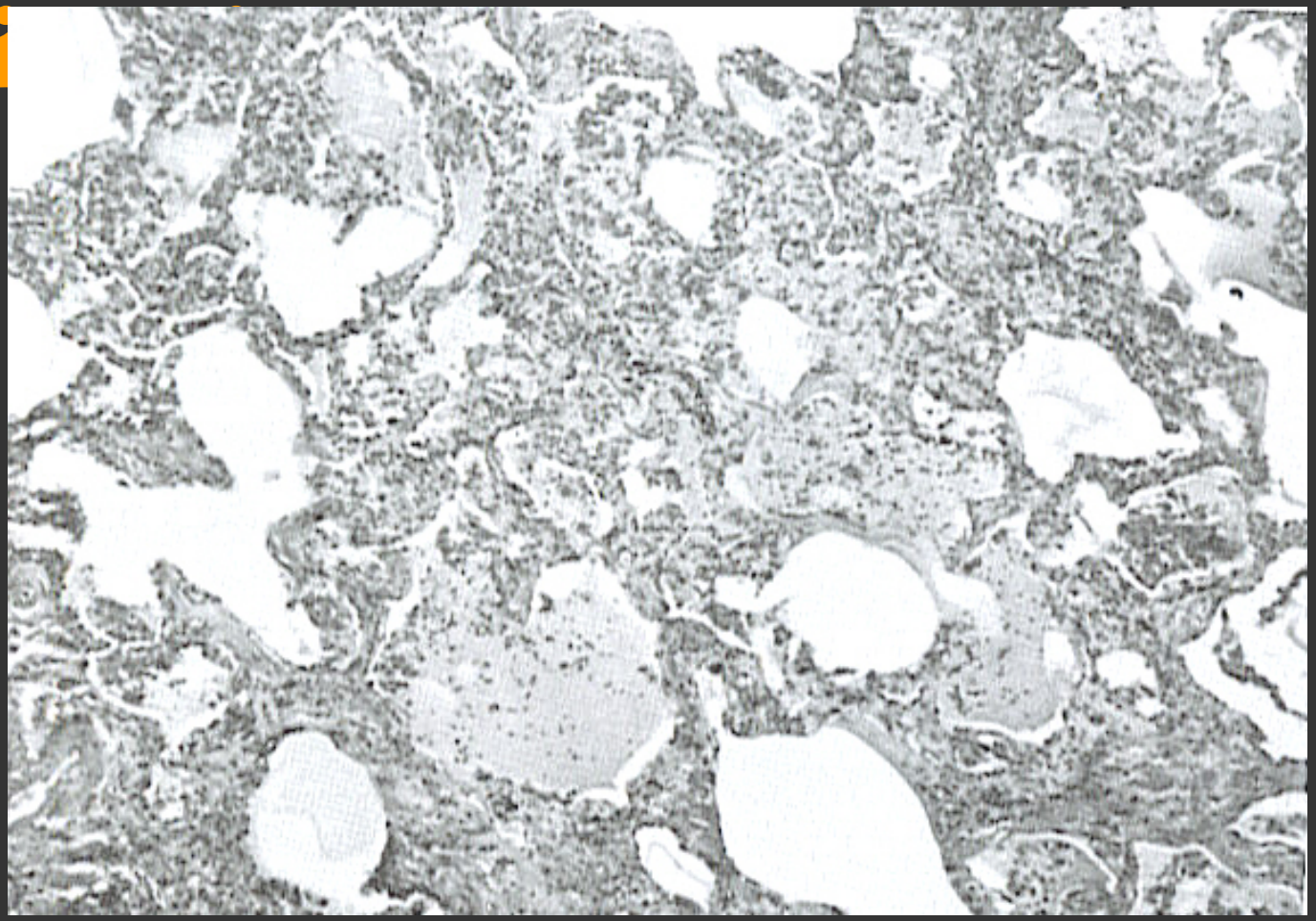
Pathogenesis- Chronic Silicosis

Classic and Accelerated



Source: ACCP Pathology Slide Set, No. 36-2

Pathogenesis- Acute Sili



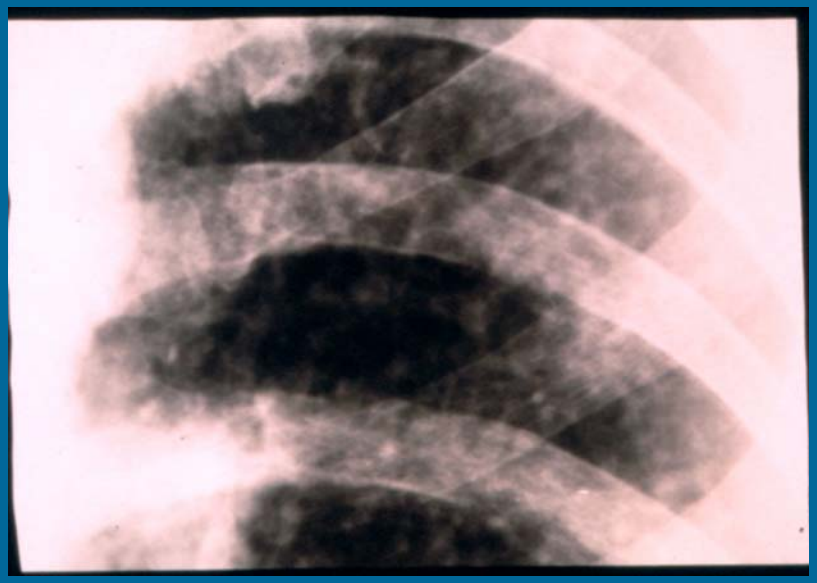
Source: Silicosis. Weber and Banks. In: Textbook of Clinical Occupational & Environmental Medicine. Rosenstock & Cullen, eds., 1994, page 270.

Chronic Classic silicosis

- Simple silicosis
  - Development predominantly in the upper lobes
  - Rounded opacities

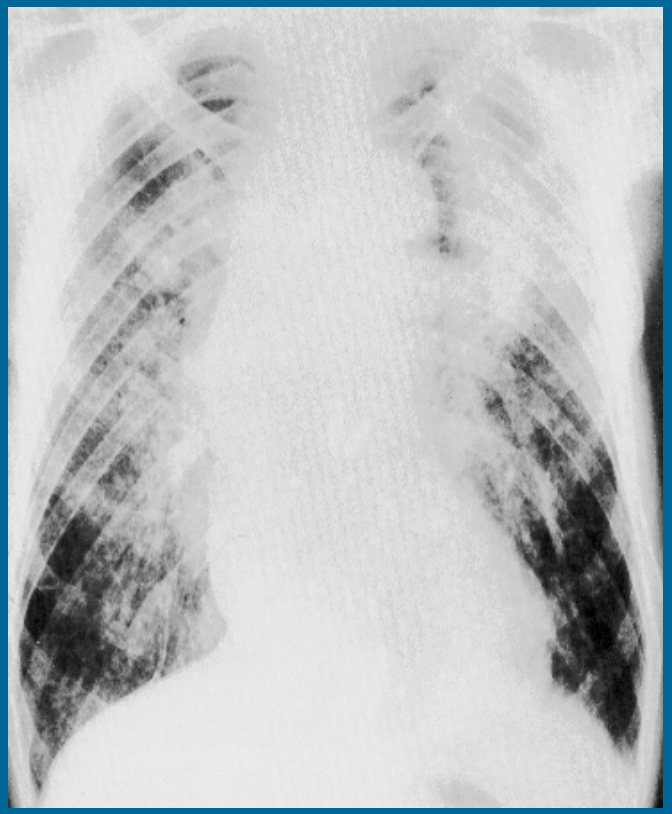
    < 1cm in diameter

    seen on chest x-ray
  - Enlarged hilar lymph nodes with peripheral calcification – eggshell calcification



Chronic Classic Silicosis

- Progressive Massive Fibrosis
  - A massing of small rounded opacities in upper lobes
  - Large opacities > 1 cm
  - Hila retract upward & lower zones become hyperinflated & appear emphysematous



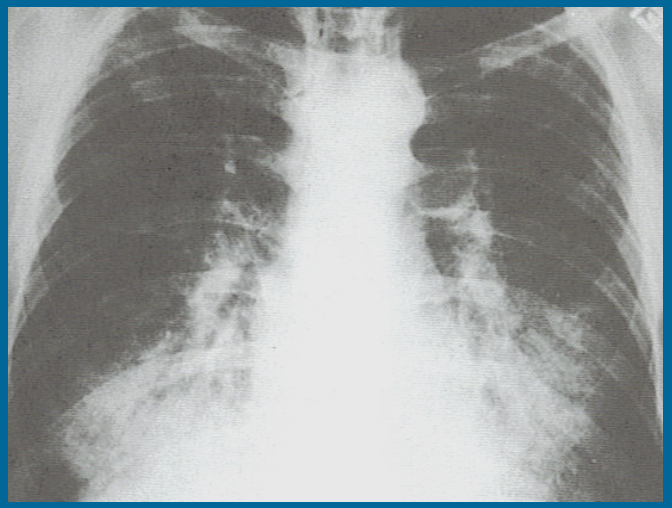
Source: Pathology of Occupational Lung Disease, 2nd ed. A Churg, FHY Green, 1998, pg. 163

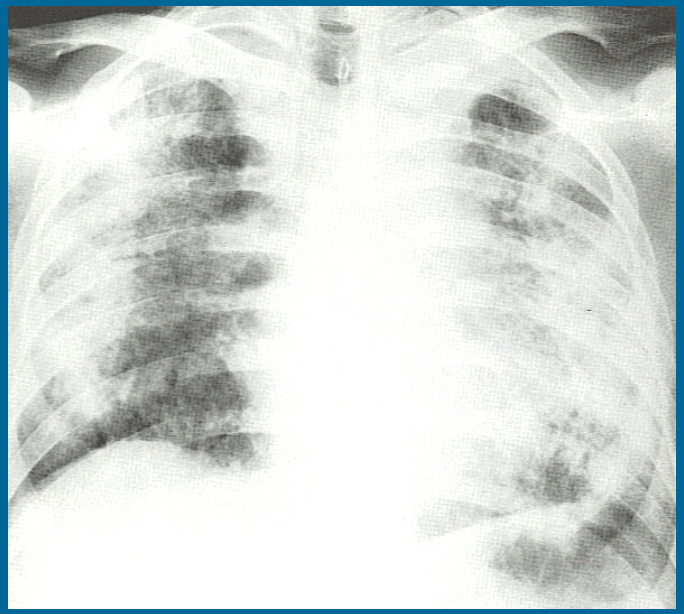
Accelerated Silicosis

- Characterized by same features as chronic classic silicosis
- Time from initial exposure and development of radiographic findings and symptoms and change in pulmonary function much shorter
- Rapid progression to PMF with severe respiratory impairment

Acute Silicosis

- **Radiographic Evidence**
  - Diffuse alveolar infiltrate
  - Air bronchograms
  - Ground glass appearance
  - Hilar & mediastinal lymphadenopathy
  - Bullae formation
  - Air trapping
  - Volume loss
  - Cavity formation





Source: Top: Environmental & Occupational Medicine, 2nd ed., Rom W, 1992, pg. 353.
Bottom: Diagnosis of Diseases of the Chest, Fraser & Pare, 1970, pg. 923.

Chronic silicosis

Simple silicosis

- Symptoms
  - Often no symptoms
  - Chronic productive cough may be due to industrial bronchitis from dust exposure
- Physical exam
  - Normal breath sounds
  - Course breath sounds with co-existing bronchitis

Chronic silicosis

Progressive Massive Fibrosis

- Symptoms
  - Range from chronic productive cough to exertional dyspnea
- Physical exam
  - Diminished breath sounds
  - Prolonged expiration
  - Clubbing rare

Acute silicosis

- Symptoms
  - Irritative cough – sometimes productive
  - Weight loss
  - Fatigue
  - Dyspnea
- Physical exam
  - Crackles heard on auscultation

Chronic silicosis

Pulmonary Function Testing

- **Simple silicosis**
  - **Normal lung function**
- **PMF**
  - **Severe restriction**
  - **Mixed obstructive/restrictive defect**
  - **Loss of pulmonary compliance**
  - **Hypoxemia**

Diagnosis

The Occupational History

- What kind of work do you do?
- Do you think your health problems are related to your work?
- Are your symptoms better or worse when you are at home or at work?
- Are you now or have you previously been exposed to dust, fumes, chemicals, radiation or loud noises?

LS Newman. N Engl J Med 1995; 333:1129

Diagnosis

- History of silica exposure
- Radiographic evidence consistent with silicosis
- Absence of other illnesses that mimic silicosis

Silicotuberculosis

- Prevalence
  - 5.3% in workers with x-ray evidence of silicosis
  - 25% in workers with acute or accelerated silicosis
  - As high as 75% among South African gold miners

Silicotuberculosis

- Diagnosis difficult
  - TB infection can be walled off in the lung by the silica induced fibrosis
  - False negative acid-fast-staining sputum smear may occur
- Radiographic changes seen with TB infections can mimic advanced cases of silicosis

Silicotuberculosis

- Diagnosis
  - Presence of chest x-ray changes of a worker with silicosis over a short period of time indicates superimposed TB infection until proven otherwise
  - Annual PPD
- If results become positive without clinical evidence of active TB, the patient should be treated with 1 year of INH

Silica Exposure & Cancer

- Crystalline silica deposited in lungs causes epithelial & macrophage injury and activation and persistent inflammation
- Human subjects exposed to dust containing crystalline silica showed an increase in the levels of sister chromatid exchange and chromosomal aberrations in peripheral blood lymphocytes
- Animal studies have shown gene mutations and tumor formation as a result of marked and persistent inflammation and epithelial proliferation

Silica Exposure & Cancer

- IARC
  - **Crystalline silica inhaled in the form of quartz or cristobalite from occupational sources *is***

carcinogenic to humans (Group 1)

Silica Exposure & Autoimmune Disease

- First described in 1953 by Caplan
  - Unusual radiologic changes in the lungs of Welsh coal miners who had pneumoconiosis
- Since then the autoimmune disease linked with crystalline silica exposure
  - Rheumatoid arthritis, scleroderma, SLE, some small vessel vasculitides

Silica Exposure & Autoimmune Disease

- Inhalation of crystalline silica particles leads to chronic immune activity and fibrosis
- Studies have shown that crystalline silica can be mobilized from lungs to other organs - lymph nodes, spleen, and kidney
- Silicosis has been linked to an increase in autoantibodies, immune complexes, and excess production of immunoglobulins, even in the absence of a specific autoimmune disease

Silica Exposure & Autoimmune Disease

- **Possible Mechanisms**
  - **May be result of adjuvant (a substance that enhances an immune response to an antigen) effect on antibody production**
  - **Cell death by necrosis and apotosis (an active process involved in gene regulation)**
  - **Host susceptibility and genetic differences may explain why all workers exposed to silica do not develop immune disorders may**

Silica Exposure & Renal Disease

- Epi studies
  - Statistical significance between silica exposure and several renal diseases
  - An increasing standardized rate ratio for acute and chronic renal disease with increasing cumulative crystalline silica exposure and an excess of end-stage renal disease incidence (highest for glomerulonephritis)

Silica Exposure & Renal Disease

- Intensity of exposure to silica dust may be more important than cumulative exposure or duration in the development of autoimmune diseases
- Study crystalline silica exposure most strongly associated with ESRD and median exposure was below the OSHA permissible exposures levels

Silica Exposure & Renal Disease

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Treatment

- **Prevention/ Prevention/Prevention**
- **Workers at risk for progression of disease and TB infection**
- **Yearly chest x-ray and PPD**
- **Flu and pneumococcal vaccine**
- **Aggressive treatment of TB infections**
- **Dyspnea treated with inhaled bronchodilators**
- **O_2 for cor pulmonale, hypoxemia, pulmonary hypertension**

Prevention

- **Occupational Health Surveillance**
  - **Gather information on cases of occupational illness and injury and workplace exposures**
  - **Condense, refine, and analyze the data**
  - **Disseminate analyzed data to workers, unions, employers, governmental agencies, public**
  - **Plan and execute interventions – primary prevention – based on the analyzed data**

Prevention

- **Occupational Sentinel Health Event**
  - **“A disease, disability, or untimely death which is occupationally related and whose occurrence may: 1) provide the impetus for epidemiologic or industrial hygiene studies; or 2) serve as a warning signal that materials substitution, engineering control, personal protection, or medical care may be required”**

Prevention

- State-based Surveillance
  - Sentinel Event Notification Systems for Occupational Risk (SENSOR)
  - Many state based silicosis surveillance projects
  - May help in case investigations
  - Many states have a legal requirement to report a case of silicosis to the appropriate state agency

Prevention

- **Hierarchy of Controls**
- **Engineering**
  - **Substitution, control hazard at source (wet process), improved ventilation**
- **Administrative**
  - **Rotating workers**
- **Personal Protective Equipment**
  - **Respirators**

Prevention - Regulation

- Federal Coal Mine Health and Safety Act of 1969 (Coal Act)
- Federal Mine Safety and Health Act of 1977 (Mine Act)
  - MSHA
- Occupational Safety and Health Act of 1970
  - NIOSH
  - OSHA

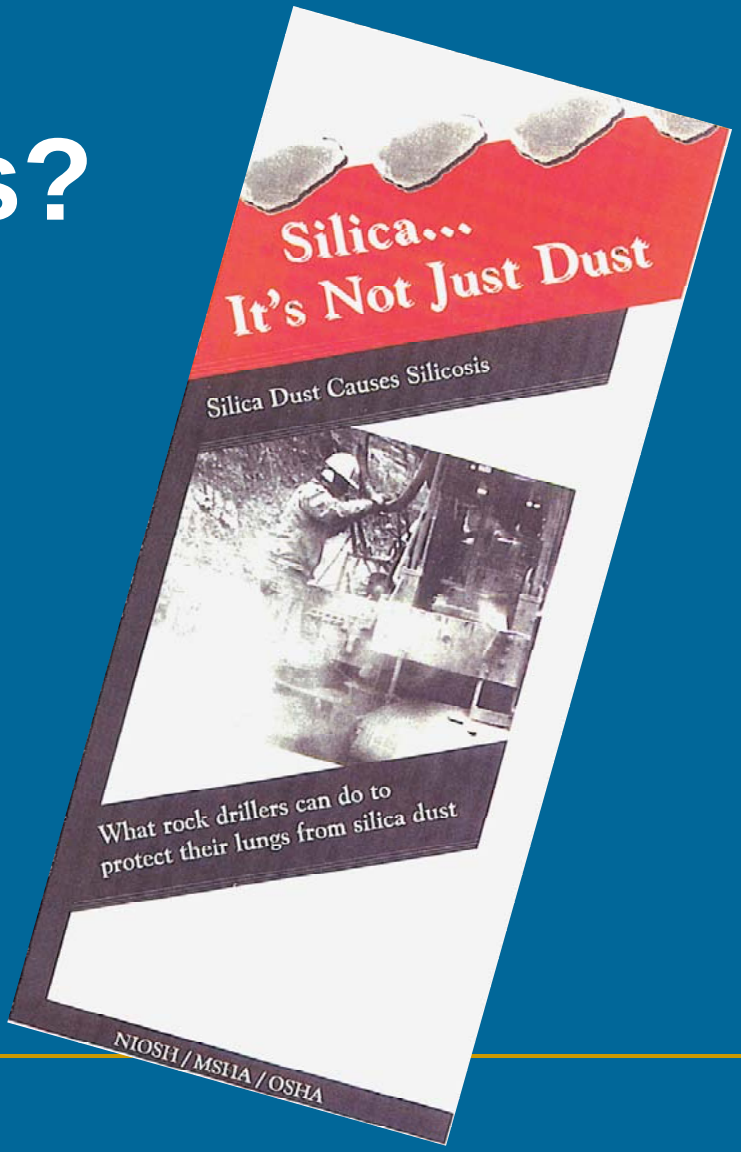
Prevention - Regulation

- **Current OSHA PEL for respirable silica**
  - **10 mg/m^3 / %SiO_2 + 2 for 8-hour TWA**

- **Current NIOSH REL for respirable silica**
  - **50 *u*g/m^3 TWA for up to 10 hours/day during a 40 hour workweek**

SILICA: The Deadly Dust

- Any Questions?



“The way to dusty death. Out, out, brief candle!
Life’s but a walking shadow, a poor player
That struts and frets his hour upon the stage And then is heard no more.”

Shakespeare

Macbeth

Act V, Scene V

SILICA: THE DEALY DUST

Pre and Post Test Questions

1. Silica (silicon dioxide- SiO_2) is formed from the two most abundant elements in the earth's crust, oxygen and silicon and is the earth's most abundant mineral and workers exposed to airborne dust levels of respirable crystalline silica (0.5 – 5.0 micrometers in diameter) are at risk for respiratory and other diseases.

    a. True
    b. False

2. Occupational exposure to respirable crystalline silica is associated with a number of respiratory diseases, including:
   (Circle all correct answers)

    a) silicosis (acute, accelerated, and chronic)
    b) progressive pulmonary fibrosis
    c) chronic obstructive pulmonary disease (e.g., chronic bronchitis, emphysema)
    d) lung cancer
    e) asthma
    f) tuberculosis

3. Occupational exposure to respirable crystalline silica is also associated with a number of other diseases, including:
   (Circle all correct answers)
    a) rheumatoid arthritis,
    b) scleroderma,
    c) systemic lupus erythematosus (SLE)
    d) small vessel vasculitides
    e) rapid progressive glomerulonephritis
    f) nephrotic syndrome
    g) end-stage renal disease.

4. In the United States, it is estimated that 200,000 miners and 1.7 million workers outside the mining industry have potential exposure to silica dust.

    a. True
    b. False

5. The reasons for taking an occupational & environmental health history are: (Circle all correct answers)

    a) To make an accurate medical diagnosis, determine appropriate treatment and prognosis
    b) To determine causality and establish the basis for compensation
    c) To institute primary preventive measures to protect other workers and other individuals in the community
    d) To prevent exacerbation of underlying preexisting lung disease by occupational and environmental agents.

6. The clinical diagnosis of silicosis is dependent on the recognition that silica exposure has been adequate to cause the disease. The occupational history is central to determining the type and extent of exposures that a worker has experienced. What questions would you ask?
(Circle all correct answers)

    a) What kind of work do you do?
    b) Do you think your health problems are related to your work?
    c) Are your symptoms better or worse when you are at home or at work?
    d) Are you now or have you previously been exposed to dust, fumes, chemicals, radiation or loud noises?

7. Some of the other pulmonary diseases that mimic silicosis on chest x-ray include: (Circle all correct answers)

    a) miliary TB
    b) COPD
    c) Histoplasmosis
    d) Sarcoidosis

8. International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) has concluded that crystalline silica inhaled in the form of quartz or cristobalite from occupational sources is carcinogenic to humans (Group1).

    a. True
    b. False

9. The main goals of occupational health surveillance are:
(Circle all correct answers)

a) To identify the incidence and prevalence of occupational illness and injury for determining control and research priorities and strategies and to evaluate the effectiveness of interventions;
b) To identify individual cases of work-elated disease or injury in order to identify other individuals from the same or similar workplace or individuals with the same or similar exposures who may be at risk for illness or injury;
c) To identify new associations between occupational hazards and the resultant injury or illness.

10. Occupational diseases such as silicosis and injuries are preventable. Hierarchy of controls refers to the concept of fixing the workplace to prevent exposures to hazards. Engineering controls are always the first and preferred method of hazard control.

a. True
b. False

.

SILICA: THE DEALY DUST

Pre and Post Test Questions *(Answers underlined)*

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EVALUATION

Silica: The Deadly Dust

PROGRAM OBJECTIVES: At the end of the session, participants will be able to:

1. Use occupational/environmental exposure history taking techniques in their daily medical practice.
2. Evaluate the risks of silica exposure among workers.
3. Describe health screening and primary prevention practices for silica exposed workers.

PLEASE CIRCLE YOUR RESPONSE

TITLE: PHYSICIAN PA NP RN

OTHER_____

This program met the stated objectives:	STRONGLY DISAGREE				STRONGLY AGREE	
COMMENTS:	1	2	3	4	5	
Information from this activity will be incorporated into my medical practice:	STRONGLY DISAGREE				STRONGLY AGREE	
COMMENTS:	1	2	3	4	5	
Please rate the effectiveness of the presenter:	NOT EFFECTIVE				VERY EFFECTIVE	
COMMENTS:	1	2	3	4	5	

Table 1.--NIOSH-recommended respiratory protection for workers exposed to respirable crystalline silica

Condition	Minimum respiratory protection* required to meet the Condition NIOSH REL for crystalline silica (50 µg/m³)**
Less than or equal to 500 $\mu g/m^3$ (10 x REL)***	Any air-purifying respirator with a high-efficiency particulate filter
Less than or equal to 1,250 $\mu g/m^3$ (25 x REL)	Any powered, air-purifying respirator with a high-efficiency particulate filter, or Any supplied-air respirator equipped with a hood or helmet and operated in a continuous-flow mode (for example, type CE abrasive blasting respirators operated in the continuous-flow mode)
Less than or equal to 2,500 $\mu g/m^3$ (50 x REL)	Any air-purifying, full-facepiece respirator with a high- efficiency particulate filter, or Any powered, air-purifying respirator with a tight-fitting facepiece and a high-efficiency particulate filter
Less than or equal to 50,000 $\mu g/m^3$ (1,000 x REL)	Any supplied-air respirator equipped with a half-mask and operated in a pressure-demand or other positive-pressure mode
Less than or equal to 100,000 $\mu g/m^3$ (2,000 x REL)	Any supplied-air respirator equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode (for example, a type CE abrasive blasting respirator operated in a positive-pressure mode)
Planned or emergency entry into environments containing unknown concentrations or concentrations less than or equal to 500,000 $\mu g/m^3$ (10,000 x REL)	Any self-contained breathing apparatus equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode,**** or Any supplied-air respirator equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode in combination with an auxiliary self-contained breathing apparatus operated in a pressure-demand or other positive-pressure mode****
Firefighting	Any self-contained breathing apparatus equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode****
Escape only	Any air-purifying, full-facepiece respirator with a high- efficiency particulate filter, or Any appropriate escape-type, self-contained breathing apparatus

* Only NIOSH/MSHA-approved equipment should be used.
** These recommendations are intended to protect workers from silicosis; only the most protective respirators are recommended for used with carcinogens.
*** Assigned protection factor (APF) times the NIOSH REL. The APF is the minimum anticipated level of protection provided by each type of respirator.
**** Most protective respirators.

Lecture Notes – Silica- The Deadly Dust

SLIDE 1

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University of New Mexico Health Sciences Center
Ken Silver, PhD
Department of Environmental Health
East Tennessee State University

SLIDE 2

This presentation is made possible by a grant from the Association of Occupational and Environmental Clinics and the National Institute for Occupational Safety & Health.

SLIDE 3

Silica
Silica (silicon dioxide- SiO_2) is formed from the two most abundant elements in the earth's crust, oxygen and silicon and is the earth's most abundant mineral. SiO_2 occurs in a non-crystalline (amorphous) or a crystalline form. Crystalline silica is found in seven forms (polymorphisms), of which quartz, cristobalite, and tridymite are the most common. The quartz form is an abundant component of soil and rock; the term is often used to refer to crystalline silica. Silica is either free (unbound to other minerals) (quartz has a high amount of free silica) or combined with other minerals and are called silicates (i.e. asbestos, talc).

SLIDE 4

Silica
It is when the crystalline silica-containing rock and sand is used or processed (i.e., mining, milling, quarrying, drilling, sand blasting, tunneling operations, foundry and boiler work, pottery and glass making operations) and work with concrete, brick, block, mortar, tile grout, and numerous materials made with silica and then disturbed such as sawing, hammering, crushing and grinding that workers can be exposed to airborne dust levels of respirable crystalline silica (0.5 – 5.0 micrometers in diameter) that can cause respiratory and other diseases.

SLIDE 5

Silica
Occupational exposure to respirable crystalline silica is associated with a number of respiratory diseases, including silicosis (acute, accelerated, and chronic), progressive pulmonary fibrosis, chronic obstructive pulmonary disease (e.g., chronic bronchitis, emphysema), and lung cancer, and places workers with silicosis at higher risk for tuberculosis and atypical (non-tuberculosis) mycobacterial disease.

SLIDE 6

Silica
Although the most common health effect of silica exposure are the lung diseases occupational exposure to respirable crystalline silica is also associated with a number of other diseases, including systemic autoimmune diseases, such as rheumatoid arthritis, scleroderma, systemic lupus erythematosus (SLE), and some of the small vessel vasculitides and with renal diseases i.e. rapid progressive glomerulonephritis, nephrotic syndrome, and end stage renal disease.

SLIDE 7

Silica- Historical Overview
Silicosis – a spectrum of pulmonary diseases was first reported by ancient Greeks. Hippocrates reported that miners developed dyspnea with exertion. Bernardo Ramazzini, the father of occupational medicine, recognized the relationship between dust exposure in the mining trades and the development of dyspnea among miners. Although the prevalence peaked during the late 19th century in the industrial countries of the world, developing countries still have outbreaks of the disease among exposed workers and the developed countries have not totally eliminated this preventable disease.

SLIDE 8

Silica- Historical Overview
Silica exposure and its health effects were responsible for the largest industrial disaster in American history. The events took place in Gauley Bridge, West Virginia during the Great Depression from 1930 - 1932. A power company was building a hydroelectric project along the New River and the Kanawha River in southern West Virginia and was building a tunnel to divert water near the town of Gauley Bridge, WV.

The subcontractor for the tunnel construction project recruited workers from the rural hill country of WV and from out of state. Many workers out of work and suffering during the Great Depression came to work at the construction site. Black workers from the South were brought in and paid as little as 30 cents an hour.

Working conditions in the tunnel were very bad. Drilling operations with no suppression of the dust and with no ventilation exposed hundreds of workers to high concentrations of dust. The crystalline silica (quartz) content of the rock was greater than 90%. As the project continued increasing numbers of workers became short of breath and died within only a few months of exposure. It was estimated that 169 black workers who died in the tunnel were buried in a mass grave in nearby fields. It is estimated that more than 475 workers – both black and white- died during the project and that 1,500 workers contracted silicosis and were disabled from the disease.

SLIDE 9

Silica- Historical Overview

In 1936 New York Congressman Vito Marcantino held hearings on the disaster. The public outcry led to increasing attention on the health hazards of silica exposure.

Years later historians discovered the blueprints for the tunnel project. The original plans called for a 28 foot diameter tunnel. However, when the companies involved in the project found that the rock was almost pure silica, they decided to build a wider tunnel to mine the silica and sell it to the local glass making industry. However, workers were not warned of the health risks and were provided no protection from the dust exposure.

In New England workers involved in the granite stone-cutting in the tombstone industry were another group of workers who contracted silicosis at a high rate. The expression “carving your own tombstone” came from this group of workers. Public concern about silica exposure and the health risks to workers led to the creation of some of the first state based occupational health and safety agencies in Massachusetts and Vermont in the 1940’s.

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Silicosis: Crude mortality rates by states

In the United States, it is estimated that 200,000 miners and 1.7 million workers outside the mining industry have potential exposure to silica dust. Better dust suppression and ventilation systems have decreased the number of cases in the United States. However, new cases are still reported both in the developed

countries as well as the developing countries and silicosis remains a disease among many groups of workers. Rosenman et al. reported in 1997 of 577 US workers who were identified with silicosis between 1987 and 1995 through a state surveillance system.

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Silicosis
Silicosis is a pulmonary disease caused by the inhalation of silica particles of respirable size (0.5 – 5.0 micrometers in diameter). The presentation and severity of the disease depends on multiple factors – concentration of free silica dust exposure, physical characteristics and innate fibrogenic properties of the dust (fraction of crystalline silica), and the duration of dust exposure. Host factors such as cigarette smoking, underlying disease and genetic characteristics of the worker may also play a factor in the disease presentation.

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Silicosis
Silicosis- 3 clinicopathologic types

Chronic (classic) silicosis is the most common presentation of silicosis. The disease results from low to moderate exposure to dust containing respirable crystalline silica (typically <30% crystalline silica) for 15-20 years or more.

Accelerated silicosis, a form of classic silicosis, results from higher levels of crystalline silica dust exposure over a period of five to ten years.

Acute silicosis is the least frequent but the most devastating form of the disease. The disease results from overwhelming excessive concentrations of free crystalline silica dust exposure for as little as a few months to a few years.

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Pathogenesis
Pathogenesis of silicosis begins with inhalation of crystalline silica particles. Particles less than 3 micrometers and greater than 0.5 micrometers have the best chance of entering and being retained in the pulmonary acini. The interaction between the silica particle and the alveolar macrophage (the main phagocytic cell in the alveolar space) starts the process of silicosis. Inhaled silica particles are phagocitized by macrophages – the alveolar macrophages become activated and an intense inflammatory response ensues – the macrophages secrete mediators (interleukin-1, macrophage-derived growth factor, fibronectin,

tumor necrosis factor) that perpetuate the inflammatory response and initiate the process of fibrosis. The macrophage ultimately ruptures and dies releasing the unaltered silica particle into the pulmonary interstitium to be taken up by another macrophage. The recurrent cycle of macrophage phagocytosis, cell death, release of cellular enzymes, and the reuptake of silica perpetuates the inflammatory and fibrotic process. Recent studies suggest that cell injury may be a more crucial factor than cell death in the pathogenesis of silicosis.

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Pathogenesis- Chronic Silicosis – Classic and Accelerated
The ongoing inflammatory process produces the silicotic nodule, the histologic hallmark of silicosis. Silicotic nodules usually form near the small bronchioles. Nodules begin to form by an arrangement of dust laden macrophages surrounded by a reticulum of fibrous tissue. The central zone of the silicotic nodule is a mixture of hyalinized connective tissue and silica dust. It is surrounded concentrically by fibrous tissue in an onionskin like pattern. Active inflammation and fibrosis occurs at the periphery of the nodule. As the periphery expands the central region enlarges involving and destroying small airways, pleura, and blood and lymph vessels.

Depending on the dust burden and the rate of development of the disease, nodules may continue to develop after exposure ceases.

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Pathogenesis- Acute Silicosis
The histological pattern of acute silicosis differs from chronic silicosis – classic and accelerated. Silicosis nodules are rarely seen or if seen poorly developed. The interstitium is thickened with inflammatory cells. There is alveolar filling with proteinaceous material consisting of phospholipids or surfactant. The histiologic appearance resembles idiopathic alveolar proteinosis. The process occurring in a background of overwhelming crystalline silica dust exposure has also been called silicoproteinosis.

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Chronic Classic Silicosis
In chronic silicosis the nodules tend to develop more predominantly in the upper lobes. In some workers low level silica exposure may be cleared and deposited in the lymph nodes. On chest radiograph calcified regional hilar lymph nodes may the only abnormality noted. However, workers with significant silica dust exposure will have rounded opacities on chest x-ray. The rounded opacities

distributed in the upper lung zones are less than 1 cm in diameter. The hilar lymph nodes are often enlarged and may have peripheral calcification described as eggshell calcification.

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Chronic Classic Silicosis
Progressive massive fibrosis results from the conglomeration of small rounded opacities. The chest roentgenograph reveals large opacities (> 1 cm in diameter) most predominantly in the upper lung zones and on a background of extensive small rounded opacities. The confluence of the nodules usually begins peripherally and migrates centrally. As the fibrous masses enlarge the hila retract upward and the lower lung zones become hyperinflated and appear emphysematous.

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Accelerated Silicosis
Accelerated silicosis is characterized by the same features as chronic classic silicosis except that the time from initial exposure and development of radiographic findings and symptoms and change in pulmonary function are much shorter. The chest x-ray may show radiographic evidence in as little as four years after initial exposure. There is also a rapid progression to PMF with severe respiratory impairment.

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Acute Silicosis
Acute silicosis, the most aggressive form of silicosis, has chest radiograph findings that typically reveal diffuse alveolar infiltration and obliteration usually accompanied by air bronchograms. There is a ground glass appearance and the typical small rounded opacities of chronic (classic) silicosis are usually not seen. The progression seen on the chest x-ray is usually rapid. The areas of alveolar filling will progress to large masses. There may also be enlargement of hilar and mediastinal lymph nodes, bullae formation, air trapping and volume loss. There may also be cavity formation with marked pleural thickening.

The top chest x-ray shows acute silicosis in the early stages. Air space densities with bronchograms are seen in both lower lung zones.

The bottom x-ray shows acute silicosis in a late phase with marked loss of lung volume with much of the lung parenchyma replaced by a course reticular pattern with confluence and conglomeration.

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Chronic Silicosis
Patients with simple silicosis may not have any symptoms. Some workers will complain of a chronic productive cough – these symptoms may be due to industrial bronchitis from dust exposure. The physical exam often reveals normal breath sounds. There may be course breath sounds in those patients with co-existing bronchitis. Occasionally there may be crackles (rales) and scattered wheezes.

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Chronic Silicosis
Patients with progressive massive fibrosis (PMF) will have symptoms that range from chronic productive cough to exertional dyspnea and may progress to respiratory failure. As the disease advances and there are more emphysematous areas breath sounds decrease. Narrowing within or near airway walls caused by silicotic nodules may produce prolonged expiration on auscultation. Patients may develop cor pulmonale, hypoxemia and pulmonary hypertension.

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Acute Silicosis
Patients with acute silicosis will have a rapid onset of chest symptoms and progressive respiratory impairment that will often lead to death due to respiratory failure. Patients present with irritative (occasionally productive) cough, weight loss, fatigue, dyspnea and occasionally pleuritic pain. On physical exam crackles are usually present due to alveolar and airway fluid. Patients will develop rapidly cyanosis, symptoms of cor pulmonale, and respiratory failure. Survival after the onset of symptoms is typically less than 2 years.

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Chronic Silicosis – Pulmonary Function Testing
Lung function in classic, simple silicosis is often normal. Multi-factorial effects of cigarette smoking, the type of dust involved (such as a mixture of dusts), the dose of the dust and the duration of exposure, and the presence of other pulmonary diseases such as tuberculosis may contribute in the alteration of an individual patient's pulmonary function. Pulmonary function testing in workers with advanced (PMF) disease will show severe restriction or mixed pattern of

obstructive/restrictive defect, loss of pulmonary compliance, hypoxemia and a reduction in diffusion capacity as a later finding in PMF**.**

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Diagnosis – The Occupational History

The clinical diagnosis of silicosis is dependent on the recognition that silica exposure has been adequate to cause the disease. The occupational history is central to determining the type and extent of exposures that a worker has experienced. Initial estimates of the worker's exposures are often based on qualitative information supplied by the worker in descriptions of workplace conditions and work practices.
A quick survey of work history, possible exposures, and symptoms related to work is helpful in determining if a more extensive occupational and environmental history should be taken. The important questions to start with are:

- What kind of work do you do?
- Do you think your health problems are related to your work?
- Are your symptoms better or worse when you are at home or at work?
- Are you now or have you previously been exposed to dust, fumes, chemicals, radiation or loud noises?

If there are any positive responses to these questions then a more extensive occupational and environmental health history should be conducted. A more extensive history should include a list of all jobs (including summer, part-time, and military jobs) possible or known exposures, length of time of exposures, work in industries such as mining, milling, quarrying, drilling, sand blasting, tunneling operations, foundry and boiler work, pottery and glass making operations, types of work procedures such as grinding, sawing, drilling, crushing materials made with silica, any use of protective equipment, and knowledge of any other fellow workers with similar illnesses.

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Diagnosis
The diagnosis of silicosis rely on three requirements: 1) a history of silica exposure; 2) radiographic evidence that is consistent with silicosis; 3) the absence of other illnesses that mimic silicosis. Some of the other pulmonary diseases that mimic silicosis include miliary TB, fungal infection (i.e. histoplasmosis), or sarcoidosis. A workplace history to help determine the length of employment (duration of exposure), exposure measurements if available, workplace control measures such as wetting down the dust or exhaust ventilation, and whether the worker wore respiratory protection or not is important.

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Silicotuberculosis
The association between silicosis and tuberculosis has been recognized since the 16th Century. In the US the prevalence of TB infection in a 1960 review of patients with radiographic evidence of silicosis was 5.3%. The overall rate has declined in the last number of years but still remains as high as 25% in workers with acute or accelerated silicosis. In countries with endemic rates of TB, such as South Africa, the prevalence has been reported as high as 75% among gold miners.

The risk of infection with the Mycobacterium tubercule increases as the radiographic changes seen with silicosis advance. Tuberculosis appears to be more prevalent among workers whose silicosis is attributed to pure silica rather than a mixed-dust exposure.

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Silicotuberculosis
Diagnosis can be difficult. The TB infection can be walled off in the lung by the silica induced fibrosis. A false negative acid-fast-staining sputum smear may occur. The constitutional symptoms seen with TB; fever, fatigue, dyspnea, and weight loss, can be seen in workers with worsening silicosis even without a TB infection. In addition, radiographic changes seen with TB infections can mimic advanced cases of silicosis.

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Silicotuberculosis
The presence of chest x-ray changes of a worker with silicosis over a short period of time indicates superimposed TB infection until proven otherwise.

Patients with silicosis should have an annual PPD skin test. If results become positive without clinical evidence of active TB, the patient should be treated with 1 year of INH. Some experts advocate longer treatment, even lifelong, with anti-tuberculosis prophylactic therapy due to the extensive, irreversible damage to the silicotic nodule by the TB organism.

Patients with silicosis are also at risk for atypical (non-tuberculosis) mycobacterial diseases and are not as easily treated as typical M. Tb. Examples of atypical (non-tuberculosis) mycobacterial infection in silicosis: M. kansasii, M. intracellulare.

SLIDE 29

Silica exposure and cancer

Crystalline silica deposited in the lungs causes epithelial and macrophage injury and activation. Crystalline silica enters the interstitium and the regional lymph nodes causing persistent inflammation. In-vitro studies have shown that crystalline silica can stimulate release of cytokines and growth factors from macrophages and epithelial cells and that these factors contribute to disease. Crystalline silica stimulates release of reactive oxygen and nitrogen intermediates from a variety of cell types

A human study on subjects exposed to dust containing crystalline silica showed an increase in the levels of sister chromatid exchange and chromosomal aberrations in peripheral blood lymphocytes.

Some animal studies have shown gene mutations in rat lungs following quartz exposure. Tridymite was tested in only one study, where it induced sister chromatid exchange in co-cultures of human lymphocytes and monocytes.

Increasing in-vitro and in-vivo evidence suggests that the rat lung tumor response to crystalline silica exposure is a result of marked and persistent inflammation and epithelial proliferation. Other pathways such as a role for crystalline silica surface-generated oxidants or a direct genotoxic effect are not ruled out; however, at present, there is no convincing evidence for these alternative pathways.

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Silica exposure and cancer

In 1997 the International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) reviewed all the research literature on the cancer risk of crystalline silica exposure and published an updated monograph in 1997. Possible differences in carcinogenic potential among polymorphs of crystalline silica were considered. Some studies were of populations exposed principally to quartz. In only one study (that of United States diatomaceous earth workers) was the exposure predominantly cristobalite. Studies of mixed environments (i.e. ceramics, pottery, refractory brick) could not delineate exposures specifically to quartz or cristobalite. Although there were some indications that cancer risks varied by type of industry and process in a manner suggestive of polymorph-specific hazards.

Seventeen cohort and five case-control studies were reported on ore miners potentially exposed to silica dust. The majority of these studies reported an

elevated mortality for lung cancer among silica-exposed workers. However, in only a few ore mining studies were confounders such as other known occupational respiratory carcinogens taken into account (I.e. diesel exhaust, polycyclic aromatic hydrocarbons, cadmium, radon or arsenic).

Among quarry and granite workers there were six cohort studies available for review. These studies provide important information on cancer risks because the workplace environments were generally free of reported exposures to potentially confounding agents (e.g., radon). All studies revealed lung cancer excesses. Direct quantification of silica dust exposure concentrations in relation to lung cancer risk was not conducted in any of these studies, mainly due to sparse occupational hygiene measurement data. However, some studies provided indications of exposure-response associations when surrogate dose data, such as duration of employment and category of exposure, were used. For example, findings for lung cancer include a nearly twofold mortality elevation among long-term granite shed workers in Vermont, United States, an eightfold elevation among sandstone workers in Copenhagen, Denmark, and a relative risk of roughly 3.5 among crushed granite stone workers in the United States with long duration of exposure and time since exposure onset. One study of German slate quarry workers indicated a more prominent relationship between employment duration and lung cancer among workers with silicosis than among workers without silicosis. The Working Group regarded radiographic evidence of silicosis as a marker of high exposure to silica.

In studies of the refractory brick and diatomaceous earth industries provided consistent evidence of increased lung cancer with overall relative risks of about 1.5. In refractory brick and diatomaceous earth plants, the raw materials (amorphous or crystalline silica) are processed at temperatures around 1000. In ceramic and pottery manufacturing plants, exposures are mainly to quartz, but where high temperatures are used in ovens, potential exposures to cristobalite may occur. In a cohort study of British pottery workers, lung cancer mortality was slightly elevated; a nested case-control analysis of lung cancer did not show an association with duration of exposure, but indicated a relationship between lung cancer mortality and average and peak exposures in firing and post-firing operations, with relative risks of approximately 2.0. In an Italian case-control study, apart from a fourfold increase in lung cancer in registered silicotics, there was a small increase in lung cancer for subjects without silicosis.

There were only three large cohort studies of foundry workers where silica dust or silicosis were considered as risk factors for cancer. One study from Denmark found a slightly elevated risk of lung cancer in silicotics compared with non-silicotics. Two studies, one from the United States and one from China, yielded conflicting results for lung cancer. The Chinese study suggested positive associations of silica with both lung cancer and stomach cancer, although there remained a potential for confounding by exposures to polycyclic aromatic

hydrocarbons. The United States study did not demonstrate an association of lung cancer with cumulative silica exposure.

The vast majority of studies on registered silicotics reported excess lung cancer risks, with relative risks ranging from 1.5 to 6.0. Excesses were seen across countries, industries and time periods.

For the evaluation of crystalline silica, there were a large number of studies across the world. Not all of the studies demonstrated excess cancer risks. However, in view of the relatively large number of epidemiological studies that have been undertaken and, given the wide range of populations and exposure circumstances studied, some non-uniformity of results would be expected. The Working Group concluded that overall the epidemiological findings support increased lung cancer risks from inhaled crystalline silica (quartz and cristobalite) resulting from occupational exposure. The observed associations could not be explained by confounding or other biases.

The conclusions of the Working Group on cancer and silica exposure concluded:
There is sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources.
There is inadequate evidence in humans for the carcinogenicity of amorphous silica.
There is sufficient evidence in experimental animals for the carcinogenicity of quartz and cristobalite.
There is limited evidence in experimental animals for the carcinogenicity of tridymite.
There is inadequate evidence in experimental animals for the carcinogenicity of diatomaceous earth.
There is inadequate evidence in experimental animals for the carcinogenicity of synthetic amorphous silica

Crystalline silica inhaled in the form of quartz or cristobalite from occupational sources is carcinogenic to humans (Group 1).
http://www.iarc.fr

SLIDE 31

Silica exposure and autoimmune disease
Respiratory complications from crystalline silica exposures have been known for centuries, but the link of silica exposure and autoimmune disease has been more recent. In the 1950s, Caplan first described unusual radiologic changes in the lungs of Welsh coal miners who had pneumoconiosis. In a subsequent study of these miners, Miall et al. (1953) found that the rheumatoid lesions in the lung were predictive for rheumatoid arthritis (subsequently called Caplan's syndrome). Since that time many studies have examined the link between crystalline silica

exposure and development of autoimmune diseases. Among the systemic autoimmune diseases, occupational exposure to crystalline silica exposure has been linked to rheumatoid arthritis, scleroderma, systemic lupus erythematosus, and some of the small vessel vasculitides.

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Silica exposure and autoimmune disease
Crystalline silica particles are ingested by alveolar macrophages and result in inflammation and activation of fibroblasts. The digested crystalline silica destroys the macrophages, and the crystalline silica is again digested by new macrophages. This repeated process leads to chronic immune activity and fibrosis. Studies have shown that crystalline silica can be mobilized from the lungs to other organs, including lymph nodes, spleen, and kidney. Silicosis and mineral dust pneumoconiosis have been linked to an increase in autoantibodies, immune complexes, and excess production of immunoglobulins, even in the absence of a specific autoimmune disease. Many of the cases of autoimmune disease were first discovered during screening of silica-exposed workers or workers who were being treated for silicosis. It was not clear in these cases whether the silicosis was a pathologic process that may predispose some individuals to develop autoimmune disease or whether the opposite was true, that the autoimmune disease may predispose some individuals to develop silicosis in the lung.

Long latency periods (15-25 years) have been reported between first silica exposure and the development of autoimmune diseases. However, in a 1987 study of granite workers, Klockars et al. (1987) documented individuals who developed autoimmune disease in a shorter period of time. The authors published results of a follow up of 35 workers exposed to silica who had developed rheumatoid arthritis. Of the workers, 13 had 5 years of exposure before the onset of rheumatoid arthritis. For 7 of the workers, latency ranged from 1 to 12 years. This study was also interesting in that 20 of the 35 miners had normal chest x-rays at the time of the onset of rheumatoid arthritis.

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Silica exposure and autoimmune disease
The mechanism of crystalline silica in the development of autoimmune diseases may be a result of the adjuvant effect on antibody production (An adjuvant is a substance that enhances an immune response to an antigen). The development of silicosis is dose dependent, but no studies have determined a dose-response or threshold effect of crystalline silica as an adjuvant. Genetic differences and susceptibility to autoimmune diseases may vary the characteristics and extent of

the inflammation caused by silica exposure. Crystalline silica can also cause cell death by necrosis and apoptosis (an active process involving gene regulation).

Apoptosis is enhanced by silica and at levels where an acute toxicity is not detected. The sFas ligand, a type II membrane protein that induces apoptosis, is elevated in silicosis patients. The elevation of sFas levels have been reported in silicosis patients who have slight shortness of breath, normal partial pressure of carbon dioxide, or normal partial pressure of oxygen and have been classified with slight respiratory disorders. Tomokuni et al. (1997) speculated that the severity of respiratory involvement may not occur to the same degree as the abnormalities and elevation of apoptosis-related molecules in silicosis patients.

Tomokuni et al. (1999) found no significant correlation of duration of exposure to crystalline silica dust and the serum levels of sFas. Host susceptibility may explain why all workers exposed to silica do not develop autoimmune disorders.

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Silica exposure and renal disease

A statistically significant association between crystalline silica exposure and several renal diseases has been reported in epidemiologic studies. Steenland et al. (2001) reported an increasing standardized rate ratio for acute and chronic renal disease with increasing cumulative crystalline silica exposure and an excess of end-stage renal disease incidence (highest for glomerulonephritis). The reported cases of glomerulonephritis in patients with crystalline silica exposure show rapid progression and are associated with positive antineutrophil cytoplasmic antibodies (ANCA) with or without systemic vasculitis, such as Wegener granulomatosis or microscopic polyangiitis. pANCA is strongly associated with microscopic polyangiitis and rapidly progressive glomerulonephritis (RPGN). In a recent study (Gregorini et al. 1997), crystalline silica-exposed patients with positive ANCA showed mainly a pANCA pattern. Not all silica-exposed patients with pANCA-positive RPGN reported by Gregorini et al. (1993) had pulmonary silicosis. Gregorini et al. (1997) reported immune abnormalities in patients with silicosis and in silica-exposed patients with no evidence of lung disease.

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Silica exposure and renal disease

The intensity of exposure to silica dust may be more important than cumulative exposure or duration in the development of autoimmune diseases. In a study of silica-exposed gold miners, Calvert et al. (1997) investigated end-stage renal disease (ESRD) and exposures. They suggested that crystalline silica exposure was most strongly associated with ESRD (especially ESRD caused by

glomerulonephritis) and that the median exposure of the cohort was below the OSHA permissible exposures levels (indicating a lower level of exposure needed to start the disease process).

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Silica exposure and renal disease
Effects of silica are thought to be either immune-complex mediated or a result of direct toxic effects of the silica on the glomeruli.

Studies have shown that crystalline silica can be mobilized from the lungs to other organs, including the kidney, lymph nodes, and spleen.

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Treatment
Prevention/Prevention/Prevention

Workers with silica exposure are at risk for both progression of the disease process and for the development of tuberculosis. It is recommended that workers with silicosis have a yearly chest x-ray and PPD. Influenza vaccination should be encouraged every year and pneumococcal vaccine encouraged.

If the silicosis patient develops TB it should be treated aggressively. The most recent treatment guidelines are available from the CDC. “Treatment of Tuberculosis”, American Thoracic Society, Center for Disease Control and Prevention, and the Infectious Diseases Society of America, MMWR; 52: 1-77.

Patient with dyspnea and obstruction may be treated with inhaled bronchodilators. O2 therapy is indicated in patients with cor pulmonale, hypoxemia, and pulmonary hypertension.

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Prevention – Occupational Health Surveillance
Occupational health surveillance is the systematic monitoring of health events and exposures in working populations for the prevention and control of occupational hazards and the associated illness and injury. Occupational health and all other public health surveillance systems are comprised of four essential sections:
•To gather information on cases of occupational illness and injury and workplace exposures;
•To condense, refine, and analyze the data;

•To disseminate the analyzed data to workers, unions, employers, governmental agencies, and the public; and
•To plan and execute interventions – primary prevention – based on the analyzed data to alter the factors that contribute to adverse health events and hazards.

The main goals of occupational health surveillance are:
•To identify the incidence and prevalence of occupational illness and injury for determining control and research priorities and strategies and to evaluate the effectiveness of interventions;
•To identify individual cases of work-related disease or injury in order to identify other individuals from the same or similar workplace or individuals with the same or similar exposures who may be at risk for illness or injury;
•To identify new associations between occupational hazards and the resultant injury or illness.

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Prevention
The concept of the occupational sentinel health event was put forth in 1983 by Rutstein (Harvard) and investigators at NIOSH for use in occupational health surveillance. They defined the occupational sentinel health event as “A disease, disability, or untimely death which is occupationally related and whose occurrence may: 1) provide the impetus for epidemiologic or industrial hygiene studies; or 2) serve as a warning signal that materials substitution, engineering control, personal protection, or medical care may be required”.

An occupational sentinel health event should trigger an investigation (an investigation similar to infectious disease outbreaks) that would identify other workers, both current and previous workers, who have had exposure to silica and are at risk for the development of silicosis. It is a public health issue to evaluate and educate workers about the health hazards of silica exposure.

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Prevention
State-based surveillance

Sentinel Event Notification Systems for Occupational Risk (SENSOR) There are many state based silicosis surveillance projects that are helpful in case investigations and many states that have a legal requirement to report a case of silicosis to the appropriate state agency. The NIOSH resource website for silicosis surveillance programs is:
http://www.cdc.gov/niosh/topics/surveillance/ORDS/StateBasedSurveillance/SENSORSilicosis.htm

State Reporting Guidelines for Silicosis

State health departments should encourage physicians, including radiologists and pathologists, as well as other health-care professionals, to report all diagnosed or suspected cases of silicosis. These reports should include persons with: A. A physician's provisional or working diagnosis of silicosis. **OR** B. A chest radiograph interpreted as consistent with silicosis. **OR** C. Pathologic findings consistent with silicosis. State health departments should collect appropriate clinical, epidemiologic, and workplace information on reported persons with silicosis as needed to set priorities for workplace investigations.

Surveillance Case Definition for Silicosis

A. History of occupational exposure to airborne silica dust. * AND EITHER OR BOTH OF THE FOLLOWING: B1. Chest radiograph or other imaging technique interpreted as consistent with silicosis. **B2. Pathologic findings characteristic of silicosis. ***

* Exposure settings associated with silicosis are well characterized and have been summarized in several reviews. The induction period between initial silica exposure and development of radiographically detectable nodular silicosis is usually >10 years. Shorter induction periods are associated with heavy exposures, and acute silicosis may develop within months following massive silica exposure.

** Cases can be classified as nodular or acute. Common radiographic findings of nodular silicosis include multiple, bilateral, and rounded opacities in the upper lung zones; other patterns have been described. Since patients may have mixed dust exposure, irregular opacities may be present or even predominant. To be considered consistent with silicosis, radiographs of nodular silicosis classified by using the ILO classification system should have small opacity profusion categories of 1/0 or greater by the International Labour Organization classification system. If the largest opacity is >1 cm in diameter, progressive massive fibrosis [PMF] (also known as 'complicated' silicosis) is present. A bilateral alveolar filling pattern is characteristic of acute silicosis and may be followed by rapid development of bilateral small or large opacities.

*** Characteristic lung tissue pathology in nodular silicosis consists of fibrotic nodules with concentric "onion-skinned" arrangement of collagen fibers, central hyalinization, and a cellular peripheral zone, with lightly birefringent particles seen under polarized light. In acute silicosis, microscopic pathology shows a periodic acid-Schiff positive alveolar exudate (alveolar lipoproteinosis) and a cellular infiltrate in the alveolar walls.

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Prevention

Hierarchy of Controls

Occupational diseases and injuries are preventable. Hierarchy of controls refers to the concept of fixing the workplace to prevent exposures to hazards. Engineering controls are always the first and preferred method of hazard control. Substitution of a hazardous material with a less hazardous one is a method of hazard control. However, substitution is not always feasible. For example, in a mine or quarry silica is naturally occurring in the rock and there is not a product substitution. However, abrasive work can substitute the use of sand (sand blasting) with products that do not contain silica.

An engineering method that would work at a mine or quarry would be controlling the hazard at the source. Using a wet process can control a dust hazard. Local exhaust ventilation is also another method to control hazards at their source in addition to dual suppression and process isolation.

When all engineering methods have been utilized but there is still potential for hazard exposure the next step in the hierarchy of controls is administrative controls. Rotating people in and out of areas with hazards would reduce the time that any one worker would be exposed.

When the first two methods have failed to protect a worker completely from a hazard personal protective equipment (PPE) controls are employed. PPE can be used for infrequent short-term exposures while other methods to control the hazards are being implemented. As a long-term measure for hazard control PPE is the last resort. Often the use of PPE, such as respirators, requires medical evaluations, special fit testing, and education for the worker. In addition, depending on the exposure and if there are other hazards in addition to the silica some respirators may not provide adequate protection. Determination of the correct respirator to use include determination of contaminant(s); physical, chemical, and toxicological properties of the contaminant(s); NIOSH recommended exposure limit (REL), OSHA permissible exposure limit (PEL), American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV), State-OSHA exposure limit, American Industrial Hygiene Association (AIHA) Workplace Environmental Exposure Limit (WEEL), or other applicable occupational exposure limit; expected concentration of each respiratory hazard; immediately dangerous to life or health (IDLH) concentration; oxygen concentration or expected oxygen concentration; eye irritation potential; and environmental factors, such as presence of oil aerosols.

Education of workers on the health risks of silica dust exposure and methods for control of exposures are an important part of prevention

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Prevention- Regulation

Federal Coal Mine Health and Safety Act of 1969 (Coal Act) was historically the most comprehensive and stringent federal legislation to govern the US mining

industry. The Coal Act dramatically increased federal enforcement in US coal mines. Both surface and underground coal mines were within its regulatory scope, requiring two annual inspections of every surface coal mine and four at every underground coal mine. The Coal Act also required monetary penalties for all violations, and established criminal penalties for knowing and willful violations. The safety standards for all coal mines were strengthened, and health standards were adopted. The Coal Act included specific procedures for the development of improved mandatory health and safety standards, and provided compensation for miners who were totally and permanently disabled by the progressive respiratory disease caused by the inhalation of fine coal dust pneumoconiosis or "black lung".

In 1973, the Secretary of the Interior, through administrative action, created the Mining Enforcement and Safety Administration (MESA) as a new departmental agency separate from the Bureau of Mines. MESA assumed the safety and health enforcement functions formerly carried out by the Bureau to avoid any appearance of a conflict of interest between the enforcement of mine safety and health standards and the Bureau's responsibilities for mineral resource development.

The Federal Mine Safety and Health Act of 1977 (Mine Act) amended the 1969 Coal Act and consolidated all federal health and safety regulations of the mining industry, coal as well as non-coal mining, under a single statutory scheme. The Mine Act strengthened and expanded the rights of miners, and enhanced the protection of miners from retaliation for exercising such rights. Mining fatalities dropped sharply under the Mine Act from 272 in 1977 to 86 in 2000. The Mine Act also transferred responsibility for carrying out its mandates from the Department of the Interior to the Department of Labor, and named the new agency the Mine Safety and Health Administration (MSHA). Additionally, the Mine Act established the independent Federal Mine Safety and Health Review Commission to provide for independent review of the majority of MSHA's enforcement actions.

The mission of the Mine Safety and Health Administration (MSHA) is to administer the provisions of the Federal Mine Safety and Health Act of 1977 (Mine Act) and to enforce compliance with mandatory safety and health standards as a means to eliminate fatal accidents; to reduce the frequency and severity of nonfatal accidents; to minimize health hazards; and to promote improved safety and health conditions in the Nation's mines

More information on MSHA can be obtained at the website: http://www.msha.gov/

The Occupational Safety and Health Act of 1970 created both NIOSH and the Occupational Safety and Health Administration (OSHA). OSHA is in the U.S. Department of Labor and is responsible for developing and enforcing workplace

safety and health regulations. NIOSH is in the U.S. Department of Health and Human Services and is an agency established to help assure safe and healthful working conditions for working men and women by providing research, information, education, and training in the field of occupational safety and health.

NIOSH provides national and world leadership to prevent work-related illness, injury, disability, and death by gathering information, conducting scientific research, and translating the knowledge gained into products and services. NIOSH's mission is critical to the health and safety of every American worker.

The National Institute for Occupational Safety and Health (NIOSH) is the federal agency responsible for conducting research and making recommendations for the prevention of work-related injury and illness. NIOSH is part of the Centers for Disease Control and Prevention (CDC) in the Department of Health and Human Services.

The Institute's responsibilities include:
•Conducting a focused program of research to reduce injuries and illnesses among workers in high-priority areas and high-risk sectors, including mining, agriculture, construction, and health care.
•Implementing and maintaining a system of surveillance for major workplace illnesses, injuries, exposures, and health and safety hazards.
•Increasing prevention activities through workplace evaluations, interventions, and recommendations.
•Providing workers, employers, the public, and the occupational safety and health community with information, training, and capacity to prevent occupational injuries and illnesses.

More information on NIOSH may be obtained at the website:
http://www.cdc.gov/niosh/homepage.html

The Occupational Safety and Health Act of 1970 was passed by Congress "to assure, as far as possible, every working man and woman in the country safe and healthful working condition". The OSH Act delegated responsibility to the Secretary of Labor for the development of workplace health and safety standards. The Occupational Safety and Health Administration (OSHA) was established within the US Department of Labor as a separate agency to develop standards and enforcement.

OSHA's mission is to assure the safety and health of America's workers by setting and enforcing standards; providing training, outreach, and education; establishing partnerships; and encouraging continual improvement in workplace safety and health.

Under Section 18 of the OSH Act states are encouraged to develop and operate state based job safety and health programs. OSHA approves and monitors State

plans and provides up to 50 percent of an approved plan's operating costs. States must set job safety and health standards that are "at least as effective as" comparable federal standards. (Most States adopt standards identical to federal ones.) States have the option to promulgate standards covering hazards not addressed by federal standards. States must conduct inspections to enforce its standards, cover public (State and local government) employees, and operate occupational safety and health training and education programs. In addition, most States provide free on-site consultation to help employers identify and correct workplace hazards.

More information on OSHA can be obtained at the website: http://www.osha.gov/

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Prevention- Regulation

The current OSHA permissible exposure limit (PEL) for respirable crystalline silica is 10 mg/m3 /% silica + 2 in an 8-hour time-weighted average (TWA). The NIOSH recommended exposure limit (REL) is 50 *u*g/m^3 as a TWA for up to 10 hours/day during a 40-hour workweek.

OSHA is proposing change to the regulations for exposure limits for silica in 2004-2005.

Table 1 describes the NIOSH-recommended respiratory protection for workers exposed to respirable crystalline silica.

Table 1. NIOSH-recommended respiratory protection for workers exposed to respirable crystalline silica

Condition	Minimum respiratory protection* required to meet the Condition NIOSH REL for crystalline silica (50 µg/m³)**
Less than or equal to 500 $\mu g/m^3$ (10 x REL)***	Any air-purifying respirator with a high-efficiency particulate filter
Less than or equal to 1,250 $\mu g/m^3$ (25 x REL)	Any powered, air-purifying respirator with a high-efficiency particulate filter, or Any supplied-air respirator equipped with a hood or helmet and operated in a continuous-flow mode (for example, type CE abrasive blasting respirators operated in the continuous-flow mode)
Less than or equal to 2,500 $\mu g/m^3$ (50 x REL)	Any air-purifying, full-facepiece respirator with a high- efficiency particulate filter, or Any powered, air-purifying respirator with a tight-fitting facepiece and a high-efficiency particulate filter
Less than or equal to 50,000 $\mu g/m^3$ (1,000 x REL)	Any supplied-air respirator equipped with a half-mask and operated in a pressure-demand or other positive-pressure mode
Less than or equal to 100,000 $\mu g/m^3$ (2,000 x REL)	Any supplied-air respirator equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode (for example, a type CE abrasive blasting respirator operated in a positive-pressure mode)
Planned or emergency entry into environments containing unknown concentrations or concentrations less than or equal to 500,000 $\mu g/m^3$ (10,000 x REL)	Any self-contained breathing apparatus equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode,**** or Any supplied-air respirator equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode in combination with an auxiliary self-contained breathing apparatus operated in a pressure-demand or other positive-pressure mode****
Firefighting	Any self-contained breathing apparatus equipped with a full facepiece and operated in a pressure-demand or other positive-pressure mode****
Escape only	Any air-purifying, full-facepiece respirator with a high- efficiency particulate filter, or Any appropriate escape-type, self-contained breathing apparatus

* Only NIOSH/MSHA-approved equipment should be used.
** These recommendations are intended to protect workers from silicosis; only the most protective respirators are recommended for used with carcinogens.
*** Assigned protection factor (APF) times the NIOSH REL. The APF is the minimum anticipated level of protection provided by each type of respirator.
**** Most protective respirators.

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Any Questions?

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