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Respiratory Diseases Among Dust Exposed Workers

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1. Introduction

Airborne contaminants occur in the gaseous form (gases and vapours) or as aerosols. Aerosols may exist in the form of airborne dusts, sprays, mists, smokes and fumes. In the occupational setting, all these forms may be important because they relate to a wide range of occupational diseases. Airborne dusts are of particular concern because they are well known to be associated with classical widespread occupational lung diseases such as the pneumoconiosis, chronic obstructive pulmonary disease, occupational asthma, etc. Occupational exposure to dust particles occurs everywhere but is especially prevalent in low- and middle-income countries. Table 1 shows some examples of the types of dust found in the work environment.

TYPE OF DUST	EXAMPLES
mineral dusts	free crystalline silica, coal and cement dusts
metallic dusts	lead, cadmium, nickel, and beryllium dusts
other chemical dusts	many bulk chemicals and pesticides
organic and vegetable dusts	flour, wood, cotton and tea dusts, pollen
biohazards	viable particles, moulds and spores

Table 1. Common types of dust in the work envrionment

2. Commonest type of occupation dust particles

2.1 Silica

Silica, also known as silicon dioxide (SiO_2), is formed from silicon and oxygen atoms. It has a melting point of 1,600°C and is a colorless, odorless, and noncombustible solid. Since oxygen and silicon make up about 75% of the Earth, the compound silica is quite common in surrounding environment[1]. Silicates comprise about 25% of known minerals, nearly 40% of the common minerals, and well over 90% of the earth's crust[1]. It is found in many rocks, such as marble, sandstone, flint and slate, and in some metallic ores. Silica can also be in soil, mortar, plaster, and shingles.

Silica occurs in three forms: crystalline, microcrystalline (or cryptocrystalline) and amorphous (non-crystalline). "Free" silica is composed of pure silicon dioxide, not combined

with other elements, whereas silicates (e.g. talc, asbestos, and mica) are SiO_2 combined with an appreciable portion of cations. Crystalline silica exists in seven different forms (polymorphs), depending upon the temperature of formation. The main 3 polymorphs are quartz, cristobalite, and tridymite. Quartz is subdivided into alpha and beta forms. In nature, most quartz is alpha-quartz and alpha-quartz comprises the bulk of crystalline silica. Quartz is the second most common mineral in the world. Amorphous forms of silica, such as opal, diatomaceous earth, silica-rich fiberglass, fume silica, mineral wool, and silica glass (vitreous silica), are generally considered as less harmful[2].

Occupational exposure to crystalline silica can occur in any workplace situation where airborne dust, containing a proportion of crystalline silica, is generated. Industries where crystalline silica is present include quarrying, mining, mineral processing (eg drying, grinding, bagging and handling), slate working, stone crushing and dressing, foundry work, brick and tile making, some refractory processes, construction work, including work with stone, concrete, brick and some insulation boards, tunnelling, building restoration and in the pottery and ceramic industries. Figure 1 shows some of the job processes that generate and disperse large quantities of respirable silica dusts into the air. Silica dust is an inhalation hazard. Workers may be at risk of silicosis from exposure to silica dust when high-velocity impact shatters the sand into smaller, respirable (< 0.5 to 5.0 μm in diameter) dust particles. From recent reports, more than 23 million workers are exposed to crystalline silica in China

Fig. 1. Job processes that generate and disperse respirable silica dusts into the air

and more than 10 million in India, as well as over 3 million workers in Europe and at 1.7 million in the United States[3].

2.2 Asbestos

Asbestos is a set of six naturally occurring silicate minerals exploited commercially which possess high tensile strength, flexibility, resistance to chemical and thermal degradation, and electrical resistance. Six minerals are defined by the United States Environmental Protection Agency as "asbestos" including those belonging to the serpentine class chrysotile and those belonging to the amphibole class amosite, crocidolite, tremolite, anthophyllite and actinolite[4]. They have different physical and chemical properties but share a fibrous form. Mineralogists have taken a particle with a length-to-breadth ratio (aspect ratio) of 10:1 or more to be a fibre. In milled asbestos most of the particles have aspect ratios that range from 5:1 to 20:1 or more and, in the case of chrysotile , mostly greater than 50:1. In medical and environmental literature a regulated fibre has been defined as a mineral particle with a length which is at least three times greater than its diameter, of length greater than 5 micrometers and diameters less than 3 micrometres. There are essentially two major varieties of asbestos viz. serpentine and amphibole. Table 2 shows the species and varieties[5].

SPECIES	VARIETY
Chrysotile	Serpentine
Anthophyllite	Amphibole
Amosite	Amphibole
Actinolite	Amphibole
Tremolite	Amphibole
Crocidolite	Amphibole

Table 2. Asbestos

Asbestos is used for insulation in buildings and as ingredient in a number of products, such as roofing shingles, water supply lines, fire blankets, plastic fillers, and medical packing, as well as clutches and brake linings, gaskets and pads for automobiles. Table 3 illustrates certain kinds of work involve high exposure to asbestos. All forms of asbestos are

work involve high exposure to asbestos	
asbestos mining and milling	manufacture of asbestos tiles
building demolition	manufacture of asbestos fabrics
manufacture of brake linings	drywall installation
shipbuilding trades	drywall removal
insulation work in construction	other asbestos removal
plasterers	firefighting
pipe fitters	asbestos tile setters
railroad workers	aluminum plant workers

Table 3. Certain kinds of work involve high exposure to asbestos

carcinogenic to humans, and may cause mesothelioma and cancer of the lung, larynx and ovary. Asbestos exposure is also responsible for other diseases, such as asbestosis (fibrosis of the lungs), pleural plaques, thickening and effusions.

Currently, about 125 million people in the world are exposed to asbestos at the workplace. According to the most recent WHO estimates, more than 107 000 people die each year from asbestos-related lung cancer, mesothelioma and asbestosis resulting from exposure at work. One in every three deaths from occupational cancer is estimated to be caused by asbestos. In addition, it is estimated that several thousand deaths annually can be attributed to exposure to asbestos in the home[6].

2.3 Coal mine dust

Coal is a valuable and plentiful natural global resource. It is found throughout the world. Coal is classified into four main types or ranks (anthracite, bituminous, subbituminous, and lignite), depending on the amounts and types of carbon it contains and on the amount of heat energy it can produce. Coal is mined by two methods: surface mining and underground mining. The choice of mining method is largely determined by the geology of the coal deposit. Underground mining currently accounts for a bigger share of world coal production than opencast. Coal mine dust is a mixture that contains more than 50 substances. The mineral content depends on the particle size of the dust and the coal seam. The most commonly found minerals in coal mine dust include kaolinite, illite, calcite, pyrite and quartz (silica). Dust from high rank coals usually contains more silica particles than dust of lower rank coals. Most workplace exposure to coal dust occurs during mining; however exposure can also occur during handling of the mined product during cleaning and blending processes or bulk handling at large coal fired facilities[7].

3. Main respiratory diseases related to occupational dust particles

3.1 Pneumoconiosis

Pneumoconiosis is an occupational lung disease and a restrictive lung disease caused by the inhalation of dust. A longer, factitious term is pneumonoultramicroscopicsilicovolcanoconiosis. Depending upon the type of dust, the disease is given different names:

1. Coal worker' pneumoconiosis(CWP): caused by inhaling coal dust;
2. Asbestosis: caused by inhaling asbestos;
3. Berylliosis: caused by inhaling beryllium;
4. Kaolin pneumoconiosis: caused by inhaling china clay;
5. Siderosis: caused by inhaling iron oxide;
6. Silicosis: caused by inhaling silica dust;
7. Metallic pneumoconiosis: caused by inhaling barium, cobalt, tin, tungsten dust;
8. Talc pneumoconiosis: caused by inhaling talc dust;
9. Popcorn pneumoconiosis: caused by inhaling fumes produced when manufacturing microwave popcorn

Some more types of pneumoconiosis include graphite pneumoconiosis, carbon black pneumoconiosis, talc pneumoconiosis, cement pneumoconiosis, mica pneumoconiosis, aluminosis, electric welder pneumoconiosis, foundry worker's pneumoconiosis.

3.2 Silicosis

Silicosis is a form of pneumoconiosis caused by inhalation of crystalline silica dust, and is marked by inflammation and scarring in forms of nodular lesions in the upper lobes of the lungs[6] (Figure 2).

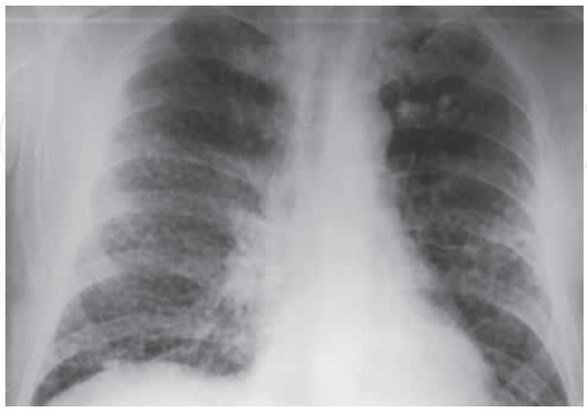

Fig. 2. Silicosis showing as nodular mass on a chest x-ray

Silicosis is the commonest occupational lung disease worldwide. It occurs everywhere but is especially prevalent in low- and middle-income countries. China is the country with the largest number of silicosis patients, with more than 500,000 cases in records from 1949 to 2010. During 1991 to 2010, more than 6,000 new cases and more than 24,000 deaths occurred annually. The problem is particularly acute in small-scale mines in China[3]. High risk of silicosis also reported in other countries. The proportions of gold miners with silicosis increased from 0.03 to 0.32 for black miners and from 0.18 to 0.22 for white miners in a 33-year period in South Africa. Among ornamental stone carvers in Brazil, the prevalence of disease remains over 50 percent. Although U.S. silicosis mortality declined between 1968 and 2002, silicosis deaths and new cases continue to occur, even in young workers[6].

The most common form of silicosis (chronic) will often develop between 15 to 45 years after first exposure, but certain rare forms of the disease can occur after a single heavy dose or heavy exposures to a very high concentration of silica in a short period of time. Workers with Silicosis may have following symptoms: Shortness of breath following physical exertion, severe and chronic cough, fatigue, loss of appetite, chest pains and fevers.

Silicosis is generally divided into three types as below:

1. Acute Silicosis: Occurs after heavy exposure to high concentrations of silica. The symptoms can develop within a few weeks or as long as 5 years after the exposure.
2. Chronic Silicosis: Occurs after long term exposure (over 10 years) of low concentrations of silica dust. This is most common form of the disease, and is often undetected for many years because a chest X-Ray often will not reveal the disease for as long as 20 years after exposure. This type of the disease severely hinders the ability of the body to fight infections because of the damage to the lungs, making the person more susceptible to other lung illnesses, including tuberculosis.
3. Accelerated Silicosis: Occurs after exposure to high concentrations of silica. The disease develops within 5 to 10 years after exposure.

The development of silicosis is associated with content of free silica in the dust, type of silica, concentration of dust, dispersion, years of exposure, prevention and individual factors. The cumulative dose of silica (respirable dust concentration x crystalline silica content x exposure duration) is probably the most important factor for the development of silicosis[8].

Pathology

Pathological varieties of silicosis include simple (nodular) silicosis, progressive massive fibrosis, silicoproteinosis (acute silicosis) and diffuse interstitial fibrosis.

Alveolar macrophages engulf inhaled free silica particles and enter lymphatics and interstitial tissue. The macrophages cause release of cytokines (tumor necrosis factor-α, IL-1), growth factors (tumor growth factor-β), and oxidants, stimulating parenchymal inflammation, collagen synthesis, and, ultimately, fibrosis.

When the macrophages die, they release the silica into interstitial tissue around the small bronchioles, causing formation of the pathognomonic silicotic nodule. These nodules initially contain macrophages, lymphocytes, mast cells, fibroblasts with disorganized patches of collagen, and scattered birefringent particles that are best seen by polarized light microscopy. As they mature, the centers of the nodules become dense balls of fibrotic scar with a classic onion-skin appearance and are surrounded by an outer layer of inflammatory cells. In low-intensity or short-term exposures, these nodules remain discrete and do not compromise lung function (simple chronic silicosis). But with higher-intensity or more prolonged exposures (complicated chronic silicosis), these nodules coalesce and cause progressive fibrosis and reduction of lung volumes (total lung capacity, ventilatory capacity) on pulmonary function tests, or they coalesce, sometimes forming large conglomerate masses (called progressive massive fibrosis).

Silica quartz crystals in lung tissue can be observed under polarised light microscopy. Figure 3 illustrates a slide under polarised light microscopy of lung tissue containing crystalline silica quartz. The white spots represent silica crystals in the specimen of lung tissue. The silica crystals present in the lung tissue are of different size and represent

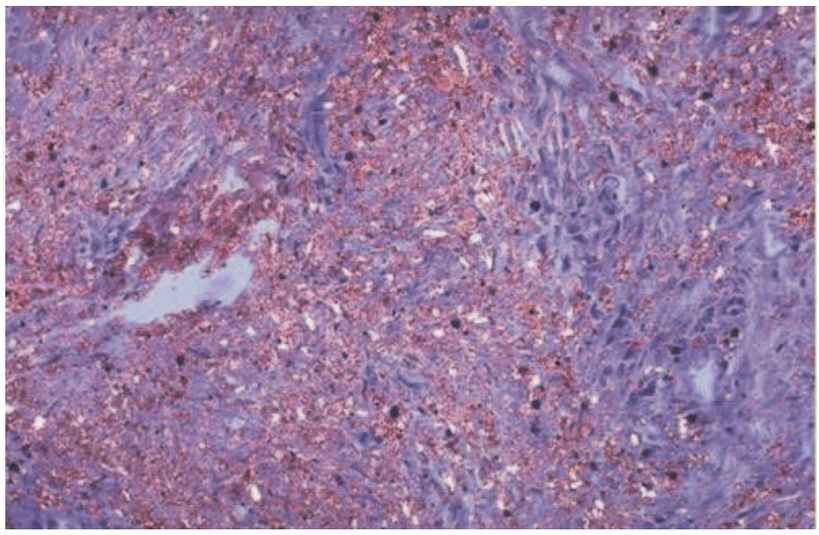

Fig. 3. Lung tissue observed under polarized light microscopy containing crystalline silica

therefore a typical picture of silica crystal distribution in lung tissue of a worker exposed to crystalline silica.

The primary feature that develops in lungs of silica quartz exposed workers is nodule formation in the upper zones of the lung[9]. Nodule formation is usually the result of many years of exposure to relatively low levels of dust that contain silica quartz[10]. Figure 4 represents a photo of silicotic nodule. The classical silicotic nodule is usually located in the area of the respiratory bronchiole.The nodule is composed of reticulin fibres in the periphery and collagen fibresin the center. Fibroblastic activity is usually evident around the periphery of the concentric lesion[11].

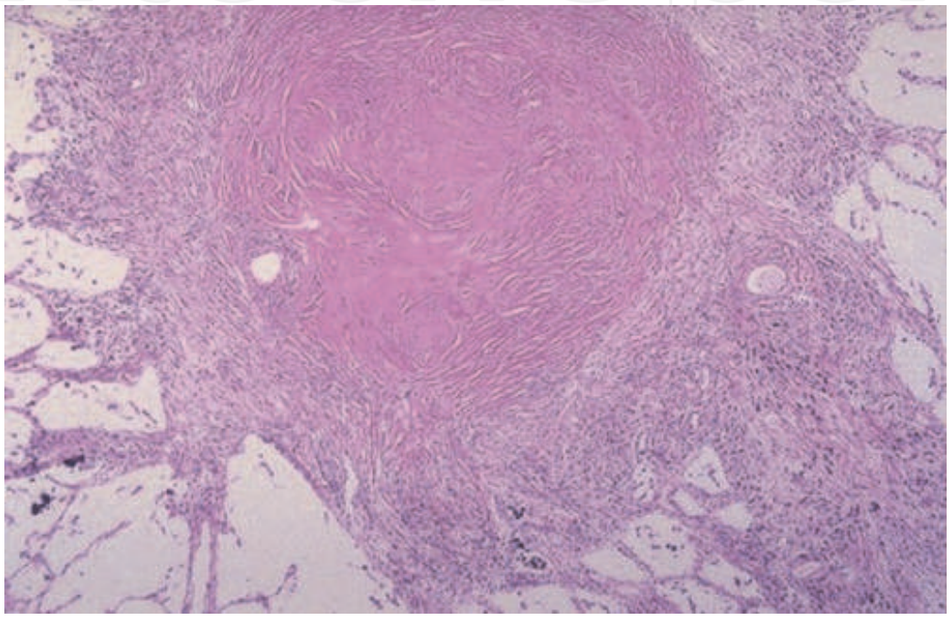

Fig. 4. A microscopic photo of a typical silicotic nodule containing collage fibres in a whorled pattern

The airway and blood vessels are frequently destroyed by being entrapped in the fibrotic nodule. Silica particles are difficult to identify in tissue section by polarized light microscopy. Therefore, special techniques involving high resoluton microscopy are required. It appears that the extent of lesion bears little association with amount of silica present[11].

Diagnosis

The diagnosis of silicosis generally rests upon history of substantial exposure to silica dusts and compatible radiological features, together with the exclusion of other competing diagnoses, like miliary tuberculosis, fungal infections, sarcoidosis, idiopathic pulmonary fibrosis, other interstitial lung diseases, or carcinomotosis.

History: The individual may report a history of exposure to silica dust. Although initially there may be no symptoms, symptoms may eventually include difficulty breathing, shortness of breath, a cough (either dry or productive), and/or chest tightness.

Physical exam: Auscultation (listening to breath sounds through a stethoscope) may reveal changes in breath sounds that may indicate obstruction in the upper lobes of the lung. Wheezing only occurs when other conditions such as bronchitis or asthma are present. In chronic complicated silicosis or subacute silicosis, right-sided heart failure (cor pulmonale) may be noted. Rales are often heard.

Tests: Lung tissue changes in progressive silicosis are often detected by chest x-ray before they cause any symptoms. Pulmonary function tests will be used to evaluate lung function and confirm the presence of lung disorders. These may include spirometry and lung volume measurement to detect any restriction of normal lung expansion or obstruction of air flow, peak flow measurement to detect narrowing of the airways, and diffusing capacity to assess the efficiency of gas absorption into the blood. Arterial blood gases (ABGs) are performed to assess the efficiency of gas exchange in the lungs by measuring oxygen and carbon dioxide (CO2) in arterial blood. CT scanning may also be useful for identifying lung nodules. It's generally accepted that the advent of high-resolution computed tomography (HRCT) has been the major diagnostic technique which is more sensitive than conventional radiography in detecting nodular lung parenchymal changes, progressive massive fibrosis, bulla, emphysema, pleural and hilar changes in silicosis. Qualitative and quantitative parameters on HRCT may also be used as indirect measures of functional impairment in silicosis. Therefore, HRCT has been widespread used. Sputum (phlegm) may be cultured to identify any causative organisms and to rule out tuberculosis.

Treatment

Damage to the lungs from silicosis is irreversible; there is no standard treatment other than reducing symptoms and treating complications. Lung tissue changes due to silicosis are often detected by a chest x-ray before they cause any symptoms. If dust exposure is stopped at this point, further progression of the disease can sometimes be prevented.

The disease is otherwise treated symptomatically. Appropriate drug therapy may be given to control symptoms; these may include drugs to reduce inflammation (anti-inflammatory), antibiotics to treat or prevent infections, and drugs to widen the airways in the lungs (bronchodilators). Sleeping in a semi-upright position may help reduce shortness of breath. Because smoking can aggravate the symptoms of silicosis and increase the risk of lung cancer, people with the condition who smoke cigarettes are urged to quit. In severe or advanced cases, a lung transplant may be required[12].

Prevention

Silicosis are preventable. In 1995, the ILO/WHO Global Program for the Elimination of Silicosis (GPES) was established by a joint ILO/WHO committee. GPES is encouraging and supporting countries with silica hazard to establish their national action programs to control silicosis.

It is recommended to assess the potential of silica exposure before a job begins, especially in the industries where silicosis cases were reported before[13]. Periodic respirable silica monitoring should be performed in all industries involving silica exposure. Currently enforced or suggested permissible exposure limits (PEL) for respirable silica are between 0.025 mg/m^3 and 0.35 mg/m^3 in different countries [14-16]. The current standards have not been confirmed by epidemiology studies to be fully protective. For example, the

quantitative risk assessments by NIOSH predicted excess lifetime risks of 19/1000 for lung cancer mortality, 54/1000 for lung disease other than cancer and 75/1000 for radiographic silicosis with exposure at the current US Occupational Safety and Health Authority standard for respirable cristobalite dust (about 0.05 mg/m3) over 45-year working lifetime[17].

For workers in workplaces with high dust levels, administrative measures can also be used to reduce exposure to silica dust e.g., by cutting short their working hours or job rotation. Exposure control at the worker level includes training and education on work practices, and personal protection. Personal protection equipment such as respirators is a good solution for short duration tasks. Respirators can be used in combination with engineering controls. However, they should only be considered as the last resort for routine full shift protection. They cannot be heavily relied upon because they may not be fully effective in workplaces with high dust concentrations. NIOSH recommends the use of half-facepiece particulate respirators with N95 or better filters for exposure to crystalline silica at concentrations less than or equal to 0.5mg/m^3 [18].

Regular medical evaluation may detect adverse health effects among exposed workers before disease reaches advance stages[13].Medical evaluation commonly includes respiratory questionnaires, physical examination, chest radiography and spirometry. There is no universal standard as to how frequent such evaluation should be performed, because the decision may be influenced by past and current respirable silica concentration, dust particulate characteristics and economic conditions. The WHO recommends routine evaluation every 2–5 years, ideally 'life-long' for workers exposed to silica dust[19]. American College of Occupational and Environmental Medicine (ACOEM) recommended evaluation at baseline and after 1 year, then 3-yearly for the first 10 years and 2-yearly thereafter when silicosis is the major concern and respirable silica levels are below 0.05 mg/m^3[20]. Biomarkers of early disease could potentially benefit prevention efforts and clinical diagnosis. While a number of biomarkers have shown some promising results, none of them have been validated fully for clinical use so far[21]. The occurrence of a new case of silicosis is a sentinel health event to prompt a thorough evaluation of silica exposure levels and control measures in workplace[22]. In addition to reporting new cases, occupational health doctors or hygienists should periodically analyze health records from all workers in an industry or plant and assess the effects of prevention activities.

3.3 Chronic obstructive pulmonary diseases

In the past few years a new definition has been presented by Global Initiative on Obstructive Lung Disease (GOLD) and by a Task Force of the American Thoracic Society (ATS) and the European Respiratory Society (ERS). Both GOLD and ATS/ERS state that "COPD is a disease state characterized by airflow limitation that is not fully reversible. The airflow obstruction is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles and gas." The ATS/ERS definition also state that COPD is both preventable and treatable and that COPD is systemic disease[23].

Epidemiology

According to WHO estimates, 80 million people have moderate to severe chronic obstructive pulmonary disease (COPD). More than 3 million people died of COPD in 2005,

which corresponds to 5% of all deaths globally. In 2002 COPD was the fifth leading cause of death, and expected to become the third leading cause of death globally by 2030, trailing only ischemic heart disease and cerebrovascular disease[24].

During 2000--2005, COPD was the underlying cause of death for 718,077 persons overall aged >25 years in the United States. In 2005, approximately one in 20 deaths in the United States had COPD as the underlying cause[25]. COPD has a prevalence of 4 to 10 percent in adults in populations in whom lung function has been measured. The National Health Interview Survey, an annual survey of approximately 40,000 United States households, has yielded an estimate of 10 million adults in the United States with a physician-based diagnosis of COPD. Other estimates, such as that from the Third National Health and Nutrition Examination Survey (NHANES III), that included spirometry along with questionnaires and a physical examination, done between 1988 and 1994, have yielded even more impressive prevalence figures. According to NHANES III, COPD affects 23.6 million adults in the United States, of whom 2.4 million have severe disease. Thus, approximately 10 percent of the United States adult population might be classified as having COPD, and of this group about 10 percent have advanced disease[23].

According to data published by the Chinese Ministry of Health, COPD ranks as the fourth leading cause of death in urban areas and third leading in rural areas [16]. The overall prevalence of COPD in China was 8.2% (95% CI, 7.9–8.6) according to GOLD diagnostic criteria. The crude prevalence of COPD was the highest in Chongqing and lowest in Shanghai among urban areas and was highest in Guangdong and lowest in Liaoning and Shangxi among rural areas. The COPD prevalence was significantly higher in rural areas compared with urban areas[26]. Both crude and age-adjusted COPD mortality rates have fluctuated but have displayed a decreasing trend from 1990 which is probably because of improved management of COPD, upgraded technologies, and awareness of the disease[24].

Risk factors

There are two types of risk factors for COPD: host factor and exposures (table 4).

ENVIRONMENT EXPOSURES	HOST FACTORS
Smoking	Genetic mutations
Occupational exposures	Airway hyperresponsiveness
Air pollution	Reduced lung growth
Childhood respiratory infections	
Low socioeconomic status	

Table 4. Common risk factors for COPD

Cigarette smoking is the major risk factor for COPD. However, relevant information from the literature published within the last years, either on general population samples or on workplaces, indicates that about 15% of all cases of COPD is work-related[27]. A 1989 study of black goldminers showed that the risk of chronic airflow limitation increases with duration of underground exposure and is an effect that is independent of the presence of silicosis. A study of white South African gold miners showed that the forced expiratory

volume in one second (FEV1), and the FEV1/FVC ratio, adjusted for age, height, and tobacco smoking, decreased with increasing cumulative respirable dust exposure, in both smokers and non-smokers. The average cumulative dust exposure attributables loss in lung function[28].

Pathology

COPD includes two main diseases: bronchitis - in which inflammation of the bronchi (tubes carrying air to and from the lung) both narrows them and causes chronic bronchial secretions. Chronic bronchitis is defined by the presence of cough and sputum production on most days for three or more months of the year for two or more consecutive years[29]; and emphysema - a permanent destructive enlargement of the airspaces within the lung without any accompanying fibrosis of the lung tissue. Asthma may also be included within the term COPD if there is some degree of chronic airway obstruction.

In COPD, inflammation causes direct destruction of lung tissues and also impairs defense mechanisms used to repair damaged tissues. This results in not only destruction of the lung parenchyma, but also mucus hypersecretion, and airway narrowing and fibrosis.

A wide range of inflammatory cells and mediators are involved in the pathogenesis of COPD, namely neutrophils, macrophages, and CD8+ T cells in different areas of the lung.

Overall, COPD pathogenesis can be summarized as resulting from a combination of genetic susceptibility combined with environmental exposures which lead to inflammatory processes that disrupt the balance of proteases and antiprotease. These abnormal inflammatory mechanisms result in tissue destruction, airway inflammation and remodeling, and ultimately airway limitation. These imbalances and the presence of inflammation may result in a "positive feedback loop," in which inflammation induces these imbalances, and the imbalances promote more inflammation. Once the inflammatory responses are set in motion, three types of damages to the lung occur: disruption of the alveolar walls, mucus hypersecretion contributing to airway obstruction, and fibrosis of the bronchioles.To support the inflammation mechanism further, a study shows there is a stepwise increase in alveolar inflammation has been found in surgical specimens from patients without COPD versus patients with mild or severe emphysema. As part of the peripheral airway system, the bronchioles are the major site of airway obstruction in COPD[30].

Diagnosis

The diagnosis of COPD, classification of its severity, and progression of the disease can be monitored with spirometry. A test that measures the forced expiratory volume in one second (FEV1), which is the greatest volume of air that can be breathed out in the first second of a large breath. Spirometry also measures the forced vital capacity (FVC), which is the greatest volume of air that can be breathed out in a whole large breath. Normally, at least 70% of the FVC comes out in the first second (i.e. the FEV1/FVC ratio is >70%). A ratio less than normal defines the patient as having COPD. More specifically, the diagnosis of COPD is made when the FEV1/FVC ratio is <70%. The GOLD criteria also

require that values are after bronchodilator medication has been given to make the diagnosis, and the NICE criteria also require FEV1%. According to the ERS criteria, it is FEV1% predicted that defines when a patient has COPD, that is, when FEV1% predicted is < 88% for men, or < 89% for women[31]. Once airflow obstruction is established, the severity of the disease is classified by the reduction of FEV 1 compared with a healthy reference population. Table 5 shows the widely used GOLD classification of COPD severity based on the FEV1.

STAGE	CHARACTERISTICS
I Mild COPD	FEV_1 80% predicted
II Moderate COPD	FEV_1 50% - 79% predicted
III Severe COPD	FEV_1 30% - 49% predicted
IV Very Severe COPD	FEV_1 < 30% predicted or < 50% predicted with room air Pao_2 < 60 mmHg (8.0KPa)

Table 5. Classification of COPD severity

On chest x-ray, the classic signs of COPD are overexpanded lung, a flattened diaphragm, increased retrosternal airspace, and bullae[32]. A high-resolution computed tomography scan of the chest may show the distribution of emphysema throughout the lungs and can also be useful to exclude other lung diseases.

Treatment

Directions about the management and prevention of work-related diseases [33-35], can be applied to COPD as well. Physicians should attempt to understand the patient's occupational exposure and whether he/she has been adequately trained in the dangers of these exposures and how to manage them. Removal of the respiratory irritants and substitution of non-toxic agents are the best approach because they eliminate the work-related COPD hazard. If substitution is not possible, ongoing maintenance of engineering controls, such as enclosure of the industrial process and improving work area ventilation, are useful. Administrative controls (e.g., transfer to another job or change in work practices), and personal protective equipment (e.g., masks or respirators) should be mentioned, although less effective in decreasing exposures to respiratory tract irritants.

3.4 Asthma

Occupational asthma is a lung disorder in which substances found in the workplace cause the airways of the lungs to swell and narrow, leading to attacks of wheezing, shortness of breath, chest tightness, and coughing.

Causes and prevalence

Though the actual rate of occurrence of occupational asthma is unknown, it is suspected to cause 2 - 20% of all asthma cases in industrialized nations. In the USA, OA is considered the most common occupational lung disease[33]. At present, over 400 workplace substances have been identified as having asthmagenic or allergenic properties. Their existence and

magnitude vary from region to region and the type of industry and can be as varied as wood dust (cedar, ebony, etc.), persulfates (Hairsprays), zinc or even seafood like prawns. In south-eastern Nigeria, a study was done to determine the magnitude of the problem among woodworkers exposed to high level of wood dust. Five hundred and ninety one woodworkers were selected using a stratified random sampling. The prevalence of occupational rhinitis was 78%, while that of asthma was 6.5%. As period of woodwork increased the prevalence of rhinitis and asthma increased (rhinitis: chi2 trend = 53.015, df = 1, P = 0.000; asthma, chi2 trend = 19.721, df = 1, P = 0.000). It demonstrates that the prevalence of rhinitis and asthma in woodworkers was high and significantly increased with years of working as a woodworker[34].

Occupations at risk

The riskiest occupations for asthma are: adhesive handlers (e.g. acrylate), animal handlers and veterinarians (animal proteins), bakers and millers (cereal grains), carpet makers (gums), electronics workers (soldering resin), forest workers, carpenters and cabinetmakers (wood dust), hairdressers (e.g. persulfate), health care workers (latex and chemicals such as glutaraldehyde), janitors and cleaning staff (e.g. chloramine-T), pharmaceutical workers (drugs, enzymes), seafood processors, shellac handlers (e.g. amines), solderers and refiners (metals), spray painters, insulation installers, plastics and foam industry workers (e.g. diisocyanates), textile workers (dyes) and users of plastics and epoxy resins (e.g. anhydrides)[35].

Mechanism

Even if the precise causative mechanism of occupational asthma is unknown, several mechanisms have been proposed, i.e. immunological, pharmacological and genetic mechanisms, and airway and neurogenic inflammation. More than one mechanism may be operative in occupational asthma. Whether various mechanisms are involved in occupational asthma induced by different agents is also unknown. An agent which causes asthma may be considered as "inducer" (i.e. causing reversible airway bronchoconstriction associated with long-lasting airway hyperresponsiveness to nonspecific and/or specific agents) or as "inciter" (i.e. triggering asthma attacks)[36]. Among the mechanisms proposed in the pathogenesis of occupational asthma, the immunological one plays a key role[35].

Diagnosis

Diagnosis of OA is a process and has to be done over a period of time. First, the patient's occupational and clinical history is taken and his symptoms are charted (Charting is usually done at the end of a typical work week and within 24 hours of the occurrence of symptoms in order to get objective information). Once this has been established, the following diagnostic methods are used:

1. Blood tests to look for antibodies to the substance;
2. Bronchial provocation test (test measuring reaction to the suspected allergen);
3. Chest x-ray;
4. Complete blood count;

5. Peak expiratory flow rate;
6. Pulmonary function tests.

Treatment

According to the Canadian Centre for Occupational Health and Safety (CCOHS), better education of workers, management, unions and medical professionals is the key to the prevention of OA. This will enable them to identify the risk factors and put in place preventive measures like masks or exposure limits, etc.

Avoiding exposure to the substance which causes you asthma is the best treatment. The best option is to change your jobs, or using a respiratory device to protect yourselves is an alternative option.

Anyone diagnosed with Asthma will have to undergo medical treatment. This is complementary to either removing or reducing the patient's exposure to the causal agents.

3.5 Pulmonary tuberculosis

Pulmonary tuberculosis, or TB, is a communicative disease caused by the bacterium *Mycobacterium tuberculosis* and, less frequently, *M.bovis*. Lesions most often occur in the lungs.

Species of *Mycobacterium* are characterized by unusual "acid fast" staining properties, slow growth, relative resistance to chemical disinfectants, and ability to survive for decades with cells in the infected animal. The few studies of TB as an occupational hazard suggest that physicians, nurses, medical laboratory workers, and miners are at increased risk of TB.

The symptoms of active TB of the lung are coughing, sometimes with sputum or blood, chest pains, weakness, weight loss, fever and night sweats. Tuberculosis is treatable with a six-month course of antibiotics.

Epidemiology

Roughly a third of the world's population has been infected with M. tuberculosis, and new infections occur at a rate of one per second. In 2007, an estimated 13.7 million people had active TB disease, with 9.3 million new cases and 1.8 million deaths; the annual incidence rate varied from 363 per 100,000 in Africa to 32 per 100,000 in the Americas[37]. In 2007, the country with the highest estimated incidence rate of TB was Swaziland, with 1200 cases per 100,000 people. India had the largest total incidence, with an estimated 2.0 million new cases[37]. According to the statistic data released by the Chinese Ministry of Health in July 2011, the recorded cases of pulmonary tuberculosis is 112647 which is the second in the recorded cases of notifiable disease, and 170 deaths.

However, few studies of TB incidence among various occupational have been reported. Therefore, only general and somewhat unsatisfactory comments can be made about TB as an occupational hazard.

Miners and others who work in poorly ventilated areas are more likely to be infected by a fellow worker who has TB than a person who works in a well-ventilated areas[38].

Silicotuberculosis

Both silica dust exposure and silicosis are risk factors for TB. Tuberculosis in a person with established silicosis is termed silicotuberculosis. The risk of developing TB increases with duration of exposure to silica dust even in the absence of silicosis. The presence of silicosis increases the risk of pulmonary TB approximately four fold, with the risk rising as radiological become more severe. This increased risk of TB associated with silicosis is lifelong, continuing after silica exposure ceases.

The presence of silicosis in the lungs can be modify the natural history of TB and may alter its radiological appearance. The interaction of TB and silicosis is very damaging to the lung, unless the TB is diagnosed and treated early.

Pathology

Infection with *Mycobacterium tuberculosis* results most commonly from infected aerosol exposure through the lungs or mucous membranes. In immunocompetent individuals, this usually produces a latent/dormant infection, only about 5% of these individuals later show evidence of clinical disease.

TB infection begins when the mycobacteria reach the pulmonary alveoli, where they invade and replicate within the endosomes of alveolar macrophages[39]. The primary site of infection in the lungs is generally located in either the upper part of the lower lobe, or the lower part of the upper lobe[39]. Bacteria are picked up by dendritic cells, which do not allow replication, although these cells can transport the bacilli to local (mediastinal) lymph nodes. Further spread is through the bloodstream to other tissues and organs where secondary TB lesions can develop in other parts of the lung (particularly the apex of the upper lobes), peripheral lymph nodes, kidneys, brain, and bone[40].

Workers exposed to silica are more likely to have TB because silica interferes with the function of the pulmonary macrophages[38].

Diagnosis and Treatment

The diagnosis of tuberculosis is confirmed by the growth of *Mycobacterium tuberculosis* from culture of sputum, CSF, urine, lymph nodes, or other infected tissue. If necessary, the patient should have a positive tuberculin shin test.

The goal of treatment is to cure the infection with drugs that fight the TB bacteria. Treatment of active pulmonary TB will always involve a combination of many drugs (usually four drugs). All of the drugs are continued until lab tests show which medicines work best. The most commonly used drugs include: Isoniazid, Rifampin, Pyrazinamide and Ethambutol.

Prevention

Transmission of TB can be prevented by the rapid identification and treatment of persons with disease and by the identification and treatment of those persons infected but not yet diseased.

As indicated above, chronic inhalation of dust particles has been linked for decades with lung diseases such as silicosis and silicotuberculosis. Also studies have suggested that dust particles may increase risk of lung cancer as well as some other diseases.

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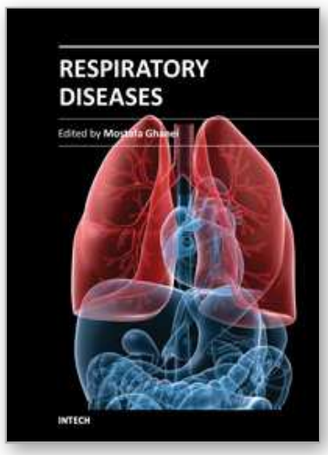

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Medicine is an ever-changing science. In this regard, Respiratory medicine is not an exception and has been evolving during recent years. As new research broadens our knowledge, advanced methods for diagnoses are better understood, providing genetic and underlying pathophysiology of diseases and new clinical experiences. Consequently, publications of new resources along with revisions of previous ones are required. The book Respiratory Diseases brings practical aspects of pulmonary diseases. It contains the result of years of experience through expert clinicians in this field from different scientific centers. The respiratory diseases are discussed according to epidemiology, pathology, diagnosis, treatment, and prognosis. It includes updated resources of the pathogenesis and some molecular aspects of the aforementioned diseases and is recommended reading for all clinicians and medical students, especially pulmonologists, to access highlighted respiratory diseases in this book.

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