INTRODUCTION

Work related pulmonary disorders occur because of repeated and long-term exposure to certain irritants on the job can lead to an array of lung diseases that may have lasting effects, even after exposure ceases.

Certain occupations, because of the nature of their location, work, and environment, are more at risk for occupational lung diseases than others. Contrary to a popular misconception, coal miners are not the only ones at risk for occupational lung diseases. For instance, working in a car garage or textile factory can expose a person to hazardous chemicals, dusts, and fibers that may lead to a lifetime of lung problems if not properly diagnosed and treated ***(American Lung Association).***

Occupational lung diseases are the primary cause of occupation-associated illness in the U.S. based on frequency, severity, and preventability of the illnesses. Most occupational lung diseases are caused by repeated, long-term exposure, but even a severe, single exposure to a hazardous agent can damage the lungs. Occupational lung diseases are preventable.

Smoking can increase both the severity of an occupational lung disease and the risk of lung cancer.

Occupational exposures, especially to dust and fumes, have been identified as potentially significant risk factors for COPD ***(Blanc and Toren, 2007).***

There are hundreds of studies looking at occupational dust and/or fume exposure. Excellent summaries of the literature were conducted by the American Thoracic Society in 2003 ***(Balmes and Becklake, 2003).***And again in 2010 ***(Eisner and Anthonisen, 2010),*** Detailed review of the epidemiologic and experimental evidence for general occupational exposures on COPD risk).

In both cases they concluded that occupational exposures were important risk factors for COPD. Diesel exhaust may be one of the fumes being referred to; however, this specificity is often not available.

Five recent studies have examined the effects of general occupational gas, dust, or fume exposure. In a US population based case-control study, two methods were used to assess exposure to vapors, gases, dust, and fumes: a question asking specifically if each job involved exposure to any of these four entities, and a job exposure matrix (JEM) based on an occupational history and expert review ***(Blanc and Eisner, 2009).***

Work-related asthma is the most commonly reported occupational lung disease in the United States ***(Petsonk et al., 2002).*** Occupational exposures can trigger asthma exacerbations in asthmatic workers or induce asthma in a previously healthy worker. Approximately 7.5% of all US adults have a diagnosis of asthma ***(CDC, 2002).*** In the US, there are an estimated 14.6 million work absence days due to asthma annually ***(Mannino et al., 2002).*** Of adults with incident asthma, an estimated 15% is attributable to workplace exposures ***(Blanc et al., 1999).***

Occupational asthma is defined as a form of asthma that is generally caused by immunological sensitisation to a (specific) agent inhaled at agents or synthetic chemicals. These occupational 'asthmogens' may be macromolecules of biological origin, metallic.

Interstitial lung diseases (ILDs) caused by exposure to agents encountered in the workplace (occupational ILD) are an important and preventable group of illnesses. Many different agents are reported to cause occupational ILD, some well described and others poorly characterized, and the list of causative agents continues to expand. Once thought of as the ''pneumoconiosis'' the list of known causes of occupational ILD extends well beyond coal, asbestos, and silica. There are also less common pneumoconioses caused by nonfibrous silicates (such as

talc, kaolin or mica) or other minerals. The clinical, radiologic, and pathologic presentations of occupational ILD are similar to non occupational variants because of the lung's limited repertoire of responses to injury ***(Beckett et al., 2000).***

Aim of the Work

The aim of this study was to determine the contribution of occupational exposures to the burden of pulmonary lung diseases among a railway workers (in Cairo).

LITERATURE REVIEW

Humans are subjected to risks from particles through different routes. Inhaling particles has a more intense and rapid effect, as particles can enter into the blood via the respiratory system. The surface area in the respiratory system is hundreds of times larger than the total area of the human skin. Also, the size and shape of different parts of the respiratory system are quite suitable to deposit particles.

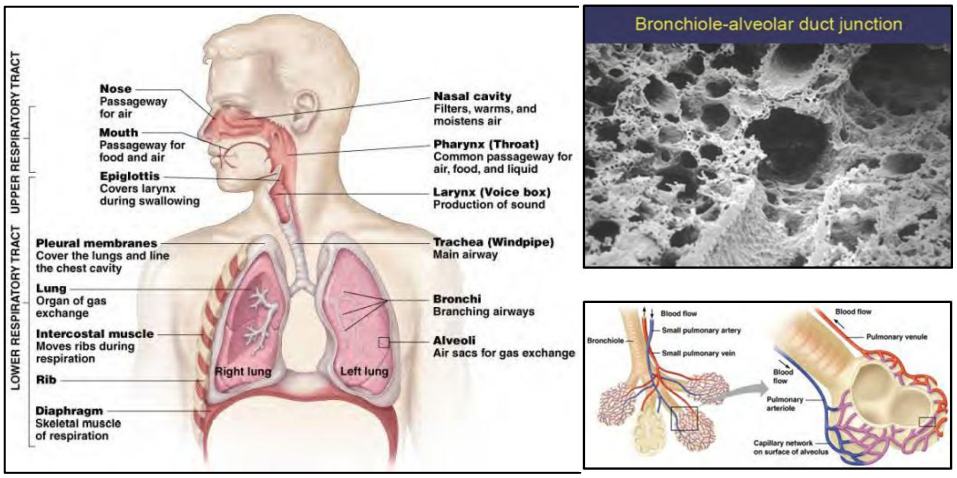

Figure (1): This figure shows different part of upper respiratory tract and lower respiratory tract. A real image of alveolar shape.

Due to increasing industrialisation and urbanisation, the prevalence of respiratory disorders has increased. Occupational respiratory disorders have been recognised for centuries ***(Bates, 1972).*** Industrial dust, smoke and fumes and poor working environment have been recognised as important causative factors in increasing the prevalence of chronic bronchitis among industrial workers. Age, smoking habit, duration and type of exposure at working

sites, nutritional and socio-economic status, etc. are contributory factors ***(Viswanathan et al., 1965; Roland et al., 1987).***

The Inhalable Fraction:

In the early 1990s, the ISO, ACGIH and CEN reached a general agreement to define the inhalable fraction, the thoracic fraction and the respirable fraction of particles. The inhalable fraction of airborne particles in ambient air, which can penetrate the respiratory system via the mouth or nose, was called the inhalable fraction. The inhalable fraction was defined based on equal to 100 μm in aerodynamic diameter. The thoracic fraction refers to the fraction of inhalable particles that pass the larynx and penetrate into the conducting airways. This fraction was defined based on equal to 10 μm in aerodynamic diameter. A portion of inhalable particles could reach the deepest part of the lungs and alveoli. This was referred to as the respirable fraction and was defined as equal to 4 μm in aerodynamic diameter. Particles penetrate and can deposit in different parts of the respiratory system depending on their size. **Tager (2005)** has summarised the particle size criteria for penetration and deposition in the respiratory system. His results are shown in Table 1.

Table (1): The respiratory tract penetration of particles in various sizes ***(Adopted from Tager, 2005)***

Particle size range(μm)	Level of penetration
≥11	Do not penetrate
7-11	Nasal passages
4.7-7	Pharynx
3.3-4.7	Trachea and primary bronchi (1^{st})
2.1-3.3	Secondary bronchi (2^{nd}- 7^{th})
1.1-2.1	Terminal bronchi (8^{th})
0.65-1.1	Bronchioles (9^{th} -23^{rd})
<0.65	Alveolar ducts (24^{th}-27^{th}) and alveoli

Particle sources in railway:

The railway workers exposed to a variety of irritants and smoke containing carbon particles, metal fumes from antimony, tin, lead, oxides of iron and nitrogen, aldehydes, sulfur dioxide, hydrogen chloride etc., which cause widespread bronchial narrowing by directly injuring the airway mucosa and causing inflammatory swelling and excessive secretions and /or by stimulating rapidly adapting irritant receptors ***(Bates, 1972).*** Table 2 shows the summary of particle sources in railway. Rail vehicles, different stationary processes, air circulation, and passenger and rail staff can be taken into account as the sources of particles.

Table (2): A summary of different particles sources in railway ***(Abbasi et al., 2011)***

Sources	Sub classification	Examples
Rail Vehicles	Exhaust (engine) emission	Diesel exhaust
	Non – exhaust (engine) emission	Wheel-rail contact Braking process Interaction of third rail and contact shoe Interaction of contact strip and overhead line Spraying sand to increase wheel-rail adhesion Erosion by air turbulence which is caused by a running rail vehicle (Piston effect)
Stationary process (Maintenance & construction)	Direct	Tunneling Rail cutting Rail welding Tamping process
	Indirect	Volatilization of oil and other lubricants Volatilization of cleaning material
Air circulation	Natural airflow	Moving and transferring particle emission from road transport Natural erosion of masonry structure
	Forced ventilation	Moving and transferring particle emission from road transport
Passengers and Rail staff	Human activities	Smoking in the platforms Smoking in the rail vehicle
	Others	Particle shed by passengers‘ clothes Degrading perishable materials and garbage

Diesel exhaust is a complex mixture of particulate matter (PM) and gases, and includes particles <1.0 μm diameter with mutagenic and polycyclic aromatic hydrocarbon (PAH) carcinogenic compounds adsorbed to a carbon core and ultrafine particles made up of condensed organics. Recent attention has focused specifically on diesel exhaust exposure as an important occupational risk factor. Individuals in a variety of occupations, including transportation (eg, railway workers) are routinely exposed

to diesel exhaust. There is no one accepted measure specific to diesel exhaust. However, work in an exposed job, as determined by industrial hygiene assessment or expert review; or self- reported exposure to occupational dust and fumes or specifically to diesel exhaust, have been used in the epidemiologic literature to assess associations with several types of lung disease ***(Kittelson et al., 2002).***

Welding is performed in railway operations wherever metal pieces need to be fused or where metal cracks must be repaired. The "trucks," bolsters, brake-beam hangers, and equalizer bars on railroad cars and various parts of locomotives frequently require welding. The welding process produces fumes. The composition and quantity of the fumes depend on the rods and the metal. Generally, however, the elements cadmium, iron, zinc, lead, chromium, nickel, manganese, and copper, as well as silicates, have been identified in the fumes. Ozone, nitrogen peroxide, and carbon monoxide are also found ***(Beaumont and Weiss, 1981).*** The fumes are known to cause or contribute to several types of lung disease, including cancer. The studies establishing an elevated risk of lung cancer among welders have not gone so far as to identify a specific carcinogen in welding fumes. This may be due to the wide range of substances that may be in the fumes. It should be remembered, however, that known lung carcinogens have been identified in these fumes ***(Beaumont and Weiss, 1981).***

Siderosis is an accumulation of iron particles in the lungs. The particles are inhaled in the form of iron oxide, which is produced from the melting metal an core of the welding rod. After being inhaled, most of the particles are absorbed by macrophages, the lungs' scavenger cells, and transported to the lymphatic system. Lung biopsies, however, have shown iron particles in the alveoli and respiratory bronchioles where gas exchange occurs. Siderosis can be seen in chest X-rays taken of the afflicted welder. The changes in the lungs are usually seen as nodular densities throughout the entire lung field, but the heaviest concentrations are seen in the middle third of the lungs. The X-ray changes resemble silicosis, a lung disease that is caused by inhaling silicon dioxide ***(Artfield and Ross, 1978).***

Silica, also known as silicon dioxide (SiO2), is formed from silicon and oxygen atoms. The compound silica is quite common in surrounding environment. Silicates comprise about 25% of known minerals. 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. Occupational exposure to crystalline silica can occur in any workplace situation where airborne dust, containing a proportion of crystalline silica, is generated. 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. Silicosis is the commonest occupational lung disease worldwide. Chronic silicosis will often develop between 15 to 45 years after first exposure. Pathological varieties of silicosis include simple (nodular) silicosis, progressive massive fibrosis, silicoproteinosis (acute silicosis) and diffuse interstitial fibrosis ***(WHO, 2007).***

Asbestos has been widely used by railway in many forms, including as insulation on steam and diesel locomotives. Railway repair shops and buildings frequently contained many types of asbestos products. Steam engines were covered from end to end with asbestos insulation. The engine rooms of diesel engines contained asbestos pipe covering on many parts of the engine. Many diesel engines had steam generators that were covered with asbestos that engineers were exposed to. It has been used in boiler insulation, steam generators, pipe covering, cabooses, gaskets, electrical panels, wallboard, plaster and brake shoes, just to name a few. As a result, many railway workers may have been exposed to this toxic and carcinogenic substance and not even known it. Some even brought it home on their work cloths exposing family members who handled their laundry.

Asbestos causes a form of fibrosis of the lungs known as Asbestosis. This disease involves a process where healthy lung tissue is replaced with scarred lung tissue. Asbestosis is what is known as a "latent disease." This means it does not develop at the time a person is exposed and can take as long as 30 to 50 years to show up. Symptoms normally manifest themselves in the form of shortness of breath or a dry cough that does not clear. Individuals may have lung damage due to asbestos and have no symptoms at all. Many workers mistakenly relate breathing changes to advanced age but they are in reality caused by lung damage from asbestos. Mesothelioma is a rare but fatal cancer that is caused by the intensive use of asbestos up to the late 1980s. Although some treatments are used to attempt to prolong the victim's life, none have been effective at curing this devastating disease. We have seen Mesothelioma cases in railroaders as young as age 58. Life expectancy of a mesothelioma patient is usually only 6-12 months from the date of diagnosis. The number of annual cases is predicted to increase steadily until 2020 in the old member states, and perhaps even later in the new member states ***(Hodgson et al., 2005).***

Work-related Asthma:

Asthma may be caused or made worse by work. People with work-related asthma often have more symptoms at work and improve away from work (on weekends and vacations). Many different exposures at work can cause occupational asthma. In addition, people who already have asthma may have work-exacerbated asthma due to asthma triggers at work, such as irritants, allergens, and temperature or humidity extremes ***(Petsonk, 2002).***

Definition:

The Global Initiative for Asthma (GINA) defines Asthma as:

Asthma is a heterogeneous disease, usually characterised by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breathing, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation ***(GINA, 2015).***

Occupational exposures can trigger asthma exacerbations in asthmatic workers or induce asthma in a previously healthy worker. Approximately 7.5% of all US adults have a diagnosis of asthma ***(CDC, 2004).*** In the US, there are an estimated 14.6 million work absence days due to asthma annually ***(Mannino et al., 2002).*** Of adults with incident asthma, an estimated 15% is attributable to workplace exposures ***(Blanc & Toren, 1999).***

Work-related asthma is often under recognized and misdiagnosed. Present practice often lacks the rigorous objective medical testing necessary to diagnose asthma and to document its relationship to the workplace ***(Rosenman et al., 1997).*** Medical history and physical exam lack both the sensitivity and specificity of diagnostic tests for occupational asthma ***(Malo et al., 1991),*** necessitating the use of objective testing for diagnosis. Delayed diagnosis can lead to a worsened prognosis ***(Paggiaro et al., 1994).*** Improvements in both diagnosis and management benefits the worker and employer such that years of productive work are not lost and any potential medical, legal or compensation issues are clarified.

Work-Related Asthma

1. Work aggravated asthma
2. Occupational asthma
    a. Occupational asthma with latency
    b. Occupational asthma without latency

Also known as Reactive Airways Dysfunction Syndrome (RADS) or irritant-induced asthma

Work-related asthma can be divided into two general groupings: Occupational asthma (OA) and work aggravated asthma (WAA). Occupational asthma is further subdivided into OA with latency and OA without latency. OA without latency is also termed Reactive Airways Dysfunction Syndrome or 'irritant-induced asthma ***(Bernstein, 2002).***