MODERN OCCUPATIONAL DISEASES DIAGNOSIS, EPIDEMIOLOGY, MANAGEMENT AND PREVENTION

Editor:
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Bentham Books

Modern Occupational Diseases Diagnosis, Epidemiology, Management and Prevention

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ISBN (Online): 978-981-5049-13-8

ISBN (Print): 978-981-5049-14-5

ISBN (Paperback): 978-981-5049-15-2

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First published in 2022

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FOREWORD

Fifty years ago the Occupational Safety and Health Act went into effect promising every worker the right to a safe job. Workplace hazards result in 5, 333 traumatic injuries and 95,000 occupational diseases (2019, U.S.) from chemical exposures, major fires, construction collapses, infectious disease outbreaks, workplace assaults, mine and stonework dust exposure, agricultural or forestry accidents, among others. There is no comprehensive surveillance system for occupational illnesses. The cost of injuries and illnesses is estimated at $250-330 billion per year. Latino and Black workers face an increased risk of injuries and illnesses compared to their white co-workers; "foreign-born" were two-thirds of the Latino workers killed on the job in 2019. In 2019, nearly 3.5 million workers across all industries had work-related injuries and illnesses that were reported by employers. Health care workers and meat-packing workers had hazardous exposure to the novel coronavirus as essential workers. Occupational diseases are preventable. It is imperative that we need to have new knowledge on occupational diseases and safety to control and prevent work-related diseases and fatality. For the working environment of our industrialized countries and worker' health and safety, a detailed and well referenced textbook is essential, and this book provides such information.

Dr. Ki Moon Bang has studied the epidemiology of occupational diseases for his 30-year career. He began at the Rocky Mountain Center for Occupational and Environmental Health at the University of Utah with an analysis of the pulmonary function of miners exposed to trona dust. He was involved in numerous studies of workers exposed to gases, asbestos, silica, coal mine dust, and toxic chemicals. He had the special experience of studying coal miners and other occupations at the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention in Morgantown, West Virginia. As professor of epidemiology and occupational health, he has taught epidemiology and occupational health to graduate and medical students at Howard University College of Medicine, West Virginia University School of Medicine, and Emory University Rollins School of Public Health. He has recruited the leading occupational medicine academicians to cover all of the salient topics: silicosis, obstructive lung disease, mesothelioma, asthma, musculoskeletal disorders, coal worker's pneumoconiosis, arsenic, and new work-related disorders emerging in the 21st century for this textbook.

This textbook will serve as very useful reference for occupational healthcare professionals including occupational medicine specialists, epidemiologists, industrial hygienists, and graduate students in public health. Of interest to a wide audience, this book also will enhance education of trainees in occupational disease and will serve as an up-to-date resource for practicing occupational medicine and other health professionals. This textbook will shed light on the current state of our understanding of occupational disease; it will look toward the future when reduced exposures and enforcement of rigorous policies will eliminate the burden of occupational diseases in the workplace. Lastly, occupational and environmental health professionals are on the front lines of communication of the importance to decarbonize the global economy to lessen the effects of global warming on workers and their families.

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PREFACE

Occupational disease is certainly one of the most important public health problems of the 21st century. Occupational disease still accounts for much preventable illnesses and injuries in the workplace. It is important to incorporate modern knowledge of disease epidemiology and cutting-edge diagnostic methods and treatment with the most recent developments in the management and prevention methods to better control work-related diseases and injuries. This book provides comprehensive coverage of all aspects of selected occupational diseases and injuries. It offers accurate, current information on the history, causes, diagnosis, management and prevention of occupational diseases.

This book is intended to serve as a useful guide for all those who are interested in occupational diseases. These include occupational medicine specialists, general practitioners, medical students, graduate students in public health, occupational health nurses, or colleagues and professionals in occupational health and safety — in other words, for all who have committed themselves to do the best practice for the occupational health of working people. The application of this book is not limited to the medical profession. It is equally needed by those responsible for control and prevention of occupational diseases, including managers at companies and government agencies responsible for the working environment of the industrialized country.

This book was assembled by a group of renowned scholars with special expertise in occupational diseases, addressing important occupational diseases including asbestosis, silicosis, work-related asthma, occupational cancer, and other diseases. This book was organized into 14 chapters. All chapters have been fully updated and enhanced to provide well-organized, practical, and relevant information. Each chapter highlights the latest research findings, the cutting-edge diagnostic methods, etiology, and the most recent developments in the management and prevention of occupational disease and injuries. Chapter 1 addresses the importance of occupational diseases in the 21st century. Chapter 2 discusses general methods for epidemiology and use of epidemiology in occupational diseases. Chapters 3-8 cover preventable occupational diseases including asbestosis, silicosis, coal worker's pneumoconiosis, occupational chronic obstructive pulmonary disease, work-related asthma, and acute respiratory infections. Chapters 9 and 10 cover occupational cancers including malignant mesothelioma and up-to-date review of the occurrence and causes of occupational cancer and prevention. Chapter 11 discusses the importance of causation of occupational diseases and the role of the International Agency for Research on Cancer and reviews silica dust as a known human carcinogen. Chapter 12 discusses health effects of arsenic. Chapter 13 discusses fatal injuries related to falls from elevated work sites and prevention strategy for safety in the construction industry. Chapter 14 reviews the etiology and risk factors, risk assessment, prevention, and surveillance of work-related musculoskeletal disorders. Case studies have been incorporated in these chapters to expand on the relevant issues. This book is therefore a detailed, comprehensive, and well reference text.

Preparing this book has been a challenging experience in my occupational epidemiology career. I was fortunate to have eminent authors in occupational diseases or safety who have contributed their chapters to the book. I wish to thank them for their great efforts on their chapters. I would like to acknowledge Dr. William Rom, Dr. David Christiani, Dr. Kenneth Rosenman, and Dr. John Parker for their support and encouragement. Finally, I would like to dedicate this book to my wife, Hanok who saw several months of weekends disappear into this effort and my two sons, Drs. Sam Bang and David Bang.

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CHAPTER 1

Occupational Disease in the 21st Century: COVID-19, Climate Change, and the Fourth Industrial Revolution

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Abstract: The beginning of the 21st century is experiencing tremendous social, political, and technological changes, combined with unprecedented climate change. Recent trends towards non-standard employment, growing economic and health disparities, and the decline of unions have served to undercut worker health and safety protections. Traditional workplace hazards remain important and preventable contributors to injuries and illness while new and/or newly recognized work factors are also becoming apparent. To meet the needs of the changing times, the traditional focus of occupational safety and health on industrial hazards is shifting toward a more holistic framework that incorporates other work stressors, underlying disparities, and the interactions with non-work factors. In this chapter, the major issues affecting workers today are examined through the two defining public health crises of our time, the COVID-19 pandemic and climate change. Initial lessons and observations from these global challenges inform the direction occupational safety and health will take to protect workers and prepare for an uncertain future.

Keywords: COVID-19, Climate change, Heat stress, Health disparities, Health, Occupational disease, Occupational safety, Work organization.

INTRODUCTION

Though frequently overlooked, work is a key social determinant of health, impacting health beyond traditionally recognized occupational illnesses and injuries. Factors such as job security, work environment and exposures, as well as job demands all contribute to an individual's overall health. These work factors change as technology and the economy change. As work factors evolve, so do the ways work impacts health.

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The beginning of the 21st century is a time of unprecedented social and technological change. Economic trends of the past fifty years towards more flexible and temporary employment and more fragmented industries and organizations have served to undercut several worker protections, just as the future of work itself is on the verge of major transformations. To meet the needs of workers during this period of growth, the traditional focus of occupational safety and health on industrial exposures is shifting toward a more holistic framework that incorporates non-industrial indoor air exposures, psychosocial stressors, underlying disparities, and interactions with non-work factors.

In this chapter, the major issues affecting workers today are examined through the two defining public health crises of our time: the COVID-19 pandemic and climate change. First, we review the impact of non-standard work and modern work organization on worker health, as well as how work contributes to health disparities overall. The COVID-19 pandemic is then presented with a focus on its effects on worker health, including the disparity in health outcomes between "essential" and "non-essential" workers. Next, we examine how climate change is impacting occupational safety and health, both in terms of worker health (*e.g.* the effects of heat stress and air pollution on vulnerable workers) as well as in hastening larger changes within the field in terms of future planning and preparedness.

Finally, we discuss how lessons learned from current occupational health challenges highlight priority areas for the field going forward. Rapid changes in science, technology, economic patterns, and the environment itself creates uncertainty and volatility, and carry the potential to threaten worker safety. Occupational medical surveillance and research, policy and regulation, training and education all require investment and updating to meet the challenges facing worker protection in the 21st century. Selected priority areas are briefly reviewed.

THE CHANGING ECONOMY/CHANGING STRUCTURE OF WORK

Bernardino Ramazzini (1633-1714), considered the founder of occupational medicine, observed that work and health are inextricably linked [1]. As society and technology evolve, the types and conditions of work change, with resultant impacts on health and disease. In 2016, world leaders met at the World Economic Forum in Davos, Switzerland, to discuss the emerging era of rapid change in work, science, and technology known as the Fourth Industrial Revolution [2].

Briefly, the three Industrial Revolutions that preceded the current period were defined by the advent of steam power and mechanization in the mid-1700s, the introduction of electric power and mass production in the latter half of the 1800s, and the incorporation of electronic devices and computers to automate production

at the end of the 1900s. Each of these revolutions fundamentally changed society and the world of work, and introduced new occupational health and safety challenges, as well as the resurgence of well-known hazards that previously had been better controlled. Table **1** summarizes major occupational health challenges of the 21st century.

Table 1. Major 21st century inter-related occupational health challenges.

Persistent, recurrent, newly recognized workplace hazards
Changing structure of work - non-standard work, precarious employment, loss of unions
Growing economic and health disparities
Inadequate occupational safety and health regulations and/or inadequate enforcement of existing regulations
Inadequate funding for occupational health education and research
Omission of data on work, workplace hazards in healthcare administrative databases, electronic medical records
Climate change (rising temperatures, extreme weather events, droughts, floods)

The Fourth Industrial Revolution is unique due to the speed and breadth of changes and the development of new emerging technologies such as artificial intelligence, robotics, the internet of things, autonomous vehicles, 3D printing, nanotechnology, biotechnology, materials science, energy storage, and quantum computing [3]. The number and scope of these disruptive advancements make it difficult to predict how the Fourth Industrial Revolution will unfold and what the precise impact will be on work and society.

The anticipated changes in occupational medicine that will accompany the Fourth Revolution will likely impact work structures that expanded following WWII. The initial decades following WWII were ones of general economic stability and prosperity through the Western world, and it was in this context that the standard employment relationship developed [4]. Defined as permanent, full-time, year-round employment with comprehensive job-related benefits as well as strong employment-related rights and protections, including the right to collective representation, this standard employment relationship continued into the mid-1970s. Starting in the 1970s, multiple factors, including the economic "oil shocks", worldwide recession, political shifts towards less government regulation and less union representation, began the trends towards reduced employment protections, the increased use of outsourcing and temporary workers and greater income disparities.

The working environment has continued to move away from the traditional post-WWII employment structure in recent years, attenuated by the Great Recession of

CHAPTER 2

Occupational Epidemiology

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Abstract: The development of occupational epidemiology has been steadily accelerating, regardingmethodology and the number of studies being conducted. This chapter reviews the general application of occupational epidemiology and illustrates some of the various studies reported in the literature to assist in the practical application of the epidemiologic approach. The epidemiologist uses analytic tools such as the case-control method to examine the complexity of variables to understand workplace exposures to disease. The analytic methods used to examine epidemiologic data have become more sophisticated over the past several decades as the focus of occupational epidemiology has shifted to the detection of early health effects associated with low-level exposures. In the future, epidemiologists need to collaborate more effectively with toxicologists, environmental scientists and biostatisticians to improve the collection of exposure data and develop more precise methods for estimating exposure that account for metabolism and excretion of toxic materials. Continuous improvement of epidemiologic analytic methods and prevention of occupational disease and surveillance are needed.

Keywords: Analytic Study, Case-Control Study, Cohort Study, Cross-Sectional Study, Descriptive Study, Ecologic Study, Meta-Analysis, Molecular Epidemiologic Study, Occupational epidemiology, Surveillance.

INTRODUCTION

Occupational epidemiology is the study of the distribution and determinants of work-related disease and injuries in the workplace. Occupational epidemiologic studies have become an important and integral part of occupational medicine. Occupational epidemiology has grown rapidly since the late 1970s. Recent developments in occupational epidemiology include the integration of epidemiologic courses into occupational medicine training, methodologic development, and access to computers and statistical software packages. A primary goal of occupational epidemiology is to determine what kinds of expo-

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sures in the workplace are associated with certain diseases or injuries to develop the best prevention strategy for occupational disease.

Employment in professional and business services was 9.9 million in the United States in 2020, according to the report from the Bureau of Labor Statistics [1]. In 2019, 2.8 million illnesses and injuries were reported in private industry workplaces, resulting in a rate of 2.8 cases for every 100 full-time workers [2] compared to 6.7 million illnesses and injuries reported in 1993 [3]. These changes in occupational workforces and health statistics have recently led to decrease in the United States.

The occupational epidemiologic approach to a particular disease is intended to identify high risk subgroup within the population and determine the effectiveness of the subsequent preventive measures. In principle, the epidemiologic study of work-related disease does not differ from other aspects of epidemiologic research. This chapter reviews the general application of occupational epidemiology and illustrates some studies reported in the literature. It is intended to assist in the practical application of the epidemiologic approach.

USE OF EPIDEMIOLOGY IN OCCUPATIONAL DISEASE

It was the science of epidemiology that demonstrated the serious health risks associated with asbestos, radiation, coal dust, and other occupational exposures. The rapid growth in the use of potentially hazardous materials has been accompanied by numerous observations of serious health effects in humans due to occupational exposures. An example of early occupational epidemiology came from the observations in 1879 of an increased occurrence of lung cancer among miners in Schneeberg [4]. Some decades later, an excess of bladder cancer among German aniline workers was reported [5]. An example of modern epidemiologic study is workers at plastic manufacturing plants who handled the gas vinyl chloride and were discovered in 1974 to be developed hepatic angiosarcoma, unusual cancer [6]. At about the same time, infertility and an extreme decrease in spermatogenesis were discovered in workers at a California plant producing dibromochloropropane [7]. Some advances have occurred to improve the work environment and avoid dangerously uncontrolled industrial plant emissions. However, there are still many reported occupational diseases and injuries that could be prevented in the workplace.

Epidemiologic principles can be readily applied to occupational studies. Occupational studies are designed according to the study objectives. In general, there are three major types of epidemiologic: descriptive, analytic, and experimental [8]. Most studies in occupational epidemiology are analytic rather than descriptive.

Descriptive Study

It is used to characterize person, place, and time. What is the age, sex, race, occupation, industry, socioeconomic status, and other personal characteristics of people who get a particular disease? Where does the disease occur? Does temporal variation or seasonal fluctuation exist?

Analytic Studies

It determines the etiologic factors associated with a disease by calculating estimates of risk. What exposures do people with the disease have in common? What is the degree of the increased risk by exposure? Analytic methods are available to control for known confounders, but unknown ones are free to distort risk estimates.

Experimental Study

It involves a search for strategies to alter the natural history of the disease. Examples are intervention trials to reduce risk factors, screening studies aimed at identifying the early stages of the disease, and clinical trials of different treatment modalities to improve prognosis. The experimental study has the advantage of randomization, a procedure that distributes both known and unknown between the test and control groups [9].

Occupational epidemiologic studies include observational studies and it is the most common one. The major types of observational studies in epidemiology are cohort, case-control, and cross-sectional designs. Other types of study designs are ecologic studies, meta-analysis, occupational surveillance, and recently developed molecular epidemiologic studies.

Occupational epidemiology has been used for testing a specific hypothesis. The specific hypothesis means in principle that “a causes b” can be tested through either a follow-up or a case-control study [10]. If the disease is rare, a case-control study is appropriate. If the exposure is rare, a follow-up study is more efficient. If both the exposure and the disease are common, both designs are feasible, and the decision depends on the availability of data, possibility of tracing records, financial resources, length of study periods, and other factors.

For establishing causal relationships, several criteria have been proposed to evaluate whether a positive association in epidemiologic studies indicates causality. The most important criteria are strength, consistency, biologic gradient, biologic plausibility, and temporality [11]. The strength of an association is the magnitude of the relative risk in the exposed group compared to that of the control

CHAPTER 3

Asbestosis

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Abstract: Asbestos, due to its unique physical properties and abundance, was widely used in commercial applications at the beginning of the 20th century. By the 1930s, reports of respiratory illnesses in workers with occupational exposure to asbestos began to surface. Inhalation of asbestos leads to a localized inflammatory response that attempts to clear inhaled asbestos fibers. This inflammatory response and retained asbestos fibers are central to the pathogenesis of asbestosis-related pulmonary disease. Asbestos causes a range of pulmonary diseases ranging from benign, incidental pleural abnormalities to progressive, fatal pulmonary fibrosis to malignant neoplasms of the lung and pleura. The benign manifestations of asbestos exposure, the focus of this chapter, can be grouped into pleural and parenchymal diseases. Asbestosis is a fibrotic, parenchymal disease caused by asbestos exposure. After several decades from initial asbestos exposure, patients develop dyspnea, exercise intolerance, and hypoxia with restrictive physiology on pulmonary function testing similar to other interstitial lung diseases. The most common pleural manifestation is pleural plaques, which are localized areas of pleural fibrosis that are often found incidentally. While normally asymptomatic, they are a marker of asbestos exposure. Other pleural manifestations tend to be symptomatic and include diffuse pleural thickening and acute benign pleural effusion. This chapter discusses pathogenesis, clinical presentation, radiographic and physiologic manifestations, and management of benign asbestos lung diseases.

Keywords: Asbestosis, Pleural Plague, Pleural Thickening, Pneumoconiosis, Round Atelectasis.

BACKGROUND

Asbestos, a generic term for fibrous magnesium silicates, is uniquely lightweight, durable, abundant and inexpensive to mine. It is resistant to fire, water, and corrosive acids. The fibers can be woven into a cloth, blended with other materials, or spun into a thread. These unique properties lead to widespread commercial applications with the second industrial revolution and two world wars. By the middle of the last century, it was used in buildings and ships for

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insulation, fireproofing, and electrical systems, in cars and planes as part of brakes, clutches, and gaskets, and household products such as ironing board covers, electric blankets, and coffee pots.

Despites numerous uses and industrial benefits, the harms of asbestos use were as early as 1924 when William Cooke published a case report in the British Medical Journal on an asbestos factory worker who died of pulmonary fibrosis and tuberculosis. Over the next few years, additional case reports of pulmonary fibrosis in asbestos workers were published and the disease was named asbestosis by Thomas Oliver [1]. In 1930, a British medical inspector, Edward Merewether, concluded that occupational asbestos dust exposure, especially to high concentrations for a prolonged time, was an occupational risk to asbestos workers and could result in pulmonary fibrosis that is disabling and fatal. He went on to conclude that dust control measures could prevent the disease [2]. This led to national regulations limiting asbestos dust exposure in the United Kingdom in 1931.

While reports of lung cancer in people exposed to asbestos date back to 1935, the association between asbestos exposure and lung cancer was not recognized until 1955 with the publication of Richard Doll's work on lung cancer death in asbestos workers. Doll found that men who worked for at least 20 years in asbestos dust-containing environments had 10 times the rates of death from lung cancer compared to what is expected in the general population [3].

Simultaneously in the 1950s, J C Wagner, Christopher Sleggs, and Paul Marchand from South Africa noticed cases of malignant pleural mesothelioma in patients from the crocidolite mining district of Griqualand West. At this time, mesothelioma was a controversial diagnosis that often required a full autopsy to rule out malignant disease elsewhere and was not known to be associated with asbestos exposure. In a seminal 1960 paper, Wagner and his colleagues reported a series of 33 patients with diffuse pleural mesothelioma who had exposure to crocidolite asbestos [4]. Over the next several years, there were additional reports of mesothelioma in patients exposed to asbestos. Initially, the reports were confined to workers with high occupational levels of exposure, but by 1970 it was understood that low levels of asbestos exposure could cause mesothelioma. This discovery as well as an appreciation for how widespread asbestos exposure was outside of the work place helped lead to the passage of the Occupational Safety and Health Act of 1970 in America. Prior to 1970, America had not regulated asbestos, and its use in the country was widespread.

Today despite knowledge of the health risks of the asbestos described above, asbestos mining and usage continues mainly in the Asia Pacific region and

developing countries. Approximately 90% of asbestos is used in cement building materials [5]. According to the WHO, 125 million people are exposed to asbestos at the workplace [5]. As of 2020, the largest asbestos mining countries were Russia, Kazakhstan, and China [6].

BENIGN PARENCHYMAL MANIFESTATIONS

Asbestosis

Asbestosis is defined clinically as a syndrome of interstitial pneumonitis and parenchymal fibrosis due to exposure to asbestos fibers that are associated with dyspnea, bibasilar rales, and restrictive physiology on pulmonary function testing. In the section below, important clinical and pathologic aspects of asbestosis are discussed.

Pathology

In the early stage of disease, lesions are characterized by fibrosis and cellular reaction in the first-order respiratory bronchioles. Distinct lesions of fibrosis, referred to as peribronchiolar fibrosis, are found within the walls of the respiratory bronchioles and can involve adjacent septi. The cellular reaction is macrophage predominant and peribronchiolar. Together, these peribronchiolar processes can narrow and obstruct the airway lumen. More distal, type II alveolar epithelial cell proliferation is enhanced and buds of loose connective tissue are found within the alveoli. In the interstitium, lymphocyte collections and smooth muscle proliferation can also be seen. As the disease progresses, second and third-order respiratory bronchioles become affected as fibrosis becomes more widespread. With advanced disease, the fibrosis becomes diffuse and leads to architectural distortion and honeycombing of the lung. The pathologic changes in asbestosis are similar to many other fibrotic interstitial lung diseases and can only be differentiated by the presence of asbestos bodies and uncoated asbestos fibers in the lung [7, 8].

Pathogenesis

Asbestos fiber deposition occurs in the small airways at bifurcations and in the respiratory bronchioles and alveoli. Once deposited, fibers are taken up by type I alveolar epithelial cells and move to the interstitium. This causes an accumulation of alveolar macrophages within the peribronchiolar interstitium, alveolar ducts, and alveolar spaces, referred to as macrophage alveolitis. This macrophage alveolitis allows for clearance of asbestos fibers without fibrosis of the surrounding lung. If the macrophage alveolitis fails to clear all asbestos fibers, persistent inflammation and fibrosis occur. As such, the degree of fibrosis relates

CHAPTER 4

Silicosis

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Abstract: Silica refers to the compound SiO_2, which can be found as amorphous or in a variety of crystalline forms. The most common crystalline form is quartz, one of the most abundant minerals in the Earth's crust. Other less common forms found in nature include cristobalite and tridymite. Small particle aerosols, including crystalline silica, can be generated by many activities carried out in industries such as construction, manufacturing, and mining. Respirable crystalline silica (RCS) refers to particles small enough to remain suspended in air and be inhaled into the deep lung. Inhaling sufficient amounts of RCS causes a fibrosing interstitial lung disease called silicosis. It also causes or is a risk factor for a spectrum of diseases, including lung cancer, chronic obstructive pulmonary disease, chronic renal disease, increased susceptibility to tuberculosis, and various autoimmune diseases. These adverse outcomes can be prevented by recognizing potentially hazardous conditions and taking steps to control RCS exposures. Unfortunately, despite being preventable, silicosis continues to occur in many settings, including recent outbreaks in emerging settings. In the USA, recently-promulgated regulations by the Occupational Safety and Health Administration (OSHA) provide a comprehensive set of interventions to control RCS exposures and provide RCS-exposed workers with health surveillance for early detection of silicosis. Current treatment options for those with silicosis are limited and primarily consist of avoiding further exposure and symptomatic management, so primary prevention is extremely important.

Keywords: Acute silicosis, Autoimmune disease, Chest radiograph, Chronic renal disease, Chronic obstructive pulmonary disease, Cristobalite, Epidemiology, Exposure, High-resolution chest computed tomography scan, Lung cancer, Lung transplantation, Occupational Safety and Health Administration, Progressive massive fibrosis, Quartz, Respirable, Silica, Silicosis, Surveillance, Tridymite, Tuberculosis.

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INTRODUCTION

Pulmonary silicosis is an interstitial lung disease caused by inhaling particles of inorganic mineral dust containing crystalline silica into the lungs [1 - 3]. Inhaling sufficient amounts of crystalline silica causes or is a risk factor for a spectrum of diseases, including lung cancer, chronic obstructive pulmonary disease (COPD), chronic renal disease, increased susceptibility to tuberculosis (TB), and various autoimmune diseases [3 - 6]. These adverse outcomes can be prevented by recognizing potentially hazardous conditions and taking steps to control respirable crystalline silica (RCS) exposures. Unfortunately, despite being preventable, silicosis continues to occur in many settings, including recent outbreaks in emerging settings. In the USA, recently-promulgated regulations by the Occupational Safety and Health Administration (OSHA) provide a comprehensive set of interventions to control RCS exposures and to provide RCS exposed workers with health surveillance for early detection of silicosis that can help to identify remediable gaps in prevention [5]. Current treatment options for those with silicosis are limited and primarily consist of avoiding further exposure and symptomatic management, so primary prevention is extremely important.

RESPIRABLE CRYSTALLINE SILICA (RCS)

Silica is a term describing the compound SiO_2. It exists in amorphous and crystalline forms. In amorphous forms, SIO_2 molecules are not arranged in a fixed pattern or structure. In crystalline forms, the SiO_2 molecules are arranged in fixed patterns. Different polymorphs of crystalline silica have molecules of SIO_2 arranged into different structures. There are a number of polymorphs or forms of crystalline silica, but the 3 major ones found in natural settings are quartz, cristobalite, and tridymite. Quartz is found in many types of rocks and is one of the most abundant minerals in the Earth's crust. Cristobalite and tridymite are far less abundant in natural settings but can be found, typically in volcanic rock [4, 7]. However, cristobalite and tridymite can be generated and create hazards in industrial settings where amorphous silica or quartz are exposed to intense heat. An important example occurs in foundries where castings are made by pouring molten metal into amorphous silica-containing clay molds.

The term respirable refers to particle size. Respirable particles are small enough to remain suspended in air and be inhaled into the deep lung. They are conventionally considered to have a diameter of 4 μm or less. OSHA method ID-142, which is used to assess exposure to RCS, uses a sampler that captures about 50% of particles of 3.5 μM and none that are 10 μM or larger [8].

EXPOSURES TO RCS

Because crystalline silica, in particular quartz, is so widely distributed in nature, many types of activities can cause exposure to RCS by aerosolizing dust from natural sources such as rocks or soil or from silica-containing manmade materials such as bricks or concrete. Examples include cutting, drilling, grinding, tunneling, demolition, mining and quarrying. Exposures can also occur from engaging in activities such as abrasive blasting with sand. Because industrial processes that involve heating materials that contain quartz or amorphous silica can generate tridymite and cristobalite, they can also be associated with RCS exposures. For example, cristobalite exposures can occur in foundries when clay molds that have been in contact with molten metal are removed from metal castings.

Worldwide, millions of workers are exposed to RCS. Estimates include 2 million in Brazil exposed at least 30% of the time [9], 3.2 million in the European Union [10], and 10 million at risk in India [9]. OSHA has provided an extensive list of occupations and industries in the USA where exposure to RCS occurs and has estimated that about 2.3 million employees are at risk for exposure and thus are covered by OSHA's RCS standard [5].

A 1998 U.S. publication evaluating RCS air sampling data collected by OSHA during regulatory compliance inspection noted that some of the highest RCS dust concentrations occurred in construction (masonry, heavy construction, and painting), iron and steel foundries (casting), and in metal services (sandblasting, grinding, or buffing of metal parts). The industry that was found to have the highest percentage of workers (6%) exposed to at least the National Institute for Occupational Safety and Health (NIOSH) recommended exposure limit (REL) was the cut stone and stone products industry [11].

New sources of RCS exposure in work settings continue to emerge [12]. Recent examples include denim sandblasting [13], artificial stone countertop fabrication [14], and natural gas extraction by hydraulic fracturing [15]. In addition, RCS exposure related to contemporary mining methods has played an important role in pneumoconiosis among U.S. coal miners [16].

PATHOGENESIS OF SILICOSIS

RCS-induced toxicity is complex and basic mechanisms of cellular damage have been addressed in recent excellent reviews [17 - 19]. Briefly, cellular targets after intrapulmonary deposition of RCS particles likely include alveolar macrophages and respiratory epithelial cells. RCS can be directly cytotoxic to cells because of its ability to generate free radicals in an aqueous solution, resulting in lipid peroxidation and DNA damage [20]. Freshly-fractured RCS particles with

CHAPTER 5

Coal Workers Pneumoconiosis

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Abstract: Coal Worker's Pneumoconiosis (CWP) was thought to be an archaic disease, but after an initial decline because of the Coal Mine Health and Safety Act in 1969, there has been a resurgence of this disease in the 21st century. For centuries, miners have been exposed to varied types and degrees of coal mine dust. Lung diseases in coal miners are caused by the inhalation, retention, and tissue reaction to the mixed constituents of this dust, which include carbon, silica, and silicates. Respirable dust particles of less than 5 microns are deposited in the proximal and distal airways and the smaller particles are deposited in the alveoli. The tissue reaction to these particles results in a variety of pathologic lesions, including coal macules, silicotic nodules, mixed dust pneumoconiosis, interstitial fibrosis, progressive massive fibrosis, bronchitis, and emphysema. These disorders are recognized primarily through occupational exposure history and characteristic radiographic imaging. With a latency of approximately 20 years, cumulative lifetime exposures appear to be most predictive of the disease severity. Prevention of these diseases should be the primary focus of the industry, the workforce, and the public health agencies. In the US, federal programs of screening and surveillance are in place and active. The treatment of these disorders as with other chronic respiratory conditions, is focused on vaccinations against respiratory infection, bronchodilator therapy when indicated, supplemental oxygen therapy when required, pulmonary rehabilitation programs, smoking cessation, vigilant observation for chronic respiratory infections, and if necessary, lung transplantation should be considered as the last resort.

Keywords: Antifibrotics, Black Lung, CMDLD, CWP, ILO, Lung transplantation, NIOSH, PMF, Pneumoconiosis, Prevention, Pulmonary rehabilitation, Resurgence, Sea coal, Vaccinations.

INTRODUCTION

Historical Background

Coal miners have been exposed to the adverse health effects of coal mine dust since the 1600s. Coal has been used as a heat source since the Bronze Age 3000 to

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4000 years ago. The Romans in Britain burned coal before 400 CE. The first reference to coal mining in Great Britain or elsewhere was made in A.D. 852. Before underground coal was mined, people collected the surface outcroppings. It is said that London's first coal arrived by sea in 1228, it was named sea-coal because it was delivered by the waves, it was not until the advent of the mechanical pump and the introduction of ventilation that underground mining became possible [1]. Historically the Chinese are credited with the first underground mining. Underground mining grew quickly as proper methods for ventilating fresh air and toxic gases from the mines, as well as effectively pumping water from the mines, were put into place. As of 1866, in Scotland, there were 472 coal mines, 41000 miners and 12 million tons of coal produced yearly.

In the United States, coal mining became a major industry in the 1830s and the earliest mines were established in Eastern Pennsylvania, Western Maryland and what is now West Virginia. In the latter part of the nineteenth century underground bituminous mines came into existence in these regions. It was in the late nineteenth and early twentieth century that large underground coal deposits were found in Illinois, Indiana, Utah, and Colorado. Back then, the growing steel industry in Pittsburgh and Cleveland generated a huge demand for coal leading to the import of labor from Europe. Historically, lung disease in miners has been referred to as miners' asthma, phthisis, anthracosis, and miners' black lung in Scotland. Shortly after the turn of the nineteenth century, Laennec talked about "melanosis of lung" and separated it into four categories: melanotic masses enclosed by cysts, melanotic masses lying encysted, melanosis in which the black matter infiltrates into the lung and melanosis with deposits on the lung surface. In 1831, the first report of ' black lungs' attributed to employment in coal mines was published [2]. He described a 59-year-old man who had been employed as a miner for 10 or 12 years and was hospitalized for generalized anasarca. He soon died from progressive heart failure. The lungs were examined, and a picture consistent with progressive massive fibrosis (PMF) with cavitation of these large lesions on a background of simple coal workers' pneumoconiosis (CWP) was described. In 1884 Marshall [3] concluded that: 'The true explanation of the origin of this disease in coal miners seems to be, that it is in consequence of the inhalation of fine coal dust, and its deposition in the substance of the lung. That coal may float through the air in particles sufficiently fine to be inhaled without immediate irritation and that it is thus inhaled is a matter of common observation'. For the rest of the 19th century, there was considerable controversy and disagreement regarding the impact on coal dust inhalation on the survival of miners. A series of studies in the 1960s showed that the prevalence of CWP varied by region of the country, the type (rank)of coal mined [4], and the duration and intensity of exposure [5, 6]. Overall data showed that the mortality of coal miners only minimally exceeded the mortality of the healthiest men, such as farmers or

agricultural laborers. This lack of coaldust effect on survival was thought to be attributable to better mine ventilation and lesser exposure to coal dust. Early in the 20th century, the understanding of lung disease in miners again changed. In 1915 Collis [7] theorized that coal dust did not produce pneumoconiosis but was a marker that showed the path by which dust enters and travels in the lung. He attributed dust disease in miners to silica inhalation (associated with working of thinner seams with likely silica contamination) and reported the disease that developed to be silicosis. This view failed to consider increasing mechanization in the mines and deepening of the mines, with the resulting increase in coal dust generation. The adverse effects of these factors more than compensated for the positive effect of increased mine ventilation [1, 8].

Coal

Coal's formation is credited to the accumulation of vegetable matter covered by a sedimentary rock that is subjected to pressure and temperature over the ages. This causes the physical and chemical properties of the matter to change. The matter dries becomes warmer. and loses oxygen content, all the while increasing the relative carbon content [9]. The first step in this conversion of vegetable matter to coal is the formation of peat a moist, spongy material. Approximately 100-ft (30-m) accumulation of peat compresses to form a 1 ft-wide coal seam. In the simplest terms, coal is comprised of moisture, pure coal, and mineral matter [10]. The terms brown coal and black coal refer to coals of low and high rank, respectively. Rank describes the extent of change from vegetation to mineral-free coal, it is directly related to the percent of the carbon in coal. The type of coal relates to the plant materials which form the coal. The grade refers to the purity of the coal - or the amount of inorganic material (including ash and sulfur) released in the burning of coal. Other important descriptive terms include quantitative measurement of coal moisture (the percentage of water that is contained in coal and released with moderate heat) and the amount of volatile matter (that percentage of substances, mainly gases and coal tar, lost when a sample is well heated). In the 21st century, coal is not the primary energy source, with petroleum and natural gas used increasingly as a substitute. In 1900, coal dominated the world for supplying its energy needs, and 94% of the world's energy requirements were met by it. By 1967, coal utilization had fallen to 40% and by 1978, coal was only responsible for 31% of fuel use [9]. In the US, electric power generation accounts for the utilization of more than 80% of the coal produced each year [11]. At present, coal continues to remain one of the important energy sources across the globe see (Fig. **1**). In 2019, 7921 million tons (Mt) of coal were produced worldwide [12], primarily mined by two methods: surface or 'opencast' mining, and underground mining. It is because of its ongoing utility and continued mining we see its effect on the health of the miners.

CHAPTER 6

Occupational Chronic Obstructive Pulmonary Disease

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Abstract: Chronic Obstructive Pulmonary Disease (COPD) is a clinical syndrome defined as non- or incompletely reversible airflow obstruction associated with persistent lower respiratory symptoms such as dyspnea, cough, and excessive sputum production. The present definition probably includes more than one disease. Despite being largely preventable, COPD is often a disabling disease with accelerated longitudinal lung function loss and systemic comorbidities and is presently the third leading cause of death and one of the most important health care expenditures worldwide. While most cases are unquestionably related to tobacco smoking, it has long been suspected and is also now fairly well established that occupational and environmental exposures, as well as a variety of other factors, contribute to its causation. Most recent estimates place the fraction of COPD causation by occupational exposures at ~15% and ~30% overall and among nonsmokers, respectively. The disease occurs in workers exposed to vapors, gases, dust, and fumes at their longest-held job, and in several occupations that include miners, agricultural, cotton/textile, and construction workers, food, drink, and tobacco processors, and bus drivers, among others. There is evidence of an additive effect of occupational exposures and cigarette smoking. There is presently no evidence that treatments differ from those in widely accepted guidelines, except for the occupational interventions for primary, secondary, and tertiary prevention discussed throughout this book. This is in large part due to treatment trials having required a fairly heavy smoking history and disregarded patients' occupations. Improved appraisal of the etiological contribution of occupational exposures should lead to progress towards disease elimination.

Keywords: Chronic obstructive pulmonary disease, Chronic bronchitis, Disability, Emphysema, Mineral dust, Occupational exposures, Population attributable fraction, Spirometry.

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a clinical syndrome presently defined as non- or incompletely reversible airflow obstruction associated with persistent lower respiratory symptoms such as dyspnea, cough, and excessive sputum production [1, 2].

Despite being largely preventable, COPD remains a leading cause of mortality, morbidity, disability, and health care expenditures worldwide. Having caused 3.23 million deaths (about 5.8% of the total) throughout the world in 2019, COPD is the third leading cause of death, overall and in the block of 110 upper- and low-middle income economies, with an increasing trend observed from 2010 to 2019 [3]. In the United States, 6.4% of the US population self-report a diagnosis of COPD [4], and it is the fourth leading cause of death [5]. In 2010, total costs in the United States attributable to COPD and its sequelae were estimated at US$32.1 billion (projected to increase to US$49.0 billion in 2020), and total absenteeism costs at US$3.9 billion, adding to a total burden of COPD-attributable costs of US$36 billion [6]. As expected, there is considerable variation but a similar burden across countries [7]. Estimates from the Medical Expenditure Panel Survey data between 2011 and 2015, placed at 5 billion US dollars the cost of inpatient treatment of COPD among US workers aged at least 18 years who were employed at any time during the survey year [8]. Based on an estimate of workplace exposures as contributing up to 50% of COPD cases, that report stated that government workers, police officers, firefighters, correctional officers, and administrative assistants resulted in the highest per-person cost, with the caveat that this may be the result of greater access to medical care in these occupations. Finally, the study also concluded that uninsured workers spent less and raised the possibility of forgoing care as a cause for this finding [9].

There are extensive and very comprehensive reviews on the pathophysiology, clinical presentation, diagnosis, and treatment of COPD, to which the reader is referred for a more in-depth review [1, 2, 10, 11]. While summarizing some of those topics for context, this chapter will focus on occupational COPD, reviewing the historical evolution of this concept and the epidemiological data supporting such association.

PATHOGENESIS AND PATHOPHYSIOLOGY

As the pathophysiology is diverse and still remains to be fully elucidated, COPD is quite likely to encompass more than one disease entity [10, 12]. Not surprisingly, COPD has received multiple and sometimes contradictory definitions over the years, and the need to unify the terminology across multiple countries has become increasingly obvious since the 1950s [13]. More recently,

the Global Initiative for Chronic Obstructive Lung Disease (GOLD) was established in 1998 and tasked an international multidisciplinary panel with defining the disease and providing guidelines for its diagnosis and treatment. Since the initial report in 2001, GOLD has periodically updated its reports, posted them on the Internet (https://goldcopd.org/), and disseminated them in the medical literature [1, 14].

COPD is presently understood as a disease with three main pathophysiological components: (1) chronic bronchitis, classically defined by the presence of productive cough for at least 3 months of the year and at least 2 years [15]. Chronic bronchitis has been attributed primarily to proximal airway inflammation and mucous gland hyperplasia, as indicated by both surgical pathology [16], and quantitative chest CT measurements [19]; (2) emphysema, defined as the enlargement of the airways distal to the respiratory bronchioles. Emphysema is primarily a pathological finding [18], which translates into a quantitative chest CT reduction in lung attenuation [19, 20]; and (3) small airway disease, consisting of inflammatory involvement of the distal airways, which, in turn, are traditionally defined as those with an internal diameter of less than 2 mm [21 - 23]. Small airway disease is also a largely surgical pathology definition, difficult to ascertain by lung physiology or chest imaging. The presence and relative proportion of these three components vary from one individual to another. As COPD is consistently associated with a number of comorbid diseases, it has even been hypothesized to represent a systemic chronic inflammatory condition [24].

COPD is a heterogeneous syndrome caused by distinct pathophysiological processes including innate and adaptive T_H1 type immune response to toxicants, microbes, or autoimmunity; persistent T_H2 inflammation; protease-antiprotease imbalance or antiprotease deficiency [25]; oxidative stress, and other mechanisms affecting the airways, alveoli, or both, resulting in diverse clinical presentations, responses to treatment, and patterns or progression [2].

Inextricably and unquestionably causally associated with tobacco smoking, it has been clear for several decades that not all smokers develop COPD [26]. A sizable minority of COPD cases occur in lifetime nonsmokers. Investigations have sought to understand the role played by other environmental (*e.g.*, indoor air pollution from biomass burning [27] or second-hand smoke, or outdoor air pollution [28]) and occupational causative factors, as well as likely genetically-determined susceptibility factors [25, 29], factors or events affecting lung development in early life [30], dysanapsis (airway caliber underdevelopment in relation to that of the lung alveoli) [31, 32], accelerated aging or senescence [33], or lung diseases throughout life, including asthma [34], tuberculosis [35 - 37], rheumatoid arthritis [38, 39], human immunodeficiency virus [40], and viral and other infections.

CHAPTER 7

Work-Related Asthma

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Abstract: Work-related asthma is a common condition that affects men and women who work in a wide range of industries. Adults can develop new-onset asthma after a latency period of months to years of exposure where they become immunologically sensitized or after an acute exposure that causes bronchial wall damage. Adults can also experience the aggravation of pre-existing asthma that may have developed in childhood but becomes worse after exposure at work to respiratory irritants. Exposure to over 300 substances., including chemicals, metals, insects, animals, plants, or fungi, have been identified that cause new-onset asthma. There are thousands of substances, as well as cold air or stress, that can aggravate pre-existing asthma. Guidelines have been developed for prompt recognition and diagnosis of work-related asthma because ongoing exposure after the onset of asthma symptoms is associated with a poorer prognosis. Both primary and secondary prevention have a role in reducing the occurrence and morbidity of the condition. The field has continued to advance with the recognition of an increased number of etiological agents, an understanding of the pathophysiology, an understanding of the prognosis and factors associated with a better prognosis, and the initiation of work on the interaction with genetic variability. Awareness of the disease by clinicians and the promulgation of allowable air standards by regulatory agencies that protect against the development of asthma at work will be essential to reduce the burden of this disease.

Keywords: Asthma, Diagnostic guidelines, Epidemiology, Prevention, Prognosis.

INTRODUCTION

Work-related asthma (WRA) is caused by exposure to a variety of substances in the workplace. For primary prevention, to reduce the occurrence of the disease, it is important to identify the various substances that cause WRA, and control exposure to these substances. For secondary prevention, to improve the prognosis of those who, despite primary prevention, develop WRA, it is important to conduct workplace medical monitoring for early diagnosis. This chapter will describe the pathophysiology, diagnosis, epidemiology, hazard control, diagnosis,

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and treatment of WRA. The chapter begins with a case report of a patient who died from WRA.

Case Report

A man in his mid-forties with a 10 year history of asthma had an acute asthma attack while at work. Normally he worked with an assistant, but on the morning of his acute asthma attack, he had sent his helper to do other work while he installed a spray-on truck bed liner in a van. His helper returned 20 minutes later to find the work completed, the equipment turned off and the individual was gasping for breath on his knees outside the building. The helper immediately took the individual to an urgent care clinic. The individual collapsed at the door of the urgent care clinic. A nurse at the clinic began cardiopulmonary resuscitation (CPR). An ambulance arrived nine minutes later. Despite CPR and transport to a nearby hospital, he was pronounced dead 46 minutes later. Wheezing was noted on auscultation while he was being bagged during resuscitation. On microscopic examination of his lungs at autopsy, there was mucus in his airways with numerous eosinophils in his airways and his mucosa. The bronchial basement membrane was thickened with hyperplasia of the mucus glands. There were aggregates of pigment laden macrophages in the peribronchial alveoli. He was also noted to have diffuse pan lobular moderate to severe pulmonary emphysema, and diffuse and heavy anthracosis. His heart showed coronary arteriosclerosis, calcification, myocardial hypertrophy, and perivascular fibrosis. The medical examiners cause of death was "Asthmatic reaction due to inhalation of chemicals." The deceased had a history of allergies. He had never been hospitalized for respiratory problems. He had had three medical visits in the year prior to his death: 1) a laceration of his hand; 2) low back pain; and 3) symptoms of shortness of breath and the medical record from that last encounter, when he had symptoms of shortness of breath, indicated he inhaled "chemicals" two days prior while working with a bed liner and wasn't wearing a breathing pack. He was exposed for ten minutes and, within 10-15 minutes, couldn't catch his breath. He received a nebulizer treatment and was given 40 mg of prednisone and antibiotics for seven days, cough syrup with codeine and an albuterol inhaler. His regular asthma medication consisted only of an albuterol inhaler. He had never had pulmonary function testing. He had smoked two packs of cigarettes per day but was tapering down. The deceased had worked as the manager at a small auto detailing facility, which included himself and two employees. The shop did vehicle detailing, rustproofing and spray-on truck bed liners. The deceased was the only individual who did the spray-on truck bed lining. After he died, his coworkers mentioned that the manager had had difficulty breathing after previous spray liner applications. The deceased used a positive pressure respirator with supplied fresh air while spaying on the bedliner [1].

The spray-on bedliner was a two component system of methylene diphenyl diisocyanate (MDI) and polyol. The components were mixed during the spray process. When mixed, polyurethane was formed, which provided abrasion resistance, insulation, and a watertight seal to the bed of the truck. The work was performed inside because no moisture can contact the bed liner during application. Isocyanates have historically been the most common cause of work-related asthma. This case report illustrates the most severe consequence of a healthcare provider not adequately addressing a patient's increased respiratory symptoms associated with work. The response by the health care provider after the visit for respiratory problems prior to the patient's death was limited to acute treatment. No action was suggested to the patient on managing future exposure. The results of studies on individuals who have developed asthma from exposure to isocyanates conclude that those individuals should no longer be exposed to isocyanates [2]. At the minimum, the manager should not have applied the bedliner and probably should no have worked at the facility.

Work-related asthma encompasses both new onset asthma and aggravation of pre-existing asthma from work exposures/conditions (Fig. **1**).

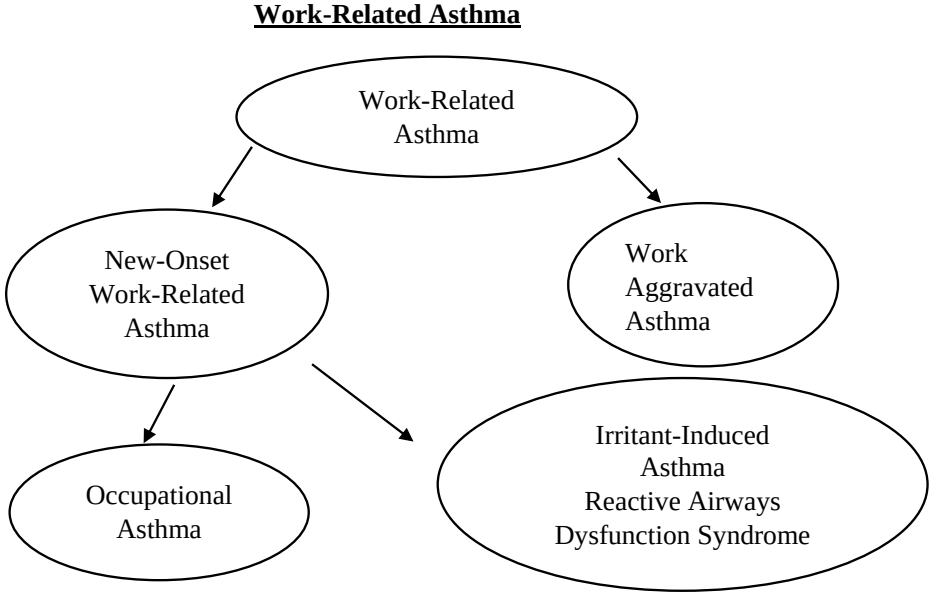

Fig. (1). Classification of work-related asthma (WRA).

New onset asthma can be caused by exposure to an irritant or a substance that causes sensitization. Over 300 substances have been identified where exposure at

CHAPTER 8

Acute Respiratory Infections: Diagnosis, Epidemiology, Management, and Prevention

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Abstract: Acute respiratory infections (ARI) are infectious diseases of the respiratory tract caused by viruses, bacteria, and atypical bacteria. They range in severity and even mild cases may cause a significant reduction in workplace productivity. ARIs commonly occur in outbreaks and disproportionally impact workers in occupations where workers are in close proximity to co-workers, members of the public, or where they reside in densely populated housing. High-risk workers include those in the healthcare sector, protective service, food and meat processing, service, and education industries. A person can become infected by inhaling virus-laden aerosols, having virus-contaminated sprayborne drops impinge on exposed mucous membranes, and touching contaminated surfaces followed by self-inoculation. More than one transfer process may be involved in the transmission, and the dominant route may differ for different causative agents, environments, and activity patterns. Preventing ARI transmission in the workplace must be holistic in approach and begin with anticipation and recognition of potential risks, reinforced by the continuous evaluation and implementation of control strategies. Control measures should be layered and multiple routes of transmission should be addressed. Controls should be adapted to the specific workplace and the ARI to prevent pathogen introduction, rapidly detect cases, and promptly eliminate exposure. Prevention and control can be accomplished by promoting vaccination, improving ventilation and air cleaning, providing paid sick leave, flexible working conditions, and work-from-home options. Promoting hand sanitation and providing appropriate personal protective equipment are important but never sufficient in isolation. Occupational health professionals should partner with workplace engineers and human resource departments to design effective programs.

Keywords: Acute respiratory infections, Coronaviruses, COVID-19, Employee Health Services, Infectious Diseases, Influenza, MERS, occupational Health, Respiratory Syncytial Virus, Rhinovirus, SARS, SARS-CoV-2, Workplace.

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INTRODUCTION

Acute respiratory infection (ARI), defined as the presence of two or more respiratory symptoms, including fever, cough, sore throat, nasal congestion, or rhinorrhea, include a broad range of infections including influenza like illnesses (ILI). ARIs are primarily upper respiratory infections of the nasopharynx and upper intra-thoracic airways caused by a variety of organisms, including viruses, bacteria, and atypical bacteria. Mild to moderately symptomatic infections are most frequent and often referred to as the "common cold." However, many of the agents responsible for ARIs can cause illnesses with a wide range of severity from completely asymptomatic to incapacitating generalized symptoms, asthma exacerbation, and involvement of other organs, including pneumonia, gastrointestinal symptoms, and rarely myopericarditis. ARIs are common throughout the year, with epidemics predominantly in the winter months in temperate regions [1] during interpandemic periods. A major global health concern is the potential for influenza and coronaviruses to cause pandemics with high attack and mortality rates.

ARIs have been dominant contributors to the overall burden of disease globally [2] but have declined somewhat, as have other infectious diseases in the past two decades [3]. However, they remain part of the top ten causes of years lost to death (YDL) and disability-adjusted life years (DALY's) for children under 10 and adults over 75 [3]. Furthermore, the socioeconomic risk factors for all diseases such as household air pollution, unsafe water, sanitation, and handwashing are still quite prevalent even though they are improving globally [4]. These risk factors can leave communities vulnerable to infectious diseases, especially ARIs.

ILI, characterized as fever with sore throat or cough, is the focus of syndromic surveillance with virologic surveillance focused on influenza virus detection. Annual influenza epidemics have resulted in between 12, 000 and 60, 000 deaths between 2010 and 2016 in the U.S. and disproportionately impact workers who frequently interact with the public, who are in proximity with their co-workers [5], and who reside in densely populated communities and housing. A recent review of the contribution of the workplace to the overall burden of lung disease found that the workplace contributed to 10% of community-acquired pneumonia, which was the only acute respiratory infection included in the review [6]. The COVID-19 pandemic caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) caused an acute illness of the respiratory tract that spread rapidly throughout the world after its first identification in December 2019 in Wuhan, China.

The COVID-19 pandemic demonstrated the dire consequences of inadequate pandemic preparedness coupled with a fragmented public health system that was slow to acknowledge and address the central role of aerosols in transmission [7] and integrate lessons learned from modeling emergence of previous coronaviruses [8] to predict the severity and scope of the pandemic. This resulted in significant morbidity and mortality, particularly for informal and precariously employed individuals [9, 10], and huge economic disruptions globally. In our view, ARI prevention demands cooperation, and the full integration, of employee occupational health service providers, urgent or primary care providers, and the public health system from the local to the national and international levels. It also requires a better understanding of the routes of transmission and an ability to incorporate recent scientific advances into public health policy.

EPIDEMIOLOGY

Broadly, ARIs can be caused by various organisms, including bacterial or viral pathogens. Bacterial pathogens are less common causes of ARIs and can include *Streptococcus pneumonia, Mycoplasma pneumonia, Haemophilus influenza*, *Chlamydophila pneumonia, Coxiella burnetii* and *Legionella pneumophila*, especially in immunocompromised individuals [11, 12]. Common viral ARI causative agents include rhinoviruses, respiratory syncytial virus (RSV), influenza virus, parainfluenza virus, human metapneumovirus (HMPV), adenovirus, and coronaviruses [13]. Though most viral ARIs are self-limiting, their clinical syndromes can vary widely in severity. Large prospective studies [14, 15] have shown that viruses contribute more than other infectious agents to morbidity and mortality due to ARIs. Furthermore, viral ARI may lead to more severe respiratory complications such as exacerbations of asthma or chronic obstructive pulmonary disease (COPD), bacterial sinusitis, or pneumonia. ARIs can occur in epidemics and can spread very rapidly within populations or across the globe, as in the case of influenza pandemics of 1918, 1957, 1968, and 2009 and the COVID-19 pandemic that began in 2019.

Of the known viral agents of ARI, ribonucleic acid (RNA) viruses particularly pose a great challenge because of their biological diversity and their ability to adapt rapidly. The evolution of RNA viruses into new variants or strains is difficult to anticipate through current technology as they often adapt quickly to evade vaccine and innate immune protection. This is because, during replication, the process of copying RNA in most RNA viruses does not involve proofreading as deoxyribonucleic acid (DNA) replication does, and so RNA viruses have a propensity to accumulate errors leading to a cloud of viral variants facilitating rapid evolution. This also increases the likelihood of RNA viruses crossing inter-

CHAPTER 9

Malignant Mesothelioma

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Abstract: Malignant mesothelioma often develops because of exposure to asbestos. The global incidence and mortality of mesothelioma remain unclear because many developing countries do not report it or the data they report are unreliable. Asbestos usage increased over centuries and reached its peak in 1970s. By the 90s the use of asbestos had been banned or tight regulated in many western countries, including the U.S. Asbestos usage continues to increase exponentially in developing countries. In addition to occupational asbestos exposure, environmental exposure to asbestos and other mineral fibers can cause mesothelioma. Asbestos carcinogenesis is largely caused by the chronic inflammatory process driven by the extracellular release of HMGB1 by mesothelial cells and macrophages. In addition, germline mutations of the BRCA-associated protein 1(BAP1) gene and less frequently of other tumor suppressor genes and DNA repair genes can cause or predispose to mesothelioma. Germline BAP1 mutant carriers develop additional malignancies during their lifetime. Fortunately, several of these malignancies, including mesothelioma, are less aggressive than their sporadic counterparts. BAP1 is also the gene most frequently mutated somatically in sporadic mesothelioma underscoring the critical role of this gene in suppressing mesothelioma growth. The preventive measure aimed at reducing occupational exposure to asbestos and environmental exposure to various carcinogenic fiber effectively reduces the incidence of mesothelioma. Carriers of germline BAP1 mutations benefit from close follow-up for early cancer detection. Because they may be susceptible to asbestos carcinogenesis, they should avoid trades or living in areas where carcinogenic fibers may be present.

Keywords: Asbestos, BRCA associated protein-1(BAP1), Carcinogenesis, Chemotherapy, Environmental exposure, High mobility group protein B1(HMGB1), Immunohistochemistry, Imaging study, Immunotherapy, Malignant mesothelioma, Naturally occurring asbestos, Occupational exposure, Radiation therapy, Surgery.

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EPIDEMIOLOGY

Incidence in the United States and Worldwide

Diffuse malignant mesothelioma (MM) is rare and almost always fatal disease [1]. Approximately 3,200 incident cases are registered, and 2,500 deaths are reported annually in the United States [2, 3]. The incident rate differs in states from 1 case per 100,000 persons to 2-3 cases per 100,000 persons depending on the presence of asbestos industries [2]. It has been estimated that mesothelioma may cause about 28,000 to 43,000 deaths/year worldwide: however, these estimates are imprecise because data from most developing countries are either lacking or unreliable [4 - 7]. Median survival for asbestos-induced mesothelioma is 6-18 months, depending on histology [3]. However, an increasing number of mesotheliomas is being diagnosed in carriers of germline mutations of BAP1 or of other tumor suppressor genes: these mesotheliomas are rarely associated with asbestos exposure and are much less aggressive: patients have a median survival of 5-7 years, about 20% of them survive > 10 years, and some >20 years [1]. Mesothelioma is usually resistant to chemotherapy and radiation therapy, and it cannot be entirely excised surgically; therefore, treatment options are limited [8 - 10].

The countries with the highest age-standardized incidence rates of malignant mesothelioma are Australia, Belgium, and Great Britain, with incidence rates are of about 30 cases per million [11]. The second group of countries with a high incident rate of around 10-20 cases per million includes several European countries, France, Germany, Italy, Netherland, and Scandinavia.

A strong correlation between mesothelioma incidence and asbestos consumption has been observed in many studies [4, 5, 12]. Moreover, in some areas with large shipyards or large asbestos-cement factories, the mortality rates of mesothelioma are higher than in areas without these industries. From 1995 to 2003, countries with the highest consumption of asbestos worldwide were Russia, China, Thailand, Brazil, India, Kazakhstan, Iran, and Ukraine [1, 13]. However, data about the incidence of mesothelioma in these countries are not available [1]. In addition, many countries, especially developing countries that still use asbestos, do not report the incidence of malignant mesothelioma deaths [14].

History and Current Situation of Asbestos Usage and Occupational Exposure

Asbestos, a commercial name used to identify 6 different types of mineral fibers, is a Greek word meaning “unquenchable”. Of the six mineral fibers, crocidolite, amosite, tremolite, anthophyllite, and actinolite belong to the amphibole group and chrysotile belongs to the serpentine group [1].

Asbestos, due to its characteristic of strength, flexibility and resistance to heat, has been widely used for thousands of commercial products such as roofing, flooring, thermal and electrical insulation, cement pipe, gaskets, brake pads, coating and caulking compounds, textiles *etc* [15, 16]. In Italy, the commercial use of asbestos began in the early 1800s to manufacture fabrics. Soon after asbestos was broadly used for fire-resistant roofing, wall materials, and steam pipes worldwide. In the United States, commercial asbestos production in 15 states has been recorded since 1900. In 1910, the United States became the country consuming the highest amount of asbestos in the world: as much as 55% of asbestos was used in the United States [17]. The usage of asbestos kept growing until mid-1970s in the U.S. and worldwide as the automotive and construction industries kept developing products containing asbestos. These products included brake shoes and clutches, cement, flooring, packings and gaskets, and thermal and electrical insulation [13, 18].

Although in the past decades, more than 50 countries have banned the use of asbestos, the usage of asbestos has increased in Asia and in countries in the Middle East countries. Approximately 2 million metric tons of asbestos are used yearly in the world [13, 17]. In the United States, the use of asbestos has not been entirely banned, but the amount of consumption is less than 0.1% of peak consumption in 1973 [18]. There is a long latency of 30-50 years from the time of asbestos exposure to patients developing mesothelioma.

Chrysotile is the most commonly used type of asbestos used commercially. It has been estimated that chrysotile accounts for over 90% of all asbestos commercially used globally, followed by crocidolite, amosite, and anthophyllite. Chrysotile is the only type of asbestos still used in the United States. Chrysotile is a hydrated magnesium silicate, the chemical formula is $Mg_3Si_2O_5(OH)_4$. Several studies indicate that chrysotile, compared to other asbestos fiber types, is less carcinogenic [19 - 21]. However, the World Health Organization (WHO), US Environmental Protection Agency (EPA), the International Agency for Research on Cancer (IARC), and the National Toxicology Program (NTP) identify all asbestos types as human carcinogens [15].

(Fig. **1**) shows asbestos bodies present in a biopsy stained with an iron (Prussian blue stain) of a MM patient who had worked in a large asbestos manufacturer in Italy.

CHAPTER 10

Occupational Cancer

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Abstract: This chapter provides an up-to-date review of the occurrence and causes of occupational cancer based on epidemiologic studies and discusses the epidemiology of occupational cancer, the characteristics, research priorities, prevention, and surveillance. Epidemiologic methods have been very successful in documenting cancer risks associated with agents. Epidemiologic data is useful when an exposure-response relationship can be demonstrated. Examples of agents for which epidemiologic studies provide evidence of an exposure-response relationship include benzene and myelogenous leukemia. Vinyl chloride causes liver cancer which is an example of associations between single agents and rare histologic types of cancer. It is more difficult to conduct epidemiologic studies to identify cancer risks associated with complex mixtures. Studies of diesel exhaust, lung cancer, and metal machining oils are cited as having employed advanced industrial hygiene and epidemiologic methods for studies of complex mixtures. At present, less than 20 known occupational carcinogenic agents have been evaluated based on studies in humans and animals by the International Agent for Research on Cancer. Furthermore, exciting developments in epidemiologic and animal studies will contribute to identifying additional carcinogenic agents in the workplace. New biologic markers of exposure and cancer-related outcomes must be identified and integrated into epidemiologic studies. Because epidemiologic data regarding the carcinogenicity of many exposures are not available, research methods to evaluate and improve the predictive value of animal and *in vitro* systems must be developed. A complete understanding of occupational cancer trends will be required further to research occupational cancer risks and means of prevention.

Keywords: Cancer Surveillance, Cancer Research, Carcinogens, Dose-response, Histologic Types, Latency Period, Morbidity, Mortality, Medical Screening, Occupational Cancer, Prevention, Threshold Limit Values.

INTRODUCTION

Cancer is a major cause of morbidity and mortality worldwide. Occupational carcinogens were among the first human carcinogens to be identified, and the causal carcinogens between occupational exposures and some human cancers

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have been established. Occupational carcinogens include chemical substances, physical agents, and microbiological agents that are present in the workplace. Occupational carcinogens may cause a significant increase in a particular type of cancer in the exposed working population. Although the number of known occupational carcinogens to humans is limited so far, the prevention of occupational cancer and the protection of workers against exposure to carcinogens is needed. This chapter provides an up-to-date review of the occurrence and causes of occupational cancer based on human epidemiologic studies, occupational cancer characteristics, research priorities, prevention, and cancer surveillance.

MAGNITUDE OF OCCUPATIONAL CANCER

Cancer is the second leading cause of death in the United States [1]. The American Cancer Society estimated 1,898,160 new cancer cases in 2021 based on data from the Surveillance, Epidemiology and End Results (SEER) program of the National Cancer Institute [2].

The proportion of occupational cancer to exposure is difficult to estimate precisely. Adequate data that allow calculation of the number of exposed individuals or levels of exposure are not available. Two possible approaches to calculating the proportion of cancer due to occupation [3] include the following:

1. Calculation based on exposure to occupational factors. In this case, it is necessary to know (a) the total population exposed to the hazard, (b) the duration and level of exposure, (c) confounding variables, and (d) the increased relative risk associated with exposure at different doses levels.

2. Calculation based on evaluating the cause of cancer at each site. It is probably more appropriate to examine cancers at each site and calculate the proportion due to defined or suspected factors. From each calculation, it may be possible to establish the upper limits of the proportion of cancers that could be predominantly due to occupational factors.

As the investigation in the United Kingdom showed that only 6% of cancers might be occupationally related to exposures in the workplaces, while 88% are due to other lifestyle factors [3]. In 1991, Vineis and Simonato evaluated the proportion of cancers attributable to occupation [4]. The proportion ranged from 1-5% when considering only exposure to asbestos and 40% for subjects exposed to ionizing radiation. Three-quarters of occupational cancers among men are found in the lungs [5]. Between 13-27% of lung cancers are attributable to some form of occupational exposure and in particular, asbestos exposure [6]. Up to 25% of bladder cancer cases in the general population can be attributed to occupational

exposure [4, 7, 8]. These estimates were determined by the prevalence of exposed individuals within the general population and the the proportion of individuals exposed within the group under consideration. Doll and Peto have estimated that about 4% of all cancer deaths can be attributed to occupational exposures [5]. Leigh and coworkers estimated that 6-10% of all cancers have occupational origin [9]. In the United States, this would present 113,890-189,816 new cases and 36,514-60,857deaths due to occupational exposure in 2021 (Tables **1** and **2**).

Table 1. Estimated incidence of occupational cancer, United States, 2021.

Sites	Estimated Number of New Cancer*	% New Cancer Due to Occupation**	Estimated Number of New Cancer Due to Occupation
Lung	235,760	10	23,576
Prostate	248,530	1	2,485
Skin	115,320	6	6,919
Bladder	83,730	7	5,861
Leukemia	61,090	7	4,276
All sites	1,898,160	6-10	113,890-189,816

*Estimated new cancer cases by the American Cancer Society, 2021 [2].
**Estimated percentage by Richard Doll and Richard Peto,1981 [5].

Table 2. Estimated number of deaths due to occupational cancer, United States, 2021.

Sites	Estimated Number of Cancer Deaths*	% Due to Occupation**	Estimated Number of Deaths due to Occupation
Lung	131.880	10	13,188
Prostate	11,540	1	1,154
Skin	34,130	6	2,048
Bladder	23,660	7	1,656
Leukemia	171,200	7	1,204
All sites	608, 570	6-10	36,514-60,857

*Estimated deaths of cancer by the American Cancer Society, 2021 [2].
**Estimated percentage by Richard Doll and Richard Peto,1981 [5].

HISTORY OF OCCUPATIONAL CANCER

An occupational carcinogen was first reported more than 200 years ago. In 1775, Sir Percival Pott reported the first occupational cancer, scrotal skin cancer, among chimney sweepers in England who were heavily exposed to soot [10]. In 1822, Paris reported excess scrotal cancer among Cornish smelter workers [11]. Over the years, other chemicals were found to cause cancer after occupational exposures, including aromatic, amines, asbestos, arsenic, benzene, vinyl chloride,

CHAPTER 11

The Role of IARC in Causation of Occupational Diseases: Case Study of the Carcinogenic Evaluation of Crystalline Silica

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Abstract: The importance of causation of occupational diseases and the role of the International Agency for Research on Cancer (IARC) are discussed in this chapter. As a case study, the process by which silica dust was judged a known human carcinogen by IARC is reviewed. Silicosis is a chronic occupational lung disease known to be caused by inhaling crystalline silica, and the pulmonary cancer risk after the diagnosis of silicosis is a part of the IARC review of evidence. Laboratory animal evidence and mechanistic findings supporting IARC evaluation are also described. There remains a need to explore the association between silica exposure and other nonlung tumors, especially gastrointestinal cancers. The Occupational Safety and Health Administration (OSHA) developed a new regulatory standard that lowered the permissible exposure to 50ug/m3 in 2016. OSHA labeled silica as a known human carcinogen because of the IARC assessment. Occupational medicine leaders need to address several current silica dust problems such as silicosis/coal workers pneumoconiosis among coal miners, acute silicosis and auto-immune diseases among countertop workers, and intervention programs to lower silico-tuberculosis among South African miners. Future research studies need good silica dust monitoring estimates and high-quality industrial hygiene samples to evaluate the associations between silica exposure and many diverse diseases.

Keywords: Artisanal gold mining, Auto-immune ailments, Countertop manufacturing, Crystalline silica, History of causation assessment, Human cancer assessment, International Agency for Research on Cancer, Silico-tuberculosis, Silicosis, Lung cancer, National Institute for Occupational Safety and Health, Occupational diseases, Occupational Safety and Health Administration, Quartz epidemiology, Prevention, Quartz exposure in coal mines.

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INTRODUCTION

This chapter reviews the history of the development of criteria for establishing causation in epidemiology and occupational medicine. As an example of this process, the author discusses the epidemiology evidence leading to the International Agency for Research on Cancer's (IARC) evaluation of crystalline silica as a known human carcinogen and how that process has led to advances in knowledge in occupational medicine. This examination will assess the impact of IARC's evaluation of crystalline silica on subsequent research and occupational medicine policy.

DETERMINING CAUSE AND EFFECT IN OCCUPATIONAL MEDICINE AND PUBLIC HEALTH

The medical profession has struggled to identify the causes of diseases throughout human history. As medicine grew more sophisticated and reliant on scientific advances over the past 170 years, knowledge about cause-and-effect relationships became crucial to the development of appropriate treatments and public health prevention strategies. This is especially true in occupational and environmental health, as discussed by Goldsmith and Rose [1, 2]. For example, cholera, an environmental, infectious disease with high mortality rates, caused 19th-century physicians grave concern. The most prevalent view or theory in the first half of the 1800s was that *miasmas* (meaning "bad airs") close to the ground caused cholera and other infectious diseases. When comparing cholera mortality rates in cities at various elevations above sea level, it was clear that death rates for cholera declined as the distance from sea level rose. Thus, the data at that time seemed to support the prevailing miasma theory.

Dr. John Snow (1813-1858), a London physician noted for his work in anesthesia (his fame and place in medicine was linked with giving Britain's Queen Victoria chloroform anesthesia during childbirth), had a life-long interest in the study of cholera [3]. He rejected the miasma theory for transmission of cholera and instead hypothesized that cholera was spread *via* water, not by air. His early observations of cholera suggested that a then-unknown causal agent entered the body by mouth, multiplied within the gastro-intestinal tract, and then was spread to others by the fecal-oral route. He further deduced that the transmission of the disease was due to drinking water contaminated with raw sewage containing cholera in some form.

When a severe cholera epidemic broke out in 1854 in London, Snow saw an opportunity to evaluate his hypotheses concerning water and cholera when he was asked to investigate the cause of a spike in new cases of the disease. Snow, relying on logic and his previous study of earlier cholera outbreaks, concluded

that the sharply localized epidemic pointed to a contaminated pump or water well. At the time, the local Broad Street water pump was a favorite location to get household water. Snow investigated 83 cholera deaths in the Golden Square area of London, found a leaking sewer pipe within a few feet of the Broad Street pump, and discovered "that there (has) been no particular outbreak or prevalence of cholera in this part of London except among the persons who were in the habit of drinking the water of the (Broad Street) pump well" [3]

All of these facts provided very strong circumstantial evidence for water transmission of the *cholera vibrio*. Snow persuaded the local parish authorities to "take the handle off the (Broad Street) pump," thus preventing more cholera cases by stopping the public from using contaminated water from the neighborhood's favorite well. An environmental scientist and engineer ahead of his time, Snow was able to argue persuasively for causal intervention (regulation) without knowing the exact organism (*cholera vibrio*), without knowing the precise way it was transmitted (from person to person *via* drinking fecal-contaminated water), without understanding the discipline of bacteriology, and without detailed knowledge of mid-19th century London's water and sewer systems.

Following Dr. Edward Jenner's successful use of the cowpox virus to produce immunity to smallpox, first published in 1798 [4], Henle (1840) and Koch (1882) developed a set of postulates to demonstrate infectious disease causation (specifically for pulmonary tuberculosis transmission) [5, 6]. The Henle-Koch postulates set forth the following criteria (modified by Lilienfeld and Stolley, 1994) to be met before an agent could be considered the cause of disease [7]:

1) The organism (bacteria, virus) must be found in all cases of the disease.

2) One must isolate the organism from patients with the disease and successfully culture it.

3) When the purified culture is inoculated into a susceptible animals or human subjects, it must reproduce the disease [7].

While the Henle-Koch postulates produced a certain rigor and discipline to study infectious diseases, these postulates were problematic in their requirement that a particular illness must have only one cause and that a particular cause should result in only one disease. However, they did enable the germ theory of disease in the 19th century to achieve dominance in medicine over other theories, such as humors and miasmas.

Evans enlarged the Henle-Koch postulates to address questions of multi-causal and non-infectious diseases as well as infectious illnesses [8]. Each of Evans's

CHAPTER 12

Health Effects of Arsenic

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Abstract: Arsenic is a naturally occurring element with exposures in various work settings. Arsenic exposure can occur environmentally, particularly through drinking contaminated water and ingestion of some foods. The most toxic forms are inorganic arsenic, iAs (trivalent, pentavalent), and its metabolites, as well as the highly toxic arsine gas, the latter causing hemolysis. There are also organic arsenicals in food, particularly seafood, of little or no known toxicity. Inorganic arsenic is well absorbed through ingestion and respiration and is quickly cleared from the blood, distributed throughout the body (including across the placenta), metabolized in the liver, and excreted in the urine with metabolites monomethylarsonic acid (MMA) and dimethylarsinic acid (DMA). Most of it is excreted with a half-life of days. Inorganic arsenic disrupts numerous enzyme systems, causes oxidative stress and induces alterations in gene expression. Acute severe poisoning, rarely seen in occupational settings, is life-threatening, usually presenting with gastrointestinal symptoms and severe diarrhea that can progress to cardio-pulmonary collapse, requiring treatment in intensive care, with chelating medication. Chronic iAs exposure can lead to characteristic skin lesions, increased cancer risks (particularly skin, lung, bladder), and other cardiovascular, neurological, endocrine and reproductive adverse health effects. Assessment involves history, physical exam and urine arsenic (can be a spot sample corrected for creatinine), speciating the sample for inorganic species. This urine arsenic biomarker assesses current exposures. Treatment and prevention focus on identifying and eliminating or decreasing exposure, both in the workplace and environment.

Keywords: Arsenic, Arsine, Arsenobetaine, Arseno sugars, Arseno lipids, Dimethylarsinic acid (DMA), Monomethylarsonic acid (MMA), Inorganic arsenic.

INTRODUCTION

Arsenic is a naturally occurring metalloid element that is distributed in the earth's crust and water. It exists in various forms (elemental, gaseous as arsine, organic and inorganic (As^{3+} [trivalent, or arsenite] and As^{5+} [pentavalent, or arsenate]

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as well as various organic forms [1 - 4]. Elemental arsenic rarely exists in nature, is insoluble in water and bodily fluids and is considered non-poisonous [1]. Arsine, a hydride gas (AsH_3) is a gas, a hemolytic agent. Organic arsenicals vary in toxicity. Arsenobetaine (found in fish and crustaceans) and arsenosugars in seafood and marine algae are generally considered of little to no toxicity, whereas melarsoprol, an organoarsenic medication, is highly toxic [1, 5].

OCCUPATIONAL AND ENVIRONMENTAL EXPOSURES

Workers are exposed to inorganic arsenic (iAs) while performing tasks in various settings and circumstances, including [6, 7]:

- Smelters: iAs Inorganic Arsenic (iAs) occurs naturally in rocks that also contain copper and lead. Heating these ores creates arsenic trioxide dust (As_2O_3), which can be recaptured. Workers are exposed through working in the smelter and cleaning and maintaining equipment.
- Coal-burning power plants burning arsenic-rich coal can create arsenic deposits in the fly and bottom ash, with exposure occurring during boiler/furnace maintenance.
- Production of chromated copper arsenide (CCA) which is used as a wood preservative ("pressure treated" wood). Currently its major use in the U.S. is for industrial applications (such as utility poles) since its residential use was voluntarily discontinued in 2003.
- Inorganic Arsenic is used in the manufacture of gallium arsenide chips and circuit boards used in electronics, aerospace, and telecommunications.
- Industries involved in the manufacturing of some glass, ceramic, metal alloys, or optical products may expose workers through contact with source materials, final products, maintenance, clean up or disposal.
- Arsenic trioxide has been used as a chemotherapeutic agent since about 2000 to treat patients with promyelocytic leukemia [8, 9]. Potential worker exposures may include those in pharmaceutical manufacturing and those involved in the delivery of the medication (such as pharmacists and nurses).
- Several pesticides and herbicides containing arsenical agents have been banned by the U.S. Environmental Protection Agency (EPA), but may still be used in other countries [10].
- Arsine gas can be formed when arsenic-containing metals undergo acid washes. Unintentional exposures have also occurred during the refining of ores that contain arsenic. Arsine is also used as a doping agent in the semiconductor industry and in the manufacture of crystals for fiberoptics or computer chips [11]

There are also exposures to iAs from environmental sources, such as ingestion of contaminated drinking water (as from wells with mobilized arsenic from geological sources or from mining/industrial contamination) and food (such as rice [12], ingestion of arsenic-containing supplements, or herbal medications or breathing contaminated air from volcanic activity or nearby industrial sources [2, 7, 13 - 15].

ROUTES OF EXPOSURE, METABOLISM, AND EXCRETION

Soluble forms of iAs are well absorbed through both ingestion and inhalation. Most trivalent and pentavalent forms of arsenic dissolved in water have about 90% gastrointestinal absorption, whereas less soluble compounds, like arsenic trioxide is less well absorbed [1]. Systemic absorption *via* respiration depends on the particle size, and the solubility of the arsenical compound. Large non-respirable particles that are cleared from the nasopharyngeal tract can be swallowed and then absorbed from the gastrointestinal tract. Minimal skin absorption occurs through intact skin but may occur with chronic applications or damaged skin [1].

Absorbed arsenic is taken up by the red blood cells, and about 90% is quickly distributed, and then excreted in the urine, usually with a clearance from blood with an elimination half-life ($t_{1/2}$) of 1-2 hours. There is a second phase that occurs over the next week with a $t_{1/2}$ of about 30 hours, and a third phase that occurs over 10 or more days with a $t_{1/2}$ estimated at 300 hours [1]. The rapid clearance from the blood makes blood an unsuitable media for biomonitoring for worker surveillance and is used mostly for assessment in cases of acute poisoning.

Once absorbed, iAs is distributed to the liver, kidney, muscle, skin (including hair), and the brain and other tissues. Arsenic also crosses the placenta and can accumulate in the fetus [1]. Metabolism, *via* methylation, occurs predominately in the liver, and to a lesser degree in kidneys, testes and lungs. Pentavalent arsenic is first reduced to trivalent arsenic, which then undergoes the methylation steps to form monomethylarsonic acid (MMA) and then dimethylarsinic acid (DMA), followed by excretion of these intermediates in the urine [1, 15]. MMA may be more toxic than trivalent arsenic, and trivalent is more toxic than pentavalent [1]. Trivalent arsenic avidly binds to sulfhydryl groups (proteins, glutathione, cysteine) interfering with numerous enzyme driven systems, including those involved with cellular respiration, gluconeogenesis and glucose uptake and glutathione metabolism, as well as inducing oxidative stress and alterations of gene expression [1, 4].

Organic forms of arsenic are found in marine life and can be ingested and absorbed [4]. Arsenobetaine, the predominant arsenic species in most fin fish and

CHAPTER 13

Construction: Accessing and Working on Elevated Work Surfaces Safely

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Abstract: This chapter provides general information and educational resources that can explain methods to safely access elevated worksites in the construction industry and develop teaching and training tools from the provided content. Fatality data are presented to emphasize the dangerous nature of construction work at elevations. These data verify that falls from elevations are still the primary cause of fatal injuries in the construction industry. The NIOSH Fatality Assessment and Control Evaluation (FACE) program is highlighted throughout the chapter. The NIOSH FACE database of fatality reports identifies risk factors and recommendations for mitigating future fatal injuries. Additionally, NIOSH research activities are discussed that relate to fall prevention and protection. The activities discussed are a sample of popular fall protection techniques available to construction workers. They emphasize creating a safe working environment using ladders, scaffolds, and lifts through proper training and awareness. Proper planning, training, and practice can reduce the potential of fatal fall-related incidents from occurring.

Keywords: Construction, Falls, Fatalities, Hazards, Ladders, Lifts, Roofs, Safety, Scaffolds, Surveillance, Training.

INTRODUCTION

This chapter intends to provide general information and educational resources that the reader could use in a variety of ways, for example: (a) to access elevated work sites safely in the construction industry and (b) to develop teaching and training tools from the provided content. Fatality data are presented to emphasize the dangerous nature of construction work at elevations. In addition, research activities related to fall prevention and protection and the Fatality Assessment and Control Evaluation (FACE) program conducted by the National Institute for Occupational Safety and Health (NIOSH) are presented.

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Construction workers are exposed to many hazards on a daily basis. Working at elevations with the potential to fall to a lower level predominates in the industry. The Construction Industry has been considered one of the most dangerous industries in the U.S [1]. Roughly 20% of all work-related deaths occur in construction, and falls account for close to 40% of those fatalities. The need for fall protection is required for a variety of occupations, such as roofers, painters, structural iron and steelworkers, sheet metal workers, facade installers, and laborers while working at elevations. Safety precautions and relevant training about moving from a lower level to an elevated level are particularly significant in reducing fatal falls to a lower level.

There are four distinct segments in the construction industry: commercial-industrial, heavy-highway, bridge, and residential. This chapter focuses on the commercial-industrial and residential industries. They both utilize multiple methods of reaching elevated levels, such as ladders, scaffolds, mast climbing work platforms (MCWPs), and aerial lifts. The workers may require different areas of safety training depending on the method used. Despite the differences, it is important to note that there are elevating techniques and training programs similar to both industries, such as the proper use of extension ladders.

Both segments must be concerned about falls from elevations. Residential buildings can sometimes involve smaller or individually-owned companies that may have less access to the proper safety equipment and training programs and might be less likely to be unionized. A commercial building is usually larger scale, may or may not involve unions and typically has more in-depth training opportunities and greater access to safety equipment. Safety precautions for both residential and commercial construction that follow the training procedures about moving from a lower level to an elevated level and vice versa are particularly significant in reducing fatal falls to a lower level.

There is a lot of crossover between necessary construction skills, but a construction specialist in one segment is not a specialist in the other. These two segments differ at the core of their building materials. In the U.S., residential building typically uses wood, and commercial building uses steel as their main materials. There are numerous potential hazards on both residential and commercial building projects. NIOSH has reported that residential construction contractors are typically lacking in access to safety programs and in utilizing fall protection equipment [2]. According to the Occupational Safety and Health Administration (OSHA), workers engaged in residential construction six feet or higher must use a conventional fall protection system, such as guardrails, a personal fall arrest system (PFAS), or safety nets [3]. There are certain additional worker protection requirements depending on the location of the task being

performed, for example, leading edges, the slope of the work platform, and guarding of the edges and surface of the work platform.

Training opportunities are delivered in a multitude of ways or environments such as: Toolbox Talks, classroom settings, conferences, Train the Trainer courses, and online media like the OSHA fall prevention training guide for employers to use to train workers [4].

EMPLOYMENT IN U.S. – SELECTED DEMOGRAPHICS

The data presented in this section are to show the trends in the workforce as it pertains to age and race/ethnicity over a six-year period, 2014 to 2019 [5]. Historically, employment within the Construction industry has a higher percentage of workers of Hispanic Ethnicity compared to all other industries, but the annual trends follow a similar pattern to the general working population in the U.S [6]. Construction workers are increasing in age annually, similar to the general working population [7]. Trends of the overall US employment and construction employment often follow the economy. Unfortunately, the breakdown by age and race/ethnicity is not available for the construction sector.

In this section, the authors describe the most recent U.S. employment data available to understand the trends overall that have been historically similar to the Construction industry. The total U.S. employment in private industry has grown steadily from 135.7 million to 148.3 million workers over the period from 2014 to 2019. Similarly, employment in the construction sector has grown steadily from nine million to almost 11 million workers. During those six years, the construction workforce as a percentage of all U.S. employment ranged from 6.6% to 7.3%. The steady increase in construction workers, from 9.0 million to 10.85 million, represents a 21% increase in employment over the six-year period (Table **1**).

Table 1. Total Employment for U.S. Private Industry, Construction Sector, and Two Occupations, 2014 – 2019. Data collected from References [10, 13, 16, 19, 22, 24].

-	2014	2015	2016	2017	2018	2019
Total U.S. Employment, Private industry	**135,722,500**	**137,931,000**	**141,550,000**	**142,988,500**	**146,263,500**	**148,300,000**
Total construction workers	9,024,000	9,281,000	9,765,000	10,400,000	10,659,000	10,850,000
Percent of Total	6.6%	6.7%	6.9%	7.3%	7.3%	7.3%
Roofing Workers	178,000	189,000	208,000	201,500	184,500	200,000

CHAPTER 14

Work-related Musculoskeletal Disorders

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Abstract: In most industrialized countries, work-related musculoskeletal disorders (WMSDs) are a major occupational health problem resulting in productivity loss, employee absenteeism, and high workers' compensation and healthcare costs. Understanding the etiology and control of WMSDs and associated risk factors is imperative for reducing the burden of this problem. This chapter is organized by five topics on WMSDs: (1) the problem and surveillance of WMSDs; (2) the etiology of WMSDs and their risk factors; (3) risk assessment methods for job-related physical risk factors; (4) risk intervention effectiveness; and (5) ergonomic guidelines and standards for the prevention of WMSDs. The authors focus on the breadth of the scientific knowledge and literature pertaining to WMSDs for occupational safety and health professionals interested in learning about the field of ergonomics. This chapter also provides anticipated future challenges in the areas of surveillance, risk interactions, risk assessments, and intervention evaluations. The research agenda for WMSDs published by the National Occupational Research Agenda (NORA) Musculoskeletal Health Cross-Sector (MUS) Council in 2018 is recommended as supplementary reading for the future direction of WMSD research.

Keywords: Work-related musculoskeletal disorders, Ergonomics, Occupational safety and health, Surveillance, Biomechanics, Vibration, Pathomechanics, Psychosocial factors, Job risk assessment.

BACKGROUND

Work-related musculoskeletal disorders (WMSDs) constitute a heterogeneous group of disorders [1, 2] that involve the tendons, nerves, joints, muscles, and circulatory system. These disorders have been given many names over the years,

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including musculoskeletal disorders, musculoskeletal injuries, overuse injuries, repetitive strain injuries, repetitive motion injuries, cumulative trauma disorders, overuse syndrome, soft tissue disorders, and occupational overuse syndrome. All of these terms broadly describe the nature of these disorders.

Systematic reviews of the etiology and risk factors of WMSDs have suggested the importance of the workplace factors contributing to musculoskeletal disorders (MSDs) [1, 3, 4]. MSDs are characterized as work-related diseases, as opposed to occupational diseases. As described by the World Health Organization (WHO), "work-related" diseases are multifactorial, indicating the contribution of numerous risk factors toward the causation of these diseases [5]. Unlike work-related diseases, occupational diseases possess a direct cause–effect relationship between hazards and disease. The Occupational Safety and Health Administration (OSHA) defines an event or exposure in the work environment as work-related if it "either caused or contributed to the resulting condition or significantly aggravated a pre-existing injury or illness. Work-relatedness is presumed for injuries and illnesses resulting from events or exposures occurring in the work environment…"

Burden of WMSDs

In the United States, WMSDs are a frequent cause of lost workdays due to injury [6]. These disorders are the most prevalent occupational disorder, not only in the United States but globally. The World Health Organization (WHO) [7] identifies musculoskeletal conditions as the leading contributors to disability worldwide, affecting people across the life-course. Although the past decade has seen a decline in the year-to-year number of cases of WMSDs [8], the proportion of occupational injuries and illnesses remains consistent. An estimated one-third of occupational injuries and illnesses are attributable to WMSDs [9, 10]. Data from the U.S. Bureau of Labor Statistics (BLS) on non-fatal injuries and illnesses in the private industry reveal that between 2011 and 2019, the proportion of WMSDs involving days away from work (DAFW) in all industries was between 30% and 34.5% [6].

WMSDs have been associated with high costs to employers due to absenteeism, lost productivity, and increased health care, disability, and workers' compensation costs [11 - 13]. Recent data showed that workplace overexertion injuries (that is, WMSDs) cost an estimated $15.1 billion a year, accounting for about 25% of total workers' compensation costs in the United States [14]. Low back disorders (LBDs) are the largest contributors to the total workers' compensation cost. The total health care expenditures incurred by individuals with LBDs alone in the United States have reached $90.7 billion a year [15].

In addition to the high costs, WMSDs also are related to reductions in quality of life and functional status [16, 17].

WMSD Case Definitions

Although various surveillance systems all enable the quantification of WMSDs, their WMSD case definitions differ. Therefore, it is important to identify and understand the WMSD case definition of each system, including the inclusion and exclusion criteria for the worker population. This chapter defines WMSDs on the basis of both the clinical nature of the disorders themselves combined with the events or exposures that caused the injuries. Nature of injury for WMSDs includes both acute injuries caused by traumatic incidents or chronic conditions classified as illnesses. Broadly speaking, most WMSD surveillance definitions include injuries or illnesses caused by overexertion. For example, these events or exposures include strenuous or repetitive work, bodily reactions from prolonged exposure to awkward postures, and exposure to vibration. Excluded from our definition are musculoskeletal symptoms or diagnoses (such as sprains or strains) caused by slips, trips, or falls; struck-by, caught-in, or crushed-by incidents; transportation incidents; violence; and fires or explosions. Although medical treatment of injured workers for low back pain, for example, may be identical, irrespective of cause, understanding what event or exposure caused the WMSD is important for primary prevention strategies, return-to-work decisions, and work restrictions. WMSD definitions in different surveillance systems limit eligibility to the diagnosis of either soft-tissue injuries or non-traumatic, chronic diagnoses such as rotator cuff syndrome or carpal tunnel syndrome while excluding injuries likely caused by acute trauma (such as sprains). In some cases, WMSD case definitions are limited to specific body parts or body regions.

For example, within the U.S. Department of Labor, the WMSD case definition adopted by OSHA differs from the case definition used by the U.S. BLS Survey of Occupational Injuries and Illnesses. The OSHA definition reads, “Musculoskeletal disorders (MSDs) affect the muscles, nerves, blood vessels, ligaments, and tendons. Workers in many different industries and occupations can be exposed to risk factors at work, such as lifting heavy items, bending, reaching overhead, pushing and pulling heavy loads, working in awkward body postures and performing the same or similar tasks repetitively.”

However, since 2011, the BLS definition has been, “Musculoskeletal disorders (MSDs) include cases where the nature of the injury or illness is pinched nerve; a herniated disc; meniscus tear; sprains, strains, tears; hernia (traumatic and nontraumatic); pain, swelling, and numbness; carpal or tarsal tunnel syndrome; Raynaud's syndrome or phenomenon; musculoskeletal system and connective

SUBJECT INDEX

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