Published in final edited form as:
*Arch Environ Occup Health*. 2019 ; 74(1-2): 15–29. doi:10.1080/19338244.2018.1532387.

# NIOSH’s Respiratory Health Division: 50 years of science and service

**Kristin J. Cummings**[a], **Doug O. Johns**[a], **Jacek M. Mazurek**[a], **Frank J. Hearl**[b], and **David N. Weissman**[a]

[a]Respiratory Health Division, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Morgantown, WV, USA

[b]Office of the Director, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Washington, DC, USA

## Abstract

The year 2017 marked the 50th anniversary of NIOSH’s Respiratory Health Division (RHD). RHD began in 1967 as the Appalachian Laboratory for Occupational Respiratory Diseases (ALFORD), with a focus on coal workers’ pneumoconiosis. ALFORD became part of NIOSH in 1971 and added activities to address work-related respiratory disease more generally. Health hazard evaluations played an important role in understanding novel respiratory hazards such as nylon flock, diacetyl, and indium-tin oxide. Epidemiologic and laboratory studies addressed many respiratory hazards, including coal mine dust, silica, asbestos, cotton dust, beryllium, diesel exhaust, and dampness and mold. Surveillance activities tracked the burden of diseases and enhanced the quality of spirometry and chest radiography used to screen workers. RHD’s efforts to improve scientific understanding, inform strategies for prevention, and disseminate knowledge remain important now and for the future.

### Keywords

Epidemiology; occupational lung disease; respiratory diseases; workers

## Introduction

The year 2017 marked the 50th anniversary of a US research and service unit currently known as the Respiratory Health Division (RHD) of the National Institute for Occupational Safety and Health (NIOSH). RHD began in 1967 as the Appalachian Laboratory for Occupational Respiratory Diseases (ALFORD), a US Public Health Service laboratory based in Morgantown, West Virginia and focused in particular on coal workers’ pneumoconiosis (CWP) (see Figure 1). In this article, we discuss RHD’s evolution over 50

CONTACT Kristin J. Cummings, cvx5@cdc.gov, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, 1095 Willowdale Road, MS 2800, Morgantown, WV 26505, USA.

Disclosure statement
No potential conflict of interest was reported by the authors.

years, from a regional to national emphasis, and from a concentration on CWP to a portfolio that includes a broad range of exposures and diseases.

The 1960s were marked by increasing recognition of the importance of environmental exposures to health, both in general and in the workplace. The 1968 Farmington, West Virginia mine disaster, an explosion that killed 78 miners, as well as activism on behalf of miners, contributed to the passage of the Federal Coal Mine Health and Safety Act of 1969 (Coal Act), which mandated a number of measures to protect coal miners. Responsibilities assigned to ALFORD included a national study of CWP, an autopsy program, and coal workers' surveillance.

One year later, the Occupational Safety and Health (OSH) Act of 1970 was enacted to reduce injury and illness in general industry. The OSH Act authorized the formation of NIOSH, a scientific agency, and ALFORD became part of the new organization. ALFORD expanded in scope from a primarily medical unit to a multidisciplinary group doing clinical, epidemiological, basic laboratory, and other research. ALFORD had access to authorities assigned to NIOSH under the OSH Act, including the ability to enter workplaces to conduct research. In 1973, NIOSH (and ALFORD) joined what is now called the Centers for Disease Control and Prevention (CDC).

In 1977, after a reorganization of the NIOSH-Morgantown facility, ALFORD became the Division of Respiratory Disease Studies (DRDS). It contained an Office of the Director overseeing research and services provided by Clinical, Environmental, Epidemiological and Laboratory Investigations Branches, as well as an Examination Processing Branch which managed surveillance services offered to coal miners and the B Reader certification program. The Environmental Investigations Branch was a new addition, led and staffed in part by industrial hygienists who had transferred to Morgantown after closure in 1976 of NIOSH's Western Area Laboratory for Occupational Safety and Health, which was located in Salt Lake City, Utah. DRDS began carrying out health hazard evaluations (HHEs) in specific workplaces, first in mines and later in other settings where respiratory hazards were of concern.

In 1996, another reorganization moved most of the investigators in the Laboratory Investigations Branch to a new NIOSH Health Effects Laboratory Division. The reorganization also moved respirator research and certification activities from NIOSH's Division of Safety Research into DRDS; these were subsequently moved to NIOSH's National Personal Protective Technologies Branch when it was established in 2001. In 1999, DRDS simplified its structure to an Office of the Director overseeing a Field Studies Branch, a Surveillance Branch, and a Laboratory Investigations Branch. In 2015, DRDS's name was changed to RHD, reflecting the division's extensive engagement in public health service activities other than research studies to protect against work-related respiratory disease. The new name also reflected the importance of optimizing workers' respiratory health by addressing the range of hazards encountered at and away from work. In addition to the name change, the Laboratory Investigations Branch was absorbed into the Field Studies Branch, leaving the current structure of Office of the Director, Field Studies Branch, and

Surveillance Branch. Past and current Directors of ALFORD, DRDS, and RHD are shown in Table 1.

## Respiratory disease in coal miners

Preventing respiratory disease in coal miners has been a major focus since the beginning of RHD. Inhaling coal mine dust can cause a range of pulmonary interstitial diseases, including CWP, silicosis, mixed dust pneumoconiosis, and dust-related diffuse fibrosis (which can be mistaken for idiopathic pulmonary fibrosis), as well as chronic obstructive pulmonary disease (COPD).[1] A major early effort was to carry out a National Study of Coal Workers' Pneumoconiosis (NSCWP), included as a requirement within the Coal Act. Ultimately, four rounds of the NSCWP were carried out between 1969 and 1988.[2–4] Data provided by these studies helped to support development of a NIOSH Criteria Document evaluating relationships between respirable coal mine dust exposure and risk of adverse health effects and recommendations for preventing respiratory diseases caused by inhalation of coal mine dust.[5,6]

The 1969 Coal Act also authorized NIOSH to pay for coal miners' autopsies. The National Coal Worker's Autopsy Study (NCWAS) was developed as a service program for the family members of deceased underground coal miners to determine if CWP or other lung diseases were present at death.[7] Analysis of 6103 miners' autopsies processed by the program from 1971 through 1996 demonstrated a positive impact of coal dust regulation.[8] Although the program is no longer active, tissue samples collected over the years remain useful for research on issues such as genetic susceptibility factors for pneumoconiosis.[9]

The Coal Act established requirements for chest radiographic surveillance of coal miners. Surveillance is managed by a program now called the Coal Workers' Health Surveillance Program (CWHSP). RHD developed regulations specifying how the program operates, which can be found in the US Code of Federal Regulations at 42 CFR Part 37. Under the program, miners are offered screening at first entry into coal mining and then at approximately 5-year intervals during their mining careers. Mine operators contract with participating medical facilities, which obtain medical tests and send them to RHD. Chest radiographs are then classified for the presence and severity of changes of pneumoconiosis using a standardized classification system maintained by the International Labor Organization (ILO).[10] Classifications are done by physicians called B Readers, who are certified by NIOSH as proficient in the ILO classification system. Miners are notified of their results and, if they have qualifying radiographic changes, of their eligibility to transfer to low dust jobs as specified in the US Code of Federal Regulations at 30 CFR Part 90.

CWHSP has played a major role in tracking the burden of respiratory disease in coal miners and in detecting recent increases in pneumoconiosis affecting coal miners. Findings from the program over the past decade have highlighted CWP as a contemporary emerging issue. A 2005 report identified regional "hot spots" of rapidly progressive pneumoconiosis, particularly in certain parts of Appalachia.[11] This led to supplementation of the CWHSP with targeted active mobile outreach to hot spot areas (see Figure 2). Data from CWHSP and mobile outreach efforts confirmed an increase in pneumoconiosis after the late 1990s.[12]

Potential reasons hypothesized for the increase include longer working hours, increased exposure to respirable crystalline silica, and less effective control of dust, possibly related to challenges faced by small mines and mining of thin coal seams surrounded by rock.[12] CWHSP findings also identified marked increases in prevalence of progressive massive fibrosis (PMF), the most severe form of pneumoconiosis in coal miners, after the late 1990s.[13] Additional surveillance efforts motivated by these CWHSP findings led to reports of large case clusters of coal miners with PMF receiving care at clinics in eastern Kentucky and western Virginia.[14,15] Furthermore, as recently published in this journal, CWHSP data demonstrated low (<15%) participation among eligible miners in the Part 90 transfer option.[16]

RHD's many efforts to document the effects of exposure to coal mine dust and the burden of disease among US coal miners have had important impacts. In part due to recognition that respiratory disease continued to be a contemporary health issue for coal miners, MSHA promulgated regulations in 2014 requiring enhanced protections.[17] These included lowering the respirable coal mine dust exposure limit, introducing requirements for frequent assessment of airborne dust concentrations using continuous personal dust monitors, adding spirometry to health surveillance to assess for loss of lung function, and expanding the health surveillance requirements to surface miners, who have been found to be at risk of lung disease including silicosis.[18–20]

## Silica, silicosis, and lung cancer

Studies of silicosis in the dusty trades began early in the history of the US Public Health Service with studies of six industrial groups including the Vermont granite industry.[21] A later reassessment of the Vermont granite workers suggested the initial dust exposure limit for crystalline silica at 10 million particles per cubic foot.[22] Researchers in RHD continued and expanded on the study of the Vermont granite industry,[23] including other cohorts of dusty trades workers.[24] Costello confirmed previous studies' findings, showing that death rates from silicosis and tuberculosis were the major health threats in the years before 1940, and also found excessive mortality rates from lung cancer in stone shed workers who had been employed prior to 1930, and hence had been exposed to high levels of granite dust. Amandus' studies[24] found excesses in lung cancer among workers in the North Carolina dusty trades silicosis registry that could not be explained by exposures other than silica. To explore the silica-silicosis-cancer relationship, RHD entered into an international partnership with the US National Cancer Institute (NCI) and the Tongji Medical University (TMU) in Wuhan, China, to study Chinese workers exposed to silica-containing dusts. The study included exposure surveys of 29 mines and factories in China, evaluation of work histories and chest radiographic records from the miners and workers,[25] and a study to compare the Chinese method of chest radiographic classification for pneumoconiosis with the ILO standards.[26] The study found that cancer risks were 22% higher among those workers who had silicosis.

In his 1991 keynote address to the American Industrial Hygiene Conference, then NIOSH Director J. Donald Millar said that the continued existence of silicosis is an "occupational obscenity".[27] He termed it an occupational obscenity because we already know, without needing further research, how to prevent deaths from this disease. Taking this charge from Dr. Millar, RHD's staff built partnerships with leaders at the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA), and began a coordinated national initiative to control exposure to crystalline silica. During field studies, RHD industrial hygienists asked workers why they did not take precautions from exposure to silica. The reply was often, "it's just dust". In response, RHD, working with MSHA and OSHA, began a campaign with the slogan, "*If it's silica, it's not just dust*".[28] Researchers in RHD issued three new documents to warn against the hazards of silica for abrasive blasters,[29] rock drillers,[30] and construction workers.[31] Ultimately, the RHD efforts were instrumental in producing the 2002 NIOSH Hazard Review on the Health Effects of Occupational Exposure to Crystalline Silica and contributed to the issuance of a new OSHA standard for crystalline silica.[32,33]

## Health hazard evaluations

RHD's participation in NIOSH's HHE Program (https://www.cdc.gov/niosh/hhe/default.html) has provided an important mechanism for recognizing and responding to emerging occupational respiratory health issues. An HHE is a workplace evaluation conducted by NIOSH in response to a request from an employer, three or more employees, or a labor union. As part of this program, NIOSH also provides technical assistance to other government agencies evaluating workplace hazards. The primary outcome is a set of recommendations for a particular workplace on hazard reduction and illness prevention. Since 1970, RHD has carried out approximately 2,300 HHEs at workplaces across the country. In addition to the site-specific service component, some of these HHEs have allowed RHD to characterize novel respiratory hazards also relevant to other workplaces.

Starting in the 1990s, RHD carried out a series of HHEs addressing a newly recognized condition called flock workers' lung disease. This interstitial lung disease was first described in nylon flock workers, who had a unique pattern of lymphocytic bronchiolitis.[34–36] In flocking, fibers measuring about 1 mm in length by 10–20 mm in diameter are cut from long filaments and applied to adhesive-coated substrates to create velvet-like materials used in a wide variety of products. Given the relatively large size of these fibers, the process was not thought to pose an inhalational hazard. However, an HHE conducted at a nylon flocking plant with two cases of disease demonstrated the presence of elongated respirable-sized nylon particles in the workplace air generated as fragments from nylon filaments during the cutting process;[37,38] experimental studies confirmed the dust's toxicity.[39] Subsequent health hazard evaluations at six other flocking facilities documented associations between respirable dust levels and adverse health effects including respiratory symptoms and reduced lung function parameters.[40,41] These evaluations also expanded the scope of concern beyond nylon: one of the facilities exclusively used rayon flock,[41] whereas the others used a variety of synthetic materials (nylon, rayon, polyester, and acrylic) in flocking processes.[40]

RHD conducted multiple health hazard evaluations that led to the recognition and characterization of the inhalational toxicity of chemical components of flavorings. In 2000, the Missouri Department of Health and Senior Services requested technical assistance in evaluating a microwave popcorn facility where a cluster of former workers with a rare lung disease, obliterative bronchiolitis, had been identified.[42] RHD demonstrated an inverse relationship between air levels of the Α-diketone diacetyl, a butter flavoring chemical, and the lung function of current workers.[43] *In vivo* studies confirmed that the butter flavoring vapors, generally, and diacetyl, specifically, were toxic to the respiratory epithelium.[44,45] HHEs at five other microwave popcorn facilities suggested risk of occupational lung disease across this industry.[46] Later HHEs highlighted the risk in other industries: bakery mix production;[47] flavoring manufacturing;[48,49] and coffee processing.[50–52] They also identified a trend towards substitution with a related Α-diketone, 2,3-pentanedione, which was found in experimental studies to have respiratory toxicity comparable to that of diacetyl.[53] These HHEs led to a 2003 NIOSH Alert on flavorings and stimulated NIOSH's establishment of recommended exposure limits (RELs) for diacetyl and 2,3-pentanedione in 2016.[54,55]

Another example relates to RHD's work on indium lung disease. Indium-tin oxide (ITO) is a sintered material used in the production of displays, touch screens, solar panels, and architectural glass. In 2009, RHD responded to an HHE request from the management of an ITO production facility where two workers had developed the rare disease pulmonary alveolar proteinosis, subsequently identified as part of a spectrum of disease manifestations related to indium exposure.[56,57] RHD found evidence of evolving lung function abnormalities that appeared to be more common in workers with higher indium exposures.[58] This HHE led to a multidisciplinary NIOSH research study that characterized the *in vivo* toxicity and physicochemical properties of production materials,[59,60] as well as the quantitative relationship between indium exposure and early biomarkers of indium lung disease.[61,62]

## Laboratory-based studies

Although most of RHD's laboratory-based activities were moved to another part of NIOSH in 1996, there is a significant history of laboratory-based research in RHD. Teams within the division were dedicated to conducting laboratory research involving physiological, pathological, biochemical, immunological and microbiological approaches to investigate occupationally induced lung disease, with the goals of establishing mechanisms and informing risk assessment. Major emphasis was placed on experimental models of silicosis, CWP, occupational asthma, byssinosis, fungal diseases, asbestosis, and mutagenesis, among others. Examples of laboratory-based contributions include descriptions of the pathology of CWP,[63] asbestos-associated diseases of the lungs and pleura,[64] and diseases associated with exposure to silica and non-fibrous silicate minerals.[65] Another important contribution was a method for extracting and quantifying endotoxin in dust samples, which played an important role in epidemiologic investigations of endotoxin as a potential etiologic agent in lung disease.[66] A prominent example of mechanistic research documented the enhanced toxicity of freshly fractured silica particles, which had important implications for the extremely hazardous nature of activities such as abrasive blasting with silica sand.[67]

RHD continues to maintain several research laboratories dedicated to conducting original inhalation exposure assessment research and providing support for field investigations by developing novel and innovative approaches for the sampling and analysis of chemical and biological agents. Recent examples of this research include development of methods for measuring diacetyl and 2,3-pentanedione in air,[68] and quaternary ammonium compounds in air and on surfaces,[69] assessing 3-dimensional printer emissions,[70] and characterizing fungi and fungal products in settled dust samples.[71]

## Epidemiological investigations

### Byssinosis

Byssinosis is an airways disease with features of both asthma and COPD that occurs with exposure to cotton dust. In the early 1970s, the prevalence of byssinosis among US cotton workers was estimated at 20%.[72] RHD contributed to a 1974 criteria document that summarized the available evidence and recommended lowering the standard from 1 to 0.2 mg/m$^3$.[73] OSHA’s 1978 Cotton Dust Standard incorporated NIOSH’s recommendations, and included provisions for medical monitoring that effectively set national standards for spirometry.[74] Despite this progress, concerns remained about the potential for byssinosis to occur at exposure levels below the standard. In a series of remarkable experimental field studies, RHD demonstrated an exposure–response relationship between airborne endotoxin (but not dust concentration) and reduced lung function, suggesting that endotoxin was responsible for the acute response to cotton dust.[75,76] The preventive effectiveness of washing cotton to reduce endotoxin concentration also was demonstrated through experimental exposure studies,[77] confirming earlier observations from RHD laboratories that were published in this and other journals.[78–80] This work contributed to a NIOSH Current Intelligence Bulletin on cotton washing methods and an amendment by OSHA to the Cotton Dust Standard.[81,82] OSHA has concluded that the Cotton Dust Standard had the effect of reducing the prevalence of byssinosis to <1%.[72]

### Occupational respiratory carcinogens

Although most work done by NIOSH addressing occupational cancer is based in other parts of the institute, RHD has made several noteworthy contributions in this area. As noted above, studies of granite workers and others in dusty trades demonstrated relationships between silica exposure and lung cancer. In addition, a 1987 report described a dose–response relationship for lung cancer mortality associated with workers’ exposure to asbestos fibers found in vermiculite mined and processed near Libby, Montana.[83] A follow-up report in 2007 confirmed increased risk for lung cancer mortality and demonstrated increased risk for cancer of the pleura in these workers.[84] These studies also documented increased mortality from nonmalignant respiratory disease and asbestosis, respectively. Finally, a large study was conducted to evaluate risk of lung cancer mortality in workers exposed to diesel exhaust at eight US nonmetal mines. A cohort mortality study found increased risk of lung cancer, esophageal cancer, and pneumoconiosis in the cohort compared to state-based rates. In addition, a dose–response relationship was shown between respirable elemental carbon exposure and lung cancer mortality.[85] A companion case-control study was also conducted to evaluate the impact of smoking and other potential confounders. It found increasing lung cancer risk with increasing cumulative respirable elemental carbon exposure. There was an interaction between smoking and respirable elemental carbon exposure such that the effect of each exposure was attenuated in the presence of high levels of the other.[86]

### Beryllium sensitization and chronic beryllium disease

Beryllium is a metal with unique properties and diverse applications. Occupational exposure to beryllium can lead to sensitization and a granulomatous lung disease, chronic beryllium disease. Evidence of sensitization and disease at exposures below the 2 mg/m$^3$ regulatory limit indicated the need to better characterize exposures, identify occupational and personal risk factors, and prevent adverse health outcomes. Since the 1990s, RHD has partnered with a major beryllium producer on a multidisciplinary research project that has substantially improved the understanding of this hazard. Noteworthy achievements include characterization of what had been an elusive exposure–response relationship,[87,88] description of genetic contributions to risk of sensitization and disease,[89–91] and demonstration of the effectiveness of a comprehensive preventive program that includes periodic medical surveillance.[92–94] RHD also examined beryllium physicochemistry and toxicology, for instance publishing in this journal the results of experimental studies of the interaction between process materials and artificial airway epithelial lining fluid.[95] RHD's research findings led to the publication in 2011 of a NIOSH Alert on preventing beryllium-related health effects and were included in a 2014 American Thoracic Society Statement on beryllium disease.[96,97] They also were cited extensively in support of OSHA's 2017 beryllium rule, which lowered the permissible exposure limit to 0.2 mg/m$^3$ and introduced requirements for medical monitoring and medical removal benefits.[98]

### Indoor environmental quality and asthma

Over the past several decades, as US shifted from a manufacturing to service economy, workplace indoor environmental quality became a major concern of the American public. Complaints about poor indoor environmental quality due to dampness and mold in schools, hospitals, offices, retail stores, and other buildings grew to be the most common reason for HHE requests to NIOSH and remain so to this day. To better understand and prevent adverse respiratory health effects of exposure to indoor dampness and mold, RHD undertook several research projects. A longitudinal investigation of a water-damaged state office building was particularly illuminating, demonstrating that new-onset asthma was associated with building occupancy, upper respiratory symptoms predicted subsequent development of lower respiratory symptoms, hydrophilic fungi and endotoxin were associated with adverse health effects, and while relocation of ill employees during remediation was health-protective, extensive remediation efforts were not fully successful in improving respiratory illness.[99–104] A semi-quantitative exposure index of the extent of water stains, visible mold, mold odor, and moisture in a space was designed and validated in multiple settings.[105–107] These efforts led to a NIOSH Alert on indoor dampness and mold,[108] and the current development of the NIOSH Dampness and Mold Assessment Tool, a practical instrument for systematic evaluation of buildings that can be used to prioritize remediation.

## Occupational respiratory disease surveillance

In addition to the previously described CWHSP, RHD has carried out a number of other surveillance activities, including population surveillance, state-based surveillance, and efforts to improve respiratory health monitoring in support of occupational respiratory health screening and surveillance programs.

## Population surveillance

Population surveillance to track the burden of work-related respiratory disease and occupational respiratory hazards in the US has been an important component of RHD efforts. These efforts have complemented, built upon, and added unique value to the surveillance activities carried out by many states, the National Center for Health Statistics (NCHS), and other federal agencies, including the Bureau of Labor Statistics (BLS), OSHA, and MSHA. Examples of large data sets that have been used by RHD include the National Health and Nutrition Examination Survey (NHANES), Behavioral Risk Factor Surveillance System (BRFSS) Asthma Call-back Survey (ACBS), and large data sets collected for administrative purposes (e.g., Centers for Medicare and Medicaid Services claims; OSHA enforcement inspections data, MSHA coal mine inspector and mine operator dust data). Large data sets collected from these and other sources have been enhanced to improve specificity in identifying factors associated with occupational respiratory diseases. The occurrence of specific diseases has been described with regard to worker demographic characteristics, industry, occupation, geography, and temporal trends. The distribution and trends in occupational exposures to agents causing respiratory diseases also have been described.

Dissemination of RHD population surveillance data has evolved over time. In addition to publications in CDC's Morbidity and Mortality Weekly Report and peer-reviewed journals, RHD published the first Work-Related Lung Disease (WoRLD) Surveillance Report in 1991.[109] This report presented data for asbestosis, CWP, silicosis, byssinosis, exposure to cotton dust, hypersensitivity pneumonitis, toxic agents, dust diseases of the lung, and CWP compensation for 1968–1987. In 2008, the seventh and final WoRLD Surveillance Report was published.[110] This report consisted of two volumes and covered a time period between 1968 and 2004. Volume I had 17 subsections, each concerning a major disease category and (where available) related occupational exposures, and one subsection concerning smoking status. Volume II had nine sections presenting data on respiratory conditions by major industrial sector, as defined by the National Occupational Research Agenda (NORA).

To continue to provide surveillance information and access to data for public health action, a webbased platform called "eWoRLD" was launched in 2008.[111] This format presents up-to-date summary tables, graphs, and figures on the occurrence of pneumoconioses, asthma, COPD, malignant mesothelioma and other respiratory conditions as well as selected related exposure data. Other online resources developed by RHD are the National Occupational Respiratory Mortality System (NORMS) and the Coal Workers' Health Surveillance Program Data Query System. NORMS is a data-storage and interactive data-retrieval system that contains national mortality data from CDC's NCHS on occupationally related respiratory diseases in the form of tables, charts, and maps.[112] With the introduction of CDC's WONDER (Wide-ranging ONline Data for Epidemiologic Research),[113] which contains publicly available mortality data for all conditions, NORMS is scheduled to be archived and closed.

### State-based surveillance

State health departments are uniquely positioned to assist NIOSH in understanding the scope of occupational respiratory disease problems and in linking surveillance to practice at the local level. RHD has worked closely with states to use clinical data sources and other data available only at the state level to identify emerging issues and prevention strategies. In 1987, targeted provider-reporting systems called SENSOR (Sentinel Event Notification System for Occupational Risk) were initiated and funded by NIOSH in association with 10 State Health Department partners.[114] SENSOR addressed limitations of provider-reporting systems such as lack of epidemiologic case definitions and lack of formal, defined networks of sentinel providers with specific responsibility for reporting selected conditions.

Six states identified either silicosis (Michigan, New Jersey, and Ohio) or work-related asthma (WRA; Colorado, Massachusetts, Michigan, New Jersey, and Wisconsin) as important problems within their states and appropriate for surveillance under the SENSOR model. WRA is a subset of asthma, and includes occupational asthma (i.e., new-onset asthma caused by factors in the workplace) and work-exacerbated asthma (i.e., preexisting or concurrent asthma worsened by factors related to work). Over time, the number of states collecting surveillance information on WRA and silicosis, and funding mechanisms varied. Currently, NIOSH receives WRA data from four states—California, Massachusetts, Michigan, and New York, and on silicosis from one state—Michigan.[115]

In 2014, NIOSH invited applications for State-Based, Occupational Health and Safety Surveillance programs.[116] Under this cooperative agreement, states conduct basic or “Fundamental” population-based surveillance for pneumoconiosis (including silicosis hospitalizations and mortality) and WRA. In addition, some states are also funded to carry out case-based or “Expanded” sentinel surveillance focused specifically on occupational respiratory diseases (e.g., WRA). State surveillance staff conduct review of medical records, and when possible, a follow-up interview with reported cases, and collect demographic, work-history, and medical information for case confirmation and classification. Putative causes of WRA are coded using the Association of Occupational and Environmental Clinics (AOEC) exposure coding scheme.[117] The results of this surveillance have identified new causes of WRA, and stimulated workplace investigations, exposure controls, and research projects to understand and prevent disease.

State-based surveillance partners published multiple annual reports, brochures, case reports, journal articles, newsletters, multi-media, and other products that track the burden of WRA and silicosis. Currently, over 400 of these products in various languages are available at the State-based Occupational Health Surveillance Clearinghouse.[118]

## Respiratory health monitoring

### Spirometry

Spirometry, a type of pulmonary function test, is inexpensive and informative, but challenging to perform correctly. RHD has had a formal role in assuring the quality of spirometry in occupational settings since the promulgation in 1978 of OSHA’s Cotton Dust Standard.[74] The standard states that “Persons other than licensed physicians, who administer the pulmonary function testing required by this section shall have completed a NIOSH-approved training course in spirometry". Approved courses must meet minimum criteria for the instruction of individuals who perform spirometry.[119] More recently, OSHA's 2016 crystalline silica standard specified that spirometry be performed by a technician with a current certificate from a NIOSH-approved spirometry course.[33] Similar requirements are in place for technicians performing spirometry as part of the CWHSP. In addition to these legally mandated roles, RHD has made substantial contributions to national guidelines for spirometry published by the American Thoracic Society.[120–122]

Managing spirometry surveillance provided to coal miners as part of the CWHSP is a relatively new responsibility for RHD.[123] A strong foundation for spirometry surveillance has been developed, including approval of participating medical facilities providing spirometry, approval of spirometry equipment to be used in surveillance, and development of infrastructure for electronic submission of spirometry tests from medical facilities to NIOSH, where they can be reviewed for quality and results reported back to miners.

NIOSH has also conducted research related to spirometry. For example, in 1999, RHD investigators used NHANES data that were collected under their over-sight to develop spirometric reference values. These reference values were widely adopted for use in interpretation of spirometry results, even in general healthcare settings.[124] Another example was work to improve approaches to using longitudinal spirometry test results to evaluate for changes over time in individuals. RHD developed Spirometry Longitudinal Data Analysis (SPIROLA) software, a program for tracking longitudinal spirometry and identifying individualized thresholds for excessive decline.[125,126] Excessive decline was shown to be associated with excess risk for mortality.[127]

## Chest radiography

Chest radiography is an important part of occupational respiratory disease surveillance for dust-induced interstitial lung diseases (pneumoconioses) caused by a range of agents, including coal mine dust, crystalline silica, and asbestos. The ILO classification system provides a standardized way to classify chest radiographs for the presence and severity of radiographic changes associated with pneumoconiosis.[10] This standardization is critical to achieving reliable, reproducible results for epidemiologic studies. ILO classification for the presence and severity of small opacities seen in pneumoconiosis is done by comparing examinees' chest X-rays with ILO standard images and determining the types of opacities (shape, size) and their level of profusion (density) relative to the standards. Over the years, much RHD work has focused on helping to keep the ILO classification system up to date, and on providing training opportunities and certification testing to help ensure the availability of physicians with documented ability to use the system.

RHD has a long history of efforts to ensure the availability of physicians able to perform ILO classification of chest radiographic images. Working in partnership with the American College of Radiology (ACR) and Johns Hopkins University, ALFORD developed procedures in the 1970s to ensure proficiency in classification of chest radiographs for pneumoconiosis and consistency across readers contributing classifications to the CWHSP. Initially, physicians could become certified to read and classify chest x-rays either by attending a course approved by ALFORD, the first of which were developed and conducted by ACR, or by submitting their classifications of six recent x-rays with increasing severity of pneumoconiosis for review by a panel of radiologists to assure proper classification according to the ILO classification system or the 1968 International Union Against Cancer (UICC)/Cincinnati system for classifying pneumoconioses.[128] These readers were subsequently designated as “first” or “A” readers and their classifications required a second, independent classification by a “final” or “B” reader,” one of a small group of radiologists, selected from among three major medical centers, with extensive experience classifying pneumoconiosis.[129,130]

During the first several years of what is now commonly referred to as the “B Reader” program, a high level of disagreement between readers was observed, which was attributed to poor film quality and inexperience among many readers in the classification of pneumoconioses, indicating the need to develop an expanded training program and proficiency examination.[131,132] Under a government contract, Johns Hopkins University developed a B Reader Certification examination, to be administered by RHD, which was first offered in late 1977.[133] Candidates who successfully pass the exam are certified for four years, and to remain certified, must pass a shorter recertification exam at any point in their fourth year of certification.

A major recent initiative involved updating the ILO classification system to enable classifying modern digital chest radiographic images instead of analog film-based chest radiographs. Prior to 2011, only analog film-based standard images were available from ILO and only analog film based chest radiographs could be classified. RHD collaborated with outside investigators to perform a study documenting that classification of digital chest radiographic images displayed on medical monitors could yield results paralleling those of analog film-based chest radiographs obtained from the same individuals and displayed on view boxes.[134] A subsequent larger reading trial confirmed equivalency of the methods.[135] Based on these findings, NIOSH worked with ILO to update the ILO classification system to allow use of examinee’s digital chest images compared to digitized ILO standard images.[10] A subsequent reading study documented that classifications of digital chest images done using the digitized ILO standard images yielded equivalent findings to older film-based approaches.[136] These efforts provided the scientific foundation for RHD to move the CWHSP to provide surveillance using digital chest images, which now account for more than 90% of the chest radiographic images submitted. A future effort involves collaborating with the ILO in an effort to update the chest images used as standards in the classification from digitized copies of analog film to modern, digitally acquired images.

With the move from using analog film to using digital images for chest radiographic surveillance, the B Reader program is making a parallel change to electronic format. The program now offers the B Reader exam in both analog and digitized format. Also, in collaboration with ACR, RHD is developing a training syllabus and B Reader certification examination using contemporary digitally acquired images.

The number of certified B Readers rose rapidly from the late 1970’s through the mid 1990’s, reaching a high of approximately 750 readers. Although certification and recertification exams continue to be offered to physicians monthly at the imaging laboratory on the NIOSH campus in Morgantown, the total number of B Readers has subsequently declined to fewer than 200 over the past two decades. Although the exact number of B Readers needed in the US is unclear, OSHA's recently promulgated silica rule will likely increase the demand for ILO classification of chest radiographs. The rule requires employers to provide medical surveillance, including periodic chest x-rays to be interpreted and classified by a NIOSH-certified B Reader, for workers exposed to respirable silica above an action level of 25 μg/m$^3$ for ≥30 days per year.[33] We anticipate that transitioning the B Reader Program's learning and certification testing materials to digital format will help to assure an adequate supply of B Readers to meet this new demand.

## Conclusions and future directions

Over the past 50 years, RHD has had many notable accomplishments that form the foundation for the division's future impact on work-related respiratory health. The initial strong focus of the division on respiratory disease in coal miners continues to be relevant with the ongoing occurrence of new pneumoconiosis cases, including severe disease such as PMF. HHEs have played an important role in identifying and characterizing novel respiratory work hazards and will remain an important mechanism for identifying sentinel outbreaks of occupational respiratory disease. Multidisciplinary approaches, including epidemiologic and laboratory studies, have been used to investigate many issues, and will continue to be critical to addressing new and emerging respiratory hazards, whether they be manmade advanced materials or naturally occurring microbial hazards. Surveillance has tracked disease trends, and new data sources, such as data from the healthcare delivery system, promise to improve our understanding of the burden of workrelated respiratory disease. Programs for occupational respiratory health monitoring have improved approaches to spirometry and chest radiography and will continue to evolve with developments in technology for respiratory health assessment. In total, RHD has been impactful and is well poised to continue to improve scientific understanding, inform strategies for prevention, and disseminate information to better prevent work-related respiratory disease into the future.

## Acknowledgments

The authors wish to acknowledge the many people who contributed to RHD's accomplishments over the past 50 years. We thank Kathleen B. Fedan and Dr. Jean M. Cox-Ganser for their thoughtful reviews of the manuscript. The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

## References

1. Laney AS, Weissman DN. Respiratory diseases caused by coal mine dust. J Occup Environ Med 2014;56l10:S18–S22.
2. Kibelstis JA, Morgan EJ, Reger R, Lapp NL, Seaton A, Morgan WK. Prevalence of bronchitis and airway obstruction in American bituminous coal miners. Am Rev Respir Dis 1973;108(4):886–893. [PubMed: 4741884]
3. Seixas NS, Robins TG, Attfield MD, Moulton LH. Longitudinal and cross sectional analyses of exposure to coal mine dust and pulmonary function in new miners. Br J Ind Med 1993;50(10):929–937. [PubMed: 8217853]

4. Graber JM, Stayner LT, Cohen RA, Conroy LM, Attfield MD. Respiratory disease mortality among US coal miners; results after 37 years of follow-up. Occup Environ Med 2014;71(1):30–39. Erratum in: Occup Environ Med. 2014;71(10):738. [PubMed: 24186945]
5. NIOSH. Criteria for a recommended standard: occupational exposure to respirable coal mine dust Cincinnati, Ohio; Washington, DC: US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 1995; DHHS (NIOSH) Publication Number 95–106.
6. NIOSH. Current Intelligence Bulletin 64. Coal mine dust exposures and associated health outcomes - a review of information published since 1995 Cincinnati, Ohio: US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 2011; DHHS (NIOSH) Publication Number 11–172.
7. NIOSH. When a coal miner dies … The National Coal Workers' Autopsy Study Morgantown, WV: US Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 1995; DHHS (NIOSH) Publication No. 95–120.
8. Vallyathan V, Landsittel DP, Petsonk EL. The influence of dust standards on the prevalence and severity of coal worker's pneumoconiosis at autopsy in the United States of America. Arch Pathol Lab Med 2011;135(12):1550–1556. [PubMed: 22129182]
9. Yucesoy B, Johnson VJ, Kissling GE, et al. Genetic susceptibility to progressive massive fibrosis in coal miners. Eur Respir J 2008;31(6):1177–1182. [PubMed: 18256065]
10. International Labour Office (ILO). Guidelines for the use of ILO international classification of radiographs of pneumoconioses, revised edition 2011 Geneva, Switzerland: International Labour Office; 2011 http://www.ilo.org/safework/info/publications/WCMS_168260/lang–en/index.htm. Accessed July 12, 2018.
11. Antao VC, Petsonk EL, Sokolow LZ, et al. Rapidly progressive coal workers' pneumoconiosis in the United States: geographic clustering and other factors. Occup Environ Med 2005;62(10):670–674. [PubMed: 16169911]
12. Laney AS, Attfield MD. Examination of potential sources of bias in the US Coal Workers' Health Surveillance Program. Am J Public Health 2014; 104(1):165–170. [PubMed: 23678894]
13. Blackley DJ, Halldin CN, Laney AS. Resurgence of a debilitating and entirely preventable respiratory disease among working coal miners. Am J Respir Crit Care Med 2014;190(6):708–709. [PubMed: 25221884]
14. Blackley DJ, Crum JB, Halldin CN, Storey E, Laney AS. Resurgence of progressive massive fibrosis in coal miners - eastern Kentucky, 2016. MMWR Morb Mortal Wkly Rep 2016;65(49): 1385–1389. [PubMed: 27977638]
15. Blackley DJ, Reynolds LE, Short C, et al. Progressive massive fibrosis in coal miners from 3 clinics in Virginia. JAMA 2018;319(5):500–501. [PubMed: 29411024]
16. Reynolds L, Halldin CN, Laney AS, Blackley DJ. Coal miner participation in a job transfer program designed to prevent progression of pneumoconiosis, United States, 1986–2016. Arch Environ Occup Health 2017;1–3. doi:10.1080/19338244.2017.1402749.
17. Mine Safety and Health Adminstration (MSHA). Lowering miners' exposure to respirable coal mine dust, including continuous personal dust monitors. Final rule. Fed Regist 2014;79(84): 24813–24994.
18. Banks DE, Bauer MA, Castellan RM, Lapp NL. Silicosis in surface coalmine drillers. Thorax 1983; 38(4):275–278. [PubMed: 6867980]
19. Piacitelli GM, Amandus HE, Dieffenbach A. Respirable dust exposures in U.S. surface coal mines (1982–1986). Arch Environ Health 1990;45(4): 202–209. [PubMed: 2169228]
20. Halldin CN, Reed WR, Joy GJ, et al. Debilitating lung disease among surface coal miners with no underground mining tenure. J Occup Environ Med 2015;57(1):62–67. [PubMed: 25563541]
21. Russell AE, Britten RH, Thompson LR, Bloomfield JJ. The Health of Workers in Dusty Trades. II. Exposure to Siliceous Dust (Granite Industry) Washington, DC: US Public Health Service, 1929; Public Health Bulletin No. 187.
22. Russell AE. The Health of Workers in Dusty Trades. VII. Restudy of a Group of Granite Workers. Washington, DC: US Public Health Service, 1941; Public Health Bulletin No. 269.

23. Costello J, Graham WGB. Vermont granite workers' mortality study. Am J Ind Med 1988;13(4): 483–497. [PubMed: 2834946]
24. Amandus HE, Shy C, Wing S, Blair A, Heineman EF. Silicosis and lung cancer in North Carolina dusty trades workers. Am J Ind Med 1991;20(1):57–70. [PubMed: 1867218]
25. Chen J, McLaughlin JK, Zhang JY, et al. Mortality among dust-exposed Chinese mine and pottery workers. J Occup Med 1992;34(3):311–316. [PubMed: 1312152]
26. Hodous TK, Chen RA, Kinsley KB, et al. A comparison of pneumoconiosis interpretation between Chinese and American readers and classifications. J Tongji Med Univ 1991;11(4):225–229. [PubMed: 1819033]
27. Millar JD. AIHCE Keynote Address: 10% Inspiration. Appl Occup Environ Hyg 1991;6(9): 742–746.
28. Kane F The campaign to end silicosis. Job Safety & Health Quarterly 1997 https://www.osha.gov/dsg/topics/silicacrystalline/endsilicosis.html. Accessed July 12, 2018.
29. NIOSH. NIOSH alert: preventing silicosis and deaths from sandblasting Morgantown, WV: US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 1992; DHHS (NIOSH) Publication Number 92–102.
30. NIOSH. NIOSH alert: preventing silicosis and deaths in rock drillers Morgantown, WV: US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 1992; DHHS (NIOSH) Publication Number 92–107.
31. NIOSH. NIOSH alert: preventing silicosis and deaths in construction workers Morgantown, WV: US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 1996; DHHS (NIOSH) Publication Number 96–112.
32. NIOSH. NIOSH hazard review: health effects of occupational exposure to respirable crystalline silica Cincinnati, OH: US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 2002; DHHS (NIOSH) Publication Number 2002–129.
33. Occupational Safety and Health Administration (OSHA). Occupational exposure to respirable crystalline silica; final rule. Fed Regist 2016;81(58): 16285–16890. [PubMed: 27017634]
34. Lougheed MD, Roos JO, Waddell WR, Munt PW. Desquamative interstitial pneumonitis and diffuse alveolar damage in textile workers. Potential role of mycotoxins. Chest 1995;108(5):1196–1200. [PubMed: 7587416]
35. Kern DG, Crausman RS, Durand KT, Nayer A, Kuhn C 3rd., Flock worker's lung: chronic interstitial lung disease in the nylon flocking industry. Ann Intern Med 1998;129(4):261–272. [PubMed: 9729178]
36. Eschenbacher WL, Kreiss K, Lougheed MD, Pransky GS, Day B, Castellan RM. Nylon flock-associated interstitial lung disease. Am J Respir Crit Care Med 1999;159(6):2003–2008. [PubMed: 10351952]
37. Burkhart J, Jones W, Porter DW, Washko RM, Eschenbacher WL, Castellan RM. Hazardous occupational exposure and lung disease among nylon flock workers. Am J Ind Med 1999;36(Suppl. 1):145–146.
38. Washko RM, Day B, Parker JE, Castellan RM, Kreiss K. Epidemiologic investigation of respiratory morbidity at a nylon flock plant. Am J Ind Med 2000; 38(6):628–638. [PubMed: 11071685]
39. Daroowalla F, Wang ML, Piacitelli C, Attfield MD, K, Kreiss K. Flock workers' exposures and respiratory symptoms in five plants. Am J Ind Med 2005; 47(2):144–152. [PubMed: 15662645]
40. Porter DW, Castranova V, Robinson VA, et al. Acute inflammatory reaction in rats after intratracheal instillation of material collected from a nylon flocking plant. J Toxicol Environ Health A 1999; 57(1):25–45. [PubMed: 10321900]
41. Antao VC, Piacitelli CA, Miller WE, Pinheiro GA, Kreiss K. Rayon flock: a new cause of respiratory morbidity in a card processing plant. Am J Ind Med 2007;50(4):274–284. [PubMed: 17370318]

42. Akpinar-Elci M, Travis WD, Lynch DA, Kreiss K. Bronchiolitis obliterans syndrome in popcorn production plant workers. Eur Respir J 2004;24(2): 298–302. [PubMed: 15332401]
43. Kreiss K, Gomaa A, Kullman G, Fedan K, Simoes EJ, Enright PL. Clinical bronchiolitis obliterans in workers at a microwave-popcorn plant. N Engl J Med 2002;347(5):330–338. [PubMed: 12151470]
44. Hubbs AF, Battelli LA, Goldsmith WT, et al. Necrosis of nasal and airway epithelium in rats inhaling vapors of artificial butter flavoring. Toxicol Appl Pharmacol 2002;185(2):128–135. [PubMed: 12490137]
45. Hubbs AF, Goldsmith WT, Kashon ML, et al. Respiratory toxicologic pathology of inhaled diacetyl in Sprague-Dawley rats. Toxicol Pathol 2008;36(2): 330–344. [PubMed: 18474946]
46. Kanwal R, Kullman G, Piacitelli C, et al. Evaluation of flavorings-related lung disease risk at six microwave popcorn plants. J Occup Environ Med 2006; 48(2):149–157. [PubMed: 16474263]
47. Day G, LeBouf R, Grote A, et al. Identification and measurement of diacetyl substitutes in dry bakery mix production. J Occup Environ Hyg 2011;8(2): 93–103. [PubMed: 21253982]
48. Cummings KJ, Boylstein RJ, Stanton ML, et al. Respiratory symptoms and lung function abnormalities related to work at a flavouring manufacturing facility. Occup Environ Med 2014;71(8):549–554. [PubMed: 24891557]
49. Kreiss K Work-related spirometric restriction in flavoring manufacturing workers. Am J Ind Med 2014; 57(2):129–137. [PubMed: 24265107]
50. Centers for Disease Control and Prevention (CDC). Obliterative bronchiolitis in workers in a coffee-processing facility - Texas, 2008–2012. MMWR Morb Mortal Wkly Rep 2013;62(16):305–307. [PubMed: 23615673]
51. Bailey RL, Cox-Ganser JM, Duling MG, et al. Respiratory morbidity in a coffee processing workplace with sentinel obliterative bronchiolitis cases. Am J Ind Med 2015;58(12):1235–1245. [PubMed: 26523478]
52. Duling MG, LeBouf RF, Cox-Ganser JM, Kreiss K, Martin SB Jr, Bailey RL. Environmental characterization of a coffee processing workplace with obliterative bronchiolitis in former workers. J Occup Environ Hyg 2016;13(10):770–781. [PubMed: 27105025]
53. Hubbs AF, Cumpston AM, Goldsmith WT, et al. Respiratory and olfactory cytotoxicity of inhaled 2,3- pentanedione in Sprague-Dawley rats. Am J Pathol 2012;181(3):829–844. [PubMed: 22894831]
54. NIOSH. NIOSH alert: preventing lung disease in workers who use or make flavorings Cincinnati, Ohio, US Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 2003; DHHS (NIOSH) Publication No. 2004–110.
55. NIOSH. Criteria for a recommended standard: occupational exposure to diacetyl and 2,3-pentanedione Cincinnati, Ohio, US Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 2016; DHHS (NIOSH) Publication No. 2016–111.
56. Cummings KJ, Donat WE, Ettensohn DB, Roggli VL, Ingram P, Kreiss K. Pulmonary alveolar proteinosis in workers at an indium processing facility. Am J Respir Crit Care Med 2010;181(5): 458–464. [PubMed: 20019344]
57. Cummings KJ, Nakano M, Omae K, et al. Indium lung disease. Chest 2012;141(6):1512–1521. [PubMed: 22207675]
58. Cummings KJ, Suarthana E, Edwards N, et al. Serial evaluations at an indium-tin oxide production facility. Am J Ind Med 2013;56(3):300–307. [PubMed: 23109040]
59. Badding MA, Fix NR, Orandle MS, et al. Pulmonary toxicity of indium-tin oxide production facility particles in rats. J Appl Toxicol 2016;36(4):618–626. [PubMed: 26472246]
60. Stefaniak AB, Virji MA, Badding MA, Cummings KJ. Application of the ICRP respiratory tract model to estimate pulmonary retention of industrially sampled indium-containing dusts. Inhal Toxicol 2017;29(4):169–178. [PubMed: 28595469]
61. Cummings KJ, Virji MA, Trapnell BC, Carey B, Healey T, Kreiss K. Early changes in clinical, functional, and laboratory biomarkers in workers at risk of indium lung disease. Ann Am Thorac Soc 2014; 11(9):1395–1403. [PubMed: 25295756]

62. Cummings KJ, Virji MA, Park JY, et al. Respirable indium exposures, plasma indium, and respiratory health among indium-tin oxide (ITO) workers. Am J Ind Med 2016;59(7):522–531. [PubMed: 27219296]

63. Pneumoconiosis Committee. Pathology standards for coal workers' pneumoconiosis. Report of the Pneumoconiosis Committee of the College of American Pathologists to the National Institute for Occupational Safety and Health. Arch Pathol Lab Med 1979;103(8):375–432. [PubMed: 378179]

64. Craighead JE, Abraham JL, Churg A, et al. The pathology of asbestos-associated diseases of the lungs and pleural cavities: diagnostic criteria and proposed grading schema. Report of the Pneumoconiosis Committee of the College of American Pathologists and the National Institute for Occupational Safety and Health. Arch Pathol Lab Med 1982;106(11): 544–596. [PubMed: 6897166]

65. Silicosis and Silicate Disease Committee. Diseases associated with exposure to silica and nonfibrous silicate minerals. Arch Pathol Lab Med 1988;112(7): 673–720. [PubMed: 2838005]

66. Olenchock SA, Lewis DM, Mull JC. Effects of different extraction protocols on endotoxin analyses of airborne grain dusts. Scand J Work Environ Health 1989;15(6):430–435. [PubMed: 2617259]

67. Vallyathan V, Castranova V, Pack D, et al. Freshly fractured quartz inhalation leads to enhanced lung injury and inflammation. Potential role of free radicals. Am J Respir Crit Care Med 1995;152(3): 1003–1009. [PubMed: 7663775]

68. LeBouf R, Simmons M. Increased sensitivity of OSHA method analysis of diacetyl and 2,3-pentanedione in air. J Occup Environ Hyg 2017;14(5): 343–348. [PubMed: 27792470]

69. LeBouf RF, Virji MA, Ranpara A, Stefaniak AB. Air and surface sampling method for assessing exposures to quaternary ammonium compounds using liquid chromatography tandem mass spectrometry. Ann Work Expo Health 2017;61(6):724–736. [PubMed: 28927165]

70. Stefaniak AB, LeBouf RF, Duling MG, et al. Inhalation exposure to three-dimensional printer emissions stimulates acute hypertension and microvascular dysfunction. Toxicol Appl Pharmacol 2017; 335:1–5. [PubMed: 28942003]

71. Park JH, Sulyok M, Lemons AR, Green BJ, Cox-Ganser JM. Characterization of fungi in office dust: comparing results of microbial secondary metabolites, fungal ITS region sequencing, viable culture, and other microbial indices. Indoor Air 2018. doi: 10.1111/ina.12470.

72. Occupational Safety and Health Administration (OSHA). Occupational exposure to cotton dust: notice of the availability of a lookback review pursuant to the Regulatory Flexibility Act and Executive Order 12866. Fed Regist 2000;65(236):76667–76669.

73. NIOSH. Criteria for a recommended standard: occupational exposure to cotton dust Rockville, MD: US Department of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1974; DHEW (NIOSH) Publication No. 75–118.

74. Occupational Safety and Health Administration (OSHA). Occupational exposure to cotton dust. Final standard. Fed Regist 1978;43(122): 27350–27463.

75. Castellan RM, Olenchock SA, Hankinson JL, et al. Acute bronchoconstriction induced by cotton dust: dose-related responses to endotoxin and other dust factors. Ann Intern Med 1984;101(2): 157–163. [PubMed: 6742645]

76. Castellan RM, Olenchock SA, Kinsley KB, Hankinson JL. Inhaled endotoxin and decreased spirometric values. An exposure-response relation for cotton dust. N Engl J Med 1987;317(10): 605–610. [PubMed: 3614274]

77. Petsonk EL, Olenchock SA, Castellan RM, et al. Human ventilatory response to washed and unwashed cottons from different growing areas. Br J Ind Med 1986;43(3):182–187. [PubMed: 3947581]

78. Kutz SA, Mentnech MS, Mull JC, Olenchock SA, Major PC. Acute experimental pulmonary responses to cardroom cotton dust. Arch Environ Health 1980; 35(4):205–210. [PubMed: 6775602]

79. Olenchock SA, Mull JC, Jones WG. Endotoxins in cotton: washing effects and size distribution. Am J Ind Med 1983;4(4):515–521. [PubMed: 6869376]

80. Olenchock SA. Gram-negative bacterial endotoxins in airborne dusts from washed and unwashed cottons. Z Erkr Atmungsorgane 1984;163(3):208–216. [PubMed: 6528651]

81. NIOSH. Current Intelligence Bulletin 56 - Washed Cotton. A review and recommendations regarding batch kier washed cotton Morgantown, WV: US Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 1995; DHHS (NIOSH) Publication No. 95–113.
82. Occupational Safety and Health Administration (OSHA). Occupational exposure to cotton dust. Direct final rule. Fed Regist 2000;65(236): 76563–76567.
83. Amandus HE, Wheeler R. The morbidity and mortality of vermiculite miners and millers exposed to tremolite-actinolite: Part II. Mortality. Am J Ind Med 1987;11(1):15–26. [PubMed: 3028136]
84. Sullivan PA. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a cohort mortality study. Environ Health Perspect 2007;115(4):579–585. [PubMed: 17450227]
85. Attfield MD, Schleiff PL, Lubin JH, et al. The Diesel Exhaust in Miners study: a cohort mortality study with emphasis on lung cancer. J Natl Cancer Inst 2012;104(11):869–883. Erratum in: J Natl Cancer Inst. 2014;106(8):dju192. [PubMed: 22393207]
86. Silverman DT, Samanic CM, Lubin JH, et al. The diesel exhaust in miners study: a nested case-control study of lung cancer and diesel exhaust. J Natl Cancer Inst 2012;104(11):855–868. [PubMed: 22393209]
87. Park JY, Virji MA, Stefaniak AB, et al. Sensitization and chronic beryllium disease at a primary manufacturing facility, part 2: validation of historical exposures. Scand J Work Environ Health 2012;38(3): 259–269. [PubMed: 21847507]
88. Schuler CR, Virji MA, Deubner DC, et al. Sensitization and chronic beryllium disease at a primary manufacturing facility, part 3: exposure-response among short-term workers. Scand J Work Environ Health 2012;38(3):270–281. [PubMed: 21877099]
89. McCanlies EC, Ensey JS, Schuler CR, Kreiss K, Weston A. The association between HLA-DPB1Glu69 and chronic beryllium disease and beryllium sensitization. Am J Ind Med 2004;46(2): 95–103. [PubMed: 15273960]
90. Snyder JA, Demchuk E, McCanlies EC, et al. Impact of negatively charged patches on the surface of MHC class II antigen-presenting proteins on risk of chronic beryllium disease. J R Soc Interface 2008; 5(24):749–758. [PubMed: 17956852]
91. Kreiss K, Fechter-Leggett ED, McCanlies EC, Schuler CR, Weston A. Research to practice implications of high-risk genotypes for beryllium sensitization and disease. J Occup Environ Med 2016;58(9):855–860. [PubMed: 27414009]
92. Cummings KJ, Deubner DC, Day GA, et al. Enhanced preventive programme at a beryllium oxide ceramics facility reduces beryllium sensitisation among new workers. Occup Environ Med 2006; 64(2):134–140. [PubMed: 17043076]
93. Thomas CA, Bailey RL, Kent MS, Deubner DC, Kreiss K, Schuler CR. Efficacy of a program to prevent beryllium sensitization among new employees at a copper-beryllium alloy processing facility. Public Health Rep 2009;124:112–124. [PubMed: 19618813]
94. Bailey RL, Thomas CA, Deubner DC, Kent MS, Kreiss K, Schuler CR. Evaluation of a preventive program to reduce sensitization at a beryllium metal, oxide, and alloy production plant. J Occup Environ Med 2010;52(5):505–512. [PubMed: 20431418]
95. Stefaniak AB, Virji MA, Day GA. Release of beryllium into artificial airway epithelial lining fluid. Arch Environ Occup Health 2012;67(4):219–228. [PubMed: 23074979]
96. NIOSH. NIOSH alert: preventing sensitization and disease from beryllium Exposure Cincinnati, Ohio, US Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 2011; DHHS (NIOSH) Publication No. 2011–107.
97. Balmes JR, Abraham JL, Dweik RA, et al. An official American Thoracic Society statement: diagnosis and management of beryllium sensitivity and chronic beryllium disease. Am J Respir Crit Care Med 2014; 190(10):e34–e59. [PubMed: 25398119]
98. Occupational Safety and Health Administration (OSHA). Occupational exposure to beryllium. Final rule. Fed Regist 2017;82(5):2470–2757. [PubMed: 28071878]
99. Cox-Ganser JM, White SK, Jones R, et al. Respiratory morbidity in office workers in a water-damaged building. Environ Health Perspect 2005; 113(4):485–490. [PubMed: 15811840]

100. Iossifova YY, Cox-Ganser JM, Park JH, White SK, Kreiss K. Lack of respiratory improvement following remediation of a water-damaged office building. Am J Ind Med 2011;54(4):269–277. [PubMed: 21413053]

101. Park JH, Cox-Ganser J, Rao C, Kreiss K. Fungal and endotoxin measurements in dust associated with respiratory symptoms in a water-damaged office building. Indoor Air 2006;16(3):192–203. [PubMed: 16683938]

102. Park JH, Cox-Ganser JM, Kreiss K, White SK, Rao CY. Hydrophilic fungi and ergosterol associated with respiratory illness in a water-damaged building. Environ Health Perspect 2008;116(1):45–50. [PubMed: 18197298]

103. Park JH, Kreiss K, Cox-Ganser JM. Rhinosinusitis and mold as risk factors for asthma symptoms in occupants of a water-damaged building. Indoor Air 2012;22(5):396–404. [PubMed: 22385263]

104. Park JH, Cho SJ, White SK, Cox-Ganser JM. Changes in respiratory and non-respiratory symptoms in occupants of a large office building over a period of moisture damage remediation attempts. PLoS One 2018;13(1):e0191165. [PubMed: 29324816]

105. Park JH, Schleiff PL, Attfield MD, Cox-Ganser JM, Kreiss K. Building-related respiratory symptoms can be predicted with semi-quantitative indices of exposure to dampness and mold. Indoor Air 2004;14(6): 425–433. [PubMed: 15500636]

106. Cox-Ganser JM, Rao CY, Park JH, Schumpert JC, Kreiss K. Asthma and respiratory symptoms in hospital workers related to dampness and biological contaminants. Indoor Air 2009;19(4):280–290. [PubMed: 19500175]

107. Cho SJ, Cox-Ganser JM, Park JH. Observational scores of dampness and mold associated with measurements of microbial agents and moisture in three public schools. Indoor Air 2016;26(2): 168–178. [PubMed: 25650175]

108. NIOSH. NIOSH alert: preventing occupational respiratory disease from exposures caused by dampness in office buildings, schools, and other nonindustrial buildings Cincinnati, Ohio: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 2012; DHHS (NIOSH) Publication No. 2013–102.

109. NIOSH. Work-related lung disease surveillance report, 1991 Morgantown, WV: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 1991; DHHS (NIOSH) Publication No. 1991–113.

110. NIOSH. Work-related lung disease surveillance report, 2007 Morgantown, WV: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 2008; DHHS (NIOSH) Publication No. 2008–143.

111. NIOSH. Welcome to eWoRLD https://wwwn.cdc.gov/eworld/. Accessed July 12, 2018.

112. NIOSH. National Occupational Respiratory Mortality System(NORMS) https://webappa.cdc.gov/ords/norms.html. Accessed July 12, 2018.

113. Centers for Disease Control and Prevention (CDC). Wide-ranging Online Data for Epidemiologic Research (WONDER) https://wonder.cdc.gov/. Accessed July 12, 2018.

114. Baker EL. Sentinel Event Notification System for Occupational Risks (SENSOR): the concept. Am J Public Health 1989;79l:18–20.

115. NIOSH. Sources of data https://wwwn.cdc.gov/eworld/Appendix/SourcesOfData. Accessed July 12, 2018.

116. Department of Health and Human Services (DHHS). State occupational health and safety surveillance program (U60) https://grants.nih.gov/grants/guide/pa-files/PAR-14-275.html. Accessed July 12, 2018.

117. Association of Occupational and Environmental Clinics (AOEC). Epidemiology tools http://www.aoec.org/tools.htm. Accessed July 12, 2018.

118. NIOSH. State-based occupational health surveillance clearinghouse https://wwwn.cdc.gov/niosh-statedocs/Default.aspx. Accessed July 12, 2018.

119. NIOSH. Spirometry training program https://www.cdc.gov/niosh/topics/spirometry/training.html. Accessed July 12, 2018.

120. American Thoracic Society. ATS statement–Snowbird workshop on standardization of spirometry. Am Rev Respir Dis 1979;119(5):831–838. [PubMed: 453705]

121. American Thoracic Society. Standardization of spirometry, 1994 update. Am J Respir Crit Care Med 1995;152(3):1107–1136. [PubMed: 7663792]

122. Redlich CA, Tarlo SM, Hankinson JL, et al. Official American Thoracic Society technical standards: spirometry in the occupational setting. Am J Respir Crit Care Med 2014;189(8):983–993. [PubMed: 24735032]

123. NIOSH. Coal workers' health surveillance program. Coal Miner Spirometry https://www.cdc.gov/niosh/topics/cwhsp/coalminerhealth.html. Accessed July 12, 2018.

124. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. Am J Respir Crit Care Med 1999;159(1):179–187. [PubMed: 9872837]

125. Hnizdo E, Yan T, Hakobyan A, et al. Spirometry Longitudinal Data Analysis Software (SPIROLA) for analysis of spirometry data in workplace prevention or COPD treatment. Open Med Inform J 2010;4: 94–102. [PubMed: 20835361]

126. Hnizdo E The value of periodic spirometry for early recognition of long-term excessive lung function decline in individuals. J Occup Environ Med 2012; 54(12):1506–1512. [PubMed: 23114387]

127. Baughman P, Marott JL, Lange P, et al. Combined effect of lung function level and decline increases morbidity and mortality risks. Eur J Epidemiol 2012; 27(12):933–943. [PubMed: 23238697]

128. Public Health Service. Specifications for medical examinations of underground coal miners. Fed Regist 1970;35(161):13206–13208.

129. Public Health Service. Specifications for medical examinations of underground coal miners. Fed. Regist 1973;38(144):20076–20081.

130. Trout ED, Jacobson G, Moore RT, Shoub EP. Analysis of the rejection rate of chest radiographs obtained during the coal mine "black lung program". Radiology 1973;109(1):25–27. [PubMed: 4783124]

131. Felson B, Morgan WK, Bristol LJ, et al. Observations on the results of multiple readings of chest films in coal miners' pneumoconiosis. Radiology 1973;109(1): 19–23. [PubMed: 4783121]

132. Morgan R, Donner M, Gayler B, Margulies S, Rao P, Wheeler P. Decision process and observer error in the diagnosis of pneumoconiosis by chest roentgenography. Am J Roentgenol Radium Ther Nucl Med 1973;117(4):757–764.

133. Wagner GR, Attfield MD, Kennedy RD, Parker JE. The NIOSH B Reader certification program. An update report. J Occup Med 1992;34(9):879–884. [PubMed: 1447592]

134. Franzblau A, Kazerooni EA, Sen A, et al. Comparison of digital radiographs with film radiographs for the classification of pneumoconiosis. Acad Radiol 2009;16(6):669–677. [PubMed: 19345120]

135. Laney AS, Petsonk EL, Attfield MD. Intramodality and intermodality comparisons of storage phosphor computed radiography and conventional film-screen radiography in the recognition of small pneumoconiotic opacities. Chest 2011;140(6):1574–1580. [PubMed: 21622551]

136. Halldin CN, Petsonk EL, Laney AS. Validation of the International Labour Office digitized standard images for recognition and classification of radiographs of pneumoconiosis. Acad Radiol 2014;21(3): 305–311. [PubMed: 24507420]

**Figure 1.**

Groundbreaking ceremony for the ALFORD building, Morgantown, West Virginia, June 29, 1969. Senator Robert C. Byrd is third from right.


**Figure 2.**
NIOSH Mobile Occupational Safety and Health Unit, used by RHD for enhanced surveillance for coal workers' pneumoconiosis.

**Table 1.**

Directors of NIOSH's Respiratory Health Division and its predecessors.

| | Years of service | Organization |
|---|---|---|
| William K.C. Morgan, MD | 1968–1974 | ALFORD[a] |
| James A. Merchant, MD | 1977–1981 | ALFORD, DRDS[b] |
| Robert E. Glenn, CIH, MPH | 1982–1988 | DRDS |
| Gregory R. Wagner, MD | 1989–2004 | DRDS |
| David N. Weissman, MD | 2005–present | DRDS, RHD[c] |

[a]Appalachian Laboratory for Occupational Respiratory Diseases.

[b]Division of Respiratory Disease Studies.

[c]Respiratory Health Division.