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Occupational History of Coal Mining and its Association with Restrictive and Obstructive Lung Disease Among Males in Two Counties in Rural Appalachian Kentucky

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Abstract

Occupational History of Coal Mining and its Association with Restrictive and Obstructive Lung Disease Among Males in Two Counties in Rural Appalachian Kentucky
By Caileigh McKenna

Objective. Appalachian (Eastern) Kentucky has a high burden of lung disease, partially attributable to significant occupational exposure to coal mine dust in the 7th largest coal-producing state in the United States. Cases of coal workers' pneumoconiosis and its most severe form, pulmonary massive fibrosis, began increasing significantly in Central Appalachia, and Appalachian Kentucky in particular, around 2000. There is a need to better understand the association between coal mine dust lung disease and coal mining exposure in Eastern Kentucky to better guide prevention efforts and operating practices.

Methods. The analysis utilized cross-sectional data from the University of Kentucky, obtained in Harlan and Letcher counties between 2015 and 2017. Multivariable Poisson regression with robust standard errors was performed to assess the association between lung disease, defined by spirometric pattern as obstructive, restrictive, or normal, and coal mining history, defined as never, underground only, surface only, and both types, adjusted for age, education, smoking level, and history of other dusty jobs. Adjusted prevalence ratios (aPR) and 95% confidence intervals (CI) were obtained. The adjusted analyses were further stratified by age and coal mining tenure.

Results. The aPR for the association between obstructive lung disease and a history of underground coal mining, surface coal mining, and underground and surface coal mining were 1.45 (95% CI: 0.94-2.25), 1.20 (95% CI: 0.71-2.02), and 1.51 (95% CI: 0.96-2.38), respectively. The aPR for the association between restrictive lung disease and a history of underground coal mining, surface coal mining, and underground and surface coal mining were 1.18 (95% CI: 0.74-1.90), 0.997 (95% CI: 0.71-2.02), and 1.51 (95% CI: 0.96-2.38), respectively.

Conclusions. The observed effects for this study demonstrated much stronger associations for obstructive lung disease compared to restrictive lung disease and for underground coal mining and both types of coal mining compared to surface coal mining. Generally, the effects of the association for the older age group and longer coal mining tenure group were stronger than the younger age group and shorter mining tenure group, respectively. The analyses were limited by a lack of statistical power and potential survival bias.

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CHAPTER 1: BACKGROUND LITERATURE REVIEW

PUBLIC HEALTH SIGNIFICANCE

Appalachia is a geographical region extending from southern New York to northern Mississippi, comprised of 423 counties in 13 states with a population of 26 million.[1] It is divided into contiguous subregions based on similarities in geographic, sociodemographic, and economic characteristics. The Central Appalachian subregion consists of eastern Kentucky, southern West Virginia, northeast Tennessee, and southwest Virginia.[2,3]

Appalachia, and Central Appalachia in particular, is impacted by long-standing and persistent health and socioeconomic inequities compared to the rest of the United States (US); however, Appalachia and other rural areas have been consistently underrepresented in health outcomes research.[4] It was not until 2004, when the Appalachian Regional Commission (ARC), a federal-state partnership formed in 1965 to promote economic development in the region, commissioned the seminal report "An Analysis of Disparities in Health Status and Access to Health Care in the Appalachian Region," that these disparities were officially documented. The report found that there were significant health disparities in the Appalachian population compared to the rest of the US, including excess mortality and disease burden. The authors noted that there was significant variability in socioeconomic factors, healthcare access, and high-risk behaviors within the region and suggested that health interventions at the local level would likely be most effective.[5,6]

Compared to the rest of the US, Appalachia has increased mortality rates from heart disease (17% higher), cancer (10% higher), chronic obstructive pulmonary disease (COPD) (27% higher), injury (33% higher), stroke (14% higher), diabetes (11% higher), and years of potential life lost (25% higher). In Central Appalachia, the rate of years of potential life lost is 69% higher than in the US overall. Among the five Appalachian subregions, Central Appalachia

has the highest rate of diabetes (13.5%), adult obesity (34.7%), physical inactivity (33.8%), and smoking (25.2%).[6]

Rural and distressed counties in Appalachia demonstrate even greater health disparities when compared to the region's metro counties and non-distressed counties, respectively. Rural dwellers account for 42% of the Appalachian population, compared to just 20% of the nation's.[6] "Distressed" is the worst of five ARC designations used to classify the economic status of Appalachian counties each fiscal year. The ARC creates a composite measure for every county in the US based on its average 3-year unemployment rate, per capita market income, and poverty rate. Each county is then ranked – from most to least economically depressed – as distressed, at-risk, transitional, competitive, and attainment. Distressed counties represent the bottom 10% of the nation's counties.[7,8] There are 81 distressed counties in Appalachia in fiscal year 2022 (October 1, 2021 to September 30, 2022), 81.5% (66) of which are located in Central Appalachia. A majority (72.2%) of the 54 Appalachian Kentucky counties are designated as distressed in fiscal year 2022. Of the 15 non-distressed counties (all designated as at-risk or transitional), 12 (80%) contain distressed areas.[9] These are areas within the county that have a median family income ≤67% of the national average and poverty rate ≥150% of the national average.[7]

Among other disparities, Central Appalachia has a disproportionately high burden of lung disease. This has been attributed to the Region's high rate of smoking, poverty, and unemployment; low educational attainment resulting in low health literacy; high percentage of rural residents; comparatively lower numbers of primary care and subspecialty providers (including pulmonologists); and significant occupational and environmental exposures to dust from underground and surface coal mining activities.[3]

Lung disease secondary to occupational coal mine dust exposure remains a significant health concern in Central Appalachia and Appalachian Kentucky in particular. The Federal Coal Mine Health and Safety Act of 1969 (The Coal Act) was passed to protect coal miners' health by establishing a mandated respirable coal mine dust exposure limit.[10] Coal workers' pneumoconiosis (CWP) and its most severe form, progressive massive fibrosis (PMF), is one of several lung diseases caused by exposure to coal mine dust. Following passage of the Coal Act, CWP and PMF prevalence in coal miners decreased steadily until around 2000, when cases of PMF began increasing in Central Appalachia, but not in other coal-producing regions. Between 1995 and 1999, the prevalence of CWP and PMF reached a nadir of 3.7% and 0.4% among active long-tenured (worked ≥25 years) coal miners and 1.5% and 0.1% among those with work tenure <25 years.[11] Between 2005 and 2009, the states with the highest prevalence of CWP were Kentucky (9%), Virginia (8%), and West Virginia (4.8%). The age-adjusted prevalence of CWP in these three states together and Kentucky alone was 4.5 and 7.6 times greater, respectively, than the prevalence in 12 other coal-producing states.[12]

COAL MINING

The history of coal production in the United States. Coal production first gained importance in the US economy during the American Industrial Revolution of the nineteenth century. During this time, it was used as the primary source of fuel for steam locomotives and in manufacturing the iron and steel that was used to build railroad tracks.[13] The demand for coal increased in the 1800s and 1900s as more uses were discovered. Coal production similarly increased during this time and into the twenty-first century.[13,14] 481 million short tons (one short ton equals 2,000 pounds)[15] of coal were produced in the U.S in 1949. Production fluctuated in the 1950s and early 1960s and then began increasing steadily from 420 million tons in 1960 to

1.1 billion tons in 1999. It peaked at 1.17 billion tons in 2008[14] and has continued to decrease since then, most significantly since 2014. One billion tons produced in 2014 decreased to 706 million in 2019 to just 535 million in 2020.[14] The 24% decrease in coal production between 2019 and 2020 has been attributed to lower natural gas prices and the coronavirus disease 2019 pandemic, which led to decreased global and national demand and resulted in extended mine closures to protect workers' safety.[16]

Coal mining by region. In 2021, coal was produced in 22 states in three major regions of the US – the Appalachian, Interior, and Western coal regions. The Appalachian coal region includes Alabama, Eastern Kentucky, Maryland, Ohio, Pennsylvania, Tennessee, Virginia, and West Virginia.[17] The region produced 138.5 million short tons of coal in 2020, representing 25.9% of the national total.[14] West Virginia is the most productive state in the region and the second most productive state overall, producing 13% of the nation's coal in 2020.[17]

The Interior coal region includes Arkansas, Illinois, Indiana, Kansas, Louisiana, Mississippi, Missouri, Oklahoma, Texas, and Western Kentucky.[17] Seventeen percent of the nation's coal is produced in the Interior region. Illinois is the most productive state in the region.[17]

The Western coal region includes Alaska, Arizona, Colorado, Montana, New Mexico, North Dakota, Utah, Washington, and Wyoming.[17] Fifty-seven percent of the nation's coal is produced here. Wyoming is the US's top coal-producing state, producing 41% of the nation's coal in 2020.[17]

In 2020, there were an average of 42,159 coal mine employees in the US. Underground coal mines employees made up 60.1% (25,356) of the workforce while the other 39.8% (16,761) worked at surface coal mines.[14]

Coal mining in Kentucky. Kentucky has historically been one of the nation's top coal-producing states, producing 24.2 million short tons of coal in 2020, 34.4% (8.3 million short tons) of which were produced in Eastern Kentucky. Kentucky's total coal production decreased by 32.7% from 2019 to 2020, mirroring the trend in the region overall. Kentucky was the 5th largest coal-producing state in the US in 2019, producing 5.1% of the US's total coal. The decline in production in 2020, however, saw Kentucky reduced to the nation's 7th largest coal-producing state, though still producing 4.5% of the US's total coal.[14]

In 2020, there was an average of 4,006 coal mine employees in Kentucky. Underground coal mine employees made up 75.4% (3,020) of the workforce, while 24.6% (986) worked in surface coal mines. The majority of Kentucky's coal mine employees (58.9% [2,359]) were employed in Eastern Kentucky.[14]

Coal mining methods. There are two main ways that coal is extracted from the earth. Surface mining is utilized when coal seams lie <200 feet underground and involves removing the overlying earth, rocks, and vegetation (overburden) to expose the coal. Underground mining is required when coal is deeper under the earth (≥200 feet).[18]

Room and pillar mining and longwall mining are the most common methods utilized in underground coal mining. The room and pillar method involves mining rooms of coal while leaving pillars in place in-between rooms to support the roof from collapse. Coal is most often mined using a continuous mining machine that cuts and frees it from the coal face and surrounding rock. As mining progresses, roof bolters follow behind, placing long steel bolts into the ceiling for further reinforcement. Longwall mining utilizes a machine to cut a wide coal face while hydraulic supports maintain the roof above the working area. As the machine progresses along the coal face, the roof is allowed to collapse in its wake.[19] In 2020, 38.1% of Kentucky's

97 coal mines and 35.6% of Eastern Kentucky's 90 coal mines were underground, while the majority were surface mines.[14]

Coal rank. Coal can be categorized according to rank, which is determined by its carbon content and the amount of heat energy it produces. In order of descending carbon content, the 4 ranks of coal are anthracite, bituminous, subbituminous, and lignite. Most coal mined in the US is bituminous and subbituminous. In 2020, all of Kentucky's 97 mines produced bituminous coal.[14] Kentucky produced 10% of the US's bituminous coal that year.[17]

PULMONARY FUNCTION AND SPIROMETRY

Spirometry. Pulmonary function tests (PFTs) assess lung function impairment in those with known or suspected lung disease. They are clinically useful as they assist in assessing the severity of lung impairment and the degree to which this impairment may affect daily functioning. Spirometry is the most commonly utilized pulmonary function test and is used to obtain lung volumes (in liters) and flow rates. It is used clinically in diagnosing lung disease, monitoring lung function and disease progression in those with known lung disease, assessing disability and impairment secondary to lung disease, and for research purposes.[20,21]

Clinically important lung volumes obtained via spirometry include total lung capacity (TLC), the volume in the lungs after a maximal inspiration, residual volume (RV), the volume left in the lungs after a maximal expiration, vital capacity (VC), the volume expired after maximal inspiration, and functional residual capacity (FRC), the volume left in the lungs following a normal quiet expiration.[21]

Flow rates are measured during forceful inspiration and expiration with maximal effort. They are quantified by forced vital capacity (FVC), the maximal volume forcefully exhaled after maximal inhalation, and forced expiratory volume in one second (FEV_1), the volume of air

forcefully exhaled in the first second after maximal inspiration. The FEV_1/FVC ratio is used to diagnose airflow obstruction.[20]

Spirometry interpretation. To assess whether spirometry measurements fall within or outside the normal range, they can be compared with established standard reference values based on birth sex, race/ethnicity, age, and height that have been obtained from large population studies of healthy nonsmoking individuals.[21] The Global Lung Function Initiative (GLI) 2012 reference values for FEV_1, FVC, and the FEV_1/FVC ratio are currently the most widely utilized standards in the literature.[22] Spirometry measurements can also be interpreted using the lower limits of normal (LLN), which classifies values as abnormal when they are less than the lower 5th percentile of the reference group value.

In general, a normal FEV_1/FVC ratio is ≥0.70, meaning that a healthy individual should expire ≥70% of the FVC during the first second of forced expiration. However, because the ratio can decrease with age, it should be considered abnormal if it falls outside the lower limit of normal for that age, regardless of its actual value. Abnormal spirometry is typically categorized as either obstructive or restrictive.[21]

Obstructive pattern of pulmonary function impairment. Obstructive spirometry occurs when there is an obstruction to expiratory airflow, typically due to increased airway resistance. This results in a decreased FEV_1/FVC ratio of <0.70, generally, or more accurately, less than the LLN calculated using standardized reference values. Maximal mid-expiratory flow rate (MMFR), the rate of airflow during the middle 50% of expiration, is also decreased. An obstructive pattern is typically seen with chronic obstructive pulmonary disease (COPD) and asthma. Emphysema, a COPD subtype, leads to destruction of the alveolar walls, decreasing radial traction during expiration, which results in decreased lung stiffness and increased

collapsibility. This leads to hyperinflation and air trapping, thereby increasing TLC, FRC, and RV.[21,23]

Restrictive pattern of pulmonary function impairment. Restrictive lung disease occurs when lung stiffness is increased, commonly due to inflammation and fibrosis in the lungs. Though expiratory airflow is normal, the decreased lung compliance limits the volume of air that can be taken in during inspiration. This results in decreased TLC, TV, VC, and FRC. The FEV_1/FVC ratio is normal at ≥0.70, generally, or more accurately, greater than the LLN calculated using standardized reference value, as is MMFR, because expiratory airflow is preserved.

A restrictive pattern is caused by two categories of disease: intrinsic and extrinsic. Intrinsic restrictive lung diseases involve the pulmonary parenchyma, whereas extrinsic restrictive lung diseases originate outside the lung, causing decreased pulmonary compliance via dysfunction of the chest wall. An intrinsic restrictive pattern is caused by a spectrum of diseases known as interstitial lung diseases (ILDs) that result from pathologic destruction of the lung tissue from varying causes. ILDs can be caused by specific exposures, most commonly occupational or environmental dusts, drugs, and radiation, or be idiopathic, like idiopathic pulmonary fibrosis (IPF) and sarcoidosis. Extrinsic causes of restrictive lung disease include neuromuscular diseases like muscular dystrophy and amyotrophic lateral sclerosis, obesity, and pleural effusion.[21,24,25]

Spirometry pattern in coal mine dust lung disease (CMDLD). Coal mine dust lung disease (CMDLD) is a heterogenous spectrum of occupational respiratory diseases caused by exposure to respirable coal mine dust. CMDLD includes coal workers' pneumoconiosis (CWP),

silicosis, mixed-dust pneumoconiosis (MDP), dust-related diffuse fibrosis (DDF), and chronic obstructive pulmonary disease (COPD).[26-30]

Pulmonary function impairment in CMDLD can be restrictive, obstructive, or mixed, reflecting the heterogeneity of the disease spectrum. CMDLD cannot be ruled out by any specific spirometric pattern. Pneumoconioses are a type of ILD caused by chronic organic or inorganic dust inhalation, and thus present with a restrictive spirometry pattern. Exposure to respirable coal mine dust causes 3 nodular pneumoconioses: CWP, silicosis, and MDP. DDF is a pattern of lung fibrosis caused by respirable coal mine dust. It is clinically and pathologically very similar to IPF, another ILD, and thus also presents with a restrictive spirometry pattern. COPD, which may also be caused by exposure to respirable coal mine dust with or without a history of smoking, causes airway obstruction, and thus presents with an obstructive spirometry pattern. A mixed spirometry pattern may be observed when one of the restrictive CMDLDs is comorbid with COPD.[19,26,31]

COAL MINE DUST LUNG DISEASE

Introduction to CMDLD. As described above, CMDLD is a heterogenous spectrum of restrictive and obstructive occupational respiratory diseases caused by exposure to respirable coal mine dust. Restrictive CMDLDs include CWP, silicosis, MDP, and DDF. COPD is the only obstructive CMDLD. Both underground and surface coal miners are at risk for developing CMDLD, which can result in respiratory impairment, disability, and premature death.[26-30,32]

CMDLD prevention and industry regulation. There is no cure for CMDLD, so prevention is the primary method of control.[27] Primary prevention is achieved by reducing miners' exposure to respirable coal mine dust. This exposure is federally regulated by the Mine Safety and Health Administration (MSHA). The enforceable air concentration limit is 1.5 mg/m^3

for surface and underground mines and 0.5 mg/m^3 for underground mine intake air and for miners with evidence of pneumoconioses (referred to as Part 90 miners).This limit applies only if respirable crystalline silica (RCS) content in the sample is <5%.[33] The National Institute for Occupational Safety and Health's (NIOSH) recommended exposure limit (REL) for coal mine dust is lower at 1 mg/m^3, expressed as a time-weighted average (TWA) of up to 10 hours. Career exposure maintained at even NIOSH's REL doesn't completely eliminate the lifetime risk of developing CMDLD. Therefore, minimizing workers' coal mine dust exposure as far below the enforceable limit as feasible is recommended.[32] This is achieved through use of engineering controls (primarily ventilation systems and water sprays) and work practices, routine monitoring of miners' dust exposure, and miner participation in NIOSH's Coal Workers' Health Surveillance Program (CWHSP).[28]

To monitor exposure levels, MSHA requires underground coal miners exposed to the highest concentrations of coal mine dust and Part 90 miners to wear continuous personal dust monitors (CPDM) for their entire work shift. MSHA inspectors use single, full-shift samples to determine a mine's compliance with the dust standard. CPDMs detect coal mine dust concentrations in real time, allowing miners to act immediately upon exposure to hazardous levels. MSHA inspects underground and surface mines four and two times a year, respectively, to ensure health and safety compliance.[34,35]

Secondary and tertiary prevention of CMDLD is achieved through NIOSH's CWHSP and Part 90 of the Federal Coal Mine Health and Safety Act of 1969 (the Coal Act). CWHSP is a free, voluntary respiratory health surveillance program offered to all surface and underground coal miners. Through the CWHSP's Coal Workers' X-ray Surveillance Program (CWXSP), underground and surface coal miners and contractors are offered health screenings that include a

chest X-ray (CXR), lung function test, respiratory assessment questionnaire, and occupational history. All coal miners are required to have an initial chest X-ray (CXR) upon employment and are eligible and encouraged to participate in CWHSP every 5 years thereafter throughout their working career, ideally ensuring development of respiratory disease is detected early. Part 90 of the Coal Act outlines procedures whereby miners with evidence of pneumoconiosis may request transfer to an area of the mine where the respirable coal mine dust concentration is maintained at ≤0.5 mg/m^3 while retaining their regular pay. Though Part 90 transfer could play a significant role in preventing progression of disease, only 14.4% of eligible miners took advantage of this option from 1986 to 2016[26-28,36-39].

RESTRICTIVE COAL MINE DUST LUNG DISEASES

Introduction. The restrictive CMDLDs include the pneumoconioses (CWP, silicosis, and MDP) and DDF.

Radiographic classification of pneumoconioses. The radiographic opacities typical of pneumoconioses have been well described in the literature and are classified using the International Labour Office (ILO) 2011 Classification of Radiographs of the Pneumoconioses, which allows for accurate, consistent, and reproducible documentation of these abnormalities for use in epidemiologic research, surveillance, and clinical care. The ILO Classification consists of 22 standard radiographs and instructions for their use, which categorize pneumoconioses as simple or complicated depending on disease severity. The chest radiographs of simple pneumoconioses are divided into four major categories (0, 1, 2, and 3) based on increasing profusion, or concentration, of small opacities (≤1 cm) in the upper, middle, and lower lung zones, as determined via comparison to the standard radiographs. Each major category is further divided into 3 subcategories, providing 12 overall categories used to classify simple

pneumoconioses. Small opacities are further identified by shape and size. Small, rounded opacities are denoted by increasing diameter as P, Q, or R, while linear/irregular opacities are denoted by increasing width as S, T, or U. Complicated pneumoconioses are defined by the presence of one or more large opacities, which exceed 1 cm in their longest dimension. Large opacities are categorized as A, B, or C based on increasing size of their longest dimension. Complicated pneumoconioses are also referred to as progressive massive fibrosis (PMF).[26,40-42]

Coal workers' pneumoconiosis (CWP). CWP is an interstitial lung disease caused by chronic exposure to respirable coal mine dust. It is generally diagnosed clinically via a history of occupational exposure, respiratory symptoms, and consistent findings on chest radiograph. Simple CWP has classically been associated with small round opacities in the upper lung zone, but the presence of irregular opacities in the lower lung zone is also common and does not rule out a diagnosis of CWP. Patients with ≥10 years of coal mine dust exposure and a clinical picture typical of CWP can be diagnosed without lung biopsy. The latency period is typically between 10 and 20 years, although a more aggressive, rapidly progressive form of CWP can develop within 5 years.[27,30,31]

Complicated CWP, or PMF, generally develops in miners with simple CWP, though it has also been observed in those without prior radiographic evidence of simple CWP[28] and can progress in the absence of continued coal mine dust exposure[43]. It is characterized by extensive fibrosis that can result in necrosis and cavitation, which significantly affects lung function. Simple CWP stage and progression to PMF are associated with cumulative dust exposure (total overall and intensity of exposure), proportion of respirable crystalline silica (RCS) in respirable dust, age, immunological factors, tuberculosis infection, and radiographic severity.[43,44]

Coal mine dust composition. Coal is a sedimentary rock formed over many years via the decomposition of organic matter. Its major component is carbon, though it also contains many other minerals, including clay minerals like kaolinite and illite, carbonates like calcite, sulfides like pyrite, and quartz. Coal mine dust is the heterogeneous mixture of coal, silica, and other airborne particulates that are generated in the process of coal mining. It is estimated that about 40-95% of coal mine dust in underground mines is comprised of coal. The amount of coal in surface coal mine dust is highly variable. Coal mine dust composition can vary considerably by geography, dust particle size, coal rank, and the composition of the rock surrounding the coal seam.[32,45]

CWP pathophysiology. Mucociliary clearance (MCC) is the physiological mechanism responsible for clearing dust, debris, and pathogens from the conducting airways. Mucus secreted from goblet cells lining the upper airways traps dust and motile cilia beat continuously to clear these secretions from the airways.[46] MCC is typically highly effective at clearing the airways when functioning normally, however, with prolonged and excessive dust exposure, this system can become overwhelmed, allowing respirable coal mine dust to enter the distal airways and lung parenchyma. Dust here is cleared by alveolar macrophages, which can similarly become overwhelmed with prolonged and excessive dust exposure. This leads to accumulation of coal mine dust and dust-filled macrophages in the walls of the small airways and interstitium.[47]

Pathologically, simple CWP is characterized by the coal macule, a lesion comprised of dust laden macrophages and minimal fibrosis that causes airspace dilation in affected respiratory bronchioles and alveolar ducts. The coal macule represents a focal emphysema that is considered a type of centrilobular emphysema. Coal macules are nonpalpable, irregularly shaped, and 1-5

mm in length. They are not visible on CXR nor are they associated with pulmonary symptoms. Continued dust exposure and impaired dust clearance leads to an increased number of coal macules and facilitates their development into larger, palpable, and fibrotic nodules. Enlargement and coalescence of these nodules to form lesions of ≥1 cm in diameter represents disease progression to complicated CWP, also known as progressive massive fibrosis (PMF).[43,47-49]

Clinical presentation. Early or mild simple CWP is typically asymptomatic.[43] As disease progresses, miners develop cough (productive or non-productive), wheezing, and shortness of breath (SOB).[28]

Silicosis. Silicosis is a pneumoconiosis caused by inhalation of respirable crystalline silica (RCS). Crystalline silica, a compound of silicon and oxygen, is a major component of Earth's crust, and most commonly found in nature as quartz. Crystalline silica is ubiquitous in the environment in soil and rocks and therefore represents a significant exposure risk in mining, among other industries. Silicosis is not usually seen as an isolated disease in coal mine workers, but rather in conjunction with CWP. As crystalline silica is found in rock, overburden, and ore, both underground and surface coal miners can be exposed during both mine construction and ore mining when crystalline silica becomes airborne during drilling, blasting, ore processing, and other disruptive processes.[43,50]

Three forms of silicosis have been described in the literature. Acute silicosis, or silicoproteinosis, is uncommon, occurs weeks to 5 years after a high dose exposure to RCS, and is rapidly progressive with poor long-term outcomes. Pathologically, the alveoli fill with eosinophilic protein rich fluid and excess surfactant secreted by hypertrophic pneumocytes. Reactive oxygen species formed from the mechanically broken silica likely induce a strong immunological reaction and lead to cellular and DNA damage. Classically, acute silicosis has

been observed with occupational sandblasting and rock drilling and in surface drillers specifically. Symptoms of acute silicosis include shortness of breath (SOB), cough, pleuritic chest pain, and constitutional symptoms like fever, fatigue, and unintentional weight loss. Acute silicosis can rapidly progress to respiratory failure, cor pulmonale, and death.[43,51,52]

Chronic silicosis, also known as nodular or classic silicosis, is the most common type. It develops after 10 or more years of chronic exposure to RCS, though it may take up to 45 years for symptoms of cough and SOB to develop. Histologically, chronic silicosis is characterized by silica nodules that may progress to interstitial fibrosis. Accelerated silicosis develops within 5-10 years of exposure, likely due to higher exposure concentration.[28,51,52]

Like CWP, chronic silicosis is classified using the ILO classification and can be characterized as simple or complicated. It can also progress to PMF. Silicosis cannot be easily distinguished from CWP radiographically or clinically, though research has shown that r-type opacities, small (≥3-10 mm) rounded opacities as defined by the ILO Classification, are more commonly associated with silicosis.[26] Silicosis is also diagnosed similarly, based on a history of significant, chronic, and often occupational silica exposure, consistent clinical presentation, and CXR with nodular opacities.[19]

Mixed-dust pneumoconiosis. MDP results from chronic exposure to both respirable coal mine dust and RCS. It cannot be distinguished from CWP or silicosis radiographically or symptomatically and can only be diagnosed pathologically.[26]

Dust-related diffuse fibrosis (DDF). DDF is a pattern of lung fibrosis associated with exposure to respirable mineral dust, including RCS and coal mine dust, that has clinical similarities to idiopathic pulmonary fibrosis (IPF) and pathologic similarities to usual interstitial pneumonia (UIP), the histopathological findings typical of IPF. These include progressive

dyspnea, clubbing, inspiratory basilar crackles, restrictive lung defect, decreased lung diffusing capacity, and honeycombing primarily in the lower lung zone. Irregular opacities are the most common finding on CXR. DDF can also coexist with nodular disease typical of the pneumoconioses discussed above. A thorough occupational history is key to differentiating DDF from IPF.[19,26,53]

OBSTRUCTIVE COAL MINE DUST LUNG DISEASE

Introduction. The only obstructive CMDLD is COPD, which includes subtypes chronic bronchitis and emphysema.

Chronic obstructive pulmonary disease (COPD). COPD is a heterogenous pulmonary syndrome characterized by airflow obstruction that is not completely reversible with bronchodilator administration. It has historically been characterized by two subtypes, chronic bronchitis and emphysema, although the current definition of COPD does not incorporate these distinctions as patients commonly present with a mix of symptoms.[54-58]

Seventy-five percent of COPD cases in the United States are caused by cigarette smoking, but exposures to indoor biomass smoke and occupational dust (including coal mine dust) and fumes account for about 25% of COPD cases in never smokers. Typically, the airflow obstruction in COPD is caused by increased airway resistance resulting from lung damage caused by abnormal inflammation triggered by cigarette smoke and other harmful exposures.[54]

Chronic bronchitis. Chronic bronchitis is a clinical diagnosis defined as chronic cough and sputum production on most days for at least 3 months per year for ≥2 years. A clinical diagnosis of chronic bronchitis does not necessarily require strict adherence to this time frame. Pathologically, it is characterized by increased mucus production secondary to enlarged mucous glands and decreased mucociliary clearance secondary to inflammation, which results in

decreased diameter of the small airways (bronchioles with <2 mm diameter). These changes account for the associated clinical symptoms but do not alone cause significant airflow obstruction. Mild airway obstruction in chronic bronchitis is due to small airway disease (inflammation/fibrosis), whereas more significant obstruction is observed with comorbid emphysema.[23]

Emphysema. Emphysema is pathologic diagnosis characterized by destruction of the alveolar walls in the lung parenchyma, leading to air space enlargement distal to the terminal bronchiole. Pathologically, it is hypothesized that the increase in inflammatory cells secondary to smoking leads to increased release of elastase and matrix metalloproteinases far above normal levels. These enzymes, which can break down components of the alveolar walls, overwhelm the normal corresponding inhibitory enzymes in the lung, resulting in overall destruction of alveolar wall. There are several pathologic emphysema types based on which area of the acinus is involved. The acinus is the functional unit of the lung and comprises the structures distal to terminal bronchiole, including the respiratory bronchioles, alveolar ducts, and alveolar sacs. Smoking and coal mine dust tend to cause centrilobular (or centriacinar) emphysema, which affects the proximal acinus (the respiratory bronchiole). Panacinar emphysema, conversely, affects the entire acinus.[23]

Clinical features. The most common symptoms of COPD are dyspnea, cough, and sputum production.

COPD and coal mining. Since the nineteenth century, coal mine dust was presumed, and later proven, to cause CWP. The term “black lung” was first coined in 1831 and CWP in 1942, when it was used to describe the lung disease caused by the dusty conditions in coal mines.[59] The understanding that coal mine dust also causes COPD is a much more recent discovery, which

was cemented around 1995, following 3 decades of research into the topic, as outlined in the seminal review by Coggon and Newman Taylor.[60] Previously, it was believed that COPD symptoms in coal miners were mostly attributable to smoking, as up to 80% of coal mining cohorts reported smoking.[61]

More recent evidence has shown that the prevalence of chronic bronchitis increases with increasing coal mining tenure and cumulative coal mine dust exposure. Chronic bronchitis in miners is associated with decreases in FEV_1. Coal mine dust causes emphysema independently of cigarette smoking and has an additive effect to smoking in causing emphysema.[26]

COAL WORKERS' PNEUMOCONIOSIS EPIDEMIOLOGY AND TRENDS

CWP epidemiology and trends. The Federal Coal Mine Health and Safety Act of 1969 (the Coal Act) was passed to improve the health and safety of American coal miners. Enacted on March 30, 1970, the Coal Act mandated respirable coal mine dust exposure limits in underground mines, established the CWHSP, and provided federal benefits for miners with CWP, known legally as black lung disease.[10] Prior to the passage of the Coal Act, coal mine explosions had resulted in many fatalities and CWP and PMF caused significant morbidity and premature mortality in coal miners.[62]

Between 1970 and 1974, the prevalence of CWP and PMF among active long-tenured (worked ≥25 years) coal miners was 32.5% and 3.3%, respectively.[62] After the Coal Act was enacted, the prevalence of CWP and PMF in all tenure groups declined significantly from 1969 through 1995. CWP cases stabilized in the late 1990s but began increasing in 2000 in miners who had worked under the Coal Act dust exposure limits for the majority or entirety of their careers. Between 1995 and 1999, the prevalence of CWP and PMF was 3.7% and 0.4% among active long-tenured coal miners and 1.5% and 0.1% among those with work tenure <25 years .[11]

Further investigation revealed that the prevalence of PMF in younger miners (<50 years old) with tenure <25 years had begun increasing significantly, particularly in the central Appalachian region, especially eastern Kentucky, western Virginia, and southern West Virginia.[62]

A 2005 study utilized CWXSP and MSHA Miner's Choice Program data from 1996 through 2002 to determine the prevalence of CWP among participating underground coal miners at the time. The authors further assessed CWP progression in miners with at least two CXRs whose most recent one showed at least category 1/1 CWP. Cases of rapidly progressive pneumoconiosis (RPP), a severe, rapidly progressing variant of CWP, were defined by an increase of more than one ILO subcategory over 5 years after 1985 and/or the development of PMF after 1985. The year 1985 was used to allow for a >10-year latency period from enactment of the Coal Act. They found that 35.4% of CWP cases were rapidly progressive and 14.8% of the RPP cases demonstrated PMF. The miners with RPP were significantly younger than those without rapid progression (48 vs. 51 years), though there was no significant difference in tenure between the groups. Further, RPP cases were significantly more likely to work in smaller mines (<50 employees) and have longer mean tenure of jobs at the face of the mine. Mapping of the data revealed clustering of RPP cases in eastern Kentucky and western Virginia. Harlan and Letcher counties, specifically, were among 25 counties where at least 40% of CWP cases were rapidly progressive.[48,49,63]

A follow up study was published in 2012 utilizing data from 2005 to 2009, obtained via the Enhanced Coal Workers' Health Surveillance Program (ECWHSP). The ECWHSP was established by NIOSH in 2005 to further investigate the previously observed increase in RPP cases among underground coal miners. The ECWHSP sent mobile units to obtain CXRs near

mines in 15 states where RPP cases had been identified. The states with the highest prevalence of CWP were Kentucky (9%), Virginia (8%), and West Virginia (4.8%). The age-adjusted prevalence of CWP in these three states together and Kentucky alone was 4.5 and 7.6 times greater, respectively, than the prevalence in the 12 other participating states. Consistent with previous research, CWP and PMF were more prevalent in smaller mines (<155 employees). Additionally, the median age of miners with CWP in Kentucky, West Virginia, and Virginia (52 years) was significantly less than that in the other states (55 years).[12]

The most recent tenure-stratified CWP prevalence estimates for underground coal miners were published in 2018. Between 2010 and 2015, CWP prevalence increased in miners of all tenure groups >10 years (10-14 years, 15-19 years, 20-24 years, and 25+ years). The national CWP prevalence in long-tenured miners and miners with 20 to 24 years of tenure was >10% and >5%, respectively. In central Appalachia, 20.6% of long-tenured coal miners had evidence of CWP. PMF was seen in 4.5% of these miners, which represented the highest documented level since 1990.[64]

The Black Lung benefits program (BLBP) was established as part of the Coal Act to provide payments to coal miners with total disability resulting from CWP.[65] As CWHSP data only represents working coal miners, BLBP data has been used to determine the prevalence of CWP among former coal miners. One study utilizing BLPB data from 2001 to 2013 found that the prevalence of category 1 CWP and advanced CWP (≥category 2) among claimants was 33.8% and 4.5%. The overall prevalence of PMF was 4% but was significantly higher in the youngest quartile (18 to 56 years old) at 5.5% compared to the oldest quartile (70 to 100 years old) at 3.3%. Additionally, in former miners aged 49 to 60, those who began working after 1970

when the Coal Act was enacted had a significantly higher prevalence of advanced CWP and PMF compared to those who began work prior to 1970 at 8.3% vs. 4%, respectively.[66]

It is likely that CWP prevalence estimates utilizing CWHSP data underestimate the true prevalence of the disease among American underground coal miners, as participation in the CWHSP is voluntary and is estimated to be between 30 and 40%. Between 2011 and 2016, only 17% of Kentucky coal miners participated in the CWHSP. This is further evidenced by discoveries of disease clusters that were not identified via the CWHSP. For example, 60 cases of PMF in active and former coal miners were diagnosed at a radiology clinic in eastern Kentucky between January 2015 and August 2016 and were brought to NIOSH's attention by the radiologist who had noted the increase in his practice.[64,67]

The cause of the increase in CWP prevalence and severity among active coal miners in the US was likely multifactorial. Several factors were proposed as potential explanations including the idea that the coal mine dust permissible exposure limit set forth by the Coal Act in 1969 was inadequate for preventing occupational disease and/or it was not being adequately enforced. The latter was supported by the increased CWP prevalence observed in smaller mines, which was hypothesized to result from smaller mines' lack of resources to adequately reduce dust exposures and prevent disease. Increased exposure to RCS resulting from thin-seam mining, which requires removal of large amounts of overlying and underlying rock, was also hypothesized to have contributed to the increase in CWP, particularly because thin-seam mining is especially common in Appalachia compared to other coal regions. Underground coal miners in Appalachia had seen a sixfold increase in the prevalence of r-type opacities (associated with silicosis) on CXR since 1980, while the prevalence in the rest of the US remained stable. Data showed that coal miner's working hours (10 or 12 hours shifts) and weekend overtime had been

increasing in the decades following the Coal Act. Longer work hours were believed to increase miners' dust exposure and reduce the time in between shifts necessary for adequate dust clearance from the lungs. Lack of participation in the CWHSP was also cited as a potential contributing factor.[36,62,66]

In 1995, NIOSH released its Criteria for Occupational Exposure to Respirable Coal Mine Dust, in which they proposed decreasing the recommended exposure limit of coal mine dust to 1 mg/m^3 (expressed as up to a 10-hour TWA) from 2 mg/m^3, which it had been for 45 years since passage of the Coal Act. Following the increase in CWP beginning in the 2000s, MSHA eventually decreased the coal mine dust permissible exposure limit to 1.5 mg/m^3 (if sample silica content is <5%) in 2014.[32,34]

RISK FACTORS FOR COAL MINE DUST LUNG DISEASE

Risk factors for CMDLD. Cumulative coal mine dust exposure is the main determinant of risk for CMDLD. As this is difficult to directly measure and estimate, variables that impact the frequency, intensity, and/or duration of coal mine dust exposure are used as proxies for estimation. These factors include employment tenure, miner age, working hours, job type, mine type, mine size, and personal use of respiratory protection. Exposure to higher concentrations of RCS and higher coal rank are also risk factors for development of CMDLD. The effect of cumulative coal mine dust exposure on emphysema, specifically, is independent of smoking, and has an additive effect with smoking.[26,31,33,36,42]

In underground coal mining, jobs at the mine face, where coal is extracted, typically experience the highest respirable dust exposure. These high-risk jobs include longwall shearer operators, jack setters, continuous miner operators, and roof bolter operators.[33] Underground coal

miners generally have a higher risk of exposure than surface coal miners because of the high dust concentration within an enclosed space, whereas outside air dilutes dust levels at surface mines.[43]

However, surface miners are still at risk of developing CMDLD. Drilling and blasting rocky overburden can lead to very high exposures to RCS for machinery operators. Although these operators typically work in enclosed ventilated cabs to minimize dust exposure, nearby surface miners out in the open may also be exposed to excessive amounts of this dust.[33,42] Among a sample of CXRs obtained between 2010 and 2011 from 2,328 working surface coal miners, 2% (46) of the 2,257 surface miners who had worked for ≥1 year had CWP, though 80% (37) of them had never worked as underground coal miners. PMF was found in 12 of the miners, 9 of whom had never worked as underground coal miners.[68]

As discussed above, increased mining of thinner coal seams in recent decades has required increased removal of surrounding rock, resulting in increased RCS exposure, which impacts CMDLD development and severity. Further, small coal mine size is also associated with increased risk, likely due to increased availability of resources that ensure compliance with methods that result in dust exposure minimization.

Coal mine dust composition likely impacts the risk of CMDLD, but due to the significant heterogeneity in coal and mine environments, no specific factors have been established as having significant risk.[33]

STUDY RATIONALE

The recent research into the resurgence of CWP and PMF in active underground coal miners has not examined what impact, if any, miners' increased RCS exposure and working hours have had on the prevalence and severity of other CMDLDs. This is likely because CWP is the only CMDLD for which there is a national surveillance program, the Coal Workers' Health

Surveillance Program (CWHSP) offered by the National Institute for Occupational Safety and Health (NIOSH).[36,37] Further, there is significantly less research into CMDLD in surface coal miners compared to underground coal miners because surface mining was not believed to cause CWP until recently, when cases of CWP and PMF were observed in surface coal miners with no history of underground coal mining.[68] In fact, surface coal miners were not eligible for participation in the CWHSP until 2014.[36]

CWP remains a significant health concern in coal miners in Central Appalachia and Appalachian Kentucky. Most recently, the prevalence of CWP and PMF among long-tenured underground coal miners in Central Appalachia between 2010 and 2015 was estimated at 20.6% and 4.5%, respectively.[64] There is a need to better understand the impact of both underground and surface coal mining on the spectrum of CMDLD, not just CWP. The purpose of this study is to perform a secondary data analysis examining the association between obstructive and restrictive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining, using data obtained between 2015 and 2017 from a rural Appalachian Kentucky mining community.

The proposed study will help capture information on recent trends in CMDLD prevalence in Appalachian Kentucky, and not just CWP. It will also examine the effects of underground and surface coal mining separately and together, which is not common in the literature. CWHSP data, which combines observations for both underground and surface coal miners, is most often used in the literature to investigate prevalence trends in coal miners. This may provide avenues for further research into the spectrum of CMDLD in all coal miners, not just underground.

CHAPTER 2. MANUSCRIPT

ABSTRACT

Objective. Appalachian (Eastern) Kentucky has a high burden of lung disease, partially attributable to significant occupational exposure to coal mine dust in the 7th largest coal-producing state in the United States. Cases of coal workers' pneumoconiosis and its most severe form, pulmonary massive fibrosis, began increasing significantly in Central Appalachia, and Appalachian Kentucky in particular, around 2000. There is a need to better understand the association between coal mine dust lung disease and coal mining exposure in Eastern Kentucky to better guide prevention efforts and operating practices.

Methods. The analysis utilized cross-sectional data from the University of Kentucky, obtained in Harlan and Letcher counties between 2015 and 2017. Multivariable Poisson regression with robust standard errors was performed to assess the association between lung disease, defined by spirometric pattern as obstructive, restrictive, or normal, and coal mining history, defined as never, underground only, surface only, and both types, adjusted for age, education, smoking level, and history of other dusty jobs. Adjusted prevalence ratios (aPR) and 95% confidence intervals (CI) were obtained. The adjusted analyses were further stratified by age and coal mining tenure.

Results. The aPR for the association between obstructive lung disease and a history of underground coal mining, surface coal mining, and underground and surface coal mining were 1.45 (95% CI: 0.94-2.25), 1.20 (95% CI: 0.71-2.02), and 1.51 (95% CI: 0.96-2.38), respectively. The aPR for the association between restrictive lung disease and a history of underground coal mining, surface coal mining, and underground and surface coal mining were 1.18 (95% CI: 0.74-1.90), 0.997 (95% CI: 0.71-2.02), and 1.51 (95% CI: 0.96-2.38), respectively.

Conclusions. The observed effects for this study demonstrated much stronger associations for obstructive lung disease compared to restrictive lung disease and for underground coal mining and both types of coal mining compared to surface coal mining. Generally, the effects of the association for the older age group and longer coal mining tenure group were stronger than the younger age group and shorter mining tenure group, respectively. The analyses were limited by a lack of statistical power and potential survival bias.

INTRODUCTION

Appalachia is a geographical region extending from southern New York to northern Mississippi.[1] It is divided into contiguous subregions based on similarities in geographic, sociodemographic, and economic characteristics. The Central Appalachian subregion consists of eastern Kentucky, southern West Virginia, northeast Tennessee, and southwest Virginia.[2,3]

Appalachia, and Central Appalachia in particular, is impacted by long-standing and persistent health and socioeconomic inequities compared to the rest of the United States (US); however, Appalachia and other rural areas have been consistently underrepresented in health outcomes research.[4] Compared to the non-Appalachian US, Central Appalachia performs worse on a variety of health, lifestyle, and social determinants measures. Central Appalachia has increased mortality (per 100,000) from heart disease, cancer, chronic obstructive pulmonary disease (COPD), injury, stroke, diabetes, and years of potential life lost; increased prevalence of diabetes, obesity, physical inactivity, and smoking; smaller numbers of primary care and specialty physicians (per 100,000); increased rate of COPD hospitalizations (per 10,000 Medicare beneficiaries); decreased median household income; increased percentage of adults living in poverty; and a lower percentage of adults with some college education.[6]

Appalachian Kentucky performs worst of all 13 Appalachian states in regard to mortality from heart disease, cancer, COPD, injury, and years of potential life lost, adult smoking prevalence, rate of COPD hospitalizations, median household income, poverty, and percentage of adults with some college education.[6]

Among other disparities, Appalachian Kentucky has a disproportionately high burden of lung disease. This has been attributed to the region's high rate of smoking, poverty, and unemployment; low educational attainment; high percentage of rural residents; comparatively

lower numbers of primary care and subspecialty providers (including pulmonologists); and significant occupational and environmental exposures to dust from underground and surface coal mining activities.[3]

Exposure to coal mine dust is a concern in Appalachian Kentucky because it has historically been one of the nation's top coal-producing states. In 2020, Kentucky produced 24.2 million short tons of coal in 2020, 34.4% (8.3 million short tons) of which were produced in Eastern Kentucky. Kentucky was the 5th largest coal-producing state in the US in 2019, producing 5.1% of the US's total coal. Production declined in 2020, however, and saw Kentucky reduced to the nation's 7th largest coal-producing state, though still producing 4.5% of the US's total coal.[14]

Chronic occupational exposure to respirable coal mine dust causes coal mine dust lung disease (CMDLD), a heterogenous spectrum of occupational respiratory diseases. CMDLD includes coal workers' pneumoconiosis (CWP), silicosis, mixed-dust pneumoconiosis (MDP), dust-related diffuse fibrosis (DDF), and chronic obstructive pulmonary disease (COPD).[26-30] Capturing the entire spectrum of CMDLD in one study is difficult, and most studies focus on one disease exclusively. Spirometry pattern is the simplest and most sensitive way to classify the entire spectrum of CMDLD.

Spirometry is the most utilized pulmonary function test and is used to obtain lung volumes (in liters) and flow rates. It is clinically useful for assessing the functional severity of lung disease. The most important spirometric measures are the forced vital capacity (FVC), the maximal volume forcefully exhaled after maximal inhalation, and the forced expiratory volume in one second (FEV_1), the volume of air forcefully exhaled in the first second after maximal

inspiration. These measures and their ratio, the FEV_1/FVC ratio, can be used to classify lung disease as obstructive or restrictive.[20,21]

Obstructive spirometry occurs when there is an obstruction to expiratory airflow, typically due to increased airway resistance, and is usually seen with COPD and asthma.[21,23] Restrictive lung disease occurs when lung stiffness is increased, commonly due to inflammation and fibrosis in the lungs. Though expiratory airflow is normal, the decreased lung compliance limits the volume of air that can be taken in during inspiration. Restrictive lung disease has both intrinsic and extrinsic causes. An intrinsic restrictive pattern is caused by a spectrum of diseases known as interstitial lung diseases (ILDs) that result from pathologic destruction of the lung tissue. ILDs may be caused by specific exposures like occupational or environmental dusts and radiation, or be idiopathic, like idiopathic pulmonary fibrosis (IPF) and sarcoidosis. Extrinsic causes of restrictive lung disease include neuromuscular diseases that impact chest wall function.[21,24,25]

Pulmonary function impairment in CMDLD can be restrictive, obstructive, or mixed, reflecting the heterogeneity of the disease spectrum. CWP, silicosis, MDP, and DDF are considered ILDs and thus present with a restrictive spirometry pattern. COPD causes airway obstruction and therefor presents with an obstructive spirometry pattern. A mixed spirometry pattern may be observed when one of the restrictive CMDLDs is comorbid with COPD.[19,26,31]

The Federal Coal Mine Health and Safety Act of 1969 (the Coal Act) was passed to improve the significant morbidity and premature mortality associated with CWP and its most severe form, progressive massive fibrosis (PMF), experienced by coal miners at the time. The Coal Act mandated respirable coal mine dust exposure limits in underground mines, which had previously been unregulated.[10,62] Following passage of the Act, the prevalence of CWP and PMF

declined significantly for 3 decades until 2000, when cases began to rise in miners from Central Appalachia, many of whom had worked under the Coal Act dust exposure limits for the majority or entirety of their careers.[11]

This trend was particularly concerning because PMF cases in Central Appalachia were occurring in younger miners with shorter working tenures than had been seen in the past and many cases were progressing rapidly at a rate not seen before.[48,49,62,63] Previously, only long-tenured coal miners, or those who worked 25 years or more, were believed to be affected by severe CWP and PMF. Kentucky was the hardest hit area in Central Appalachia. Between 2005 and 2009, the states with the highest prevalence of CWP were Kentucky (9%), Virginia (8%), and West Virginia (4.8%). The age-adjusted prevalence of CWP in these three states together and Kentucky alone was 4.5 and 7.6 times greater, respectively, than the prevalence in 12 other coal-producing states.[12]

This increase in CWP and PMF has been attributed to several factors, including the idea that the coal mine dust permissible exposure limit set forth by the Coal Act was inadequate for preventing occupational lung disease or the limit was not being adequately enforced at smaller mines with limited resources. Additionally, increased exposure to respirable crystalline silica (RCS) from thin-seam mining, which requires removal of large amounts of overlying and underlying rock, was also hypothesized to have contributed, particularly because thin-seam mining is especially common in Appalachia compared to other coal regions. Lastly, an increase in coal miners' working hours and weekend overtime has been noted in the decades following the Coal Act.[36,62,66]

The recent research into the resurgence of CWP and PMF in active underground coal miners has not examined what impact, if any, miners' increased RCS exposure and working

hours have had on the prevalence and severity of other CMDLDs. This is likely because CWP is the only CMDLD for which there is a national surveillance program, the Coal Workers' Health Surveillance Program (CWHSP) offered by the National Institute for Occupational Safety and Health (NIOSH).[36,37] Further, there is significantly less research into CMDLD in surface coal miners compared to underground coal miners because surface mining was not believed to cause CWP until recently, when cases of CWP and PMF were observed in surface coal miners with no history of underground coal mining.[68] In fact, surface coal miners were not eligible for participation in the CWHSP until 2014.[36]

CWP remains a significant health concern in coal miners in Central Appalachia and Appalachian Kentucky. Most recently, the prevalence of CWP and PMF among long-tenured underground coal miners in Central Appalachia between 2010 and 2015 was estimated at 20.6% and 4.5%, respectively.[64] There is a need to better understand the impact of both underground and surface coal mining on the spectrum of CMDLD, not just CWP. The purpose of this study is to perform a secondary data analysis examining the association between obstructive and restrictive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining, using data obtained between 2015 and 2017 from a rural Appalachian Kentucky mining community.

METHODS

Study Design and Sample. Secondary data analysis was conducted using cross-sectional data from the Mountain Air Project (MAP). The MAP was designed and implemented by researchers at the University of Kentucky (UK) to investigate the relative effects of local environmental exposures and health behaviors on the disproportionately high rates of respiratory disease in Appalachian Kentucky.

Study design, sampling methods, and data collection are described extensively in May et. al.[4] A community-based participatory research framework, incorporating extensive input from community advisory boards (CABs) in Harlan and Letcher counties in eastern Kentucky, was used in designing and implementing the MAP, a cross-sectional study which utilized hollows as a novel sampling unit. Harlan and Letcher counties were selected for the study because they are typical Appalachian Kentucky counties – rural, designated as economically distressed by the Appalachian Regional Committee (ARC), and impacted by a disproportionate burden of respiratory diseases like asthma and chronic obstructive pulmonary disorder, high smoking rates (24.9% of adults smoked tobacco in 2014-2016), and proximity to active coal mining.[4] In 2016, there were 9 active underground coal mines and 7 active surface coal mines in Harlan county and 8 active surface coal mines in Letcher county, though the counties contained many more active coal mines in the past decade.[69]

As respiratory exposures varied significantly within the counties, hollows were utilized as sampling units. Hollows are communities contained within a watershed, which is a small area of land drained by a stream. Several watersheds make up a larger river basin, the land drained by a river and its tributaries.[70] Hollows are delineated geographically by the US Geological Survey 14-digit hydrologic unit codes (HUC) (Note: hollow and HUC are used interchangeably). Ten hollows were randomly selected per tertile of a composite airborne exposure index, which incorporated several HUC measures that affect the local concentration of airborne particulate matter. These measures included miles of roadway per square mile, miles of coal haul routes per square mile, percentage of surface area occupied by abandoned underground and surface mines, percentage of surface area occupied by active underground and surface mines, and the number of

oil or gas wells per square mile. Per recommendations by the CABs, the study area was expanded to include additional areas undergoing active resource extraction.[4]

On the ground enumeration was utilized for sampling from the 4,291 residencies identified within the study area. HUCs found to contain less than 10 homes were removed and replaced with another randomly selected HUC. Hollows were prioritized for enumeration by travel time and accessibility. Excluding 176 abandoned homes, 4,115 households within 40 hollows were enumerated with a unique identifying number. The household that served as the starting point within each HUC was chosen randomly and every following first through fifth house was subsequently approached (depending on the number of homes within the HUC) until a minimum of 10 households per HUC were recruited. 1,435 (35%) of the enumerated houses were contacted and eligible to participate. 1,190 (82%) of these households consented to study participation. 972 participants, one from each household, completed a health survey and spirometry. Twenty-eight of the forty enumerated HUCs had participation rates of 80% or more.[4]

Data were collected between November 2015 and August 2017 by trained community health workers (CHWs) who administered a health questionnaire and obtained spirometry using a portable EasyOne spirometer (ndd Medical Technologies, Andover, MA). Adults with respiratory disease or symptoms were encouraged to serve as the household participant, though this was not an inclusion criterium for participation. Participants were eligible for inclusion if they were noninstitutionalized, 21 years old or older, English-speaking, and provided voluntary consent for participation. Spirometry was administered to all participants with no significant cardiovascular or cerebrovascular events in the preceding 30 days. Information on demographics, health indicators, and occupational and environmental exposures were obtained from the

questionnaire. Participants received $40 and a copy of their spirometry interpreted by a pulmonologist.[4]

The current study population was restricted to male participants, as only 6 and 9 women with interpretable spirometry had a history of underground and surface coal mining, respectively. Of the remaining 401 male study participants, 14, 32, and 51 had missing, uninterpretable, and poor quality (grade D or F) spirometry, respectively, and were removed from the sample. The final sample used for analysis had 304 male participants.

Outcome Variable. A UK board-certified pulmonologist and fellow of the American College of Chest Physicians utilized the third National Health and Nutrition Examination Study (NHANES III) method as outlined by Hankinson et. al.[71] to assess participant spirometry quality and classify pulmonary function as restrictive, obstructive, normal, or uninterpretable. The Hankinson paper derived spirometric reference values and lower limits of normal for white, Black, and Mexican Americans using a sample of never smoking NHANES III participants. Spirometry quality was assessed by the objective and validated acceptability/usability and repeatability criteria for FEV_1 and FVC measures to give a letter grade from A through F. The American Thoracic Society's (ATS) current technical standards for conducting spirometry are a modified version of this system.[20,71]

A minimum of 3 spirometry trials were initially performed for each participant. Low quality tests were readministered for 3 additional trials. The highest quality trial per participant was used to assess pulmonary function. The UK pulmonologist initially categorized spirograms as normal or abnormal. Abnormal tests were then categorized as restrictive or obstructive. Tests were designated abnormal based on the FEV_1 and FVC predictive pulmonary function comparison values and lower limit of normal calculations for participant race, sex, and age as

outlined in the NHANES III data. Normal spirometry was defined by $FEV_1/FVC \geq 0.70$ and FVC ≥80% of predicted. Restrictive spirometry was defined by $FEV_1/FVC \geq 0.70$ and FVC <80% of predicted. Obstructive spirometry was defined by $FEV_1/FVC < 0.70$. Some participants' tests were categorized as uninterpretable based on low quality. These readings were all grade C, D, or F.[71,72]

Exposure Variable. Coal mining history was categorized using participant responses to the survey questions "Have you ever worked as an underground coal miner?" and "Have you ever worked on a surface coal mine?" Coal mining exposure was then categorized into four levels: no history of coal mining for those who responded no to both questions, history of underground coal mining only for those who responded yes to the first question and no to the second, history of surface coal mining only for those who responded no to the first question and yes to the second, and history of both underground and surface coal mining for those who responded yes to both questions.

Those participants who responded affirmatively to having a history of underground and/or surface coal mining were then asked to document the total number of years they had worked as an underground and/or surface coal miner. For participants who reported a history of both underground and surface mining, their responses were added to give a combined tenure value in years.

A coal miner with a tenure of ≥25 years is considered "long-tenured" and at increased risk of developing CMDLD compared to miners with tenure <25 years. In the literature, tenure is typically stratified as such to allow for comparison of the risk of CMDLD between the two groups.[36,62,66] Due to data distribution, coal mining tenure was categorized as <20 years and ≥20 years for the purpose of this study.

Covariates. Covariates were considered for inclusion in the adjusted analysis using a directed acyclic graph (DAG) mapping the causal pathway between a history of coal mining and pulmonary function. The DAG was created with DAGitty,[73] (Figure 1) which recommended that adjustment for age, education, race, annual household income, and history of other dusty jobs was minimally sufficient for estimating the effect of coal mining exposure on pulmonary function.

Age was calculated by subtracting the date of birth from the date of spirometry. Education was categorized into three levels based on participant responses to the question "What is the highest degree or level of schooling you have completed?" Those who responded with "grades 1-5", "grades 6-8", and "grades 9-11" were categorized as having less than a high school education. Those who responded with "completed high school or GED" or "some college but no degree" were categorized as such. Those who responded with "associate degree (junior college, AA, AS)," "bachelor's degree," "graduate or professional school (masters, doctorate, MD, JD, DDS, etc.)," were categorized as associate degree or higher. Education level was considered a proxy for socioeconomic status (SES).

Annual household income was dichotomized as <$25,000 and ≥$25,000 by combining categorical responses to "Please click the choice that best describes your total combined household income for the past 12 months. This includes total income before taxes earned in the past year by all family members living with you." Although annual household income would be useful in further capturing the complexity of SES, 22.4% of the male study participants declined to answer the question, so this variable was excluded from the final adjusted models.

Smoking level (in packyears) was also included to further capture the complexity of SES. Participants were categorized as nonsmokers, low smoking level (1-20 packyears), or high

smoking level (>20 packyears). Participants' smoking status was first categorized in response to the question, "Have you smoked at least 100 cigarettes in your entire life?" Those who answered no were categorized as nonsmokers. Those who answered yes were further categorized as current or former smokers based on their response to the question, "Have you smoked cigarettes during the last 30 days?"

All current and former smokers provided the age at which they first started smoking in response to the question, "How old were you when you first started smoking cigarettes?" Former smokers provided the age at which they last smoked in response to the question, "How old were you when you last smoked a cigarette?" Smoking years for current smokers were calculated by subtracting age first smoked from current age. Smoking years for former smokers were calculated by subtracting age first smoked from age last smoked. The number of packs per day for current and former smokers was calculated by dividing the number response to, "On average, about how many cigarettes a day do/did you smoke?" by 20. Packyears were then calculated for each former and current smoker by multiplying smoking years by packs per day. Current and former smokers' smoking level was then categorized by packyears as described above. Smoking level, rather than smoking status, was used in the final adjusted models as it better captures the risk of cigarette smoking by quantifying the exposure.

Race was categorized as white, Hispanic, and Black. Race is certainly an important predictor for inclusion in the adjusted model as it impacts lung function and employment history in coal mining, in addition to significantly impacting health disparities in general. However, the male study population was overwhelmingly white with only 8 Hispanic men and 1 Black man included in the sample. Race was thus excluded from the final adjusted models. A sensitivity analysis excluding the non-white study participants was performed.

A history of other dusty jobs was categorized as yes if participants responded yes to any of the following questions: “Have you ever worked in logging or milling?,” “Have you ever worked in highway construction or repair?,” and “Have you ever worked in home or building construction?” Participants who responded no to all three questions were categorized as having no history of other dusty jobs.

Statistical Analysis. SAS 9.4 (SAS Institute, Cary, NC) was used for all analyses. Descriptive statistics for age, race, education, annual household income, smoking status, smoking level, coal mining history, coal mining tenure, and history of other dusty jobs were calculated for each outcome category. Pearson’s chi-square tests for significance were used to compare the baseline differences in race, education, annual household income, smoking status, smoking level, coal mining history, and coal mining tenure between the obstructive and normal lung function groups and the restrictive and normal lung function groups. A pooled t-test was used to compare the baseline differences in age between the groups. An alpha level of 0.05 was considered statistically significant for all analyses.

Poisson regression with robust standard errors was used for all modeling to obtain crude and adjusted prevalence ratios (cPR and aPR, respectively) and 95% confidence intervals (CI) for the association between lung disease and coal mining history. Poisson regression was chosen to derive estimates for the prevalence ratios because the outcomes of obstructive and restrictive lung disease were highly prevalent in the study population (28% and 23% demonstrated obstructive and restrictive disease, respectively). Analyses were performed separately for obstructive and restrictive lung disease, utilizing a dichotomous outcome variable with normal pulmonary function as the comparison group.

Unadjusted Poisson regression with robust standard errors was performed to determine the association between the outcome variable and coal mining history, age, education, smoking level, and history of other dusty jobs. Regardless of statistical significance, all variables were retained for the final adjusted multivariable model due to biological plausibility for their inclusion, as previously discussed.

Multivariable adjusted Poisson regression models with robust standard errors were performed to assess the association between obstructive and restrictive lung disease and coal mining history, adjusted for age, education, smoking level, and history of other dusty jobs. Adjusted PR and 95% CI were obtained.

The final adjusted multivariable model was then stratified by age at 60 years, adjusting for the same covariates, and by coal mining tenure at 20 years, adjusting for the same covariates plus coal mining tenure. Both age and coal mining tenure are used in the literature as proxies for cumulative dust exposure. Cumulative dust exposure is the main determinant of risk for development of CMDLD. It was therefore hypothesized that the effects of older age and longer coal mining tenure on the association between lung disease and coal mining history would be particularly strong.

A non-human subjects research determination from the Emory University Institutional Review Board (IRB) determined this project did not require IRB review because it does not meet the federal regulatory definition for a "clinical investigation" or as research with "human subjects." This was determined after the Emory University IRB reviewed the provided project protocol closure document from the University of Kentucky IRB, which stated that deidentified data was approved for use in secondary analyses after closure of the study. Informed consent was obtained from all participants.

RESULTS

Descriptive statistics. Among the total sample of 304 male MAP participants, 149 (49%) had normal pulmonary function measured via spirometry, while 85 (28%) and 70 (23%) demonstrated obstructive and restrictive lung disease, respectively. The mean ages for those with normal, obstructive, and restrictive spirometry were 50.9 years (±16), 60.3 years (±10.9, $p<0.0001$), and 55.3 years (±12.4, $p=0.0428$), respectively. Participants with normal, obstructive, and restrictive spirometry were 96.6%, 95.5% ($p=0.9439$), and 98.6% ($p=0.1056$) white. Of those with normal spirometry, 27% had less than a high school education, 48% had graduated high school/had a GED or had completed some college, and 25% had an associate degree or higher. Comparatively, the prevalence for these educational categories in those with obstructive disease was 41.2%, 49.4%, and 9.4% ($p=0.0062$), respectively, and the prevalence for those with restrictive disease was 25.7%, 61.4%, and 12.9% ($p=0.0822$), respectively. Forty-one percent of those with normal spirometry were nonsmokers, while 30.2% and 28.9% had a low packyear level and a high packyear level, respectively. Fifty percent of participants with normal spirometry had worked in another dusty profession, while 43.5% (p=0.3661) of participants with obstructive lung disease and 32.9% (0.0195) of participants with restrictive lung disease (Table 1).

Overall, 95 (31%) participants had no history of coal mining, 82 (27%) had a history of underground coal mining only, 59 (19%) had a history of surface coal mining only, and 68 (22%) had a history of both underground and surface coal mining. For those with normal lung function, 36.9% had no history of coal mining, 21.5% had a history of underground coal mining only, 22.2% had a history of surface mining only, and 19.5% had a history of both underground and coal mining. For those with obstructive lung function, 21.2% had no history of coal mining,

32.9% had a history of underground coal mining only, 15.3% had a history of surface mining only, and 30.6% had a history of both underground and coal mining (p=0.0106). For those with restrictive lung function, 31.4% had no history of coal mining, 31.4% had a history of underground coal mining only, 18.6% had a history of surface mining only, and 18.6% had a history of both underground and coal mining (p=0.4522) (Table 1).

Of the 32 participants with normal lung function who had a history of underground coal mining, 65.6% had a coal mining tenure of <20 years and 34.4% had a tenure ≥20 years. Of the 28 participants with obstructive lung function who had a history of underground coal mining, 39.3% had a coal mining tenure of <20 years and 60.7% had a tenure ≥20 years. Of the 22 participants with restrictive lung function who had a history of underground coal mining, 54.5% had a coal mining tenure of <20 years and 45.5% had a tenure ≥20 years (Table 2).

Of the 33 participants with normal lung function who had a history of surface coal mining, 84.8% had a coal mining tenure of <20 years and 15.2% had a tenure ≥20 years. Of the 13 participants with obstructive lung function who had a history of surface coal mining, 46.2% had a coal mining tenure of <20 years and 53.8% had a tenure ≥20 years. Of the 13 participants with restrictive lung function who had a history of surface coal mining, 76.9% had a coal mining tenure of <20 years and 23.1% had a tenure ≥20 years (Table 2).

Of the 29 participants with normal lung function who had a history of both underground and surface coal mining, 37.9% had a coal mining tenure of <20 years and 62.1% had a tenure ≥20 years. Of the 26 participants with obstructive lung function who had a history of both underground and surface coal mining, 50.0% had a coal mining tenure of <20 years and 50.0% had a tenure ≥20 years. Of the 13 participants with restrictive lung function who had a history of

underground and surface coal mining, 38.5% had a coal mining tenure of <20 years and 61.5% had a tenure ≥20 years (Table 2).

Multivariable adjusted model. The aPR for the association between obstructive lung disease and a history of underground coal mining was 1.45 (95% CI: 0.94-2.25); between obstructive spirometry and a history of surface coal mining was 1.20 (95% CI: 0.71-2.02); and between obstructive spirometry and a history of underground and surface coal mining was 1.51 (95% CI: 0.96-2.38). For restrictive lung disease, the aPR for underground coal mining was 1.18 (95% CI: 0.74-1.90); for surface coal mining was 0.997 (95% CI: 0.58-1.73); and for underground and surface coal mining was 0.97 (95% CI: 0.54-1.73) (Table 3). The aPRs for the sensitivity analysis excluding nonwhite participants were not meaningfully different. For obstructive lung disease, the aPR for those with a history of underground coal mining, surface coal mining, and both underground and surface coal mining were 1.46 (95% CI: 0.92-2.29), 1.28 (95% CI: 0.76-2.16), and 1.57 (95% CI: 0.97-2.51), respectively. For restrictive lung disease, the aPR for the same exposure groups were 1.17 (95% CI: 0.72-1.89); 1.05 (95% CI: 0.60-1.81); and 1.02 (95% CI: 0.57-1.82), respectively (Table 4).

Multivariable adjusted model stratified by age. Among participants <60 years old, the aPR for obstructive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining were 1.43 (95% CI: 0.68-3.04), 0.90 (95% CI: 0.39-2.10), and 2.35 (95% CI: 1.25-4.42), respectively. Among participants ≥60 years old, the aPR for obstructive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining were 2.39 (95% CI: 1.29-4.42), 2.47 (95% CI: 1.22-5.02), and 1.54 (95% CI: 0.75-3.15), respectively (Table 5).

Among participants <60 years old, the aPR for restrictive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining were 1.10 (95% CI: 0.56-2.16), 1.02 (95% CI: 0.53-1.95), and 0.91 (95% CI: 0.34-2.40), respectively. Among participants ≥60 years old, the aPR for restrictive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining were 1.40 (95% CI: 0.79-2.44), 0.92 (95% CI: 0.36-2.34), and 1.12 (95% CI: 0.58-2.19), respectively (Table 6).

Multivariable adjusted model stratified by coal mining tenure. Among participants with <20 years coal mining tenure, the aPR for obstructive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining were 1.24 (95% CI: 0.71-2.16), 0.83 (95% CI: 0.40-1.69), and 2.11 (95% CI: 1.27-3.50), respectively. Among participants with ≥20 years coal mining tenure, the aPR for obstructive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining were 1.67 (95% CI: 1.02-2.71), 2.01 (95% CI: 1.08-3.73), and 1.18 (95% CI: 0.67-2.07), respectively (Table 7).

Among participants with <20 years coal mining tenure, the aPR for restrictive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining were 1.13 (95% CI: 0.65-1.95), 0.98 (95% CI: 0.54-1.79), and 1.32 (95% CI: 0.58-3.03), respectively. Among participants ≥20 years coal mining tenure, the aPR for restrictive lung disease and a history of underground coal mining, surface coal mining, and both underground and surface coal mining were 1.27 (95% CI: 0.71-2.28), 1.05 (95% CI: 0.38-2.93), and 0.81 (95% CI: 0.42-1.57), respectively (Table 8).

DISCUSSION

The prevalence of obstructive lung disease was consistently higher in those with a history of underground coal mining only and those with a history of both underground and surface coal mining compared to those with no history of coal mining overall and in each age and coal mining tenure group. The effects generally appeared to be dose dependent, with a stronger effect observed for those with a history of both types of coal mining compared to those with a history of just underground coal mining, although this did not hold true for the highest risk age and mining tenure groups (age ≥60 and mining tenure ≥20 years). The prevalence of obstructive lung disease was higher in those with a history of surface coal mining compared to those with no history of coal mining overall and among those age 60 and older and those with coal mining tenure of 20 years or more, although the effects were generally not as strong compared to those for underground and both types of coal mining. There did not appear to be an association between restrictive lung disease and a history of any type of coal mining in the entire study population or in the age and tenure-stratified analyses.

Overall, the observed effects for this study demonstrated much stronger associations for obstructive lung disease compared to restrictive lung disease and for underground coal mining and both types of coal mining compared to surface coal mining. Generally, the effects of the association for the older age group and longer coal mining tenure group were stronger than the younger age group and shorter mining tenure group, respectively.

A recent analysis examined spirometric abnormalities in a population of former coal miners (n = 2,568) using data obtained from the Black Lung Clinics Program between 2019 and 2020. A majority of the miners had a history of underground coal mining (81.4%) and had worked most of their career in Central Appalachia (78.5%). About 57% of the population

demonstrated abnormal spirometry, 35% of whom had restrictive spirometry and 8.8% of whom had obstructive spirometry. Those with obstructive and mixed spirometry were categorized as having airflow obstruction and made up 21.7% of the sample.[74]

Although this study is not directly comparable to ours, restrictive spirometry is almost 4 times more prevalent than obstructive spirometry alone and 1.6 times more prevalence than obstructive and mixed spirometry together in this sample of former coal miners. It is therefore likely that the observed results for restrictive lung disease in our study represent are due to a lack of statistical power, rather than a demonstration that obstructive disease is more prevalent than restrictive lung disease among those with a history of coal mining. Those with restrictive spirometry represented the smallest outcome group with only 70 participants, and only 48 of these participants had exposure to any type of coal mining

There is no literature examining the prevalence of restrictive and obstructive spirometry in those with a history of surface coal mining only. As previously discussed, surface coal miners were not believed to be at risk of developing CMDLD for many years due to outside air dilution of respirable dust.[43] Although surface coal mining has now been recognized as an independent risk factor for development of CWP and PMF, national surveillance data on surface miners has only been collected since 2014 and studies utilizing this data in the literature typically examine underground and surface coal miners together. However, 2% (46) of a sample of 2,257 surface coal miners who had worked for ≥1 year were found to have CWP, though 80% (37) of them had never worked in an underground coal mine. PMF was found in 12 of the miners, 9 of whom had never worked in an underground coal mine.[68] While surface miners can develop CWP and PMF, as demonstrated in the above study, the prevalence of CWP and PMF in underground coal miners, especially in Central Appalachia, was found to be comparatively much higher in 2010

and 2011, when data from this study was collected. It is therefore reasonable to assume that the prevalence of both obstructive and restrictive lung disease is lower among those with a history of surface coal mining only compared to those with any history of underground coal mining, as demonstrated in our study.

Cumulative coal mine dust exposure is the main determinant of risk for CMDLD. As this is difficult to directly measure and estimate, variables that impact the frequency, intensity, and/or duration of coal mine dust exposure, like age and coal mining tenure, are used as proxies for estimation in the literature.[26,31,33,36,42] This explains the stronger effects of association that were observed for the older age group (≥60 years old) and the longer coal mining tenure group (≥20 years), as they are at higher risk of developing CMDLD due to increased cumulative dust exposure.

This analysis has several strengths. The MAP represents an immense effort to better understand the burden of lung disease in a community with unique occupational and environmental exposures that is underrepresented in health outcomes literature. Participation and follow-up rates were very high, and the study population was presumed to be representative of the area. The data was collected between 2015 and 2017 and is thus more recent than the most recently published CWP and PMF prevalence data from 2010-2015. The CABs were dedicated, hard-working and extremely involved in all stages of the project and the CHWs were extensively trained. Though time and labor intensive, this method could be used in future studies of rural populations to ensure buy-in from local communities.

There are several limitations to this study that may account for the observed findings. Most significantly, the analysis likely lacked adequate statistical power to detect a difference between exposure groups due to the relatively small sample size, even smaller exposure groups,

and use of dichotomous lung function measures rather than the continuous values for FEV_1, FVC, and the FEV_1/FVC ratio.

The second limitation was the classification of CMDLD using obstructive and restrictive spirometry. First, the NHANES III spirometric reference values utilized to classify the outcome are outdated. The GLI 2012 reference values have been preferred for use in the literature for the past 10 years. Additionally, obstructive and restrictive spirometry measures are very sensitive for detecting CMDLD (and lung disease in general), but not very specific. It is possible that a participant with a history of coal mining has a restrictive (and less likely obstructive) lung disease independent of their coal mining exposure, such as sarcoidosis. This could lead to differential misclassification of the outcome for those with exposure to coal mining, biasing results towards the null.

Survival bias is also possible. It may be that those with the most extensive or longest history of coal mining were less likely to participate in the study due to premature mortality or significant morbidity that prevented completion of spirometry. This may also explain why those with both restrictive and obstructive lung disease and a history of underground and surface coal mining have lower aPR values compared to those with a history of underground coal mining only among those 60 years and older and those with a tenure of 20 years or more.

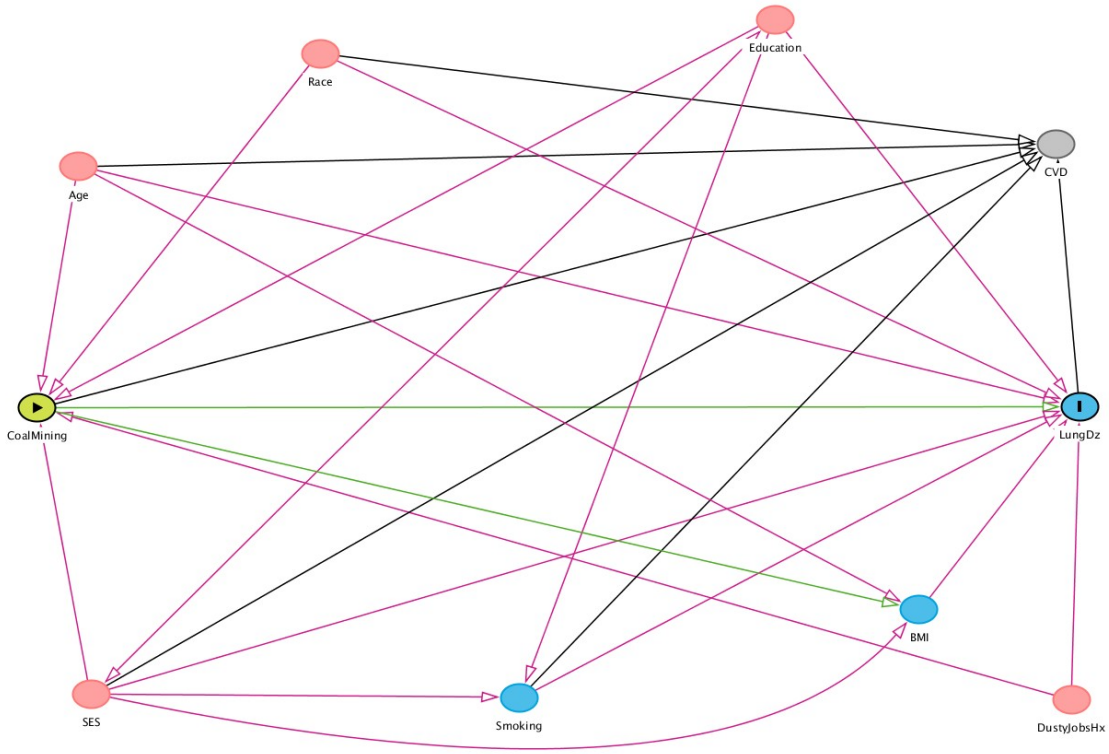
Last, selection bias is possible. Although hollows and homes were selected as randomly as possible for enumeration, input from the CABs was utilized in determining the study area and may have been biased towards communities the members were from or familiar with. Areas were also prioritized for selection based on travel time and accessibility during the winter months. Additionally, within each hollow, some randomly selected households had to be omitted from enumeration due inaccessibility or safety concerns. As always, the eligible households that

consented to participate in the study may be different from those that did not consent to participation.

Further research into CMDLD in Central Appalachia and Appalachian Kentucky is recommended to continue documenting the prevalence trends observed in the region over the past 2 decades. The most recent prevalence estimates were published in 2018, utilizing data from 2010-2015. A larger study with a specific focus on validated CMDLD in active and former coal miners is recommended to better characterize disease distribution and risk factors specific to the region and to address the potential limitations discussed above.

FIGURE AND TABLES

Figure 1. Directed acyclic graph (DAG) between a history of coal mining and obstructive and restrictive lung disease



Exposure = CoalMining = history of coal mining
Outcome = LungDz = obstructive or restrictive lung disease
CVD = cardiovascular disease
SES = socioeconomic status
BMI = body mass index
DustyJobsHx = history of other dusty jobs (logging/milling, highway construction/repair, and/or home/building construction)

Table 1. Descriptive Statistics of Male Study Participants by Spirometric Pulmonary Function (n = 304)

Characteristic	**Normal spirometry** 149 (49.0) Frequency (col %)	**Obstructive spirometry** 85 (28.0) Frequency (col %)	**P-value***	**Restrictive spirometry** 70 (23.0) Frequency (col %)	**P-value***
Age†	50.9 (±16.0)	60.3 (±10.9)	<0.0001	55.3 (±12.4)	0.0428
Race					
White	144 (96.6)	82 (96.5)	0.9439	69 (98.6)	0.1056
Hispanic	5 (3.4)	3 (3.5)		0	
Black	0	0		1 (1.4)	
Education level					
<High School	40 (27.0)	35 (41.2)	0.0062	18 (25.7)	0.0822
High school/ GED or some college	71 (48.0)	42 (49.4)		43 (61.4)	
Associate degree or higher	37 (25.0)	8 (9.4)		9 (12.9)	
Missing (1)	1	0		0	
Annual household income					
<$25K	65 (43.6)	45 (52.9)	0.0703	32 (45.7)	0.9246
≥$25	53 (35.6)	18 (21.2)		23 (32.9)	
Missing (68)	31 (20.8)	22 (25.9)		15 (21.4)	
Smoking status					
Current	61 (40.9)	13 (15.3)	<.0001	36 (51.4)	0.3363
Former	41 (27.5)	44 (51.8)		15 (21.4)	

Characteristic	Normal spirometry 149 (49.0) Frequency (col %)	Obstructive spirometry 85 (28.0) Frequency (col %)	P-value*	Restrictive spirometry 70 (23.0) Frequency (col %)	P-value*
Never	47 (31.5)	28 (32.9)		19 (27.1)	
Smoking level					
Nonsmoker	61 (40.9)	13 (15.3)		36 (51.4)	
Low (1- 20 packyears)	45 (30.2)	18 (21.2)	<.0001	9 (12.9)	0.0211
High (>20 packyears)	43 (28.9)	54 (63.5)		25 (35.7)	
Ever worked as coal miner					
No	55 (36.9)	18 (21.2)		22 (31.4)	
Underground coal mining only	32 (21.5)	28 (32.9)		22 (31.4)	
Surface coal mining only	33 (22.2)	13 (15.3)	0.0106	13 (18.6)	0.4522
Both types of coal mining	29 (19.5)	26 (30.6)		13 (18.6)	
Underground coal mining tenure (years)					
0	55 (63.2)	18 (39.1)		22 (50.0)	
<20	21 (24.1)	11 (23.9)	0.0030	12 (27.3)	0.2422
≥20	11 (12.6)	17 (37.0)		10 (22.7)	

Characteristic	Normal spirometry 149 (49.0) Frequency (col %)	Obstructive spirometry 85 (28.0) Frequency (col %)	P-value*	Restrictive spirometry 70 (23.0) Frequency (col %)	P-value*
Surface coal mining tenure (years)					
0	55 (62.5)	18 (58.1)	0.0201	22 (62.9)	0.8157
<20	28 (31.8)	6 (19.4)		10 (28.57)	
≥20	5 (5.7)	7 (22.6)		3 (8.6)	
Underground and surface coal mining tenure (years)					
0	55 (65.5)	18 (40.9)	0.0182	22 (62.9)	0.9630
<20	11 (13.1)	13 (29.6)		5 (14.3)	
≥20	18 (21.4)	13 (29.6)		8 (22.9)	
Ever worked in other dusty profession‡					
No	75 (50.3)	48 (56.5)	0.3661	47 (67.1)	0.0195
Yes	74 (49.7)	37 (43.5)		23 (32.9)	

col % = column percent (by outcome category)
*P-value obtained via Pearson's chi-square test for categorical variables and pooled t-test for age
†Mean (± standard deviation)
‡Logging/milling, highway construction/repair, and/or home/building construction

Table 2. Descriptive Statistics of Male Study Participants with a History of Coal Mining by Spirometric Pattern and Coal Mining Tenure (n = 209)

Spirometry pattern	Coal Mining Tenure	Underground coal mining 82 Freq (row %)	Surface coal mining 59 Freq (row %)	Both types of coal mining 68 Freq (row %)
Normal	Tenure < 20 years	21 (65.5)	28 (84.8)	11 (37.9)
	Tenure ≥20 years	11 (34.4)	5 (15.2)	18 (62.1)
Obstructive	Tenure < 20 years	11 (39.3)	6 (46.2)	13 (50.0)
	Tenure ≥20 years	17 (60.7)	7 (53.8)	13 (50.0)
Restrictive	Tenure < 20 years	12 (54.5)	10 (76.9)	10 (76.9)
	Tenure ≥20 years	10 (45.5)	3 (23.1)	3 (23.1)

row % = row percent (by spirometry and coal mining tenure category)

Table 3. Adjusted Prevalence Ratios Assessing Association Between Obstructive and Restrictive Spirometry and History of Coal Mining Among Male Study Participants (n = 304)

Exposure	Obstructive Spirometry (n = 85)		Restrictive Spirometry (n = 70)	
History of Coal mining	aPR*	95% CI	aPR*	95% CI
Never	Ref.	-----	Ref.	-----
Underground only	1.45	0.94-2.25	1.18	0.74-1.90
Surface only	1.20	0.71-2.02	0.997	0.58-1.73
Both	1.51	0.96-2.38	0.97	0.54-1.73

*Adjusted for age, education, smoking level, and history of other dusty jobs
aPR = adjusted prevalence ratio
CI = confidence interval

Table 4. Sensitivity Analysis - Adjusted Prevalence Ratios Assessing Association Between Obstructive and Restrictive Spirometry and History of Coal Mining Among Male Study Participants (n = 295)

Exposure	**Obstructive Spirometry (n = 82)**		**Restrictive Spirometry (n = 69)**	
History of Coal mining	aPR*	95% CI	aPR*	95% CI
Never	Ref.	-----	Ref.	-----
Underground only	1.46	0.92-2.29	1.17	0.72-1.89
Surface only	1.28	0.76-2.16	1.05	0.60-1.81
Both	1.57	0.97-2.51	1.02	0.57-1.82

*Adjusted for age, education, smoking level, and history of other dusty jobs
aPR = adjusted prevalence ratio
CI = confidence interval

Table 5. Adjusted Prevalence Ratios Assessing Association Between Obstructive Spirometry and History of Coal Mining Stratified by Age (n = 234)

Exposure	**Obstructive Spirometry** (n = 85)			
	Age <60 years (n = 40)		Age ≥60 (n = 45)	
History of Coal mining	aPR*	95% CI	aPR*	95% CI
Never	Ref.	-----	Ref.	-----
Underground only	1.43	0.68-3.04	**2.39**	**1.29-4.42**
Surface only	0.90	0.39-2.10	**2.47**	**1.22-5.02**
Both	**2.35**	**1.25-4.42**	1.54	0.75-3.15

*Adjusted for age, education, smoking level, and history of other dusty jobs
aPR = adjusted prevalence ratio
CI = confidence interval
Bold indicates a significant result

Table 6. Adjusted Prevalence Ratios Assessing Association Between Restrictive Spirometry and History of Coal Mining Stratified by Age (n = 219)

Exposure	**Restrictive Spirometry** (n = 70)			
	Age <60 years (n = 39)		Age ≥60 (n = 31)	
History of Coal mining	aPR*	95% CI	aPR*	95% CI
Never	Ref.	-----	Ref.	-----
Underground only	1.10	0.56-2.16	1.40	0.79-2.44
Surface only	1.02	0.53-1.95	0.92	0.36-2.34
Both	0.91	0.34-2.40	1.12	0.58-2.19

*Adjusted for age, education, smoking level, and history of other dusty jobs
aPR = adjusted prevalence ratio
CI = confidence interval

Table 7. Adjusted Prevalence Ratios Assessing Association Between Obstructive Spirometry and History of Coal Mining Stratified by Coal Mining Tenure (n = 234)

Exposure	**Obstructive Spirometry** (n = 85)			
	Tenure <20 years		Tenure ≥20 years	
History of Coal mining	aPR*	95% CI	aPR*	95% CI
Never	Ref.	-----	Ref.	-----
Underground only	1.24	0.71-2.16	**1.67**	**1.02-2.71**
Surface only	0.83	0.40-1.69	**2.01**	**1.08-3.73**
Both	**2.11**	**1.27-3.50**	1.18	0.67-2.07

*Adjusted for age, coal mining tenure, education, smoking level, and history of other dusty jobs
Bold indicates a significant result

Table 8. Adjusted Prevalence Ratios Assessing Association Between Restrictive Spirometry and History of Coal Mining Stratified by Coal Mining Tenure (n = 219)

Exposure	**Restrictive Spirometry** (n = 70)			
	Tenure <20 years		Tenure ≥20 years	
History of Coal mining	aPR*	95% CI	aPR*	95% CI
Never	Ref.	-----	Ref.	-----
Underground only	1.13	0.65-1.95	1.27	0.71-2.28
Surface only**	0.98	0.54-1.79	1.05	0.38-2.93
Both	1.32	0.58-3.03	0.81	0.42-1.57

*Adjusted for age, coal mining tenure, education, smoking level, and history of other dusty jobs

CHAPTER 3: FUTURE DIRECTIONS/PUBLIC HEALTH IMPLICATIONS

RECOMMENDATIONS

Future research. Further research into CMDLD in Central Appalachia and Appalachian Kentucky is recommended to continue documenting the prevalence trends observed in the region over the past 2 decades. The most recent prevalence estimates were published in 2018, utilizing data from 2010-2015. A larger study with a specific focus on validated CMDLD in active and former coal miners is recommended to better characterize disease distribution and risk factors specific to the region and to address the potential limitations of the current study.

Policy. Increasing CMDLD primary prevention would be the most effective policy intervention for decreasing its prevalence, morbidity, and mortality among American coal miners. The most obvious method to accomplish this goal would be decreasing the mandated coal mine dust concentration limit from the current value of 1.5 mg/m^3 (10-hour TWA) to 1 mg/m^3 (10-hour TWA), as has been recommended by NIOSH since they published the "Criteria for Occupational Exposure to Respirable Coal Mine Dust" in 1995.[32] This is unlikely to happen soon, or ever, as the coal mining industry is very resistant to federal regulation and has a powerful lobby. Despite the increase in CWP and rapidly progressive PMF seen in younger Central Appalachian coal miners beginning in 2000, it took until 2014 for MSHA to decrease the coal mine dust air concentration limit to 1.5 mg/m^3 from 2 mg/m^3, the level that had been established over 30 years prior. Other policy interventions that might be effective in decreasing CMDLD prevalence and disease severity include: increasing miner participation in the CWHSP via incentives or with increased convenience for miners to obtain a more complete and timely picture of trends in CMDLD prevalence; improving miners' education about, awareness of, and participation in Part 90 transfers; and targeting smaller mines for education initiatives and additional resources to ensure compliance with preventive measures.

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