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Subcommittee on Workforce Protections Committee on Education and Labor U.S. House of Representatives Breathless and Betrayed: What is MSHA Doing to Protect Miners from the Resurgence of Black Lung Disease? June 20, 2019

Chairwoman Adams, Ranking Member Byrne, and distinguished members of the Subcommittee. My name is Robert Cohen and I am the Director of the Mining Education and Research Center, and Clinical Professor of Environmental and Occupational Health Sciences at the University of Illinois at Chicago, School of Public Health. I am also Professor of Medicine at the Northwestern University Feinberg School of Medicine and Direct the Occupational Lung Disease Program at Northwestern Medicine. I direct the Black Lung Center of Excellence funded by the Health Resources and Services Administration (HRSA) and serve as the medical director of the National Coalition of Black Lung and Respiratory Disease Clinics, the coalition of Black Lung Clinic Programs also funded by HRSA. There are 60 HRSA clinic sites across 15 states which serve over 13,000 coal miners.¹

My testimony will cover three issues.

First, there is extensive evidence that there is a resurgence of Black Lung in the United States, especially in its most severe forms.

Second, there is evidence indicating that over exposure to respirable crystalline silica is an important contributing factor to this resurgence.

Third, there are short and long term policies that MSHA should adopt to stem the resurgence of black lung disease.

I. Evidence of Resurgent Severe Pneumoconiosis in U.S. Underground Coal Miners

a. Surveillance of Active Miners

The NIOSH Coal Workers Health Surveillance Program (CWHSP) offers chest x-ray screening to active surface and underground coal miners to identify those miners with early signs of disease and notify them so that they might take steps to reduce exposure and prevent disease progression. Data on chest x-ray findings from the CWHSP has shown a steady decline in miners with disease dating from the passage of the Coal Mine Health and Safety Act in 1970 to the mid-1990's, falling from 30% down to a low of 5%.² Since then, however, there has been a significant surge in the numbers of active miners with scars on their chest radiographs. The most recent data indicates that 10% of miners have changes consistent with coal workers pneumoconiosis. Of great concern is the report that one in five coal miners with 25 or more years of mining experience from Kentucky, West Virginia, and Virginia, have evidence of disease compared to one in ten nationally.² (See Figure 1.) In 2005, an analysis of CWHSP chest x-ray data showed there were areas of the country where the disease appeared to be progressing rapidly (See Figure 2). These areas were labeled, "Hot Spots". Further reports have shown this central Appalachian region to be the epicenter of severe resurgent disease.

b. Disease in Former Miners

Outbreaks of hundreds of cases of the most severe form of the disease with miners exhibiting large debilitating scars, known as Progressive Massive Fibrosis (PMF) have been reported from clinics in Kentucky and southwestern Virginia.^{3,4} As a follow up study to these outbreak reports, the University of Illinois research group (UIC) in collaboration with NIOSH analyzed data from the United States Department of Labor's Black Lung Benefits program, and for the first time ever reported national trends of PMF in thousands of former miners.⁵ This study revealed that the percent of miners' with

PMF remained below 2% of claims from the inception of the program in 1970 until 1996 when the proportion of severely diseased miners increased dramatically reaching 8.3% of claims by 2014 (See Figure 3). There have been a total of 2,474 miners determined to have disabling PMF since 1996. The proportion of PMF claims from 1970 through 2016 increased by 17% in the central Appalachian states of Kentucky, West Virginia, and most severely to 31% of claims in Virginia.

These numbers, while extremely disturbing, do not reflect the individual struggles that these miners with severe forms of the disease suffer through. Those of us who care for these miners in our black lung clinics on a daily basis see them fighting for breath, often with the support of supplemental oxygen as they fight to improve their condition by exercising in pulmonary rehab programs. These miners are relatively young, some as young as their 30's and 40's. They suffer from loss of their careers, hobbies, and ability to support their families. They suffer from early mortality in spite of our most heroic attempts to treat them including referrals for lung transplantation.^{6,7}

c. Increases in Mortality:

Recently the UIC research group in collaboration with NIOSH performed another important analysis. For the first time we used identifiers from miners who participated in the US DOL Black Lung Benefits Program and the CWHSP we obtained death certificates on 34,771 deceased former U.S. coal miners who had an average of 26 years of coal mine employment.⁸ We analyzed causes of death and found a significant increase in proportional mortality from non-malignant respiratory diseases including COPD, emphysema, and pneumoconiosis among miners aged 65–74. Those who would have worked before 1950 had a proportional mortality for these diseases of 15%, this increased to 28-32% for those working in the mines between 1950 and 1990. The proportional mortality from pneumoconiosis in younger miners (<65 years) also increased significantly from 4-5% for those working before 1950 to 7.2% for those working between 1960 and 1990. This pattern of mortality for these preventable lung diseases should not be occurring, rather we should be seeing a decrease in proportional mortality of pneumoconiosis and for non-malignant lung disease especially since miners began working in mines operating under dust control regulations mandated by the 1969 Federal Coal Mine Health and Safety Act.

d. Conclusion

I believe that the evidence from surveillance of active working miners and from former miners is convincing. There is a resurgence of coal mine dust lung disease in US coal miners. We see this in chest x-ray surveillance data, black lung claims data, and in mortality data. This resurgence is most severe in the central Appalachian states of Kentucky, West Virginia, and Virginia. There is also significant evidence that the disease is not just mild, but in fact the most severe disabling forms of this preventable disease are occurring in younger miners.

II. What is the role of silica in resurgent severe coal mine dust lung disease?

a. Radiographic evidence

Respirable crystalline silica (RCS) is present in rock strata, above, below, and often between coal seams. Cutting coal, securing the roof and ribs, developing tunnels and shafts, cutting overcasts, and removing overburden are all activities which generate fine respirable silica dust. Freshly fractured respirable silica is highly toxic and causes significantly more lung scarring than coal dust. Cumulative exposure to RCS was associated with increasing radiographic scarring and lung inflammation.^{10–13} Larger round scars on chest x-rays have been associated with silicosis,^{14–16} and the proportion of these miners with these scars has increased in central Appalachia, paralleling the increase in severe disease in these miners.^{2,17}

b. Pathologic evidence

Chest x-rays only show a size and shape of scar/opacity that is associated with silicosis. The UIC research group decided to go beyond x-ray shadows and look at the tissues of miners with this new and more aggressive form of pneumoconiosis. In 2014 we assembled a case series of 13 miners with this disease who had undergone lung biopsies, transplant, or recent autopsy in order to look more carefully at their lung tissue. These miners from West Virginia, Kentucky, and Pennsylvania, worked in jobs with heavy exposures such as continuous miner operators and roof bolters. We published the results of this work in 2015.¹⁸ Under the bright light and polarizing light microscope you can clearly see the type of dust in the scar tissue. The surprising finding was that only one of these miners had a classic coal dust form of pneumoconiosis. Six miners had silicosis, or a silica predominant mixed dust disease and four additional had an evenly distributed mixed dust. These findings were clearly an alarm for our research group. The experienced occupational pulmonary pathologists who reviewed this material had not seen such severe silicosis in coal miners. This led the UIC research group to plan two follow up investigations.

We realized that NIOSH had lung pathology material from more than 7,000 coal miners whose autopsied lungs were submitted as part of the NIOSH National Coal Workers Autopsy Study. We decided to look back at these materials, search for the severe cases, and evaluate the type of PMF that was present and see if there were changes over the decades in the pattern of disease. We classified these cases as coal dust, mixed, or silicotic type of PMF.¹⁹ This had never been done before. We examined the tissues obtained from 376 miners born between 1885 and 1961 who had an average of 33 years of mining experience. These findings were presented at the American Thoracic Society Meetings in Dallas a few weeks ago and showed that there has been a statistically significant change in the type of PMF over historical periods from only 24% of miners having a silicotic type of PMF in specimens accessioned before 1990, to more than 40% after 1990 (See Figure 4). This indicated that miners dying more recently were nearly twice as likely to have a silicotic type of disease.

c. Mineralogic Evidence

The next study we planned was an evaluation of the mineral particles present in the lung tissue of miners with RPP and PMF. This would allow us to determine if silica was playing an important role. We decided to go beyond light microscopy to field emission scanning electron microscopy and an x-ray probe to examine the particles in the tissue of 100 modern miners compared to 100 autopsied miners' lungs obtained prior to 1996. We have completed this work on the first eight miners recently diagnosed with rapidly progressive pneumoconiosis and PMF and ten historical comparisons. We found that the concentration and proportion of silica particles in the tissues nearly doubled and was statistically significant. These preliminary findings will be presented at the Society of Toxicologic Pathology next week in Raleigh, North Carolina.

d. Conclusion

I believe that the resurgent epidemic of black lung in central Appalachia is driven in significant part by excessive exposure to respirable crystalline silica. This is supported by the chest radiographic findings in surveys of active miners, the case series studies of the pathology in current miners, the change in type of PMF seen in historical cohorts of miners' lung tissues in the National Coal Workers Autopsy program, as well as our preliminary studies of the mineralogy found in the lungs of current miners compared to those from the last quarter of the twentieth century.

These findings are consistent with high exposures to silica resulting from changes in mining practices including mining thin seam mines as well as using continuous miners to develop slopes and tunnels.

III. Recommendations

The passage of the new 2014 dust rule, "Lowering Miners' Exposure to Respirable Coal Mine Dust, Including Continuous Personal Dust Monitors" which enacted new regulations to monitor and control dust levels in our nations mines was a significant step forward.²⁰ However, I believe that MSHA needs to go further and sample respirable crystalline silica more frequently than quarterly using current technologies. Current sampling technology takes days or weeks before silica exposure monitoring results are returned to the mine.

The good news is that NIOSH has developed an in-mine silica analyzer which is paired with a Personal Quartz Monitor to provide reasonable estimates of exposure to RCS, such as at the end of each shift. MSHA should require coal operators to use this technology to better understand exposures to RCS, and which will allow for a much more timely response by operators to control excessive exposures. Daily silica monitoring results could better inform MSHA inspectors on the implementation of ventilation plans or the need to place the mine on a reduced standard. This technology should be provided to miners working in mine development, and in those designated occupations associated with the most severe disease including roof bolters, continuous miner operators and helpers and long wall operators. The results of this data should be posted and made publicly available. MSHA should increase the frequency of monitoring for RCS until a tamper proof personal quartz monitor becomes available.

In addition, evidence indicates that MSHA's Permissible Exposure Limits (PEL) for coal mine dust should have a specific PEL for silica, and that this should equal the current OSHA standard of 50 micrograms/m³. The health risks caused by exposure to silica were recently reviewed by OSHA in preparation for its new final rule.⁹ The risk analysis and work done by OSHA to develop their new silica rule is directly applicable to miners exposed to this same dust.

Finally, I believe there should be enhanced surveillance for miners working in the hot spots of central Appalachia. They should be offered more frequent screening including voluntary participation

in chest x-ray and spirometry testing, every 2-3 years rather than every five years. Enhanced physiologic assessment diffusion capacity, a practical lung function test that can detect early lung damage should also be strongly considered in addition to spirometry.

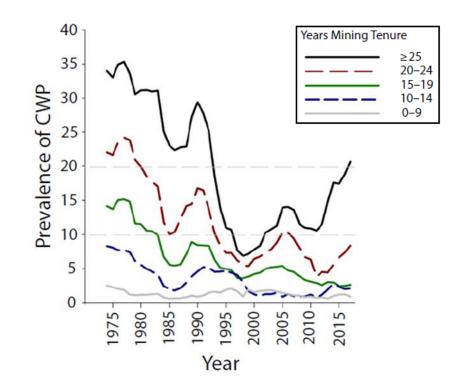
Thank you for the opportunity to provide this testimony. I am pleased to answer any questions you may have.

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Figure 1



Prevalence of Coal Workers' Pneumoconiosis (CWP) Among Those Working Underground in Central Appalachia, Coal Workers' Health Surveillance Program, Appalachia, 1970–2017

Figure 2

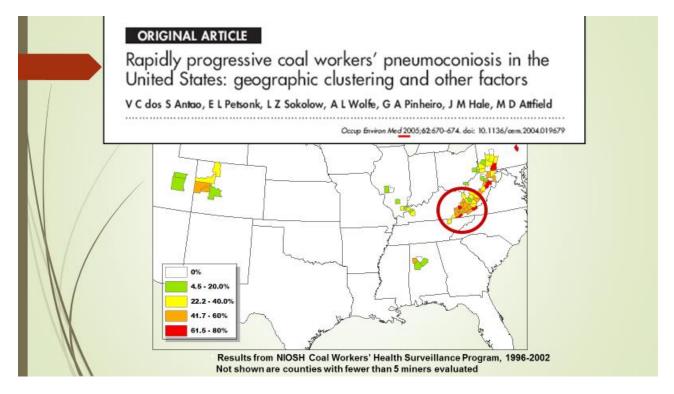


Figure 2: Geographic distribution of the proportion of miners with rapidly progressive pneumoconiosis, color coded by increasing proportion. Counties with a high proportion of cases have been labeled "HOT SPOTS". Note the clustering of Hot Spot counties in Southern WV, Eastern KY, and southwestern VA.

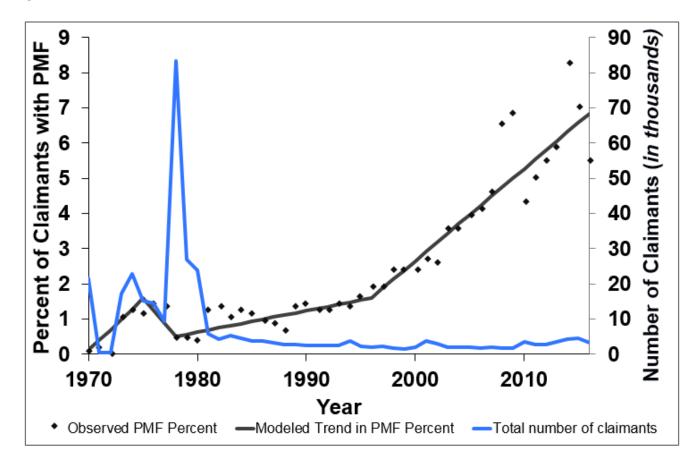


Figure 3

Figure 3. Number of claimants for Federal Black Lung Program benefits and the percentage of these claimants that received a determination of progressive massive fibrosis (PMF) during their claim process, 1970–2016. Data source: U.S. Department of Labor, Office of Workers' Compensation Programs, Division of Coal Mine Workers' Compensation.

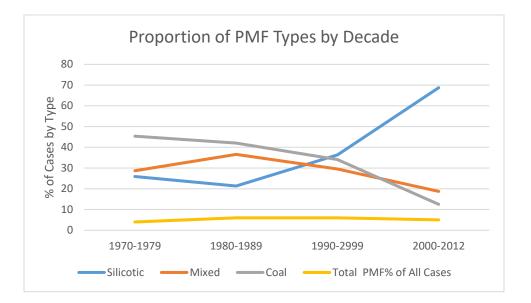


Figure 4. Proportion of PMF cases in National Coal Workers Autopsy Study by type, 1970-2012. Silicotic-type PMF - fused silicotic nodules as the predominant feature (> 75%) of the area of the lesion, mixed-type PMF - >25% but \leq 75% silicotic nodules, coal-type PMF had \leq 25% silicotic nodules.