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Trends in pneumoconiosis mortality and morbidity for the United States, 1968–2005, and relationship with indicators of extent of exposure

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Abstract. This surveillance report examines trends in selected pneumoconioses in the U.S. for 1968–2005 and their relationship with past indicators of extent of exposure. Numbers of deaths with asbestosis, silicosis, and coal workers' pneumoconiosis (CWP) were tabulated by time and age at death. Worker monitoring CWP prevalence data were tabulated by tenure group. Information on indicators of extent and intensity of exposure were obtained from various sources. Asbestosis deaths from 1968–2005 closely followed the historical trend in asbestos consumption, and appear to be declining in most age groups. Given appropriate exposure control, asbestosis could be eliminated by 2050. Silicosis deaths decreased substantially from 1968–2005, but levelled off after 1998 in all age groups, indicating a continuing occupational risk. In the anthracite coal region, CWP mortality has been declining rapidly. If there is no resurgence in the industry, CWP could disappear in that region by 2030. In the much larger bituminous region, deaths have declined over time but may be increasing among younger individuals. In addition, although CWP prevalence in working coal miners declined substantially from 1970 to 1994, it increased from 1995 to 2006. This indicates the need for increased vigilance in dust control in underground coal mining.

1. Introduction

Although silicosis and coal workers' pneumoconiosis (CWP) have been recognized occupational diseases for many centuries, the industrialization led to a substantial rise in the prevalence of these diseases and associated mortality. Similarly, the increasing use of asbestos starting in 1900 led to the epidemic of asbestosis and other asbestos-related diseases. From 1968–2005 alone, over 100,000 individuals died in the U.S. with mention of at least one of the three conditions on the death certificate [1]. The belated recognition of the burden and costs of these diseases on individuals and the state motivated the introduction of workplace control measures and airborne dust standards in the 1970s in the U.S., thus reducing exposure levels. Now that almost 40 years have elapsed from the introduction of the first major U.S. federal exposure limit (for coal dust in 1969), it is time to assess what progress has been made in eliminating pneumoconiosis and what the future might hold. Accordingly, this paper presents information on the temporal patterns of mortality in the U.S. for the three principal pneumoconioses: asbestosis, silicosis, and CWP, and examines their trends with indicators of extent exposure as well as discussing the likely impact of the compliance dust standards introduced around

the 1970s. Mortality was selected as the principal outcome variable because there are no satisfactory national morbidity data on asbestosis and silicosis. National morbidity data on CWP do exist and are included in this analysis. The findings presented here should be considered preliminary.

2. Methods

2.1. Mortality data

U.S. national data from death certificates for 1968–2005 were employed. These data were supplied from by the National Center for Health Statistics, with the cause of death coded using the International Classification of Diseases (ICD) system in force at time of death. The following ICD codes were used for each cause: asbestosis – 515.2 (ICD-8), 501 (ICD-9), and J61 (ICD-10); silicosis – 515.0 and 010 (ICD-8), 502 (ICD-9), and J62 (ICD-10); and CWP – 515.1 (ICD-8), 500 (ICD-9), and J60 (ICD-10). All mentions on the entity axis of the death certificate, i.e., both underlying and contributing causes of death, were included. Industry information, extracted from the death certificates and available on a subset of deaths from 1985–1999 for certain states was employed to aid in interpretation of the data on silicosis. All of the mortality information was derived using the National Institute for Occupational Safety and Health (NIOSH) web-based interactive mortality query system [1].

2.2. Morbidity data

NIOSH has operated a federally-mandated worker monitoring program for underground coal miners since 1970 [2]. Every working underground coal miner is eligible for free periodic chest radiographs. These are processed by NIOSH, and read by at least 2 NIOSH readers using the ILO system for classifying the pneumoconioses. Data are presented here for 1970 – 2006 by tenure group for prevalence of category 1/0 or greater small opacities.

2.3. Indicators of extent of exposure

To put the mortality trends into perspective, we compared them with surrogates of extent of exposure. These differed by outcome and were selected on the basis of the nature of the exposure to the material concerned. For asbestos, the best indicator of extent of exposure available was asbestos consumption, the logical basis being that the more asbestos consumed, the more people are exposed. Data on consumption came from a U.S. Geological Survey report [3]. Silicosis proved more troublesome, in that airborne silica dust often arises in processes incidental to the main product being extracted, made, or maintained. We examined a range of potential data sources, including employment, consumption, and production data available for much of the 20th century for various industries where silica exposure is known to occur. These included metal mining, quarries, sand and gravel, iron and steel production, and construction. For CWP, since few are exposed to coal mine dust apart from those mining the material, the surrogate for extent of exposure was number of underground coal miners. Different patterns of employment and disease occurrence led to us examining the anthracite and bituminous regions of the U.S. separately. In this, the anthracite region was defined as those counties in the eastern third of Pennsylvania, while the bituminous region encompassed all of the other coal fields in Pennsylvania and elsewhere. Employment information on coal mining came from a U.S. Energy Information Administration report [4].

3. Results

The number of asbestosis deaths reported in four age-at-death groups from 1968–2005 together with asbestos consumption is shown in figure 1. Deaths in the oldest age group (75-84) were rising but may have peaked recently. In contrast, deaths in the three younger age groups appear to be declining, with their peaks at earlier years the younger the age at death (142 and 22 for the two youngest age groups in

1986 to 92 and 15 in 2005). In general, the trends in deaths tend to match that for consumption, particularly for the older age groups.

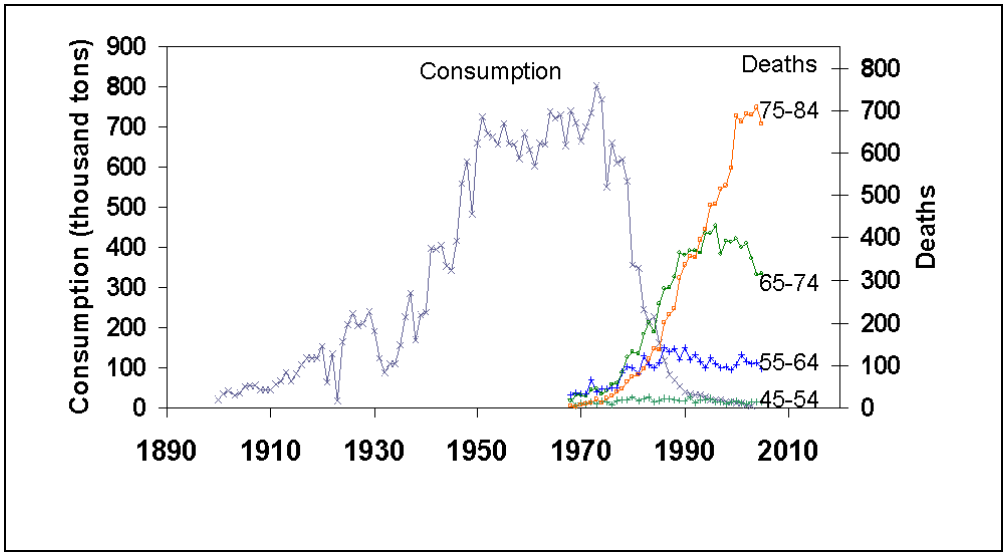

Figure 1. Asbestosis deaths by age at death (1968–2005) and asbestos consumption (1900–2003).

Figures 2 and 3 show silicosis deaths from 1968–2005 with two indicators of extent of exposure: employment in metal mining (figure 2) and employment in construction (figure 3). Fewer deaths over time are seen for all age-at-death groups. The trend in silicosis deaths appears to match employment in metal mining but not that for construction. During 1985–1999 metal mining was reported as the industry on the death certificate for 9% of the deaths, while 12% reported construction work.

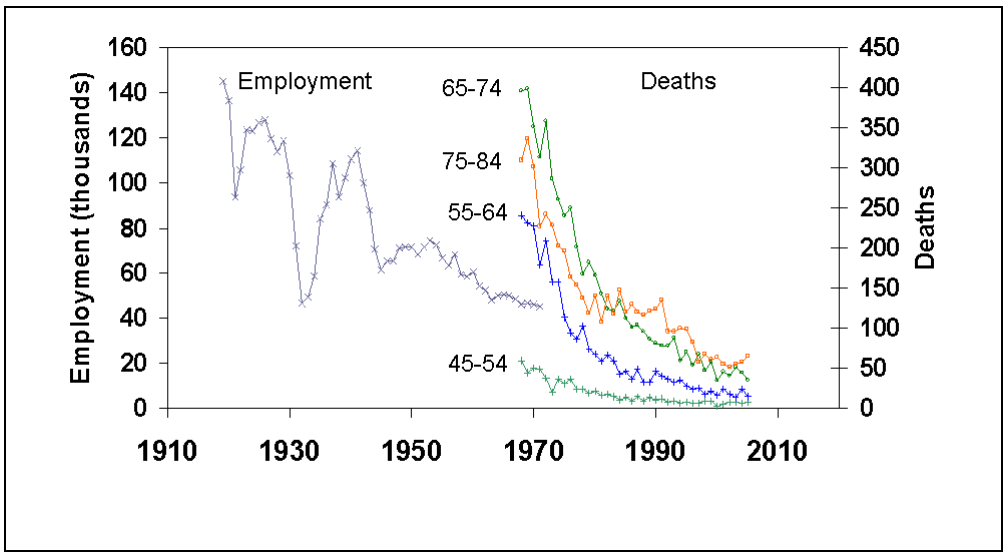

Figure 2. Silicosis deaths by age at death (1968–2005) and employment at metal mines (1919–1971). (9% of all deaths with silicosis from 1985–1999 were associated with metal mining.)

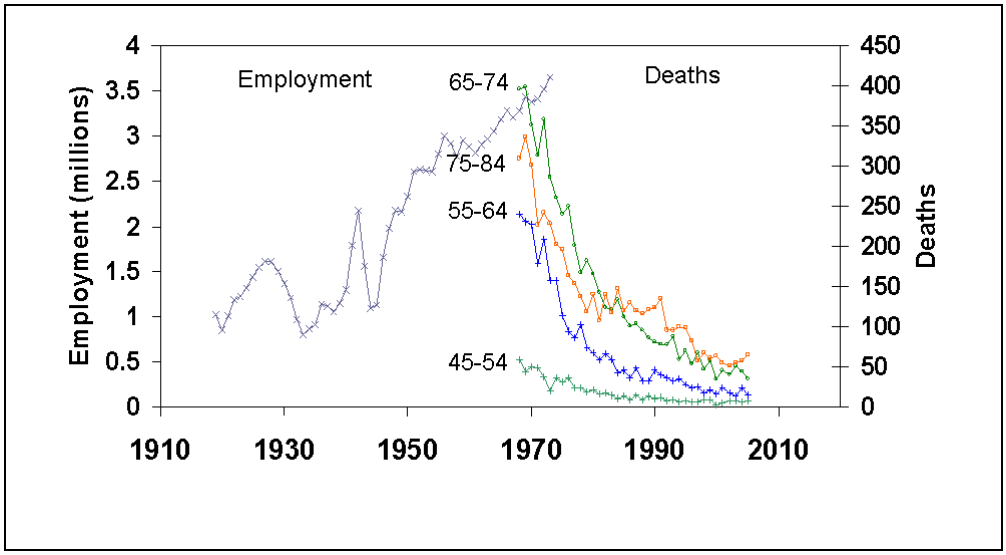

Figure 3. Silicosis deaths by age at death (1968–2005) and employment in construction (1919–1973). (12% of all deaths with silicosis from 1985–1999 were associated with construction.)

Information for CWP deaths in the anthracite region is shown in figure 4. It is clear that coal production has virtually ceased in this area and has been low for the last 30 – 40 years. Mortality in the youngest age-at-death group (45–54) is down to 0.5 cases per year with mention of CWP.

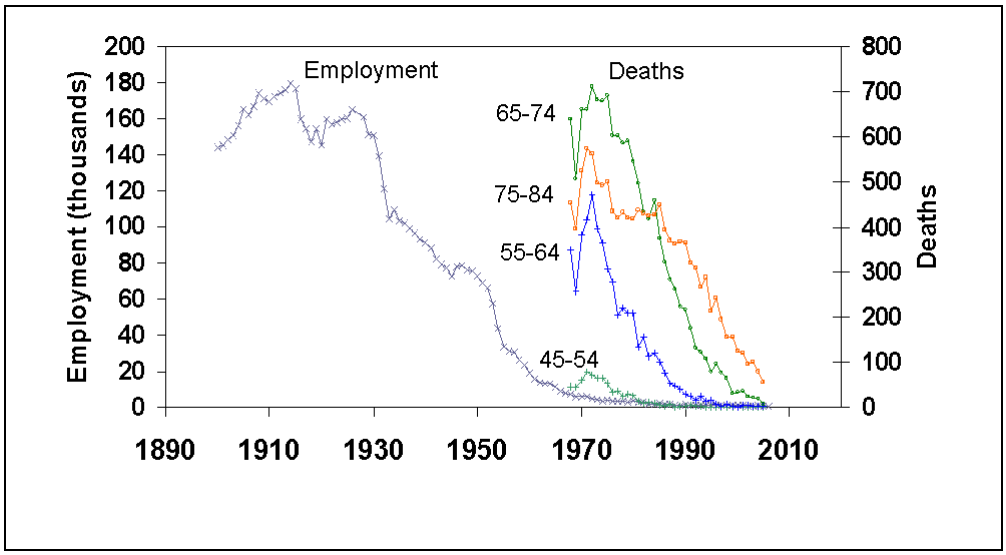

Figure 4. CWP deaths by age at death (1968–2005) and employment (1968–2006): anthracite region.

Figure 5 shows the equivalent picture for the bituminous region. As with the anthracite region, coal mining employment has fallen considerably although, unlike anthracite, there was a rise during the 1970s. Deaths with CWP rose initially in all age groups, and then fell, although there is a disconcerting indication that deaths in the younger age groups are rising.

Unlike the other outcomes studies here, national morbidity data exist for CWP. These data provide CWP prevalence data from 1970 – 2006 (figure 6). Since these are prevalence data, they reflect

changes in intensity of exposure to coal mine dust and are independent of the number of miners exposed. They therefore complement the employment data and supply additional information on which to assess the temporal changes in mortality. Overall, the decline in prevalence evident until 1995-1999 appears to reflect and vindicate the reduction in exposure mandated by the 1969 U.S. Coal Mine Health and Safety Act. However, since 1995 there has been disturbing evidence that the prevalence of CWP has been rising (figure 6). Most of these miners would have started work after the introduction of the current federal compliance limit on dust exposure.

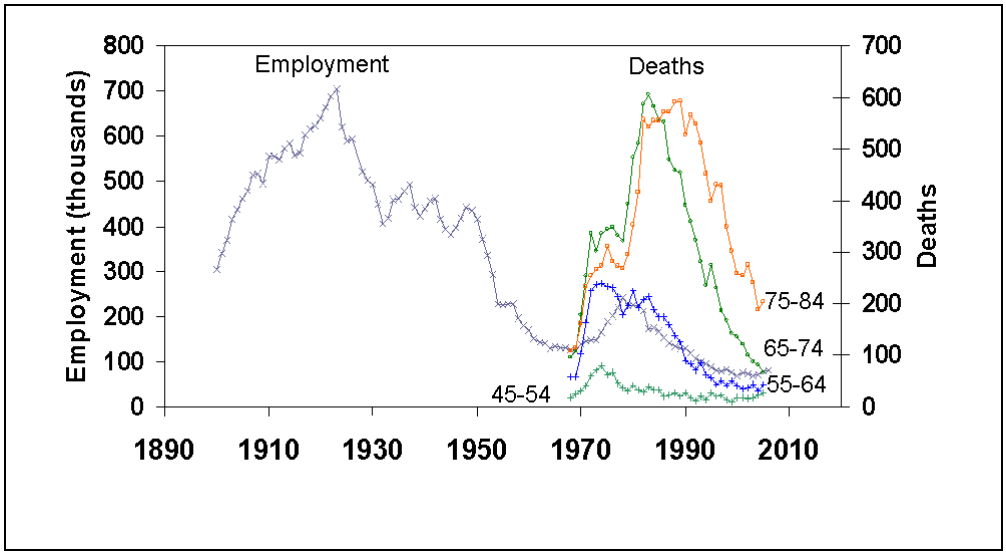

Figure 5. CWP deaths by age at death (1968–2005) and employment (1968–2006): bituminous region.

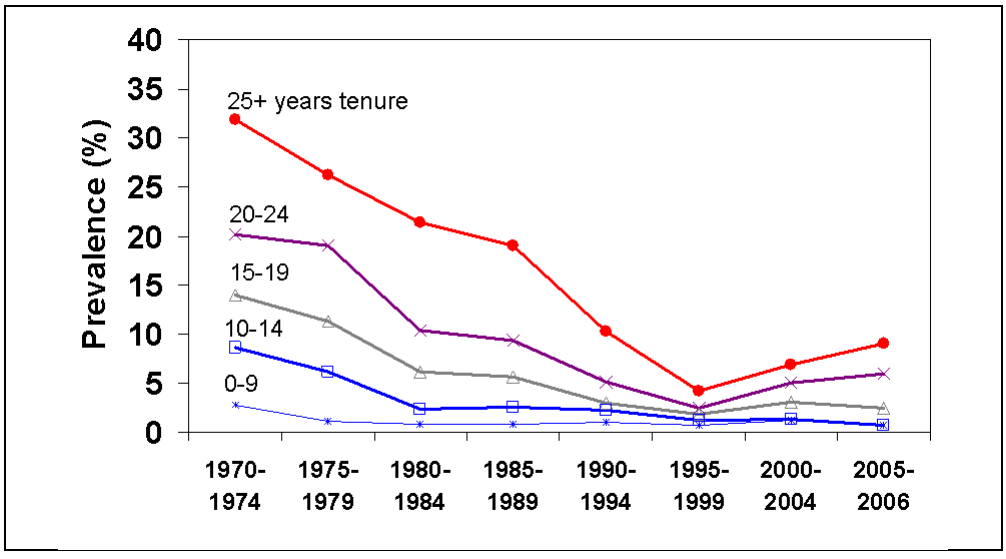

Figure 6. CWP prevalence among working coal miners (1970–2006) by tenure in mining.

No attempt was made to include deaths at age 15-44 for the three outcomes in the above description as they are too few for adequate representation. However, they confirm the general picture

for the deaths in the adjacent 45–54 year age group for each outcome – asbestosis deaths appear to be trending lower, bituminous CWP deaths appear to be rising, and silicosis deaths are stationary.

4. Discussion

The correlation observed between deaths with asbestosis and asbestos consumption, coupled with the downward trend in consumption, suggests that the disease will eventually decline in importance and could even disappear. A preliminary tentative analysis undertaken by juxtaposition of the consumption and mortality curves suggests that this might occur around 2050 for the oldest age-at-death group, and before then for the younger age groups. However, this speculation is grounded totally on the assumption that asbestos exposure arises solely from asbestos usage (consumption). Although continued manufacture and application of new asbestos-containing products is still occurring at a low level (with future asbestos consumption being contingent on efforts to ban its use [5]), it could be that the bulk of asbestos exposure to workers in the future will increasingly come from the handling of existing asbestos installations and products. As buildings, factories, and ships that incorporate asbestos in their construction reach the end of their useful life they will be demolished, and those doing that work will be at risk of exposure. Even if buildings are not demolished but are renovated or asbestos is remediated there remains a great risk of exposure. Non-adherence to airborne compliance limits will lead to overexposure and continuing future mortality from asbestosis and other asbestos-related disease. Knowing what we know now about the toxicity of asbestos and how to prevent disease, it would be tragedy if this were ignored and a new epidemic of disease came about.

The apparent correlation between past asbestos consumption and observed mortality observable in Figure 1 supports the derivation of predicted deaths for future years. Deaths for the oldest age-at-death group appear to be peaking. This could coincide with the peak in consumption around 1950. It took 40 years for consumption to decline to negligible levels after 1950, suggesting that asbestosis mortality might continue for another 40 years (i.e., to around 2050) in that age group. Since there is evidence that the peaks in mortality have already occurred for those dying at younger ages, asbestosis mortality might cease to be a major problem before then for those age groups.

Nicholson and colleagues [6] made an intensive study of asbestos exposure and its associated disease outcomes, including a detailed attempt to predict future mortality. In this, the focus was on cancer outcomes, although a small part of the report mentioned asbestosis deaths. They looked only at underlying causes and so their results cannot be compared directly with those presented here. They stated that 200 asbestosis deaths were occurring (presumably around 1980) and that the number would perhaps double during the next two decades (i.e., to around the year 2000) and decline thereafter. It is not exactly clear from where they obtained their claim of 200 deaths, since the actual number around the time the paper was published was about 100 (89 in 1979, 101 in 1980, and 101 in 1981). Nevertheless, they overstated the deaths for 1980, and understated the increase, there being 561 deaths in 2000 – a 5-fold rise. Their statement that asbestosis deaths would decline thereafter is not yet being realized since overall underlying deaths were stationary at around 550 in 2000-2005. However, the overall number is greatly influenced by the deaths occurring at age 75-84. As are seen in figure 1, downward trends in numbers of asbestosis deaths are evident in the younger age groups before 2000.

While there are logical and very pertinent indicators of extent of exposure for asbestosis (consumption) and CWP (employment), silicosis lacks such obvious surrogates. It is often a disease resulting from incidental exposures in the making or processing of other products rather than arising from the production of a silica-based product [7]. While there are obvious industries where silica is the main product (ground silica mining and production, for instance), or where there is a direct link between exposure and product (crushed stone quarrying), the link between the principal product or activity and concomitant silica exposure is less obvious in many other important situations. Such sources of silica exposure are manifold, e.g., sand blasting and road saws in construction; foundry work; ore extraction in metal mining; pottery and clay production; and grinding operations in

manufacturing. The variety of exposure situations makes it difficult to arrive at one succinct, pertinent indicator of extent of exposure to silica.

The number of silicosis deaths over time followed the trend in employment in metal mines. However, this relationship could not explain the overall pattern in silicosis deaths since only 9% of deaths were associated with work in metal mining (1985–1999), even though this was the greatest percentage associated with any specific industry. For example, 6% were associated with work in non-metal mining and quarrying. The Construction industry sector, as a whole, accounted for 12% of all deaths, but as is seen in figure 3, employment in construction shows a rise, not a fall, over time. In fact, most indicators of extent of exposure we could locate showed rises in employment or production over time. Many of these did show dips during the Great Depression years. However, anyone working at that time would likely have been born around or before 1900 and so would have died before 1980. So, while this could possible have impacted the trend in deaths for the oldest age group, it could not have impacted the similar downward trends in all of the younger age groups. Nor could all of the silica deaths come from industries for which we might not have obtained exposure surrogates. Of all deaths with silicosis between 1985 and 1999 for which we have information on usual industry, 58% reported working in mining, primary metal industries, construction, and stone, clay, glass, and concrete products [1]. These were industries for which we had information on employment, production, and/or consumption. Ultimately, then, the implication is that the reduction in deaths came about, at least in part, from reductions in worker exposures, probably both prior to and after the imposition of federal compliance limits in the 1970s.

It is possible that some CWP deaths were otherwise reported as silicosis. Some evidence for this can be found in looking at the usual industry and occupation on the death certificate, which mentioned coal mining and coal mining-related jobs in 7% of the deaths reported with silicosis. Whether these are true cases of silicosis, perhaps acquired during work on surface coal mines, or are misdiagnosed/misreported CWP is unknown. However, removal of the silicosis deaths from states where coal mining is prevalent did not eliminate the overall downward trends in deaths. Hence, the observed declines seen in figures 2 and 3 did not simply result from the downward trend in numbers with pneumoconiosis associated with reduced coal miner employment as noted for CWP below. Another possible disease-diagnosis issue relates to the recording of interstitial pulmonary fibrosis (IPF). A number of researchers have examined this issue, the results of which have been summarized by Taskar and Coultas [8]. Their conclusion is that IPF could be an occupational disease and might, in some cases, be simply a misdiagnosis of pneumoconiosis, particularly silicosis. Changes in diagnosis therefore could have impacted the trends in silicosis deaths. However, no data exist with which to evaluate this question. Since the number of deaths with silicosis appears to be currently stable, with no great evidence of continuing decline, silicosis mortality is expected indefinitely into the future. Adoption of the NIOSH recommended limit of 0.05 mg/m3 would aid in reducing this ongoing burden of disease.

For CWP, the reduction in employment in the anthracite region to the two hundred or so individuals currently employed clearly implies that few future cases of CWP will occur. An informal analysis based on past employment of deaths in the two older age groups (where there is the greatest legacy of past exposures), suggests that anthracite CWP deaths will decline to near zero about 2025 and 2037, respectively. This observation is conditional on the continuation of present trends and no revitalization of coal mining in the region. Since deaths appear to be declining faster than past employment in the anthracite industry, it may be that the above predictions are pessimistic and that an estimate of 2030 for the oldest age group is more realistic. With respect to CWP in the bituminous region, it is clear that substantial numbers of miners remain exposed. In addition, in 2008 bituminous coal mining employment was expanding. This alone, ignoring issues concerning the current effectiveness of the current federal dust compliance limit (discussed below), would lead to CWP mortality continuing indefinitely into the future. In fact, this may be the reason that mortality appears to be rising in the younger age groups, its trend following that of the increasing employment in the 1970s. Further follow-up is needed to monitor this situation.

Migration between the anthracite and bituminous regions may impact the findings for CWP. Due to the virtual demise of the anthracite coal industry, it is possible that many anthracite workers migrated to the bituminous coal fields, their deaths there artificially inflating the number of bituminous deaths and likewise underestimating the number of anthracite deaths. Some migration in the opposite direction could have occurred, although it is likely to be less. It is not known to what extent migration took place, as information on the source of work exposures is not given on the death certificate.

The employment data used in the analysis of CWP for both regions includes both surface and underground miners (data giving only underground employment has so far not been identified). The ratio of surface to underground employment has increased over time. Therefore, the number of miners experiencing the higher coal mine dust exposures more prevalent at underground mines decreased rather more rapidly than the employment trends on figures 4 and 5 indicate. Further research on this is needed.

Changes in the cultural understanding of the extent and severity of CWP in the U.S. coalfields have occurred over time. In addition, the ICD system revised its definitions of CWP substantially in the move from ICD-8 to ICD-9. In ICD-8 the principal term used was anthracosilicosis, whereas in ICD-9 the term was coal workers' pneumoconiosis. Overall, this change most likely impacted reporting in the bituminous region since it not only suggests that anthracite miners are affected by their work exposure but also implies that silica exposure is also involved in disease development. Epidemiological evidence emerging in the 1960s and 70s has since clearly shown that CWP does occur from exposure to bituminous coal mine dust, is related to cumulative exposure to that dust, and that silica exposure plays only a minor role in the causation of CWP. Increasing acceptance of the epidemiological findings may have led to the rise in reported deaths with pneumoconiosis in the bituminous region. In the anthracite region, in contrast, with its long history of coal mining (going back into the 18th century) and the higher risk of disease development from anthracite coal [9] it seems likely that CWP was recognized more fully and more promptly than in the bituminous coalfields. The ICD change in definition in 1979 probably came at a time when there was a greater appreciation of the risks that coal miners endure and so did not greatly impact the reporting of CWP in the anthracite region.

As noted above, it is also possible that some CWP was listed as silicosis (and vice versa), since the two diseases can be hard to distinguish medically. Certainly, we have evidence that coal miners have often been reported to have died with silicosis rather than CWP [10]. Possibly some or most of those cases may have worked at surface mines, but this is unknown, and further research is needed. A related issue arises from the reporting of Unspecified Pneumoconiosis. The geographical distribution of deaths with this cause is similar to that for CWP [10]. They may therefore have been CWP deaths, except that the recording physician used the shorthand 'pneumoconiosis' instead of stating CWP explicitly. Again, further research is needed to elucidate this issue.

In terms of predicted mortality for the future, the almost complete decline in the anthracite coal industry indicates that new cases of CWP should be very few and that current mortality arises from the legacy of past exposures. Some tentative extrapolation of the current temporal decline for the oldest age group suggests that mortality with CWP in the anthracite region may virtually disappear by 2030. The situation is much more complex for the bituminous coal region. Here employment appears to be cyclical, depending on the need for coal, with past increases and falls, and a current ongoing increase. Given no impact of other factors, the latter observation suggests that the number of CWP cases should similarly fluctuate in synchrony with past employment. Moreover, the observed increases in disease prevalence and mortality at younger ages do not bode well for the future.

There are several external factors that could have impacted the observed trends in deaths over time. The first is mass screening for asbestosis by legal firms and others intent on obtaining legal compensation for their clients led to the identification of many reported cases of asbestosis. Overall, the number of claims rose sharply in the mid-1990s, but declined abruptly in 2004 [11]. The recognition of the cases by this means might carry over to the later reporting of asbestosis on the death certificate. The mass screening for asbestosis has been followed by similar activities for silicosis. Therefore, the temporal trend in screening activity for compensation for these two diseases could

impact the later temporal trend in reported mortality. Unfortunately, reliable data do not exist to assess the magnitude of this effect, if any, on the predictions. Federal compensation for CWP was introduced in the 1969 Coal Mine Health and Safety Act. The immediate effect is that affected individuals applied for, and received compensation, and then retired from coal mining. It is possible that part of the reduction in prevalence seen in the worker monitoring program resulted from the loss of those individuals from the workforce. Leaving coal mining also meant an end to coal mine dust exposure and so a reduction in risk of the disease progressing. However, it seems unlikely that this would impact the reporting of CWP on the death certificate for such individuals. Rather, receipt of compensation probably increased the chance of a report of CWP on the death certificate.

Is it possible to discern whether the U.S. federal compliance dust standards introduced around the 1970s are having an effect on mortality? Clearly, workers who spent time working after the U.S. federal compliance standards were imposed should have realized some benefit, resulting in fewer pneumoconiosis deaths that might have been the case if no standards had been imposed. Is this observable in our results? Of all age-at-death groups, the oldest would be expected to be the least impacted, as their work time under the reduced dust levels is very limited. Examination of the data for that age group for silicosis and CWP shows that they have slower rates of decline compared to the other age groups. For asbestosis, they are the only group whose mortality is still rising. The faster rates of decline of deaths at younger ages compared to the older age group therefore imply that workers have benefited from the imposition of federal dust standards. Overall, the mortality data are not a very satisfactory way to examine this issue. A much better approach is to examine morbidity data, particularly disease prevalence. For CWP, there is convincing evidence that the dust standard has been effective. But, as noted above, the recent increases in CWP prevalence and mortality are disturbing and point to a failure in dust control.

5. Conclusion

This analysis has examined U.S. deaths with asbestosis, silicosis, and coal workers' pneumoconiosis over the period 1968–2005 in relation to past indicators of extent of exposure to asbestos, silica, and coal dust. The findings for asbestosis suggest that due to the legacy of past exposures, older individuals will continue to die with asbestosis until about 2050; deaths among younger individuals may virtually disappear earlier. This prediction is totally contingent upon the effective control of asbestos fiber exposures from demolition and remediation work, as well as in the small remaining manufacturing industry. There has been substantial progress in reducing the burden of mortality from silicosis. Since there is no obvious relationship with diminished employment in at-risk activities, temporal reductions in exposure intensities may well have played a major role in the reduction in mortality. However, deaths with silicosis are still occurring, and the continuing occurrence in younger individuals suggests that intense overexposures remain too frequent. Owing to the virtual cessation of anthracite mining activity, and given no resurgence in the industry, the last few deaths with CWP in that region might well occur around 2030. The situation with respect to CWP in the bituminous coalfields is mixed. There has been a long period of reduced mortality and morbidity, probably due to both reduced mining employment and better dust control. However, the last 8 years has seen a disappointing rise in disease prevalence. At the same time CWP mortality in younger coal miners appears to be rising, rather than dropping as would be expected. Clearly, attention to better dust control is necessary, coupled with a reduction in dust levels to the limit recommended by NIOSH in 1995 [12]. These activities are even more critical when it is understood that bituminous coal mining employment is increasing currently.

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