Rapid Communication

24 Years of Pneumoconiosis Mortality Surveillance in Australia

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Abstract: 24 Years of Pneumoconiosis Mortality Surveillance in Australia: Derek R. SMITH, *et al.* International Center for Research Promotion and Informatics, National Institute of Occupational Safety and Health—Asbestosis, silicosis and Coal Worker's Pneumoconiosis (CWP) represent three of the most important occupationally-related dust diseases in Australia. To gain a clear picture of pneumoconiosis trends over time, a 24-yr retrospective analysis of national mortality data was performed for the period 1979 to 2002. Over 1,000 pneumoconiosis-related fatalities occurred during this time, 56% of which were caused by asbestosis, 38% by silicosis and 6% by CWP. Between 1979 and 1981, silicosis accounted for 60% of all pneumoconiosis-related fatalities in Australia, followed by asbestosis (31%). By 2002 however, asbestosis was causing 78% of all fatalities, while silicosis accounted for only 19%. Asbestos-related mortality increased three-fold between 1979 and 2002, with a clear excess risk demonstrated among males. On the other hand, mortality rates for silicosis and CWP declined significantly during the same time period. Overall, this study suggests that pneumoconiosis, particularly asbestosis, continues to be an important occupational disease in Australia. Although progress has been made in reducing deaths due to occupational silicosis and CWP, asbestosis rates continue to rise, reflecting the long latency between dust exposure and clinical disease. Countries which continue to use asbestos products in the workplace should note the tragic legacy of this material within contemporary Australia.

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Pneumoconiosis is a broad term referring to pulmonary

fibrosis and other parenchymal changes caused by the inhalation and deposition of mineral dust into the lungs¹. Aside from lung cancer, occupational pneumoconiosis represents one of the most important causes of respiratory disability throughout the world². Due to the long latency period many individuals with early-stage pneumoconiosis are often asymptomatic³, although disease progression usually results in major disability and premature death. There is no effective treatment, and the only viable preventive strategy is reducing the amount of dust to which workers are exposed⁴. From an occupational perspective three of the most common subtypes in Australia are asbestosis, silicosis and Coal Worker's Pneumoconiosis (CWP), as they usually arise from exposure to compounds which are rare outside the work environment. Mining continues to be an important source of employment within this country⁵, and has also played a key historical role in national development⁶. As such, there are a relatively large number of current and ex-miners in Australia with a history of occupational dust exposure.

There are many good reasons for investigating pneumoconiosis death rates longitudinally, and the use of national mortality data offers substantial benefits when investigating the progress of both occupational and non-occupational diseases¹. Firstly, the data is national in scope. Secondly, it involves a large and comprehensive study population. And thirdly, national mortality information is readily available in many countries. Most importantly, mortality surveillance can provide accurate historical information on the progress of diseases; and if monitored long enough, may also be used to assess the effectiveness of preventive measures¹. The most universal method for classifying causes of death is the International Statistical Classification of Diseases and Related Health Problems (ICD), which has its origins the late 19th century⁷, and is currently overseen by the World Health Organization (WHO)⁸. Revisions are made every 10 years, and reflect the continual refinement and understanding of disease causation, both domestic and occupational. Given the accuracy of national ICD data

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and its key occupational relevance, we considered it necessary to investigate contemporary pneumoconiosis mortality in Australia.

Methods

This study utilized a retrospective analysis of Australian national mortality data from 1979 to 2002. Data was stratified according to ICD Versions 9 and 10, as specified by the WHO⁸⁾. The three most important occupational pneumoconiosis categories in Australia were as follows: *Coal Workers' Pneumoconiosis* (ICD 9: 500 / ICD 10: J60), *Pneumoconiosis due to Asbestos Dust* (ICD 9: 501 / ICD 10: J61) and *Pneumoconiosis due to Silica Dust* (ICD 9: 502 / ICD 10: J62). Case fatalities for each of the three disease classifications were extracted from the Australian Bureau of Statistics (ABS) national mortality data set, further details of which are available elsewhere⁹⁾.

The time period between 1979 and 2002 was selected for retrospective analysis, as 1979 was the first year that Australia began to code mortality data using ICD 9. There is an excellent degree of concordance between ICD 9 and ICD 10, with regard to these three dust diseases. In the ICD codes used in Australia prior to 1979 there were some important overlaps in disease categories, particularly J60 to 62, meaning that comparison with earlier data was not appropriate. The most recent information collected by the ABS was for deaths occurring in 2002, and therefore, this point was used as the termination year for surveillance. Although there were a reasonably large number of fatalities from asbestosis and silicosis, the absolute number of deaths due to CWP was small when stratified by single years. The ABS has a standard policy whereby small case numbers cannot be released, as the individuals may be potentially identifiable. This meant that case-fatality data for single

years was unavailable. To address the problem and avoid privacy issues, case fatality numbers were subsequently combined into 3-yr blocks. Accurate figures for the total Australian population during the surveillance period were obtained from the ABS national data set, grouped into 3-yr blocks and also stratified by gender.

Three-year incidence rates for each pneumoconiosis subtype were calculated as crude death rates per 1 million persons, both as a whole group and by gender. Mortality rates were established using all deaths per three-year period and the total population of Australia during the same time. As there were no females who died from CWP and only one who died from silicosis, incidence rates for these two disease categories were calculated as whole-group incidence rates (both genders) and male-only incidence rates. Deaths from asbestosis on the other hand were clearly concentrated among males, and as such, the relative risk by gender was evaluated using a mortality odds ratio (where female gender=1.0). Results were displayed as Odds Ratios (OR) with 95% Confidence Intervals (95%CI)¹⁰⁾. To most easily visualize the progression of mortality trends over time¹¹⁾, all data from this study were displayed graphically.

Results

Over 1,000 pneumoconiosis-related fatalities were recorded in Australia between 1979 and 2002. Among them, 99% of all cases were male, 56% resulted from asbestosis, 38% from silicosis and the remaining 6% from CWP. Refer to Fig. 1. The proportion of deaths caused by individual subcategories varied during the surveillance period. In the first group (1979–1981), silicosis accounted for 60% of all deaths, followed by asbestosis (31%). By 2002, asbestosis was causing 78% of all pneumoconiosis-related fatalities, while silicosis was only accounting for

Fig. 1. Pneumoconiosis-related fatalities in Australia between 1979 and 2002.

Fig. 2. Mortality due to asbestosis in Australia between 1979 and 2002.

Fig. 3. Relative Risk for asbestosis among males in Australia between 1979 and 2002.

19%. Refer to Fig. 2. There were also major differences in asbestosis mortality rates between the genders, with a clear excess demonstrated among males. The crude male mortality rate peaked at 5.3 cases per million in 2000–2002, whereas for females it was only 0.1 cases per million during the same time period. The relative risk for asbestosis among Australian males ranged from 5.1 to 7.4, with an overall increased risk of 6.5 (95% CI: 5.6–7.7) when compared to females. Refer to Fig. 3. Crude mortality rates for silicosis showed a sustained decline during the surveillance period, falling from 1.8 per million in 1982–1984 to 0.5 per million in 1997–1999. A clear excess was demonstrated among males, with their crude mortality rate declining from 3.6 per million in 1982–1984 to 1 per million in 1997–1999.

Although not as distinctive as for silicosis, crude mortality rates due to CWP also declined during the surveillance period, falling from 0.6 per million (males) and 0.3 per million (both genders) in 1988–1990, to 0.1 per million (males) and 0.07 per million (both genders) in 1994–1996. Refer to Fig. 4.

Discussion

Pneumoconiosis due to asbestos dust

Asbestosis was clearly the most important dust disease in Australia between 1979 and 2002, being responsible for over half of all pneumoconiosis-related fatalities. As with other developed countries, Australia's current situation reflects the large number of people who were previously exposed to asbestos fibers from a variety of

Fig. 4. Coal Worker's Pneumoconiosis and Silicosis mortality in Australia between 1979 and 2002.

sources. Workplace exposure was no doubt an important source, with Leigh and Driscoll¹²⁾ estimating that occupations with the highest risk include ex-mine workers from Wittenoom, power station workers and railway laborers. Non-occupational exposure represents an additional risk for those who lived near asbestos mines during their operation¹³⁾. Aside from mining, a large proportion of domestic exposure also occurred following the widespread use of asbestos-based building products and piping during the post World War 2 construction boom, many of which remain in the environment today¹⁴⁾. Although asbestos has now been officially phased out¹⁵⁾, the renovation and demolition of old buildings which still contain the fiber remains a hazardous issue in Australia.

The rapid rise of asbestos-related mortalities in Australia is similar to the United States, where asbestosis mortality almost trebled between 1979 and 1990, and continued to rise thereafter^{16,17)}. There are a few possible reasons for this phenomenon. Firstly, asbestosis requires a long lag-time to develop, meaning that workers who were exposed to fibers last century will only now begin to show signs of disease. Secondly, there is the issue of comparability between contemporary data and that collected during prior research. In recent years a more efficient disease reporting program has helped increase the awareness of asbestos-related disease in Australia, ensuring that the majority of cases are now correctly identified¹⁴⁾. This may lead to marked contrasts with earlier data sets where the true incidence of asbestos-related disease was probably underestimated, due to a general misidentification of cases, and even an unwillingness of pathologists to make such a diagnosis¹⁸⁾. Furthermore, some earlier cases may have been mistakenly classified as non-specific lung cancer, leading to a further underestimation of asbestos-related disease.

Whatever the reason, by extrapolating results from our current study, it seems certain that Australia's asbestosis mortality rate will continue to rise in future. Such predictions are similar to asbestosis-related mortality forecasts for the United States¹⁹⁾ and the United Kingdom²⁰⁾.

Pneumoconiosis due to silica dust

During the current investigation, the number of silicosis cases and their contribution to total mortality declined markedly. Between 1997 and 1999 for example, there were less than 30 registered fatalities from this disease in Australia. Silicosis has also ceased to be the major cause of pneumoconiosis in this country. Between 1979 and 1981 for example, silicosis accounted for almost two-thirds of all pneumoconiosis-related fatalities. By 2000–2002 however, this proportion had declined markedly, with silicosis constituting less than one-fifth of the total pneumoconiosis cases in Australia. A similar reduction in silicosis mortality has been noted in the United States, with the overall mortality rate for this disease declining 93% between 1968 and 2002²¹⁾. Between 1979 and 1990 for example the American silicosis mortality rate had almost halved¹⁶⁾, with further reductions noted between 1990 and 1999¹⁷⁾. By 2002, the American mortality rate was around 0.66 per million²¹⁾, a result which is similar to our current study. Although silicosis mortality is clearly declining in Australia, continued surveillance will be necessary to help monitor future trends.

Coal worker's pneumoconiosis

During the current study fewer than 100 CWP fatalities were documented over the 24-yr surveillance period. Although not as distinctive as for silicosis, CWP mortality also declined during the surveillance period. The

magnitude of this decline was large, suggesting that CWP is becoming a less prevalent occupational health issue in Australia during recent years. This result is similar to previous research from the United States²²), where continuous reductions in CWP prevalence were also noted during various surveillance programs^{22–24}). Unlike Australia, where the largest decline in absolute CWP mortality was observed between 1988 and 1996, research from the United States suggests that 1990 to 1999 was the period where considerable progress was made with regard to CWP fatality reductions¹⁷). Similar progress has also been made in other developed countries such as the United Kingdom, where the prevalence of CWP is also being actively reduced in recent years²⁵). Nevertheless, mining remains an important part of the Australian economy as we enter the 21st century. The ongoing use of underground coal mining and its important contribution to the Australian economy, suggests that some CWP-related fatalities will continue to occur in future years.

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