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Asbestosis-Related Years of Potential Life Lost Before Age 65 Years—United States, 1968-2005

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1 figure, 2 tables omitted

EXPOSURE TO ASBESTOS FIBERS CAN CAUSE asbestosis and other diseases[1] after a latency of 10-40 years from initial exposure to onset of illness. Asbestos still is used in the United States (approximately 2,200 metric tons in 2006) in certain products manufactured domestically.[2] In addition, an undocumented amount of asbestos continues to be imported in products manufactured elsewhere, and a substantial amount of asbestos remains in existing buildings and manufactured products. An estimated 1.3 million construction and general industry workers in the United States potentially are exposed to asbestos each year, mainly from manipulation of asbestos during renovation or demolition activities.[3] Also, although asbestos ore is no longer mined in the United States,[4] some U.S. mine workers might remain at risk for exposure to asbestos contained in other ores. To characterize trends in premature mortality attributed to asbestosis in the United States, CDC analyzed annual underlying cause-of-death data for 1968-2005, the most recent years for which data were available.* This report describes the results of that analysis, which indicated that annual years of potential life lost before age 65 years (YPLL) attributed to asbestosis increased 64%, from an average of 146.0 YPLL per year during 1968-1972 to 239.6 per year during 2001-2005 (regression trend for the 5-year moving average, $p<0.001$), for an overall total of 7,267 YPLL (mean per decedent: 6.2) over the entire period. These results demonstrate that asbestosis-attributable YPLL continue to occur and that efforts to prevent, track, and eliminate asbestosis need to be maintained.

For this analysis, decedents for whom the *International Classification of Diseases* (ICD) code for asbestosis was listed as the underlying cause of death were identified from 1968-2005 mortality data.† Given the occupational etiology and long latency of asbestosis, analysis was restricted to deaths of persons aged ≥25 years. Standard industry and occupation information that met CDC quality criteria was available for decedents in 26 states during the 1985-1999 period.‡ After 1999, funds for coding industry and occupation were not available, and coding at the state level ceased. The number of states reporting data in any particular year varied from 16 to 22, and the number of years of data available for any one state varied from 2 to 15. Industry and occupation were classified according to two U.S. Census Bureau coding systems.§ YPLL and mean YPLL were calculated using 5-year age groups and standard methodology.[5] A simple linear regression model was used for time-trend analysis of YPLL (using 5-year moving averages).

During 1968-2005, asbestosis was identified as the underlying cause of death for 9,024 decedents. Of these, 1,169 (13.0%) were aged 25-64 years, including one (0.1%) decedent aged 25-34 years; 17 (1.5%) aged 35-44 years; 165 (14.1%) aged 45-54 years; and 986 (84.3%) aged 55-64 years, accounting for 7,267 YPLL (mean per decedent: 6.2). The majority of asbestosis decedents aged 25-64 years were male (1,125 [96.2%]) and white (1,064 [91.0%]), accounting for 7,038 (96.8%) and 6,470 (89.0%) YPLL, respectively.

YPLL attributed to asbestosis deaths increased 64%, from an average of 146.0 per year during 1968-1972 to 239.6 per year during 2001-2005 (regression trend, $p<0.001$). YPLL varied annually, from a low of 69 (mean per decedent: 8.6) in 1973 to a high of 306 (mean per decedent: 5.9) in 1990. The rate varied annually, from a low of 0.73 per million in 1973 to a high of 2.78 per million in 1970. During 1968-2005, asbestosis deaths in Texas (85; 577 YPLL), Pennsylvania (99; 544 YPLL), New Jersey (90; 527 YPLL), and California (76; 468 YPLL) accounted for 29.9% of all decedents aged 25-64 years with asbestosis as the underlying cause of death and 29.1% of the total YPLL attributed to asbestosis.

Industry and occupation information was available for 153 (28.8%) of the 531 decedents aged 25-64 years with asbestosis as the underlying cause of death during 1985-1999. Of 54 industries reported, the greatest YPLL were in construction (244 YPLL; mean per decedent: 5.7); ship and boat building and repairing (41; mean per decedent: 5.9); and military (41; mean per decedent: 5.9). Of 59 occupations reported, the greatest YPLL were for insulation workers (112; mean per decedent: 5.9); managers and administrators, not elsewhere classified (43; mean per decedent: 7.2); and plumbers, pipefitters, and steamfitters (42; mean per decedent: 4.7).

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CDC Editorial Note: YPLL are a measure of premature mortality that emphasizes deaths occurring among younger persons during their most productive years.[5,6] Persons dying before age 65 years are considered as having years of potential work tenure lost, on the assumption that these are a worker's most productive years. During 1968-2005, asbestosis was identified as the underlying cause of death for 1,169 decedents aged 25-64 years, accounting for 7,267 YPLL. Overall, a mean of

6.2 YPLL per decedent was attributed to asbestosis during 1968-2005, indicating that, on average, decedents aged 25-64 years with asbestosis listed as the underlying cause of death died at age 58 years. Despite the decline in asbestos use and reduced exposures, the findings described in this report indicate that asbestosis-attributable YPLL continue to occur. Because asbestosis mortality typically manifests several decades after initial exposure to asbestos, much of the continuing YPLL likely is attributed to exposures experienced decades ago. During 1970-2004, the annual number of asbestosis-related deaths (based on the analysis of asbestosis deaths coded on the entity axis in multiple cause-of-death files‖) in the United States increased nearly 17-fold, from 89 (age-adjusted death rate: 0.6 per million persons aged >15 years) in 1970 to 1,493 (6.9) in 2000, and then declined slightly to 1,470 (6.3) in 2004, for an overall total of 25,413 asbestosis deaths over the entire period.[7] This slight decline in the age-adjusted death rate was attributed to several factors, including reduced use of asbestos and improved control of asbestos exposure.[8,9] Beginning several decades ago, increased awareness of the health consequences of asbestos exposure stimulated voluntary and regulatory actions by the Environmental Protection Agency and the Occupational Safety and Health Administration.[8,9]

Available data (for 153 decedents) indicated that the greatest industry-specific YPLL values were associated with work in construction and ship and boat building and repairing, which is consistent with documented past industry-specific asbestos exposures.[1] Likewise, two of the three occupations with the greatest YPLL values, insulation workers and plumbers, pipefitters, and steamfitters, are well known to have been associated with asbestos exposures.

The findings in this report are subject to at least six limitations. First, this report used a death certificate–based definition of asbestosis as the underlying cause of death. Because some deaths from asbestosis might have been attributed to other diseases (e.g., idiopathic pulmonary fibrosis) instead of to asbestosis, the findings in this report likely underestimate deaths and YPLL attributable to asbestosis. Second, complete work histories are not listed on death certificates, and the relevance of the reported usual industry and occupation to actual hazardous exposures could not be verified. Although no studies have examined the accuracy of usual industry and occupation information on death certificates specifically for asbestosis decedents, research suggests a generally good agreement of this information compared with that from other sources.[10] Third, coded information on usual industry and occupation were available for decedents in only 26 states, accounting for 28.8% of all U.S. asbestosis decedents during 1985-1999. Thus, these data might not be nationally representative for 1985-1999. Fourth, the state issuing a death certificate is not always the state in which the decedent's asbestos exposure occurred. Fifth, ICD cause-of-death codes used in this analysis changed twice during 1968-2005. However, these revisions likely did not introduce bias or affect the temporal trend in asbestosis deaths.[7] Finally, YPLL, as calculated, do not account for the full burden of asbestosis. During the period for which CDC analyzed U.S. death data, approximately 87% of the deaths with asbestosis listed as the underlying cause of death occurred in persons aged ≥65 years. Moreover, although YPLL do reflect premature mortality during the most productive years of life, YPLL do not account for all reduced quality of life or work years lost attributed to disability from asbestosis. Persons with asbestosis can live for many years with severely limited lung function and few treatment options, leading to inability to work.

The continuing occurrence of cases of asbestos in younger persons (asbestosis-attributable YPLL) underscores the need for persistent asbestosis prevention and elimination efforts. Effective primary prevention is critical because asbestos-related diseases can develop or progress even after occupational exposure ends. Guidance for persons concerned about exposure to asbestos and for health-care providers who work with patients potentially exposed to asbestos is available at http://www.cdc.gov/health/asbestos.htm. CDC continues to conduct surveillance for asbestosis and other asbestos-related deaths to follow trends and identify problems.

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REFERENCES

1. Rom WN. Asbestos-related lung disease. In: Fishman AP, Elias JA, Fishman JA, Grippi MA, Senior RM, Pack AI, eds. *Fishman's pulmonary diseases and disorders.* 4th ed. New York, NY: McGraw-Hill Medical; 2008:943-958.
2. Kelly TD, Matos GR. Historical statistics for mineral and material commodities in the United States: US Geological Survey data series 140. Reston, VA: US Geological Survey; 2007. Available at http://minerals.usgs.gov/ds/2005/140.
3. US Department of Labor. Better protection against asbestos in the workplace. Fact sheet no. OSHA 92-06. Available at http://www.pp.okstate.edu/ehs/training/oshasbes.htm.
4. Van Gosen BS. Reported historic asbestos mines, historic asbestos prospects, and natural asbestos occurrences in the eastern United States. US Geological Survey open-file report 2005-1189. Reston, VA: US Geological Survey; 2006. Available at http://pubs.usgs.gov/of/2005/1189.
5. Wise RP, Livengood JR, Berkelman RL, Goodman RA. Methodological alternatives for measuring premature mortality. *Am J Prev Med*. 1988;4(5):268-273.
6. CDC. Premature mortality in the United States: public health issues in the use of years of potential life lost. *MMWR*. 1986;35(2)(suppl):1S-11S.
7. Bang KM, Mazurek JM, Syamlal G, Wood JM. Asbestosis mortality surveillance in the United States, 1970-2004. *Int J Occup Environ Health*. 2008;14(3):161-169.
8. US Environmental Protection Agency. EPA asbestos materials bans: clarification. Available at http://www.epa.gov/oppt/asbestos/pubs/asbbans2.pdf.
9. Martonik JF, Nash E, Grossman E. The history of OSHA's asbestos rule makings and some distinctive approaches that they introduced for regulating occupational exposure to toxic substances. *AIHAJ*. 2001;62(2):208-217.
10. Steenland K, Beaumont J. The accuracy of occupation and industry data on death certificates. *J Occup Med*. 1984;26(4):288-296.

*Since 1968, CDC's National Center for Health Statistics (NCHS) has compiled multiple cause-of-death data annually from death certificates in the United States. CDC's National Institute for Occupational Safety and Health (NIOSH) extracts information on deaths from occupationally related respiratory diseases and conditions from the NCHS data and stores the information in the National Occupational Respiratory Mor-

tality System (NORMS), available at http://webappa.cdc.gov/ords/norms.html.
†ICDA-8 code 515.2 (asbestosis) for years 1968-1978, ICD-9 code 501 (asbestosis) for years 1979-1998, and ICD-10 code J61 (pneumoconiosis due to asbestos and other mineral fibers) for years 1999-2005. For years 1999-2005, decedents with ICD-10 underlying cause coded as J65 (pneumoconiosis associated with tuberculosis) or J92.0 (pleural plaque with presence of asbestos) also were included in the underlying cause-of-death tabulation for asbestosis if code J61 also was listed on the death certificate.
‡Alaska, Colorado, Georgia, Hawaii, Idaho, Indiana, Kansas, Kentucky, Maine, Missouri, Nebraska, Nevada, New Hampshire, New Jersey, New Mexico, North Carolina, Ohio, Oklahoma, Rhode Island, South Carolina, Tennessee, Utah, Vermont, Washington, West Virginia, and Wisconsin.
§Industry and occupation information from death certificates was coded on the NCHS multiple cause-of-death data files according to the 1980 U.S. Bureau of Census Index of Industries and Occupations classification system from 1985 to 1992, and according to the 1990 U.S. Bureau of Census classification system from 1993 to 1999. For the industries and occupations listed in this report, the 1980 and 1990 classification system codes and titles were the same.
‖Entity axis includes information on all of the diseases, injuries, or medical complications, as well as the location (part, line, and sequence) of the information recorded on each death certificate. "Detail Record Layout" available at http://www.cdc.gov/nchs/about/major/dvs/mcd/1998mcd.htm.

Updated Guidelines for the Use of Nucleic Acid Amplification Tests in the Diagnosis of Tuberculosis

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GUIDELINES FOR THE USE OF NUCLEIC acid amplification (NAA) tests for the diagnosis of tuberculosis (TB) were published in 1996[1] and updated in 2000.[2] Since then, NAA testing has become a routine procedure in many settings because NAA tests can reliably detect *Mycobacterium tuberculosis* bacteria in specimens 1 or more weeks earlier than culture.[3] Earlier laboratory confirmation of TB can lead to earlier treatment initiation, improved patient outcomes, increased opportunities to interrupt transmission, and more effective public health interventions.[4,5] Because of the increasing use of NAA tests and the potential impact on patient care and public health, in June 2008, CDC and the Association of Public Health Laboratories (APHL) convened a panel of clinicians, laboratorians, and TB control officials to assess existing guidelines[1,2] and make recommendations for using NAA tests for laboratory confirmation of TB. On the basis of the panel's report and consultations with the Advisory Council for the Elimination of TB (ACET),* CDC recommends that NAA testing be performed on at least one respiratory specimen from each patient with signs and symptoms of pulmonary TB for whom a diagnosis of TB is being considered but has not yet been established, and for whom the test result would alter case management or TB control activities, such as contact investigations. These guidelines update the previously published guidelines.[1,2]

Background

Conventional tests for laboratory confirmation of TB include acid-fast bacilli (AFB) smear microscopy, which can produce results in 24 hours, and culture, which requires 2-6 weeks to produce results.[5,6] Although rapid and inexpensive, AFB smear microscopy is limited by its poor sensitivity (45%-80% with culture-confirmed pulmonary TB cases) and its poor positive predictive value (50%-80%) for TB in settings in which nontuberculous mycobacteria are commonly isolated.[3,6,7]

NAA tests can provide results within 24-48 hours. The Amplified *Mycobacterium tuberculosis* Direct Test (MTD, Gen-Probe, San Diego, California) was approved by the Food and Drug Administration (FDA) in 1995 for use with AFB smear-positive respiratory specimens, and in a supplement application, an enhanced MTD test was approved in 1999 for use with AFB smear-negative respiratory specimens from patients suspected to have TB. In addition, the Amplicor *Mycobacterium tuberculosis* Test (Amplicor, Roche Diagnostics, Basel, Switzerland) was approved by FDA in 1996 for use with AFB smear-positive respiratory specimens from patients suspected to have TB. NAA tests for TB that have not been FDA-approved also have been used clinically (e.g., NAA tests based on analyte specific reagents, often called "home-brew" or "in-house" tests).[8,9]

Compared with AFB smear microscopy, the added value of NAA testing lies in its (1) greater positive predictive value (>95%) with AFB smear-positive specimens in settings in which nontuberculous mycobacteria are common and (2) ability to confirm rapidly the presence of *M. tuberculosis* in 50%-80% of AFB smear-negative, culture-positive specimens.[3,7-9] Compared with culture, NAA tests can detect the presence of *M. tuberculosis* bacteria in a specimen weeks earlier than culture for 80%-90% of patients suspected to have pulmonary TB whose TB is ultimately confirmed by culture.[3,8,9] These advantages can impact patient care and TB control efforts, such as by avoiding unnecessary contact investigations or respiratory isolation for patients whose AFB smear-positive specimens do not contain *M. tuberculosis*.

Despite being commercially available for more than a decade,[1] NAA tests for TB have not been widely used in the United States largely because of (1) an uncertainty as to whether NAA test results influence case-management decisions or TB control activities; (2) a lack of information on the overall cost-effectiveness of NAA testing for TB; and (3) a lack of demand from clinicians and public health authorities. However, recent studies showed that (1) clinicians already rely on the NAA test result as the deciding factor for the initiation of therapy for 20%-50% of TB cases in settings where NAA testing is a routine practice[4,7] and (2) overall cost savings can be achieved by using NAA test results for prioritizing contact investigations, making decisions regarding respiratory isolation, or reducing nonindicated TB treatment.[4,7]

In response to the increasing demand for NAA testing for TB and recognition of the importance of prompt laboratory results in TB diagnosis and control, ACET requested that APHL and CDC convene a panel to evaluate the available information (e.g., current practices, existing guidelines, and