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Asbestosis mortality in the USA: facts and predictions

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ABSTRACT

Background: Mortality trends in the USA show that deaths from asbestosis are increasing, while deaths related to other pneumoconiosis are declining.

Objectives: To analyse the association between asbestos consumption and asbestosis mortality trends.

Methods: In an epidemiological time series study, we used a modern computer-intensive local regression method to evaluate the relationship between asbestos consumption per capita (1900–2006) as the predictor variable and number of deaths from asbestosis (1968–2004). The predictor variable was progressively lagged by annual increments from 30 to 60 years and the goodness of fit assessed for each lag period. The model having the smallest Akaike's Information Criteria was used to derive extrapolated estimates of future mortality based on more recent asbestos consumption data.

Results: Asbestos consumption per capita reached a peak in 1951 and gradually declined until 1973, when it started to drop rapidly. In 2006, it was 0.0075 kg/person/year. There were 25 564 deaths from asbestosis over the period 1968–2004. The best-fitting model (adjusted coefficient of determination (R^2) = 99.7%) for 1968–2004 deaths from asbestosis used asbestos consumption per capita 48 years prior (1920–1956) and the log value of asbestos consumption per capita 43 years prior (1925–1961). This model predicts a total of 29 667 deaths (95% CI 19 629 to 39 705) to occur during 2005–2027 (an average of 1290 deaths per year).

Conclusions: This study demonstrates a clear association between asbestos consumption and deaths from asbestosis and indicates that asbestosis deaths are not expected to decrease sharply in the next 10–15 years.

The use of asbestos fibres on a true industrial scale began in Italy, early in the nineteenth century, with the development of asbestos textiles for fireproof apparel. In the USA, the use of asbestos only started to increase in the early 1900s. For much of the twentieth century, the USA was the leading user of asbestos. After World War II, there was a surge in consumption.[1] In the early 1970s, driven by compelling evidence about the association of asbestos with lung cancer and asbestosis, governmental agencies intervened to reduce workplace exposures. The National Institute for Occupational Safety and Health (NIOSH) published criteria for a recommended standard in 1972 and 1976. This latest document called for a recommended exposure limit (REL) of 100 000 fibres per cubic meter.[2] The Occupational Safety and Health Administration (OSHA) issued asbestos standards in 1972, 1986 and 1994. The current permissible exposure limit (PEL) is the same as the REL proposed by NIOSH.[3] In 1989, the US Environmental Protection Agency (EPA) issued a rule banning most asbestos-containing products.[4] This regulation was overturned by a court decision, and revised versions were issued in 1992, 1993 and 1994. Although asbestos is no longer mined in the USA — the last mine was closed in 2002 — imported chrysotile asbestos is still used in industrial processes. More than 2000 tons were used in 2006, especially in the manufacture of roofing products (42%) and coating and compounds (42%).[5] According to OSHA, 1.3 million workers are still exposed to asbestos in the USA.[6] Much of the current exposure in construction occurs during repair or removal work or in demolition. Asbestos is present in most primary and secondary schools and commercial buildings in the country. It is estimated that there are more than 3000 commercial products containing asbestos.[7]

Asbestosis is the interstitial pneumonitis and fibrosis caused by the inhalation of asbestos fibres. The latency period between initial exposure and the onset of disease is 15 or more years, but varies with intensity of exposure. As exposures fall or personal protection improves, latency is likely to increase. In contrast with mesothelioma, where short exposures may cause the disease, asbestosis is associated with prolonged exposures of sufficient intensity, usually over 10–20 years.[8] A recent report indicates that asbestosis deaths are clearly increasing over time, while mortality from other pneumoconiosis, such as silicosis and coal workers' pneumoconiosis, is declining.[9]

In this study we analysed the association between asbestos consumption and asbestosis mortality trends from 1968 to 2004, in order to make projections for future asbestosis mortality in the USA.

METHODS

We conducted an ecological study where asbestos consumption was used as a surrogate measure for exposure. Data on apparent consumption (1900–2006), which is defined as asbestos production plus imports minus exports minus changes to government and industry stocks, were drawn from US Geological Survey publications.[5 10] We calculated asbestos consumption per capita to avoid biases due to changes in the resident population. As denominators, we used population estimates from the US Census Bureau.[11 12] For future predictions, we used population projections from the US Census Bureau.[13]

Asbestosis mortality data were drawn from NIOSH's National Occupational Respiratory Mortality System web page, which is a compilation

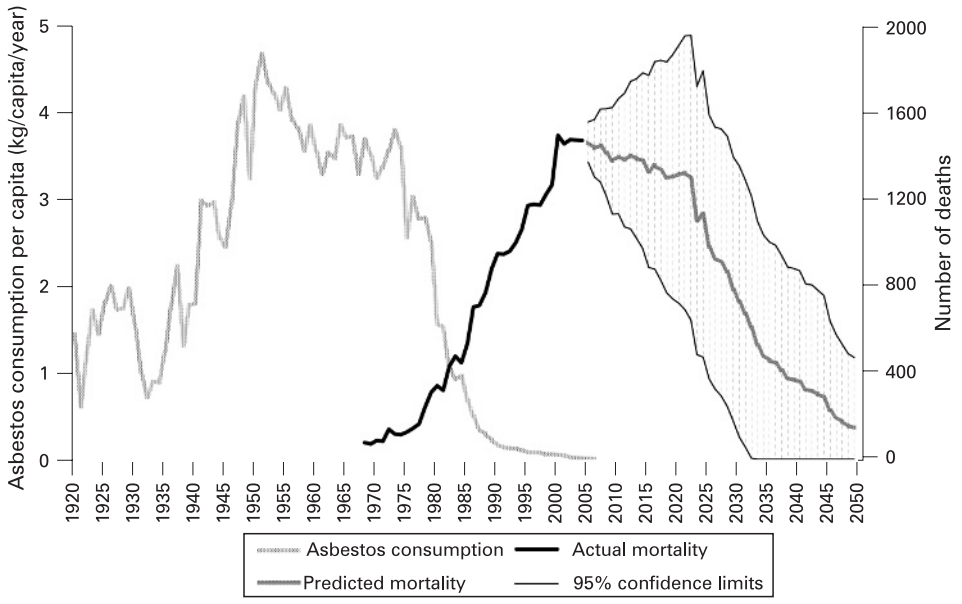

Figure 1 US asbestos consumption per capita (1920–2006), actual (1968–2004) and projected (2005–2049) deaths from asbestosis.

of national mortality data obtained annually from the National Center for Health Statistics multiple-cause-of-death records.[14] Asbestosis coding is based on the International Classification of Diseases, eighth revision (ICD-8 code 515.2 (1968–1978)),[15] ninth revision (ICD-9 code 501 (1979–1998)),[16] and tenth revision (ICD-10 code J61 (1999–2004)).[17] We extracted the number of asbestosis deaths coded as "multiple cause" (i.e., underlying and contributing causes) for all US residents 15 years and older, for the available period of 1968–2004, all states combined. We also collected these data by sex, race, and age group (15–44, 45–54, 55–64, 65–74, 75–84, and 85 years and older).

We used a generalised additive model (GAM) to evaluate the relationship between asbestos consumption per capita as the predictor variable and number of recorded asbestosis deaths. The model assumes that the numbers of deaths are Poisson random variables. GAM is a flexible non-parametric method that permits non-linear functions of independent variables and accommodates time series data.[18 19] GAMs are an extension of generalised linear models (GLMs), for example, Poisson regression, which allow a linear predictor that is a sum of smooth functions of covariates. The smooth functions used here are spline functions with automatic selection of the smoothing parameter and degrees of freedom (thin plate regression splines), which are estimated in a non-parametric manner and allow considerable flexibility to achieve a very close fit to the actual data.

This analysis accounts for an underlying time-varying Poisson mean and has been used previously in similar ecological studies.[20] For the ith year, the model can be written:

$$\text{Log}(E[\text{deaths}_i]) = f_1(\text{year}_i)+f_2(\text{asb}_{i\text{-}48})+f_3(\log[\text{asb}_{i\text{-}43}])+\text{offset}(\log(\text{pop}_i))$$

Where deaths follow a Poisson distribution, the functions f_1, f_2 and f_3 are spline functions and "asb" is asbestos consumption per capita. The term deaths_i represents the time variation in the underlying mean number of deaths from asbestosis.[21] The term year_i is simply the year in which the ith deaths occurred. An offset term, $\log(\text{pop}_i)$ corresponding to the US resident population for the years 1968–2004 was also included.

We examined a large number of models to determine the best values of the lag to use for fitting the number of asbestosis deaths. The predictor variable was progressively lagged by annual increments from 30 to 60 years and the goodness of fit using Akaike's Information Criteria (AIC) was assessed for each lag period. First the best lag term (48 years) was found for the "asb" covariate, and then several different lagged terms for the log "asb" covariate were examined. The AIC for the model with 48-year lag term is 362.5 and the model with both the 48-year lag term and the 43-year log lag term is 351.0. The p value for the chi-square analysis of deviance is <0.001 and indicates a statistically significant improvement by adding the 43-year log lag term. The model having the smallest AIC was used to derive extrapolated estimates of future mortality based on more recent asbestos consumption data. We used the package *mgcv*[21] in R software[22] to fit the model.

RESULTS

In general, asbestos consumption per capita increased steadily from 0.3 kg/person/year in 1900 to 2 kg/person/year in 1929 and presented some variation until 1938. Subsequently, there was a sharp increase in consumption until it reached a peak of 4.7 kg/person/year in 1951. After this period, consumption gradually declined to 3.8 kg/person/year in 1973, when it started to drop rapidly. Asbestos consumption in 2006 was 0.0075 kg/person/year (fig 1).

There were 25 564 asbestosis deaths recorded over the period 1968–2004, of which, 8488 (33%) were recorded as the "underlying" cause of death. Table 1 presents demographic characteristics of these decedents. The vast majority of deaths occurred among males (96.2%) and whites (93.5%). Most decedents were in the 65–74 and 75–84 age groups. Asbestosis mortality increased from 68 deaths in 1968 to a peak of 1493 deaths in 2000, and remained stable thereafter, until 2004 (fig 1).

The best-fitting model (adjusted $R^2 = 99.7\%$) for 1968–2004 deaths from asbestosis used asbestos consumption per capita 48 years prior (1920–1956) and the log value of asbestos consumption per capita 43 years prior (1925–1961). The addition of the log of the per capita consumption significantly improved model fit ($p<0.001$). Smooth spline functions (with approximately 6 and 3 degrees of freedom, respectively) were fit to both of these variables. A smooth spline function with approximately 7 degrees of freedom was fit to the variable "year".

Table 1 Demographic characteristics of 25 564 decedents with asbestosis*, among US residents aged 15 years and older, 1968–2004

Characteristic	No (%)
Sex	
Male	24 603 (96.2)
Female	961 (3.8)
Race	
White	23 908 (93.5)
Black	1512 (5.9)
Other	144 (0.6)
Age group (years)	
15–44	69 (0.3)
45–54	638 (2.5)
55–64	3375 (13.2)
65–74	8578 (33.6)
75–84	9747 (38.1)
⩾85	3157 (12.3)

*Based on the International Classification of Diseases (ICD), eighth revision (ICD-8 code 515.2 (1968–1978)), ninth revision (ICD-9 code 501 (1979–1998)), and tenth revision (ICD-10 code J61 (1999–2004)).

We employed the previous model to project future asbestosis deaths, using asbestos consumption per capita from 1960 to 2001, the log asbestos consumption per capita in 1963–2004, and US Census Bureau population projections for 2005–2049.[13] This model predicts a total of 29 667 deaths (95% CI 19 629 to 39 705) to occur during 2005–2027 (an average of 1290 deaths per year) (fig 1). Also, note that predictions for the years after 2027 involve values of asbestos consumption per capita that are outside the range of values used to fit the model but are included here only for the purpose of graphic illustration. Future predictions are of course tenuous, will be affected by unanticipated future influences and may be biased due to unobserved covariates.

DISCUSSION

This study demonstrates a clear association between asbestos consumption and deaths from asbestosis. Although this ecological relationship lacks the strengths of formal exposure–response analyses, its public health importance should not be underestimated.

Asbestos consumption has been used as a predictor of asbestos-related deaths in several studies, in different countries. Marinaccio *et al* applied an asbestos consumption model to pleural mesothelioma deaths (calculated as a percentage of pleural tumours) and predicted that between 810 and 830 deaths per year would occur in Italy in the period 2012–2020.[23]

Using data from 10 Western countries, Takahashi *et al* found a clear linear relationship between asbestos consumption and mesothelioma incidence/mortality rates, with a Spearman correlation coefficient of 0.70 ($p = 0.03$).[24] When Japan was included in the analysis, the correlation coefficient decreased to 0.52 ($p = 0.10$), given the low incidence rate of mesothelioma diagnoses in that country. With more recent data and replacing Japan with New Zealand, Tossavainen updated the previous model finding a Spearman correlation coefficient of 0.73 ($p = 0.01$).[25] More recently, Lin *et al* revisited this study design and estimated the ecological relationship between asbestos consumption and diseases associated with asbestos including 33 countries.[26] Asbestos consumption in 1960–1969 was a highly significant positive predictor of mortality in 2000–2004 for all mesothelioma in men ($R^2 = 0.74$), all mesothelioma in women ($R^2 = 0.58$), and asbestosis in men ($R^2 = 0.79$).

In 1982, Nicholson *et al* used predictions for mesothelioma mortality to estimate asbestosis deaths.[27] They calculated that the number of deaths from asbestosis in that year, approximately 200 according to the paper, would double in the following two decades and decline thereafter. The peak in asbestosis mortality actually appears to have occurred around 2002 (fig 1), but the observed number of deaths surpassed their projections. In 1982, asbestosis was recorded as the underlying cause of death in 102 cases, and reached 532 cases in 2002. An additional 326 deaths in 1982 and 941 deaths in 2002 were recorded as contributing causes of death.[14]

Also using a hypothetical equivalence between asbestosis and mesothelioma mortality, and assuming that only around 20% of deaths in men with asbestosis are attributed to asbestosis, Walker *et al* estimated the prevalence of the disease for the period 1980–2009, by 5-year intervals.[28] According to their predictions, there would be 5700 men alive with asbestosis in the period 2005–2009 and this number would triple if cases with low radiological profusion were considered. These results cannot be directly compared to our findings. However, if we assume that every case of asbestosis is recorded on death certificates as either the underlying or contributing cause of death — which is unlikely — their predictions might be plausible, since we expect more than 20 000 deaths from asbestosis to occur. Otherwise, those predictions largely underestimated the burden of asbestosis in the USA.

One of the strengths of our study is that we do not project asbestosis deaths based on another disease. We have comprehensive national mortality data; historical asbestos consumption has been available since 1900. Moreover, new modelling techniques allow us to correlate more than only point values of consumption and number of deaths in a given year, as in the previous mentioned papers.[24–26 29] Because the GAM uses multiple degree of freedom spline functions as predictors, we are able to account for differences in exposure — or its surrogate, asbestos consumption — that may have occurred over a certain period, which is more likely to represent a real-life situation. In other words, we assume that the outcome is related to consumption over several years, given appropriate latency, instead of only 1 year. In addition, the model achieved high levels of goodness of fit, which would be equivalent to an R-squared of 99.7%, highly statistically significant ($p<0.001$). This increases our confidence in the model and its results.

Nevertheless, our study presents some limitations. First of all, it is an ecological study; thus, asbestos consumption may not reflect each individual's actual exposure, especially because exposure and risk are not even across the population. Several aspects of asbestos exposure may influence the overall number of individuals that will develop asbestosis and eventually die

Main messages

- There is a clear association between asbestos consumption and asbestosis deaths as reported on death certificates.
- Asbestosis deaths are not expected to decrease sharply in the next 10–15 years.

Policy implications

- Additional burden on healthcare, productivity, and the compensation and litigation system is expected from past asbestos exposure.

with the disease: number of people exposed, level and duration of exposure, and type of asbestos used. These variables are not available on death certificates and therefore were not considered in our model. Asbestos consumption probably reflects well the number of people exposed to the fibre: in most industries, if consumption increases, it is likely that the number of workers potentially exposed will also have been increased.

Level of exposure is probably determined by a combination of amount of asbestos used and environmental control measures. If we take into account that permissible exposure limits to asbestos were stricter after the 1970s, we can assume that levels of exposure were probably lower after this period than in previous years. Similarly, since asbestos use has been phasing out in the last decades, workers may have had shorter duration of exposure in more recent decades, compared with those who worked in earlier years. In addition, imports of amphibole asbestos decreased after the 1980s.[10] This type of fibre, given its low solubility, may present a higher risk for malignant and non-malignant disease than chrysotile asbestos.[8] As a result of these factors, predictions using asbestos consumption in the last three decades may overestimate mortality.

In contrast, other factors may lead to underestimated predictions of asbestosis deaths. First, exposure to asbestos during demolition and maintenance of past asbestos installations is not recognised under the consumption figures. However, this type of exposure is likely to continue for many years and may increase the risk of asbestosis, leading to underestimated projections. Second, mortality data are subject to potential errors associated with disease diagnosis, recording and coding. If recognition of disease changed over time, too few deaths may have been reported in early years. On the other hand, increased awareness of asbestos-related disease, perhaps due to litigation, may have enlarged the number of reported cases in more recent years. Finally, differences among ICD code versions may also have a role. For instance, in the 10th revision, the rubric for code J61 is ''pneumoconiosis due to asbestos and other mineral fibers'', whereas the rubric for the eighth and ninth revisions was simply ''asbestosis''. This might have resulted in an increase in the number of recorded cases of asbestosis between the ninth and tenth revisions.

CONCLUSIONS

This study demonstrates a clear association between asbestos consumption and deaths from asbestosis as reported on death certificates. Our analyses indicate that deaths from asbestosis are not expected to decrease sharply in the next 10–15 years.

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