Estimating the Number of Asbestos-related Lung Cancer Deaths in Great Britain from 1980 to 2000

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Introduction: Inhalation of asbestos fibres is known to cause two main kinds of cancer—mesothelioma and lung cancer. While the vast majority of mesothelioma cases are generally accepted as being caused by asbestos, the proportion of asbestos-related lung cancers is less clear and cannot be determined directly because cases are not clinically distinguishable from those due to other causes. The aim of this study was to estimate the number of asbestos-related lung cancers among males by modelling their relative lung cancer mortality among occupations within Great Britain in terms of smoking habits, mesothelioma mortality (as an index of asbestos exposure) and occupation type (as a proxy for socio-economic factors).

Methods: Proportional mortality ratios for lung cancer and mesothelioma for the 20-year period from 1980 to 2000 (excluding 1981) were calculated for occupational groups. Smoking indicators were developed from three General Household Surveys carried out during the 1980s and 1990s. Poisson regression models were used to estimate the number of asbestos-related lung cancers by estimating the number of lung cancer deaths in each occupation assuming no asbestos exposure and subtracting this from the actual predicted number of lung cancer deaths.

Results: The effect of asbestos exposure in predicting lung cancer mortality was weak in comparison to smoking habits and occupation type. The proportion of current smokers in occupational groups and average age at which they started smoking were particularly important factors. Our estimate of the number of asbestos-related lung cancers was between two-thirds and one death for every mesothelioma death: equivalent to between 11,500 and 16,500 deaths during the time period studied.

Conclusions: Asbestos-related lung cancer is likely to have accounted for 2–3% of all lung cancer deaths among males in Great Britain over the last two decades of the 20th century. Asbestos-related lung cancers are likely to remain an important component of the total number of lung cancer deaths in the future as part of the legacy of past asbestos exposures in occupational settings.

Keywords: asbestos; lung cancer; mesothelioma; mortality; smoking

INTRODUCTION

Inhalation of asbestos fibres is known to cause four main respiratory diseases. These include two kinds of cancer—mesothelioma and lung cancer—and two non-malignant conditions—asbestosis and diffuse pleural thickening (Doll and Peto, 1985; Hutchings et al., 1995). Mesothelioma death statistics for Great Britain are readily available from the British mesothelioma register, which is a comprehensive source of mesothelioma mortality data derived from textual descriptions of death certificates rather than a reliance on cause of death codings (McElvenny et al., 2005). The majority of deaths occurring in any year can be assumed to result from past exposures to asbestos in occupational settings (McDonald and McDonald, 1987; Peto et al., 1995). Although lung cancer death statistics for Great Britain are also readily available, and asbestos has also been recognized as an
important risk factor for lung cancer for many years (Liddell, 2001), the number of lung cancer deaths attributable to asbestos cannot be determined directly. This is because there are a number of risk factors for lung cancer—in particular, cigarette smoking—and the resulting cases are generally clinically indistinguishable from each other.

Estimates of the number of asbestos-related lung cancers can be made indirectly from observations of numbers of excess lung cancer deaths in cohorts of asbestos-exposed workers and from population attributable risk (PAR) estimates derived from lung cancer case control studies, or from studies of associations between mesothelioma and lung cancer risk. Estimates derived from a variety of such studies across Europe were brought together and published in a review in 1999 (Albin et al., 1999). A limitation of these estimates is their focus on particular asbestos-exposed groups which makes generalization to the population level problematic.

The aim of our study was to estimate the number of asbestos-related lung cancers in Great Britain during the period 1980–2000 (excluding 1981) by fitting a generalized linear model for lung cancer mortality in relation to smoking levels and mesothelioma mortality (as an index of asbestos exposure levels) within different occupational groups that spanned the whole of the British working population.

**METHODS**

*Model form and construction of the dataset*

The observed number of male lung cancer deaths at ages 16–74 during the period 1980–2000 (excluding 1981) by occupational group was modelled in terms of smoking and asbestos exposure using Poisson regression. Deaths in the year 1981 and among those aged >74 years were excluded from the analysis due to unreliable occupation coding (Coggon et al., 1995a). The period 1980–2000 was chosen because this is the longest for which death data is available in Great Britain coded by occupation on a consistent basis. The Southampton classification of occupations was used since this groups together occupation codes within the Classification of Occupations 1980 with similar occupational hazards likely to affect mortality (Noble et al., 1995; Coggon et al., 1995b), and because mesothelioma proportional mortality ratios (PMRs) (used as an explanatory variable as an index of asbestos exposure) have already been published on this basis using data from the British mesothelioma register (McElvenny et al., 2005).

To account for differences in size and the age structure of occupational groups, the natural log of the expected number of lung cancer deaths—calculated by proportionate mortality methods—was declared as an offset in the Poisson regression model.

 Lung cancer mortality data for Great Britain by occupational group was compiled from data supplied by the Office for National Statistics (ONS) for deaths in England and Wales and by the General Register Office for Scotland for deaths in Scotland. All-cause mortality data by occupational group was obtained broken down in the same way as the lung cancer deaths in order to calculate expected numbers of lung cancer deaths. These were calculated using proportionate mortality methods (Breslow and Day, 1987) with age standardization carried out within 5-year age groups.

Quantitative measures of smoking habits among occupational groups were developed from the General Household Survey, a large-scale sample survey conducted by the ONS (UK Data Archive, 1992). Smoking variables were derived from combined data for the surveys in 1986, 1988/89 and 1990/91—those surveys for which smoking data was available coded to the Classification of Occupations 1980. Data from these surveys was obtained from the UK Data Archive website (UK Data Archive, 1989, 1990, 1992). Even when combining data for three GHS surveys, for some occupational groups the number of sample cases was small or zero resulting in very uncertain estimates of the smoking measures. Occupations where the first smoking variable, the proportion who had ever smoked, was based on <20 sample cases were therefore excluded. All smoking variables are summarized in Table A1 along with details of how they were derived.

There is no source of information providing direct measurements or estimates of asbestos exposure among occupational groups in Great Britain. However, since a large majority of mesothelioma cases result from exposure to asbestos within occupational settings and are fatal within a short time from diagnosis (McDonald and McDonald, 1987), the relative mortality of occupational groups can be used as an index of their asbestos exposure levels. [This approach was adopted by De Vos Irvine et al. (1993), for example, in their analysis of asbestos and lung cancer in Glasgow and the West of Scotland.] PMRs for mesothelioma from a recent analysis of the British mesothelioma register (McElvenny et al., 2005) were used as an index of relative asbestos exposure among occupational groups. Mesothelioma PMRs for males were available by Southampton occupation codes for the same time period as the observed and expected lung cancer deaths: 1980–2000 (excluding 1981 and deaths at ages >74 years).

Occupational groups within the Southampton classification can be categorized into ‘occupational order groups’ which bring together people with jobs of a similar social and economic status (Noble et al.,
In addition to the indicators of smoking habit and asbestos exposure, the occupational order group was included as a factor variable in the model as a proxy for socio-economic factors.

**Model fitting**

Poisson regression models were fitted using GLIM version 4 (Francis et al., 1993). Since asbestos and smoking are established risk factors for lung cancer, the mesothelioma PMR and the smoking variable that reduced the deviance by the most were included in the model at the outset. Occupational order group was found to reduce the deviance of the null model very substantially and so this was also included. Thereafter, the focus of the modelling was to incorporate additional smoking variables (and any interactions) into the initial model in order to reduce the deviance further and derive an adequate model for prediction purposes. There is some degree of overlap between some of the smoking variables. For example, there are strong correlations between the first three smoking variables: the proportion who have ever smoked any kind of tobacco, the proportion who have ever smoked cigarettes and the proportion who currently smoke cigarettes. The proportion who typically smoke more than 20 cigarettes per day is also related to the average number smoked per day. Thus, once a variable from each of these two groups of variables was included in the model, no further variables from the groups were considered.

Overdispersion in the residuals led to a deviance well in excess of the number of degrees of freedom—even after the addition of all smoking variables and a number of interaction terms. Therefore, to balance model parsimony and goodness of fit, variables which reduced the deviance by only a small amount (a few units) were excluded. Residuals for each model were analysed and overdispersion was accounted for by rescaling the variance of the response—according to the standard methodology of assuming that the variance is greater than usual by a constant factor estimated by the quotient of the deviance and degrees of freedom of a suitable model (Aitkin et al., 1989).

The adequacy of simpler nested models was then investigated via $F$-tests of deviance differences. Occupational order group is itself likely to be related to asbestos exposure through the concentration of this exposure within groups of occupations which are composed of predominantly manual workers and the fact that individual’s job histories are likely to remain within the same occupational order group rather than moving between groups. Thus, the inclusion of occupational order group in the model may factor out some of the variation that is properly ascribed to asbestos exposure. Therefore, a version of the final model which excluded occupational order group was also fitted and alternative estimates of the number of lung cancers due to asbestos derived.

**Estimating the number of lung cancers due to asbestos**

Figure 1 shows the predicted excess lung cancers due to asbestos exposure, $\hat{O}_{\text{asb}}$, and the observed lung cancer excess (i.e. the total observed lung cancers minus the predicted lung cancers for no asbestos exposure) plotted against the observed mesothelioma deaths.

The mesothelioma PMR relating to zero asbestos exposure is not equal to zero since there is evidence to suggest that there are $\sim$100 mesothelioma deaths each year in Great Britain not linked to asbestos.
exposure, with roughly equal numbers occurring in males and females (McElvenny et al., 2005). This is the so-called ‘background’ level of mortality; over the 20-year analysis period, ~1000 such male mesotheliomas would be expected (=20×50). Dividing this number by the total of 17 491 male mesotheliomas actually observed gives an approximate PMR for a hypothetical group of men with no exposure of 0.06.

For each occupation, an estimate of the excess lung cancer deaths due to asbestos exposure ($\hat{O}_{\text{asb}}$) was calculated as $\hat{O}_{\text{asb}} = \hat{O}_1 - \hat{O}_0$, where $\hat{O}_1$ is the fitted number of lung cancers and $\hat{O}_0$ is the predicted number of lung cancers for the same occupation but with no asbestos exposure (i.e. with the mesothelioma PMR set to 0.06). The total excess lung cancer deaths due to asbestos exposure (obtained by summing the $\hat{O}_{\text{asb}}$ over all occupations) and the total observed mesothelioma deaths were then used to estimate the ratio of male asbestos-related lung cancer to mesothelioma deaths over the 20-year analysis period. To allow for the fact that the analysis only included deaths at ages 16–74 and those among occupations where the proportion who had ever smoked was based on at least 20 sample cases in the GHS, the total predicted asbestos-related lung cancers ($\hat{O}_{\text{asb}}$) was adjusted by dividing by the proportion of deaths at ages 16–74 years and by the proportion of deaths among those occupations included in the analysis. The estimate of 1000 background cases was also subtracted from the observed mesothelioma total.

RESULTS

Summary statistics

There are 194 occupations within the Southampton Classification. In addition, there are 17 ‘residual’ CO80 codes that do not map to any Southampton Classification code, which results in 211 occupational groups altogether. Of these, 131 had sufficient sample cases from the GHS for inclusion in the analysis. Of the 80 excluded groups, statistically, 4 had mesothelioma PMRs significantly above average and 23 had PMRs significantly below average. Thus, the majority of occupations which were most informative in providing an index of asbestos exposure were included in the modelling.

There were 333 537 lung cancer deaths in Great Britain among males aged 16–74 during the period 1980–2000 (excluding 1981). Of these, 302 771 (91%) were among the 131 occupations selected for the analysis. The PMRs for these groups ranged from 0.382 to 1.376. The number of mesothelioma deaths in the period was much lower at 12 820 [with 12 080 (94%) of these among occupations selected for analysis], but the range of PMRs was much wider (0.084–5.025).

Overall, a high proportion of respondents from the GHS (~80%) indicated that they had regularly smoked cigarettes, cigars or a pipe at some time in the past. However, there was substantial variation in the proportion who regularly smoked at some time among different occupations (range 40–100%). Over 60% of respondents indicated that they were either current or ex-smokers of cigarettes. The overall proportion of current cigarette smokers was much lower at 35% and the proportion of heavy smokers lower still at 20%. There was a substantial amount of variation among other smoking variables across occupations.

Scatter plots of the lung cancer PMR versus the various explanatory variables (not shown) showed that there was strong positive correlation with the first four smoking variables (variables S1–S4, see Table A1), weaker positive correlation with the mesothelioma PMR (PM), the average number of cigarettes smoked per day (S5) and the proportion of ex-smokers (S7). There was weak negative correlation between the lung cancer PMR and the average time since smoking cessation (S9), and stronger negative correlation with the average age when started smoking (S8). There was substantial variation in the lung cancer PMR within and between the occupational order groups (OR), with the minimum PMR for some groups being higher than the maximum for certain others.

Poisson regression modelling

The model which formed the basis for rescaling the variance of the response included a single factor for the occupational order group (OR) and five covariates: the mesothelioma PMR (PM), the proportion of current smokers (S3), the average age at which started smoking (S8), the proportion of ex-smokers (S7) and the average number of cigarettes smoked per day (S5). The coefficients for the first three of these covariates were dependent on the occupational order group, i.e. the model included interactions between these covariates and the order group factor. This model had a deviance of 472.36 on 75 degrees of freedom. The only deficiency identified by the residual analysis was the greater than expected spread of the residuals suggesting that rescaling the variance to allow for overdispersion was a valid approach. The model, in which only the slopes for the proportion of current smokers and the average age at which started smoking were dependent on the occupation order group, was an adequate simplification. This model was further simplified by amalgamating some of the occupational order groups after carrying out t-tests of the relevant parameters and additional F-tests of the nested models. This reduced the number of occupational order groups in the final model to 12. This model had a deviance of 108.02 on 95 degrees of freedom. The form of the linear predictor and
the values of the coefficients are presented for each of the occupational order groups (together with a description of the groups) in Table A2.

The form of the model implies an exponential relationship between the predicted observed lung cancer deaths and asbestos exposure index as expressed by the mesothelioma PMR. The predicted lung cancer deaths for an individual occupation, \( i \), can be written:

\[
\hat{O}_i = k_i \cdot \exp(0.023 \times [PMR_i]),
\]

where \( i = 1, \ldots, 131 \), and \( k_i \) is a constant dependent on the expected lung cancer deaths, occupational order group and smoking measures for occupation \( i \).

However, over the range of PMR values observed, the relationship is approximately linear. The number of lung cancer deaths predicted by a simple linear relationship differs from that predicted by the exponential relationship by <1% over this range.)

Estimating the number of lung cancers due to asbestos

Occupations with more mesothelioma deaths tend to have a greater number of predicted asbestos-related lung cancers. Figure 1 suggests that the ratio of predicted asbestos-related lung cancer to mesothelioma deaths is approximately two-thirds. This was confirmed by the numerical calculation.

The total predicted asbestos-related lung cancers for men aged 16–74 among all the occupations included in the analysis was 6900. Over the analysis period, 66% of male lung cancer deaths were at ages 16–74, and the occupations included accounted for 91% of lung cancer deaths among all occupations. Thus, the rescaled estimate of the total number of asbestos-related lung cancer deaths among males during the period was \( \sim 11500 \). The total number of mesothelioma deaths among males (all occupations and ages) during the analysis period was 17491, of which \( \sim 16500 \) are likely to be due to asbestos exposure (assuming 1000 background cases). Thus, an estimate of the ratio of the number of asbestos-related lung cancer to mesothelioma deaths was \( \sim 0.7 \) for the period studied.

The form of the linear predictor and parameter estimates for the model which excluded the factor variable for occupational order group is shown in Table A2. The deviance for this model was 314.14 on 125 degrees of freedom—clearly, a much poorer fit. An equivalent chart to that shown in Fig. 1 for this model (not shown) suggests that the ratio of predicted asbestos-related lung cancer to mesothelioma deaths is \( \sim 1 \). Although the alternative model is clearly a poor fit, this result does at least indicate that the model including occupational order group may lead to an underestimate of the ratio of asbestos-related lung cancers to mesotheliomas because of an association between occupational order group and asbestos exposure. Based on this evidence, a reasonable conclusion would be that the ratio is somewhere between two-thirds and unity.

**DISCUSSION**

Discussion of the model

Previous studies have indicated that lung cancer risk in smokers is particularly related to the duration of smoking, the age at which started smoking and number of cigarettes smoked (IARC, 1986). Lung cancer risk factors other than tobacco smoking and occupational asbestos exposure were summarized in a number of recent reviews (Alberg and Samet, 2003; Neuberger and Field, 2003; Ruano-Ravina et al., 2003; Tyczynski et al., 2003). These include passive smoking; exposure (mainly occupational) to chemicals, dusts and particles/fibres, including chromates, chloromethyl ethers, nickel, arsenic, cadmium, polycyclic aromatic hydrocarbons, wood dust and silica; outdoor air pollution, including combustion-generated carcinogens and other industrial emissions; radon and ionizing radiation. In our model, only smoking and asbestos exposure were considered directly—though other factors may have been accounted for to some extent via the inclusion of occupation type (used as a proxy for socio-economic factors).

Although the smoking indicators used in our model describe the typical smoking habits of occupational groups rather than individuals, the relative sizes of the coefficient values are nevertheless broadly consistent with the existing understanding of lung cancer risk, as outlined above. For a given occupational group, the proportion of smokers and the average number of cigarettes smoked (for smokers), when taken together, provide a measure of the overall level of smoking. In our model, the proportion of current smokers was found typically to have the largest influence on the predicted number of lung cancer deaths whereas the average number of cigarettes smoked had a much weaker effect. All but two of the coefficients for the proportion of current smokers were positive (Table A2). The absolute value of both negative coefficients was relatively small and only one was statistically significant (\( P < 0.001 \)). Average age at which started smoking also had a strong effect and 9 of the 12 coefficients were negative in value. Although all three of the positive coefficients were statistically significant (\( P < 0.002 \) in each case), two were small in magnitude. The one substantive anomaly was the relatively large positive value of the coefficient for occupational order group 6 (security and protective service occupations). This appears to be due to the particular mix of occupations within this group: two of the four occupations (police and fire service personnel) have a low proportion of
current smokers, thus bringing the average down for the group as a whole. However, because the lung cancer PMR and average age at which started smoking are both close to the overall average values, this results in the coefficient for the latter being driven up.

The proportion of ex-smokers had a weaker influence relative to the other smoking variables. The inclusion of this variable along with the proportion of current smokers makes sense intuitively since taken together these variables provide a measure of the proportion of individuals who have ever smoked cigarettes, which is presumably related to the overall smoking ‘exposure’ of a particular group.

In comparison to smoking habits, the asbestos exposure of occupational groups (as measured by the mesothelioma PMR) had a much weaker influence on the lung cancer risk. (The relative lung cancer risk for a group with very high asbestos exposure (mesothelioma PMR = 5) compared to one with no exposure, given the same smoking habits, was 1.12.)

Occupational order group was also an important factor in the model. Furthermore, the strength of the effects of two of the smoking variables (proportion of current cigarette smokers and average age at which started smoking) were found to be dependent on the occupational order group. As already noted, occupational order group may itself be associated with asbestos exposure to some extent. In fact, groups of occupations may also express some characteristics of smoking habits that are not identified at the individual occupational code level. However, that order group is an important factor in the model suggests that the relative lung cancer mortality cannot be explained solely in terms of asbestos exposure and smoking. The occupational order groups may characterize some other important effects on the lung cancer risk (or effects of agents acting in combination) that are related to social class.

Although the presence of overdispersion in our analysis may to some extent have affected the reliability of inferences made after variance rescaling, e.g. F-tests when attempting to simplify models, there was no indication from residual analyses that the final model was not an adequate description of the data. The ecological fallacy is unlikely to affect this analysis since dose–response relationships between asbestos exposure and lung cancer, and between smoking and lung cancer have been demonstrated in many other contexts and studies (IARC, 1986; McDonald and McDonald, 1987). It is unlikely therefore that the effect of considering groups of workers masks some important undetected effect at the individual level.

Discussion of the results in the context of other research

Our estimate of the ratio of asbestos-related lung cancer to mesothelioma deaths of between two-thirds and one during the 20-year period 1980–2000 (excluding 1981) equates to 2–3% of the total number of male lung cancer deaths. Although this implies a substantial number of asbestos-related lung cancers, in relative terms this proportion is small in comparison to the proportion of lung cancers due to smoking. The estimate is also somewhat lower than those from other studies. In some heavily asbestos-exposed worker cohorts, there have been up to ~10 times as many excess lung cancers as mesotheliomas. Cohorts involving chrysotile exposure have generally been associated with the highest ratios and those involving crocidolite have been associated with the lowest (McDonald and McDonald, 1987). A study of lung cancer and mesothelioma incidence in the West of Scotland (De Vos Irvine et al., 1993) derived an estimate for the number of asbestos-related lung cancers in this region based on a statistical model of lung cancer mortality in terms of past asbestos exposure, controlling for smoking and other factors. The PAR for asbestos-related lung cancer for the period 1975–84 was estimated to be 5.7%, and this suggested that there were ~2 lung cancers for every mesothelioma death. The review of asbestos-related cancer in Europe (Albin et al., 1999) drew together PAR estimates from a number of studies, including the West of Scotland study. PAR estimates for asbestos-related lung cancer were found to be between 5.7 and 19%. However, the generalization of these PARs from regional studies is problematic and the authors of the study yielding the highest PAR of 19% (Karjalainen et al., 1994) suggested that a representative PAR for Finland may be less than half this value, although a subsequent study (Nurminen and Karjalainen, 2001) estimated it to be 14%. A study in the Netherlands that was judged by its authors to be representative of the national male population gave a PAR of 11.6% (van Loon et al., 1997). In addition, studies in Germany not included in the European review have given PAR estimates, for asbestos-related lung cancer among males, between 8 and 9% (Jöckel et al., 1998; Brüskewitz et al., 2000). That our estimate of the number of asbestos-related lung cancers in Great Britain is somewhat lower than the central estimate for the West of Scotland region [known to be associated with fairly high asbestos exposures resulting from the shipbuilding industry (McElvenny et al., 2005)] seems reasonable given the observations above about regional studies.

Limitations of the study

A limitation of the approach adopted in this analysis is that it is difficult to relate the indirect measures of asbestos exposure provided by the mesothelioma PMRs to actual physical cumulative exposures that could be measured in workplaces or the environment. An occupation with mesothelioma
PMR of 1 has the same level of mesothelioma mortality as the whole working population, but it is not possible to state the physical exposure to which this level of mortality is equivalent, other than to note that it must reflect some kind of average level.

A further problem relates to the type of asbestos exposure most likely to be indexed by the mesothelioma PMRs. Variations in the mesothelioma PMR among occupations will primarily reflect variations in amphibole exposures (since the majority of mesotheliomas will be due to such exposures). If chrysotile exposures are not positively correlated with these amphibole exposures, and if chrysotile is a substantial contributor to lung cancer mortality, then the model is likely to underestimate the contribution of asbestos in general. However, we think it is likely that chrysotile and amphibole exposures are positively correlated. Given that our estimate of the ratio of asbestos-related lung cancers to mesotheliomas is more in line with results from cohort studies involving amphibole exposure, this suggests that chrysotile may not be a major contributor to lung cancer at the population level—though presumably, the actual number of lung cancers due to chrysotile will still be relatively much higher than the number of mesotheliomas due to chrysotile. The fact that our estimate of the ratio is even lower than estimates from cohorts involving amosite exposure (Seidman et al., 1986; Sluis-Cremer et al., 1992) is somewhat surprising given that amosite is likely to be a major contributor to asbestos-related lung cancer. A possible explanation may be that the ratio of lung cancers to mesotheliomas is different at different exposure levels (with relatively more lung cancers at higher exposures). The exposure response models proposed by Hodgson and Darnton (2000) suggest this is the case. If, across the population as a whole, most mesotheliomas arise from exposures at levels below those in the cohorts cited above, the overall lung cancer to mesothelioma ratio will also be lower than in these cohorts.

The problem of how the asbestos exposure index is ‘calibrated’ in terms of physical exposures is exacerbated by the occupation coding on death certificates. To get the best indication of relative mortality levels within occupations (and thus for the purposes of the present analysis, the best inferred asbestos exposure levels), mesothelioma deaths should be assigned to the occupation in which exposure occurred (McElvenny et al., 2005). In practice, this is not possible. Death certificates only record the last occupation of the deceased. This, together with the fact that mesothelioma typically has a long latency period, means it is likely that in a substantial proportion of cases workers will have moved from the job in which the exposure occurred into other areas of work before the disease was diagnosed. This means that differences in mortality between occupations will be diluted—an effect which is particularly likely to affect groups associated with industries that have diminished in size due to changing patterns of work over recent years. Nevertheless, analyses of mesothelioma by occupation suggest that the occupations which are genuine sources of asbestos exposure do yield the highest PMRs (McElvenny et al., 2005). Thus, the correspondence between the mesothelioma PMR values and asbestos exposure levels is likely to be largely preserved.

Another limitation of our analysis is that no account was taken of trends in lung cancer mortality, smoking and asbestos exposure over time. In order to construct a dataset comprising measures of lung cancer mortality, smoking and asbestos exposure that were not based on very small numbers of cases for the majority of occupations, data for a number of years was combined. This means that the fitted models only provide information about males for the period for which the model applies. In other words, they do not yield information about the relationship between asbestos exposure, smoking and lung cancer that can be generalized for any situation. This is because overall lung cancer mortality and mesothelioma mortality has followed different patterns over time. Mesothelioma mortality has increased rapidly over the period of this analysis, whereas total lung cancer mortality has been gradually declining. If asbestos-related lung cancers have followed the same pattern of increase as mesothelioma deaths as a consequence of latency, then this implies that the proportion of lung cancers that are asbestos-related would have increased over the period. However, such an increase may be offset by a general reduction in smoking levels over time. It is generally recognized that there is at least some interaction between the asbestos exposure and smoking on the lung cancer risk, though there is still no consensus about the nature of the interaction (Liddell, 2001; Lee, 2001; Berry and Liddell, 2004). Thus, changes in smoking habits could have a substantial impact on the number of asbestos-related lung cancers. Indeed, this could partly account for the observation made earlier that our estimate of the ratio of asbestos-related lung cancers to mesotheliomas is lower than that observed in past cohorts involving amosite exposure (a relatively high proportion of which are likely to have been smokers).

In our analysis, the inferred asbestos exposures may have taken place over extended periods of time, and the actual periods of exposure may have been different from one group to the next. Given a similar latency for asbestos-related lung cancer and mesothelioma, these implied asbestos exposures should be broadly those which have resulted in the lung cancers observed over the same period. However,
the smoking information was derived from a different source—the General Household Survey. Derived smoking measures for occupational groups are based on information given at the time the surveys were carried out—which may be a long time after the asbestos exposures implied by the mesothelioma PMRs occurred. If smoking habits and asbestos exposures had remained constant over time, a ‘steady state’ scenario could be envisaged, whereby the proportion of current smokers and level of asbestos exposure should correlate well with the lung cancer risk. Although exposures have clearly not remained constant, provided that the relative differences in asbestos exposures and smoking habits have not changed dramatically, the fitted model should reflect a genuine relationship.

CONCLUSIONS

This analysis indicates that a substantial number of lung cancer deaths in Great Britain during the 20-year period 1980–2000 (excluding 1981) were asbestos related. The ratio of asbestos-related lung cancer to mesothelioma deaths during the period was estimated to be between two-thirds and one. This equates to an estimated 11 500–16 500 deaths among males, which is 2–3% of the total lung cancer deaths among males.

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APPENDIX

Table A1. Derived smoking variables from the General Household Survey (GHS)

<table>
<thead>
<tr>
<th>GHS variable used</th>
<th>Derived variable for analysis</th>
<th>Notes on derivation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smokever</td>
<td>S1 Proportion of respondents who have ever smoked cigarettes/cigars/pipe</td>
<td>Number answering ‘yes’ divided by the total (within each occupation)</td>
</tr>
<tr>
<td>Cigever</td>
<td>S2 Proportion of respondents who have ever smoked cigarettes (that is, proportion that are current or ex-smokers)</td>
<td>Derived from cignow and cigever: those answering ‘yes’ to either cignow or cigever divided by the total respondents (within each occupation)</td>
</tr>
<tr>
<td>Cignow</td>
<td>S3 Proportion of respondents who currently smoke cigarettes</td>
<td>Number answering ‘yes’ divided by the total (within each occupation)</td>
</tr>
<tr>
<td>Qtywkend and Qtywkday</td>
<td>S4 Proportion of respondents who currently smoke typically at least 20 cigarettes per day</td>
<td>Daily average calculated for each current smoker [= (2/7) \times \text{qtywkend} + (5/7) \times \text{qtywkday}]. Then, proportion heavy = number smoking (\geq 20) cigarettes per day divided by the total (within each occupation)</td>
</tr>
<tr>
<td>Qtywkend and Qtywkday</td>
<td>S5 Average number of cigarettes smoked per day (current smokers)</td>
<td>Daily average calculated for each current smoker [= (2/7) \times \text{qtywkend} + (5/7) \times \text{qtywkday}], and then averaged over respondents (within each occupation)</td>
</tr>
<tr>
<td>Cigtype</td>
<td>S6 Proportion of current smokers using unfiltered cigarettes</td>
<td>Number answering 2 or 3 divided by the total (within each occupation)</td>
</tr>
<tr>
<td>Cigever</td>
<td>S7 Proportion of respondents who used to smoke cigarettes but do not do so now</td>
<td>If not currently smoking, number answering ‘yes’ divided by the total (within each occupation)</td>
</tr>
<tr>
<td>Cigage</td>
<td>S8 Average age at which ‘started smoking’ (current and ex-smokers)</td>
<td>Average calculated within each occupation.</td>
</tr>
<tr>
<td>Cigstop</td>
<td>S9 Average number of years since stopping smoking cigarettes</td>
<td>Weighted average calculated for ex-smokers within each occupation based on category mid-points (12.5 years used for final category)</td>
</tr>
</tbody>
</table>
In the final model, the fitted lung cancer PMR is given by \( \exp(\eta) \) with \( \eta \) as shown in the table above for each occupational order group. In the alternative model, the fitted lung cancer PMR is given by \( \exp(\eta) \) with \( \eta \) as \( \eta = 0.372 + 0.043 \cdot [PM] + 0.917 \cdot [S3] - 0.066 \cdot [S8] + 0.219 \cdot [S7] + 0.010 \cdot [S5] \).

**REFERENCES**


