Differences in occupational mortality from pleural cancer, peritoneal cancer, and asbestos

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Abstract

Objective—To assess whether the increased risk of disease related to asbestos in occupations from the construction and engineering industries applies equally to pleural cancer, peritoneal cancer, and asbestosis.

Methods—Analysis was based on deaths among men aged 20–74 in England and Wales during 1979–80 and 1982–90. (n = 1 656 096). Information about cause of death and the last full time occupation of decedents was derived from death certificates. Proportional mortality ratios (PMRs) by occupation were calculated for each of pleural cancer, peritoneal cancer, and asbestosis.

Results—Altogether, 2848 deaths were attributed to cancer of the pleura, 362 to cancer of the peritoneum, and 281 to asbestosis. When occupations were ranked according to PMRs from these diseases, striking differences were found. The category of construction workers which included lagger had the highest mortality from peritoneal cancer (PMR 990, 64 deaths), but a PMR of only 160 (77 deaths) for pleural cancer. In contrast, several occupations with much higher mortality from pleural tumours had no excess of peritoneal cancer. PMRs for asbestosis related more closely to those for peritoneal than pleural cancer.

Conclusions—These findings suggest that the exposure–response relations for diseases related to asbestos are not all linear, and that risks of pleural mesothelioma may be underestimated by simple extrapolation from observations in cohorts with heavy exposure.

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Keywords: asbestos; mortality; mesothelioma

Occupational exposure to asbestos is a major cause of mortality from asbestosis, mesothelioma, and bronchial carcinoma. In Britain, legal controls on exposure to asbestos were first introduced in 1933, and since then have been progressively tightened. Recent analysis indicates that mortality from mesothelioma is rising in men at most ages, and suggests that overall rates will continue to increase through to the next century. It is important to establish whether this trend results from inadequate enforcement of statutory exposure limits, or whether the limits themselves are not sufficiently stringent.

One starting point is to identify the occupations associated with excess mortality from diseases related to asbestos. During the 1980s, a large proportion of male deaths from mesothelioma in England and Wales were related to work in the construction and engineering industries. It is unclear whether the increased risk in these jobs applies to mesothelioma of the pleura, peritoneum, or both. We have analysed data from the same period that indicate important differences in the relative frequency of pleural and peritoneal mesothelioma by occupation, and which may have implications for control strategy.

Method

Our analysis was based on all deaths among men aged 20–74 in England and Wales in 1979–80 and 1982–90. Data for 1981 were unreliable because of industrial action in that year by registrars of deaths. Information about the age, underlying cause of death, and most recent full time occupation of decedents was obtained from death certificates. Causes of death were coded to the ninth revision of the international classification of diseases (ICD-9), and occupations were coded initially to the Office of Population Censuses and Surveys (OPCS) 1980 classification of occupations. The occupational units defined in the OPCS classification were then aggregated into 194 larger job groups.

For each job group we calculated proportional mortality ratios (PMRs) for cancer of the pleura (ICD-9 = 163), cancer of the peritoneum (ICD-9 = 158-8 and 158-9), and asbestosis (ICD-9 = 501), with five-year age specific proportions in all occupations combined as the standard. Confidence intervals (CIs) for PMRs were based on the Poisson distribution.

Results

Over the 11 year period of study 1 656 096 deaths were recorded in men with adequately described occupations, including 2848 from cancer of the pleura, 362 from cancer of the peritoneum, and 281 from asbestosis. The table lists the job groups with significantly (P < 0·05) raised PMRs for at least one of these diseases.

The ranking of PMRs for cancers of the pleura and peritoneum was quite different. For example, construction workers not elsewhere classified (nec), a group which includes laggers, had the highest mortality from peritoneal cancer (PMR 990, 64 deaths), but a
PMR of only 160 (77 deaths) for pleural cancer. In contrast, several of the occupations with the highest mortality from cancer of the pleura—metal plate workers (PMR 709, 73 deaths), upholsterers (PMR 366, 16 deaths), carpenters (PMR 362, 167 deaths) and electricians (PMR 349, 127 deaths)—had no excess of peritoneal cancer.

Mortality from asbestosis was more closely related to that from peritoneal than pleural cancer, with the highest PMR again in construction workers nec (PMR 1592, 71 deaths). The Spearman rank correlation coefficient across the 24 job groups in the table was 0.43 for asbestosis with cancer of the peritoneum and 0.15 for asbestosis with cancer of the pleura.

**Discussion**

In this analysis we restricted attention to the three causes of death that are related most specifically to asbestos. Asbestos also causes bronchial carcinoma, but the effect is less discernible in analyses of occupational mortality because the relative risk is smaller and because associations are confounded by differences in smoking and exposure to other lung carcinogens in the workplace.

The analysis was limited by inaccuracies that are known to occur in information obtained from death certificates. Not all deaths ascribed to cancers of the pleura or peritoneum are mesotheliomas, and some mesothelioma deaths are classified as cancers of other or unspecified sites. Also, some deaths may be incorrectly attributed to asbestosis on the basis of pleural thickening or plaques, when no fibrosis is present. Moreover, data were only available on the most recent full time job of decedents, and some subjects will have been exposed to asbestos in earlier employment about which we had no information. In general, however, the effect of such errors should be to attenuate occupational associations, and they would not be expected to have a differential effect on the ranking of occupations by mortality from different diseases related to asbestos. The use of periodic medical examinations or an unusually high rate of necropsies in certain occupations might boost the detection of peritoneal more than pleural tumours, but it would not explain such large disparities in the relative frequencies of these diseases as were found. Nor would it account for the high frequency of deaths from asbestosis in some occupations with relatively low mortality from pleural cancer.

Another limitation was the use of proportional mortality rather than true death rates. Although PMRs may have been somewhat depressed or inflated by differences in the overall death rates of job groups, the effect should be similar for each of the three diseases examined, and again would not explain the contrasting ranking of jobs by PMR.

The occupations with significantly raised PMRs in our analysis were largely the same as those found previously to have high mortality from mesothelioma overall. All entail potential exposure to asbestos although in some cases the exposure is related to work in specific industries rather than a general feature of the occupation. For example, the excess mortality among welders occurred mainly in centres of shipbuilding. Many of the high risk occupations are in the construction industry where exposure has occurred from the use of asbestos in lagging and other building materials. The high PMR for cancer of the pleura in carpenters compared with many other building trades may be related to work with asbestos board. Notable for an absence of significant risk are motor mechanics, (PMR 46, 12 deaths from pleural cancer; PMR 88, three deaths from peritoneal cancer; PMR 80, two deaths from asbestosis) about whom concerns...
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have been raised because of the presence of asbestos in brake linings. It seems that this exposure has no important effect on mortality from mesothelioma or asbestosis, perhaps because the asbestos fibres are modified by heat.

The difference in the ranking of occupations by mortality from pleural and peritoneal cancer and asbestosis is striking and cannot realistically be ascribed to chance. Nor is it likely to be explained simply by differences in the types of asbestos to which occupations are exposed. Crocidolite and amosite are more potent causes of mesothelioma than chrysotile, and all of the occupations with high PMRs from peritoneal cancer could have involved exposure to crocidolite. But welders, who had a higher PMR than construction workers nec from pleural cancer, are also likely to have had exposure to crocidolite (especially those employed in shipbuilding), and yet had a deficit of peritoneal cancers.

A more plausible explanation is that the exposure-response relations for mesothelioma and asbestosis are non-linear, with the risk of pleural mesothelioma increasing relatively more steeply at low exposures but less steeply at high exposures. Where an occupation entails low exposure to asbestos, excess pleural cancer occurs but there is little effect on peritoneal cancer or asbestosis. With high exposures, the risk of pleural cancer is increased further, but that of peritoneal cancer and asbestosis goes up much more and becomes dominant. Also, the effect on occupational mortality is diluted according to the proportion of men in the job group who are exposed. For example, construction workers nec include not only lagger with very high exposure to asbestos, but also other occupations such as floor layers with minimal exposure. As a consequence, the PMRs of the group are reduced for diseases related to asbestos. In the case of peritoneal cancer and asbestosis, the risk in lagger is so high that the PMR of the job group as a whole remains highest in the ranking. The PMR for pleural cancer is lower and diluted to a level below that in occupations such as carpenters where a larger proportion of men are exposed, but at a lower level.

This hypothesis is consistent with findings on the ratio of pleural to peritoneal mesotheliomas in cohort studies of asbestos workers, where peritoneal tumours have tended to be relatively more common in cohorts with longer and heavier exposures, although with occasional exceptions. It also accords with the finding of higher fibre contents in the lungs of patients dying from peritoneal compared with pleural cancer. It is an indication for caution when extrapolating risk estimates for mesothelioma at low exposures to crocidolite and amosite from observations in cohorts with heavy exposure. In particular, the risks of pleural mesothelioma may be underestimated if a linear exposure-response is assumed. This should be taken into account when control limits are reviewed.

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