Correspondence

Metal polishing, stomach cancer, and clearing houses

SIR,—In a letter published in your June issue (p 429) Drs Mant and Mayon-White expressed the need for a “clearing house” to deal with sporadic clusters of disease. This is a matter which the Medical Division of the Health and Safety Executive and the Employment Medical Advisory Service have attempted to deal with.

Some years ago “an early warning system” was established for reporting clusters or single cases of disease where a hitherto unrecognised occupational aetiology might be postulated. This was advertised through our field force of employment medical advisers and the Society of Occupational Medicine’s Newsletter. In the ensuing two years fewer than half a dozen notifications were received, since then even fewer have been received.

The need for a workable scheme is undeniable, but the recourses required for investigation and collation of data and feedback to physicians are considerable. The success of any such venture would depend on sufficient publicity to ensure a continued awareness of the scheme.

The Health and Safety Executive is at present considering ways in which information on occupational ill health may be improved, and would be interested to hear of possible health problems associated with occupation.

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Risk assessment in the asbestos cement industry

SIR,—Hughes and colleagues have recently published (1987;44:161–74) an updated study of mortality of workers employed in two American asbestos cement manufacturing plants and have presented an estimate of lung cancer risk in the asbestos cement industry. By tracing over 95% of the workers they have corrected the serious flaw in their earlier study1 which had left the vital status of many workers uncertain. Several problems persist with respect to the interpretation of the latest report, however.

The authors have presented dose response relations for lung cancer derived by combining estimates of individual cumulative asbestos exposures with diagnoses of lung cancer obtained from death certificates. In plant 1 the slope of the relation was not significantly different from zero; nor was it significantly different from zero in plant 2, but by resorting to the technique of forcing the intercept through zero, the authors were able to obtain a “significant” slope for the regression line. The regression method they used was that of iteratively weighted least squares, and the authors of this method have stated that they consider the slope obtained by forcing the intercept through zero to be less meaningful than the unrestricted slope.2 The numerical result presented by Hughes and colleagues must thus be viewed with some caution.

As part of their discussion, the authors reviewed the findings for lung cancer and mesothelioma in seven cohorts of asbestos cement workers. They compared the results of the various studies by plotting the observed lung cancer risk by the estimated median employment duration. This method of comparison does not, however, produce a meaningful result, for, as Johnson has recently reminded readers of this journal, duration of exposure is a good surrogate for dose only when exposed individuals were subject to equal exposures for different periods.3 Exposure estimates were available for only three of the seven cohorts, and among these three, estimates were not the same. The appropriate method of comparison is to take account of the cumulative exposures of the workers. The most comparable of the cohorts are a Canadian cohort. I have studied the workers from plant 2 of Hughes et al4 since both factories were owned by the same multinational corporation and they manufactured similar products. The ratios of observed to expected deaths per fibre-year of cumulative exposure, using the figures of Hughes et al, are about 0.045 and 0.068 (United States cohorts) and 0.054 (Canadian cohort).

Despite this apparent agreement in lung cancer risk, a more careful comparison shows that there are substantial differences. The Canadian cohort comprised workers employed one year or more. Their average cumulative exposure was estimated to be 60 fibre-years/ml, within an estimated range of error of three to fivefold, and the lung cancer SMR was 512. From table 10 of Hughes et al5 I estimate that the average cumulative exposure of the plant 2 employees who worked one year or more was about 85 fy/ml and the lung cancer SMR was 180 (table 7). How can one account for the threefold difference in risk?

One may look for explanations on both sides of the dose response equation. The Canadian exposure estimates were stated to be uncertain to within a factor of three to five, so that increasing the estimated exposure to the upper end of this range could account for much of the difference. On the other hand, both plants were owned and operated by the same multi-
national corporation and they manufactured similar products. The estimates of average workplace exposure appear to be similar in both plants, so that the Canadian exposure estimates may not be greatly understated.

On the response side of the equation the matter of diagnosis is crucial, and here differences in the health care system between Canada and the United States may account for some of the disparity. Citizens of Ontario are covered by a government run health insurance scheme that pays all physician and hospital fees as well as the costs of diagnostic testing. Most of the ill workers from the Canadian factory were treated at university teaching hospitals or at hospitals around metropolitan Toronto. Blue collar workers in the southern United States may not have had access to the same quality of medical care under the free enterprise American health care system. Diagnostic information appearing on the death certificates of asbestos workers is often inaccurate and Hughes and colleagues report no effort to go beyond the death certificate to establish the causes of death. They consequently report that 13 deaths in plant 1 and 32 in plant 2 were attributed to cancers of ill defined sites or were secondary respiratory/digestive cancers. These poorly specified malignancies represent 30% of the total attributed to respiratory cancers, and, whereas the authors acknowledge that some of these cancers may have been related to asbestos, they ignored them in the risk assessment. Failure to take account of these additional tumours almost certainly leads to an underestimate of the risk of asbestos exposure.

By contrast, the sites of six of 72 tumours were unspecified by death certificates in the Canadian study but evidence was obtained to determine that among the six were two lung cancers, a mesothelioma, and a stomach cancer. Slides or pathology reports were obtained for review for 49 of the 61 lung, gastrointestinal, or mesothelial tumours in the Canadian study. This information, as well as other "best evidence" information, was used when exposure response calculations were performed using direct standardisation and comparisons internal to the cohort.

The assessment of lung cancer risk in the asbestos cement industry thus remains problematical. Although the Canadian study found significant dose response trends for the asbestos associated malignancies considered together as a group, the shape of the dose response curve for lung cancer was perturbed by the influence of competing causes of death in the higher exposure categories. In the study of Hughes and colleagues there was almost certainly underascertainment of asbestos associated cancers and lung cancer risk showed no sensible relation with estimated cumulative asbestos exposure in either of the two plants. Pending new information, the best that can be done is to consider carefully all the data currently available while bearing in mind the flaws of each study.

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Drs Hughes and Weill reply:
We are at a loss to know why Finkelstein claims that the dose response relation for plant 2 was not statistically significant unless we "resorted" to forcing the intercept through one. Contrary to his claim, when a line is fitted to these data without forcing an intercept of one (the result, as stated in our paper, was: y = 1.17 + 0.0085 x, where x is cumulative asbestos exposure in mppcf-ys), the slope was significantly non-zero: improvement chi-squared statistic (compared with fitting a constant—that is, a zero slope): 5.00, p = 0.026. We can only surmise that Finkelstein calculated the goodness of fit $\chi^2$ statistic for the fitted line, which, indeed, was not statistically significant; this test only confirms that the linear model fits our data reasonably well.

We are well aware of the issue of whether the intercept should be forced through one or not, which is why we provided the fitted equation using both approaches. With either method, the fitted line for plant 2 provides a reasonably good fit to our data and the slope is statistically significant. Therefore, contrary to Finkelstein's statement, the results concerning lung cancer risk and estimated cumulative asbestos exposure make sense for both plants in our study. In plant 2, where an excess lung cancer risk was observed, there was a generally increasing trend of risk with increasing level of estimated exposure, and this trend was statistically significant. In plant 1, where there was little or no excess lung cancer risk, no trend was observed and the observed lung cancer SMRs were consistent with random variability about the expected value of 100.

In comparing the results of the various published asbestos cement studies (table 14) we would certainly have preferred to use more detailed information on asbestos exposure than duration of exposure. Unfortunately, as we indicated, duration was the only measure available for several of these cohorts. As Finkelstein points out, the disparate results of these studies with respect to duration of exposure probably indicates that the Canadian cohort had different exposures over time compared with the other cohorts.

We are unable to reproduce Finkelstein's values for the ratio of observed to expected lung cancer deaths
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per fibre-year of cumulative exposure for the two New Orleans plants; the values should be 0.031 and 0.100. This comparison considers only a single, combined measure of observed risk; in comparing only these three cohorts more detailed information is available and should be used. Moreover, this comparison relies on the exposure estimates for the Canadian cohort, which have been questioned, since no sensible pattern of lung cancer risk with cumulative exposure was observed for that cohort. In comparing these three cohorts, attention may be restricted to workers in our plants who were employed for more than five years. With median durations of more than 15 years for these two groups, they are reasonably comparable with the Canadian cohort, with a median duration estimated as approximately ten years. Despite similar average exposure concentration estimates, the lung cancer results remain surprisingly different: SMRs of 109 and 221 for these subgroups in our plants compared with 490 for the Canadian cohort. These differences, even those between the two New Orleans plants, suggest important differences in these plants, possibly in the actual exposure concentrations or in fibre types.

With regard to causes of death in our residual cancer category, we pointed out the number of deaths for each of the principal ICD codes within this category precisely because of our concern about this group. We do not agree, however, that the “best evidence” approach is appropriate here since we are primarily interested in comparisons with general population rates based on death certificate information. Internal comparisons can show a dose response relation, thus establishing causality, but this is hardly necessary for asbestos and lung cancer today.

We may, however, consider this group in another way. Since there are ICD codes for secondary digestive/respiratory cancers and for cancer with site unspecified, comparisons were made of the observed and expected numbers, as for any other sites. In neither plant were the secondary digestive/respiratory cancers raised compared with Louisiana rates. For site unspecified among plant 1 workers, there was a small excess among the shortest term workers but no excess in the longer term and no trend with cumulative exposure. In plant 2 there were 26 cancers with unspecified (including three mesotheliomas) compared with 14.2 expected. The possible concern, therefore, is with the 11.8 excess cases with site unspecified, and whether some of these could be lung cancers. In an attempt to determine how much lung cancer dose response would be affected if some of these excess site unspecified cancers were actually lung cancers for each cumulative category of asbestos exposure the excess cases were distributed to the specific sites (lung or digestive, for example) in the same proportion as the cases of cancer with site specified. This allocation had only a minimal effect on the non-respiratory cancer SMRs. The weighted least squares regression line for lung cancer and cumulative asbestos exposure, without forcing an intercept of one, became 1.24 + 0.0075 x, for x in f/ml-y (the slope was statistically significant, p < 0.02). This compares with the reported fit of 1.17 + 0.0061 x, without allocation of these cases. Thus the cancers of unspecified sites had little effect on the dose response relation; the fit using a forced intercept of 1-0 was also not changed appreciably by allocating these cases.

Finally, we are amazed at Finkelstein’s gratuitous remarks concerning “blue collar workers in the southern United States” and the “quality of medical care under the free enterprise American health care system,” implying that misdiagnoses may be related. There is not a whit of evidence to support this socioeconomic speculation and we find such commentary inappropriate. Moreover, the State of Louisiana has long been a recognised leader in providing free medical care to its citizens; the primary facility of this system is located in New Orleans and serves as a teaching hospital for two university medical schools.

References

Asbestos related lung disease in maintenance workers

Sir,—Hilt’s recent study (1987:44:621–6) of the prevalence of non-malignant asbestos related lung disorders in a cohort of chemical industry workers exposed to asbestos highlights two important aspects of asbestos related occupational lung disease: (1) the importance of “indirect” or “secondary” exposures in the aetiology of these diseases and (2) the need for increased recognition of such exposures and monitoring of these occupational groups.

Indirect exposure to asbestos in occupational settings is becoming an increasingly recognised health risk, particularly in non-asbestos industries. For instance, Paci et al have described recently the existence of an asbestos hazard among non-asbestos textile workers in Italy, specifically among “rag sorters” working in the reprocessed textile industry. These individuals were exposed to asbestos fibres freed from polypropylene bags previously used to transport asbestos. These bags were cut and used in the textile
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