Correspondence

Correlation between fibre content of lung and disease in east London asbestos factory workers

SIR,—Wagner *et al* (1988;45:305-8) reported asbestos fibre counts in asbestos workers with various diseases and in control subjects. When increasing attention is being paid to the importance of an adequate statistical basis for inferences in medical papers,¹ it was depressing to note that their article was devoid of statistical analysis and quoted only mean values for groups being compared with no indication of variation about the mean.

Leaving aside these considerations, I have other reservations about their findings. The mean asbestos fibre count of 11.2 million (31.3% of 35.8 million) per gram of dry lung in the control group was higher than that indicated in a control group including dwellers in heavily polluted industrial cities in a previous study by Wagner and colleagues.² (In that study the mean value was not quoted but fig 1 in the paper indicates that the median value was much less than 10 million.) It was stated that the control subjects were patients undergoing surgery for lung cancer who generally lived in east London but who had no history of occupational exposure to asbestos. No details were given as to how the occupational history was obtained. Many east Londoners have had a varied career including one or more jobs with some exposure to asbestos and if the absence of exposure to asbestos was deduced from the hospital records rather than from a detailed occupational history the information must be regarded as unreliable. This possibility is supported by the mention in the discussion that less chrysotile was found in factory workers with pleural mesothelioma than in controls. It is difficult to believe that environmental pollution alone, even in east London, resulted in a higher chrysotile burden in the lungs than work in a factory in which chrysotile was used extensively.

The authors suggested that their results indicated a much greater degree of asbestosis in subjects with lung cancer than in subjects with mesothelioma. The cases considered had been referred to the pneumoconiosis medical panel after postmortem examinations had been conducted on behalf of coroners between 1976 and 1984. During this period lung cancer was eligible for compensation by the Pneumoconiosis Medical Panel only if asbestosis of the lungs was also present and death certificates did not carry the current reminder to consider the possibility of industrial disease in cases of lung cancer where there had been exposure to asbestos. Consequently, cases of lung cancer in asbestos workers in whom clinical asbestosis

had not been diagnosed were usually not reported to the coroner whereas cases of mesothelioma were more commonly reported whether or not asbestosis was present. The authors' suggestion that selection bias is unlikely to have operated is, therefore, almost certainly incorrect. That bias did occur is supported by the absence of any cases of lung cancer with the lowest grade of asbestosis, likely to be subclinical, and the absence of any cases without asbestosis. The data presented do not justify conclusions about the frequency and severity of asbestosis in patients with lung cancer and mesothelioma.

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References

- 1 Gardner MJ, Altman DG. Estimating with confidence. *Br Med J* 1988;296:1210-1.
- 2 Wagner JC, Pooley FD, Berry G, *et al*. A pathological and mineralogical study of asbestos-related deaths in the United Kingdom in 1977. *Ann Occup Hyg* 1982;26:423-31.

Wagner *et al* reply:

Rudd is correct to be concerned about our failure to report any indications of variability. These were included in draft versions of our paper but regrettably were omitted finally.

The results for mean and range of total fibres by diagnostic category, together with those for asbestos fibres alone, are given in table 1.

Unfortunately, in his interpretation of our data Rudd has erroneously equated percentage of average with average of percentages. We tabulated the average of the percentage distribution of fibre contents of the lung, in line with other authors. It is incorrect to assume that multiplying the mean fibre count by the mean percentage of asbestos fibres will yield the mean fibre count for asbestos fibres. Table 1 provides the data for asbestos fibre counts that Rudd was trying to calculate. He also assumed that the mean and median values were likely to be equal, which is well known not to be true for very skew distributions, such as fibre counts. In our study the mean asbestos fibre count in the controls was more than double the median count of 6.3 million fibres per gram of lung tissue. In this our study is concordant with the previous study by Wagner and his colleagues.

It is appropriate to calculate confidence intervals for fibre counts by using the logarithmic transformation

Table 1 Mean total and asbestos fibre content by diagnostic category expressed as millions of fibres per gram dried lung

Category	No	Total fibre count			Asbestos fibre count		
		Mean	Median	Range	Mean	Median	Range
Controls	56	35.8	27	4-179	13.8	6	0.4-161
Carcinoma lung complicating asbestosis	14	1141.7	812	172-4378	1108.3	769	160-4378
Pleural mesothelioma	9	262.9	54	11-1080	241.6	33	8-1004
Peritoneal mesothelioma	10	565.7	186	48-1908	532.1	168	30-1908
Asbestosis	3	1720-7	358	144-4661	1652.1	294	95-4568

Table 2 Lung contents of total fibre and total asbestos fibre expressed as millions of fibres per gram dried lung: geometric means (95% confidence limits)

Category	No	Total fibre	Total asbestos fibre
Controls	56	27 (20, 35)	6.5 (4.6, 9.1)
Carcinoma lung complicating asbestos	14	761 (447, 1300)	714 (360, 1415)
Pleural mesothelioma	9	83 (43, 162)	61 (26, 143)
Peritoneal mesothelioma	10	287 (153, 538)	241 (107, 542)
Asbestosis	3	621 (197, 1960)	503 (115, 2205)

and a pooled estimate for the variance. On this basis, the means and confidence intervals for total fibre contents are shown in table 2. Note that, as often happens with skew distributions, failure to use a transformation would have led to two of the confidence intervals including negative values. Clearly this is inappropriate for essentially positive data.

Turning to the occupational histories of our control subjects (who were patients undergoing surgery for lung cancer) these were indeed obtained from the routine hospital records. The hospital concerned was Rudd's own and several of the patients had been

referred for surgery by Rudd himself. His assertion that reliable occupational histories are not routinely taken regrettably detracts from the value of our controls, but it is unlikely that a large proportion of east Londoners have worked with asbestos. We believe that our 56 controls may have included just one with unsuspected occupational exposure. This case had high crocidolite and amosite fibre counts, six times higher than any other control. Overall, 42 of the 56 controls had crocidolite and amosite fibre counts of less than 1 million per gram of lung tissue, whereas all but two of the asbestos workers exceeded 10 million.

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VIIIth International Symposium on Occupational Health in the Production of Artificial Organic Fibres, Nancy, France, 10-12 October 1989

Scope and aims: health hazards in the production of artificial fibres from materials of natural or synthetic origin, mainly with their associated chemicals. The fibres production and the toxic chemicals may be