

Correspondence

Pulmonary fibrosis in asbestos insulation workers with lung cancer

SIR,—There has been discussion of the paper of Kipen *et al* (1987;44:96-100) by Rudd¹ and a response by Suzuki *et al*² but neither of these letters has dealt with the basic flaw in the design of the original investigation.

Kipen *et al* reviewed 138 cases of histologically confirmed lung cancer among 450 diagnosed in the United States-Canadian insulator cohort. The restriction to 138 was because only this number had adequate chest *x* ray films and adequate samples of pulmonary tissue for microscopic evaluation. Whether the 138 cases were representative of all the cases is open to question but let us assume that they were. The major problems were: (1) it was assumed that all interstitial fibrosis, even with rare or no asbestos bodies, was asbestosis by the authors since they equated the two terms in two places; (2) there were no unexposed control groups with "blind" evaluation of either the chest *x* ray films or the histological specimens.

Regarding the first problem, it must be recognised that agents other than asbestos cause interstitial fibrosis, perhaps most commonly cigarette smoke,³ and asbestosis has no pathognomonic *x* ray or histological signs. Therefore, the second problem, the lack of "blindly assessed" controls, becomes the most serious defect in this study. Without "blindly evaluated" controls, it is impossible to assess the contribution of asbestos to the causation of lung cancer in asbestos workers.

Among the United States-Canadian insulators investigated, the relative risk of lung cancer 20 or more years after onset of exposure may be calculated to be 4.23 from data in the paper by Selikoff *et al*⁴ using death certificate diagnoses to provide a valid comparison with the risk in the general population. From this relative risk, it is obvious that 23.6%—that is, one out of every 4.23 cases of lung cancer—would have occurred in this population without exposure to asbestos. I made this calculation based on the interval of 20 or more years from onset of exposure because 134 of the 138 cases studied by Kipen *et al* occurred 20 or more years from onset of exposure (data from their fig 3). This figure of 23.6% approximates the 18% figure in the series of 138 cases reported as having no radiographic signs of asbestosis in the pulmonary parenchyma. In view of the potential for error in the methods used, this approximation is remarkably good.

Since both asbestosis and asbestos related lung cancer have a dose response relation to asbestos, the most parsimonious interpretation of these juxtaposed data is that the cases of lung cancer without *x* ray evidence of asbestosis are the cases that would have occurred in this cohort if there had been no exposure to asbestos—that is, they were the expected number. The conclusion by Kipen *et al* that their findings "indicate the primacy of the history of exposure to asbestos, irrespective of the presence or absence of non-malignant *x* ray changes (asbestosis) when considering lung cancer possibly associated with occupational exposure to asbestos" is not warranted by the design of their study.

There is considerable circumstantial evidence that the risk of lung cancer is raised only among workers exposed to asbestos who also have parenchymal asbestosis.⁵ If this issue is ever to be settled definitively adequately designed studies must be undertaken.

W WEISS

*Emeritus Professor of Medicine,
Hahnemann University,
3912 Netherfield Road,
Philadelphia, PA, USA 19129.*

References

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- 2 Suzuki Y, Kipen H, Lillis R, Selikoff IJ. Pulmonary fibrosis in asbestos insulation workers with lung cancer. *Br J Ind Med* 1987;44:719-20.
- 3 Weiss W. Smoking and pulmonary fibrosis. *J Occup Med* 1988;30:33-9.
- 4 Selikoff IJ, Hammond EC, Seidman H. Latency of asbestos disease among insulation workers in the United States and Canada. *Cancer* 1980;46:2736-40.
- 5 Browne K. Is asbestos or asbestosis the cause of the increased risk of lung cancer in asbestos workers? *Br J Ind Med* 1986;43:145-9.

Incidence of cancer of the scrotum, 1971-84

SIR,—In 1984 Waldron *et al* reported on scrotal cancer in the West Midlands for the period 1936-76 (1984;41:445-9). They noted that the annual number of registrations appeared to be declining in the 1970s, compared with the relatively high numbers recorded in the 1960s. We can confirm that this trend has persisted having examined recent cancer registration data for the West Midlands Region (see figure). A recorded occupation was available for 109 (73%) of the 149 cases registered in the period 1970-84. Of these 109 occupations, at least 70% probably involved exposure either to mineral oils, pitch, or tar.

Annual numbers of registrations for England and