CORRESPONDENCE

Pulmonary effects of exposure to fine fibreglass: irregular opacities and small airways obstruction

Sir,—Kilburn et al (1992;49: 714–20) published a cross sectional prevalence study of workers exposed to fibreglass in an appliance manufacturing plant by means of chest x ray films and spirometry. They concluded that fibreglass used for insulating purposes seems to produce human disease similar to asbestosis. Because of the well known interobserver error in the reading of chest films, it is essential as a matter of good science that the investigation includes “blind” concurrent readings of films from unexposed controls similar to the exposed group in all respects except for the exposure being studied. Kilburn et al neglected to include such a procedure. The use of the standard films for pneumoconiosis is no substitute for proper controls as it tells us nothing about under or over-reading by the investigators.

The reading of control films intermixed with films of the exposed group without knowledge by the readers as to which group each film belongs to might have shown no differences if there was matching by at least age, sex, and smoking habits. Smoking habits should include type of tobacco use and degree as well as duration of smoking. Kilburn et al did not include any adjustment for degree of cigarette smoking in their adjustment of their radiographic results and there is no evidence of adjustment for smoking habits with regard to the readings of the chest films in their study. Consequently, their conclusion is not warranted because their method was seriously flawed. In addition, I agree with the other points made by Rossetter.1

The reply by Kilburn et al contains a section labelled “No fibrosis from cigarette smoking alone” which is full of error. They claimed that I used minifilms (70 mm) in a 1972 paper and deny the existence of Rossetter’s reference No 5 to my 1991 paper in the British Journal of Industrial Medicine! My original studies of this subject were done with minifilms and were reported in the 1960s but subsequent studies were done with the use of large films, all without my knowledge of smoking habits, and these later reports entailed the use of the 1980 International Labour Office standard films and classification. These reports were confirmatory of the early findings. Kilburn et al are apparently unaware of my papers published in 19843 and 1988.4 These include extensive reviews of relevant publications. Perhaps they have simply ignored this body of work?

It is interesting to note that Kilburn2 (Rossetter’s reference No 6) in 1981 not only denied the validity of my earlier reports on the relation between cigarette smoking and radiological abnormalities suggestive of mild diffuse pulmonary fibrosis but also castigated my publications since 1971 on the increased frequency of asbestosis in smokers compared with non-smokers. Yet in 1986 he published a study,5 the only reference in his response to Rossetter4 in which his own data confirmed this phenomenon. Neither in 1986 nor now has he referred to his 1981 error in denying a smoking effect on the frequency of asbestosis.

In his 1992 paper there were two errors which he had to admit in the correspondence: in the answer to Bender5, Kilburn et al admitted missing the concentrations of fibreglass by an order of magnitude and in the response to Weiss6, missing evidence on duration of asbestos exposure. One wonders how many other errors have gone undetected. The entire response to Rossetter’s other criticisms is woefully inadequate.

WILLIAM WEISS
(Emeritus Professor of Medicine,
Hahnemann University),
3912 Netherfield Road,
Philadelphia, PA 19129, USA.


Authors’ reply:
As an expert “over reader” the concern of Weiss for interobserver error (bias) is commendable. Attractive as the idea of blind concurrent controls may be, the absence of pneumoconiosis in 241 of 284 fibreglass workers surely met the same objective for both our readers.

Weiss is wrong concerning “any adjustment for degree of cigarette smoking in their adjustment of their radiographic results.” Our spirometric measurements were adjusted for height, sex, age, and duration (years) of cigarette smoking. The coefficient for duration is the smoking correction with the most power.12

As cigarette smoking does not produce fine irregular opacities alone but only increases the likelihood in the presence of fibres, there is no adjustment procedure for it in interpretation of the chest x ray films.

The long defence by Weiss of his single observer, non-standard readings of x ray films for asbestosis is out of place here and has been fully answered previously.3

The final insinuation that “other errors have gone undetected” is nothing but a “cheap shot”. The fibreglass levels measured without the resolution of electron microscopy are irrelevant (reply to Bender). The durations of asbestos exposure were also not contributory as the workers exposed to asbestos were removed from consideration for fibreglass causation, although this is an overcorrection.