

*The Authors' reply*

Bender is correct. The decimal point in the fibre counts, which were done by light microscopy, moved twixt mind and print. This change does not alter our perception of the conditions of exposure in the plant which emphasise in the same paragraph of the paper that 49% to 83% of the fibreglass used in the plant had diameters less than 5 µm. Most of these would be invisible to light microscopy. Moreover, no dependable relation exists to estimate total airborne fibre burdens from light microscopical counts of fibres with widely variable diameters. This puts Bender's second point in perspective. In a dense swirl of fibres that are invisible to the light microscope, the asbestos concentrations of 0.1 to 0.25 fibres/ml in four of 14 samples seem of lesser concern when no fibres were detected in the other 10 samples.

This plant was closed shortly after our study so measuring and further modelling was precluded.

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

Sir,—The paper by Kilburn *et al* (1992;49:714–20)<sup>1</sup> differs only slightly from a previously published version,<sup>2</sup> neither of which provides any evidence that “commercial spun rotary fibreglass used for insulating appliances appears to produce human disease that is similar to asbestosis” (authors' abstract). There

are some substantive questions that the authors have not considered but of which they were aware.<sup>3</sup>

#### **Radiographic changes**

Firstly, the authors assume that any appearance of radiographic change, with the International Labour Office (ILO) 1980 criteria,<sup>4</sup> shows that the person is suffering from pneumoconiosis. The ILO classification states that it does not define pathological entities and that there are no features seen in a chest radiograph that are pathognomic of dust exposure. It is descriptive of the chest radiographic appearances. Interpretation of the findings requires other relevant evidence.

This other evidence mainly concerns cigarette smoking. Weiss<sup>5</sup> has clearly shown that small irregular opacities are more prevalent among smoking workers unexposed to hazardous dust. Regrettably, Kilburn *et al*<sup>1,2</sup> failed to publish the numbers of smokers, ex-smokers, or non-smokers, although they did publish the numerator numbers of workers with any radiographic change.

It is possible, however, to determine the denominators from the various percentages published by Kilburn *et al*. Table 1 compares the prevalences of radiographic changes from the current paper with those from the earlier version, and with the prevalences from Weiss.<sup>5</sup> The prevalences of Kilburn *et al*, although differing in their two publications, agree closely with the results from Weiss.<sup>5</sup> There could be some slight discrepancy

because of the differing definitions of radiographic change. The current rates of Kilburn *et al* do not include 12 people with small opacities read as category 0/1, but do include 10 workers with pleural changes only. For neither subgroup were smoking data provided. The overall Kilburn *et al*<sup>1</sup> prevalence of any small opacities is 15.8%, very close to the Weiss<sup>5</sup> rate of 15.5%. It must be presumed that the pattern of small opacity prevalence by smoking habits for Kilburn *et al*<sup>1</sup> is close to that shown in the table.

The 35 people with radiographic change in the previous version of this paper<sup>2</sup> included two with pleural changes only. The pattern of small opacity prevalence by smoking habits for the remaining 33 people must also be close to that shown in the table.

Kilburn denies any association between cigarette smoking and small opacities,<sup>6</sup> but these data contradict his own views.

#### **Pulmonary function**

The second argument adduced by Kilburn *et al* for an effect of fibreglass is that the workers' lung function was reduced significantly. The evidence for such a reduction is based on comparison with a reference group. There are two important issues in relation to the comparison group chosen by Kilburn *et al*: non-validity of comparison group; and resulting bias.

The prediction equations were developed by Miller *et al*<sup>7</sup> from a small sample, biased towards rural dwellers from Michigan State. Of the 1738 people who actually completed the examinations, 79% were rejected for a variety of reasons. It is impossible to conclude that the remaining 369 white, non-obese, clinically normal, non-extreme people form a valid comparison group. As an example of the effect of the rejection criteria, all smokers aged over 63 years were excluded.

As a result of the exclusion rules, the prediction equations of Miller *et al*<sup>7</sup> are likely to overestimate predicted lung function, with the overprediction increasing with age. For FEV<sub>1</sub> for example, the age coefficient of Miller *et al* for males is -0.0233. This coefficient compares with -0.0292 from Knudson *et al*<sup>8</sup> and -0.033 from Cotes *et al*.<sup>9</sup> At age 25, the prediction of Miller *et al* is 2% or 9%

*Comparison of prevalences of radiographic changes by smoker group*

	Smoker group			Total
	Smokers	Ex-smokers	Non-smokers	
Kilburn <i>et al</i> <sup>1</sup> (current paper): No of workers	93	92	99	284
With small irregular opacities 1/0 or more and pleural changes	18 (19.4%)	19 (20.7%)	6 (6.1%)	43 (15.1%)
Kilburn <i>et al</i> <sup>2</sup> (previous version): No of workers	93	92	99	284
With small irregular opacities and pleural changes	21 (22.6%)	11 (12.0%)	3 (3.0%)	35 (12.3%)
Weiss <sup>5</sup> No of workers	135	-	46	181
With small irregular opacities 0/1 or more	27 (20.0%)	-	1 (2.2%)	28 (15.5%)

higher than that for Knudson *et al* or Cotes *et al*. By age 60, the Miller *et al* prediction is 10% or 23% higher. If the prediction equations of Knudson *et al*<sup>8</sup> had been used, probably none of the predicted values for forced expiratory volume in one second (FEV<sub>1</sub>) or forced vital capacity (FVC) would have been significantly reduced, even without adjustment for smoking habits. If the equations of Cotes *et al*<sup>9</sup> had been used, certainly none would have been significantly reduced.

In the present paper, Kilburn *et al*<sup>3</sup> compared the pulmonary function of 17 male current smokers with radiographic changes with that of 39 male current smokers without radiographic changes. In their earlier paper, those with radiographic changes were reported to be on average six years older, but this information is not presented again. The bias in the prediction equations for pulmonary function invalidate this comparison.

#### Other issues

The text refers to Botham and Holt<sup>10</sup> as showing that "fibreglass also causes peribronchiolar fibrosis by inhalation." That paper does not mention peribronchiolar fibrosis, and indeed it would be unlikely to do so because the study was primarily concerned with inhalation of glass powder for one day, followed up for one month, with some comparison with the effects of exposure to fibrous glass. Kilburn *et al*<sup>2</sup> failed to reference any of the long term inhalation studies of fibreglass, none of which has shown any evidence that fibrosis is caused by fibreglass exposure.<sup>11 20</sup>

The participants in this study were 284 volunteers from the "500 workers with 20 years of exposure to fibreglass." It is difficult to understand how the average duration of exposure to fibreglass could have been 19.9 years (table 2 from Kilburn *et al*).<sup>2</sup> Was the selection criterion based on duration of employment rather than on duration of exposure?

The non-smokers had higher prevalences of bronchitis and of asthma than did the smokers, with the ex-smokers having the lowest prevalence. This is so different from other studies that a discussion of this would have been appropriate. The only explanation given is that this "may reflect current and ex-smokers

with seniority relocating into jobs with less exposure to fibreglass." This is hardly an adequate discussion. It is also irrelevant if the true selection criterion was based on duration of employment.

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#### The Authors' reply

We would like to reply to Rossiter as follows:

#### No dual publication

Rossiter begins, in his first sentence, with the accusation of dual publication, which is both unkind and wrong. The proceedings of the second California thermal insulation conference were compiled and provided to the participants—not published.

#### No fibrosis from cigarette smoking alone

He proceeds by renewing his contention that Weiss, using minifilms read by magnification, found irregular opacities. Weiss did not use International Labour Office (ILO) criteria for film interpretation, and his work was done in 1972 not 1991 as implied by Rossiter's reference<sup>5</sup> and has not been replicated. Finally he apparently failed to appreciate that silver halide grains may be magnified to haziness in minifilms. The definite study in several thousand subjects with exposure to asbestos showed that smoking apparently enhances the opacities but does not create them.<sup>1</sup>