be entirely due to the association between age and the lifetime odds of having performed ripout.

Asbestos exposure (which was modelled as adjusted years of exposure) was treated differently from fibreglass exposure (ever/never had a high exposure) in the multiple logistic regression analyses. The tables and text indicate that a fibreglass exposure index based on either the adjusted years of fibreglass exposure or none/moderate/high fibreglass exposure would not have indicated any association between fibreglass exposure and chronic bronchitis.

The median duration of exposure in the "high level" fibreglass group is zero years, and 75% of this group had less than one year of experience at the "high level". It is not biologically plausible that such a fleeting exposure is responsible for symptoms of chronic bronchitis.

Work history and exposure modelling are not adequately considered. It is questioned whether the exposure models are truly able to distinguish qualitatively and/or quantitatively between the exposures of asbestos, welding, and fibreglass, given the high degree of correlation among them. No attempt was made to validate the self reported work histories (which are open to recall bias) nor to validate the models of fibreglass and asbestos exposure. More should have been done to validate the exposure modelling assumptions because the paper's conclusions are based on these assumptions (see industrial hygiene comments later).

The overall design of the survey raises important questions about the potential impact of selection bias on the study. This includes the representativeness of the results and the validity of generalising these results beyond the sample.

The survey's results are based on less than 40% of those eligible and invited to participate. It relied on data from a previous medical screening in which only 47% of those invited agreed to participate (12 454 of 26 329 sheet metal workers). Of this, 407 (47%) eligible workers were selected from United States Sheet Metal and Air Conditioning National Association locals in the southeast sun belt and west coast states. Only 333 (82%) of these 407 completed the interviews.

Unanswered, yet most important questions remain. How did survey eligibility criteria affect results? Are there health related selection factors that influenced eligibility—for example, worked in the sheet metal shop for at least 70% of his working career, did removal for at least 40% of his working career, or welding for less than 20% of his working career? What sort of self selection operated over time to eventually impact eligibility, exposure, or health?

An important industrial hygiene consideration and a major issue in this study is the assignment of "high", "medium" and "low" concentration designations. No actual airborne fibre measurements were made of the occupational tasks. Rather, exposures were derived from several published reports. Also, the questionnaire only obtained "average percentage times" spent working in four broad areas of sheet metal work—namely, shop work, welding, job site installation, and ripout. Unless the exposure history is accurate, in terms of the actual work tasks, airborne concentrations, duration of exposure, and other airborne exposures at the work site, any analysis will be of very limited value.

For example, the designation of "high" exposure was given to any fibreglass ripout operation. There were no ripout exposure concentration values referenced. One can not draw analogies from asbestos ripout operations with regard to the amount of fibre fly. A limited amount of sampling data (there is not that much fibreglass torn out) shows that fibreglass ceiling board ripout resulted in airborne fibre exposures with an average of 0·29 fibres/ml for all fibres, using the NIOSH 7400A method (which would be somewhat similar, but not identical to the method used by Balzer *et al*[4] and Fowler *et al*[5]).

When the 7400B method (respirable fibres) was used, total fibre concentration was 0·14 fibres/ml, with further analyses revealing only 0·041 fibres/ml of respirable glass fibres. For pipe insulation ripout the airborne exposure concentrations were 0·126, and 0·046 fibres/ml for all respirable fibres and respirable glass fibres, respectively.[2]

The fibre concentrations reported by Balzer, Copper, and Fowler, as well as being total fibre counts, did not differentiate between glass and other fibrous materials.[3-5] Further, the average airborne fibre diameters were well above the respirable range, suggesting that respirable fibre exposure would be lower.

Using NIOSH 7400B analytical methods, airborne average exposure concentrations for a wide variety of fabrication and installation operations including pipe insulation, range assembly, duct assembly, duct board installation, water heater assembly, and flex duct assembly ranged from 0·006 fibres/ml (duct board assembly) to 0·087 fibres/ml (general fabrication) for all fibres and 0·002 (duct board installation) to 0·071 fibres/ml (general fabrication) for glass fibres. In no instance did the 95th percentile individual concentration exceed 0·12 fibres/ml.[2] These respirable fibreglass exposure concentrations are similar to average concentrations recently noted in insulation wool manufacturing plants (all fibres, 0·03 fibres/ml)[6] and all fibres, 0·025 fibres/ml[7]) Because of these low uniform exposure values, it is not reasonable to divide the sheet metal workers' exposures into high, medium, and low categories.

It then follows that it is difficult to attribute the apparent excess of chronic bronchitis to overexposure to fibreglass. The authors are then faced with the same issues which confronted and confounded Engholm, and Von Schmalensee[3] and Engholm *et al*.[9]

Based on the data presented, the paper's conclusion that high intensity exposure to fibreglass causes chronic bronchitis is unwarranted.

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Authors' reply

Konzen makes a number of criticisms of our study's finding that sheet metal workers with chronic bronchitis were 2·28 times as likely to have performed tasks involving high level fibreglass exposure (that is, ripout of fibreglass materials). We would like to take this opportunity to clarify our methods and provide additional information.

Konzen has a concern about selection bias. He correctly notes that only 47% of invited sheet metal workers participated in the initial medical examinations from which our sample for interview was drawn. For this study, 407 workers were selected from among those 12 454 initially examined, and 333 (82%) completed a telephone interview. Forty of the 74 non-participants were decreased or otherwise lost to follow up; of those actually contacted, 90·7% completed an interview.

To look indirectly at possible selection bias, we compared baseline (medical examination) characteristics of participants and non-participants from this study; the prevalence of chronic bronchitis was 15% in both groups. Notably, the *non-participants* (rather than the participants) had spent significantly more time doing job site installation and ripout work, which generally involve more dust exposure than shop work. Thus it is unlikely that the association between chronic bronchitis and ripout exposures would be biased by participation factors.

Konzen also questions whether our selection criteria may have biased the results. We selected workers who reported at the baseline medical examination doing primarily shop work (>70% of career) or doing ripout for >40% of their careers. These selection criteria were established to obtain a range of asbestos and fibreglass exposure among participants, with shop workers having more fibreglass and less asbestos exposure, and other workers having a variety of exposures, including high level exposures to both substances. We excluded workers who reported welding more than 20% of the time, in order to exclude this exposure as a major confounder. We do not believe that there would have been exposure and health selection factors simultaneously operating among the workers we selected. An example of how such a selection bias could occur would be if workers with lung disease switched from job site installation work to (often) less demanding shop work as they developed symptoms. These workers, however, would not have worked at least 70% of their careers in the shop, and thus would not have been included in this study. We

believe that our selection methods were valid.

Konzen remarks that our final model for risk factors for chronic bronchitis is not age adjusted. We did not expect that age would be associated with chronic bronchitis after accounting for pack-years and occupational dust exposures. In fact, inclusion of age did not improve the fit of this model ($p > 0.90$) and changed the coefficients by less than 1%.

The remaining criticisms are related to our assessment of fibreglass exposure and the treatment of such exposure in the analysis.

It seems from Konzen's comments, that he does not fully understand our task based exposure data. The criticism that ". . .the questionnaire only obtained "average percentage times" spent in four broad areas of sheet metal work—namely, shop work, welding, job site installation, and ripout" is incorrect. In fact, these broad data were obtained on a questionnaire completed at the earlier medical examinations. The purpose of *this* study, done three to four years *later*, was to interview workers regarding their work histories. Participants were carefully queried regarding their percentage time exposure to an extensive set of material specific tasks. Most of these tasks involved fibreglass materials (for example, fabricating or installing fibreglass ductboard) or asbestos materials (for example, cutting transite pipe). There were also a few questions about exposure to welding tasks. Using this material specific task based history, we believe that we obtained qualitatively valid data from study participants that distinguished fibreglass from asbestos exposures. As information on tasks was not available in any historical records, we were unable to validate the self reported work histories.

The concern about recall bias is valid. If recall bias had been present, however, we would have expected to see an increased risk of chronic bronchitis for all types of fibreglass exposures, and not just for ripout exposures.

Konzen expresses concern about the correlations between exposures. As stated earlier, our selection criteria were established to obtain a range of asbestos and fibreglass exposures among participants. We stated in our paper that the correlation coefficient relating total adjusted years of asbestos exposure and total adjusted years of fibreglass exposure was 0·48. We did not detect any problems with multicollinearity in our regression analysis. The presence or absence of high level fibreglass exposure was not highly associated with total years of either fibreglass or asbestos exposure. Welding exposure was not correlated with either fibreglass or asbestos exposure.

Our exposure model employed the assignment of fibreglass related tasks into "high," "medium," and "low" exposure intensity groups. This was difficult because there is little published industrial hygiene data to describe fibreglass dust concentration associated with the tasks that sheet metal workers have performed either recently or historically. As described in the appendix of our paper, we asked six knowledgeable industrial hygienists to independently assign exposure intensity categories for the tasks. Fibreglass ripout received five "high" votes and one "medium" vote. No other task received any "high" votes, except *indirect* exposure to fibreglass ripout, which received one "high" vote.

As adjusted years of low and medium level fibreglass exposure were correlated and not considered separately in the analysis, the real issue of concern should be whether fibreglass ripout entails higher levels of exposure than the tasks that were considered to involve low and medium level exposures. The recent study by Jacobs *et al*[1] referred to by Konzen details industrial hygiene sampling results for a number of manufacturing operations involving glass wool insulation. One of these operations was removal of pipe insulation and ceiling boards. Whether measured as total fibres or as respirable glass fibres, the exposure levels during removal were about an order of magnitude higher than fibre levels for three other tasks about which we queried sheet metal workers: fabrication of ductboard; installation of ductboard; and fabrication of lined ducts (heat seal and duct liner operators). Our industrial hygienist raters assigned these three tasks to the "medium" exposure group. Thus our exposure assignments seem consistent with the results of Jacob *et al*.[1]

A question still remains, though, regarding fibre concentrations experienced during *actual* job site demolition work, where removal of ductboard, lined ducts, and fibreglass insulation may at times create much higher exposures. A recent report published by Johns Hopkins University researchers[2] sampled fibreglass exposures to sheet metal workers and other construction workers under actual field conditions. Unfortunately, they did not include demolition work among the tasks sampled.

Konzen points out that, in the regression model for chronic bronchitis, years of asbestos exposure was treated as a continuous variable whereas fibreglass exposure was treated categorically, distinguishing "high" exposure from lesser intensity exposures. In developing the regression models, duration of both asbestos and fibreglass exposures (all levels) were modelled in four category groups (none, low, intermediate, and high duration). Also, for fibreglass, high intensity exposure was tested for inclusion as a yes or no variable (high and medium level exposures to asbestos were too correlated to permit this.) Results for asbestos showed a monotonically increasing risk as duration increased, so we employed the statistically efficient continuous variable in our final model. For fibreglass, high intensity exposure was a statistically significant predictor of chronic bronchitis, whereas risk did not increase significantly with increasing duration of all fibreglass exposure. We would have liked to determine whether *duration* of high intensity exposure was predictive of chronic bronchitis, but there was insufficient spread in the data to employ this analysis.

Twenty eight per cent of the workers in the chronic bronchitis analysis had spent time doing fibreglass ripout (the only "high level" exposure). More than 80% of these persons with high level exposure had one or two *adjusted* years of high fibreglass exposure, while the remainder had up to seven adjusted years of exposure. Konzen comments that such fleeting exposures could not plausibly cause chronic bronchitis; in so commenting, he may not realise that *adjusted* years of exposure takes into account the average % time that a sheet metal worker performed the task in question. One adjusted year of exposure could result from doing fibreglass ripout work one quarter of the time for four years, 10% time over 10 years, or 2·5% (one hour a week) over a 40 year career. These are not fleeting exposures, and in fact may represent regular performance of tasks over a lifetime of sheet metal work.

We are pleased to have had this opportunity to respond to Konzen's criticisms. In summary, we believe that our conclusion is valid: in this study, high level exposure to fibreglass was associated with a more than doubled risk of chronic bronchitis. Given the lack of appropriate industrial hygiene data, our exposure model was based on *relative*, rather than absolute, exposure levels. Our study cannot consider the question of the concentration levels which engendered this risk. This should be a priority for future researchers.

1 Jacob TR, Hadley JG, Bender JR, Eastes W. Airborne glass fiber concentrations during manufacturing operations involving glass wool insulation. *Am Ind Hyg Assoc J* 1993; 54:320–6.
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Prevalence odds ratio *v* prevalence ratio

Sir,—Cross sectional studies are common in occupational epidemiology, in particular for studying exposure effects on non-fatal diseases (for example, musculoskeletal disorders). The effect measure used when presenting results from a cross sectional study is, in general, either the prevalence odds ratio (POR) or the prevalence ratio (PR). Lee and Chia (1993;50:861–2) have discussed the relative merits of these two effect measures.

Under certain stationarity assumptions on the underlying population, the prevalence odds (PO) is the product of the incidence rate (I) and the mean duration of the illness under study (D): $PO = I \cdot D$.[1] Consequently, the prevalence odds ratio and the prevalence ratio are given by:

$$POR = \frac{I_A D_A}{I_B D_B}$$

and

$$PR = \frac{I_A D_A/(1+I_A D_A)}{I_B D_B/1+I_B D_B)}$$

where I_A and D_A denote the incidence and mean duration, respectively, of a particular illness in a subpopulation classified as A (for example, A = subject to a certain exposure), and I_B and D_B are the corresponding measures in another subgroup of subjects categorised as B.

The table illustrates how the usual effect measures are related to each other under necessary stationarity assumptions. In particular, note that the risk odds ratio (OR), which is the effect measure used in a case-control study with "cumulative incidence" sampling,[2] does *not* equal the POR. Also note that the PR *neither* equals the relative risk *nor* the incidence rate ratio.

Lee and Chia argued that the PR is preferable to the POR, because the PR is "easy to interpret and to communicate", whereas the POR "lacks intelligibility". In