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 researchers² 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.

2 Corn M, Lees PSJ, Breyse PN. Characterization of end-user exposures to commercial insulation products. Johns Hopkins University, Baltimore, MD: 1992. (Final report for Thermal Insulation Manufacturers Association.)

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

Table Comparison of relative risk (RR), risk odds ratio (OR), incidence rate ratio (IRR), prevalence odds ratio (POR) and prevalence ratio (PR). An index (A or B) refers to a specific subpopulation. The hypothetical populations are assumed to fulfil necessary stationarity assumptions.

R_A^*	$R_B^\dagger$	RR	OR	IRR	$D_A^\ddagger = D_B^\S = 1$		$D_A = D_B = 4$		$D_A = 4; D_B = 1$	
					POR	PR	POR	PR	POR	PR
0.60	0.50	1.20	1.50	1.32	1.32	1.17	1.32	1.07	5.29	1.92
0.60	0.30	2.00	3.50	2.57	2.57	1.82	2.57	1.34	10.3	2.99
0.60	0.10	6.00	13.5	8.70	8.70	5.02	8.70	2.65	34.8	8.24
0.40	0.10	4.00	6.00	4.85	4.85	3.55	4.85	2.26	19.4	7.04
0.20	0.10	2.00	2.25	2.12	2.12	1.92	2.12	1.59	8.48	4.96
0.20	0.05	4.00	4.75	4.35	4.35	3.73	4.35	2.77	17.4	9.65

* R_A = Risk of developing illness in subpopulation A during 1 time unit.

† R_B = Risk of developing illness in subpopulation B during 1 time unit.

‡ D_A = Mean duration of illness in subpopulation A (time units).

§ D_B = Mean duration of illness in subpopulation B (time units).

their notation, prevalence rate ratio (PRR) is used instead of prevalence ratio (PR), which seems confusing, as prevalence and rate are different concepts. The fundamental flaw in their argumentation, however, is that they place, in some respects, cross sectional studies on an equality with longitudinal studies by considering a PR as a relative risk and a POR as a risk odds ratio when comparing the effect measures; as is seen in the table, this is certainly not true. Contrary to their conclusion, by estimating the ratio of the mean durations, D_A/D_B , a POR can easily be converted into an incidence rate ratio (under certain stationarity assumptions),¹ whereas a relation between the PR and some aetiologically understandable effect measure may be more difficult to see through.

Moreover, Lee and Chia's description is imperfect in another respect: the reader is left with the impression that the POR is the only effect measure possible to estimate under a logistic regression model; if a PR is desired instead, it can always be obtained from the estimated probabilities of study illness for different covariate patterns, based on the model.³

It should be stressed that restricted stationarity assumptions underlie the derivation of the mentioned relations between prevalence, incidence, and duration and hence limit the applicability of these known relations. As far as I know, there are no empirical studies that show to what extent departures from these assumptions may influence the effect estimates. In practice, the underlying mechanisms that affect the outcome of a cross sectional study are complex. Hopefully, recent theoretical work^{4,5} will somehow improve our ability to analyse and interpret data from cross sectional studies.

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- 1 Feeman J, Hutchison GB. Prevalence, incidence and duration. *Am J Epidemiol* 1980; 112:707-23.
- 2 Greenland S, Thomas DC. On the need of the rare disease assumption in case-control studies. *Am J Epidemiol* 1982;116:547-53.
- 3 Hosmer DW, Lemeshow S. *Applied logistic regression*. New York: Wiley, 1989.
- 4 Keiding N. Age-specific incidence and prevalence: A statistical perspective. *Journal of the Royal Soc A* 1991;154:371-412.
- 5 Alho JM. On prevalence, incidence, and duration in general stable populations. *Biometrics* 1992;48:587-92.

NOTICES

Reminder

Symposium on health hazards of glycol ethers. 19-21 April 1994, Abbay de Port-à Mousson, Nancy, France

(More details appear in *Br J Ind Med* 1993;50(November):1056) For further information contact the Symposium Secretariat, International Symposium on Health Hazards of Glycol Ethers, INRS, Avenue de Bourgogne BP 27 F54 501 Vandoeuvre Cédex France. Tel (33) 83 50 20 27, Fax (33) 83 50 20 96.

Fourth International Conference on Education and Training in Occupational Health, 24-28 April 1994, Amsterdam, The Netherlands.

The conference is organised by the Amsterdam School of Occupational Medicine, Corvu and will take place in the buildings of the Universiteit van Amsterdam. It aims at those involved in teaching professionals in the field of occupational health, safety, and wellbeing. The scientific programme consists of oral presentations, workshops, poster sessions, and, as a new element, demonstration lessons. It is built up along two lines. Firstly, the establishment, performance, and evaluation of an education and training programme. Secondly, the establishment, performance, and evaluation of an occupational health and safety programme in a company. During the whole conference there will be an information market and a sponsor market. The fee is DFL 820 (members of ICOH may register for DFL 780).

For more information, contact the Conference Office, Universiteit van Amsterdam, PO Box 19268 1000 GG Amsterdam, The Netherlands, fax +31-20-5252771 or email congres@bdu.uva.nl.

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Correction

Radiographic abnormalities and mortality in subjects with exposure to crocidolite (1993;50:902-906).

During printing fig 2 (p 905) was inadvertently changed. The correct fig 2 is given here: