Correspondence

Welding 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 time” 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 fully 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 removing fibreglass ductboard). 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.

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 sampled fibreglass exposures to sheet metal workers and other construction workers (for example, drywall installers). 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 indicator 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 effective but 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 high level 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 percent of the workers in the chronic bronchitis analysis had spent time doing fibreglass ripout (the only “high level” exposure) compared to 10% 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.


Prevalence odds ratio vs 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 such 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= ID. Consequently, the prevalence odds ratio and the prevalence ratio are given by:

\[ \text{POR} = \frac{I_D}{I_D} \]

and

\[ \text{PR} = \frac{I_D/(1+I_D)}{I_D/(1+I_D)} \]

where \( I_D \) and \( D \) 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 \( I_A \) and \( D_A \) 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, the risk odds ratio (POR), which is the effect measure used in a case-control study with “cumulative incidence sampling," 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
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 PRR 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 PRR can easily be converted into an incidence rate ratio (under certain stationarity assumptions), 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 PRR is the only effect measure possible to estimate under a logistic regression model; if a PRR is desired instead, it can always be obtained from the estimated probabilities of study illness for different covariate patterns, based on the table.°

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 will somehow improve our ability to analyse and interpret data from cross-sectional studies.

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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 fulfill necessary stationarity assumptions.

<table>
<thead>
<tr>
<th>( R_A )</th>
<th>( R_B )</th>
<th>RR</th>
<th>OR</th>
<th>IRR</th>
<th>POR</th>
<th>PR</th>
<th>POR</th>
<th>PR</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.60</td>
<td>0.50</td>
<td>1.20</td>
<td>1.50</td>
<td>1.32</td>
<td>1.07</td>
<td>1.07</td>
<td>1.32</td>
<td>1.32</td>
</tr>
<tr>
<td>0.60</td>
<td>0.30</td>
<td>2.00</td>
<td>3.50</td>
<td>2.57</td>
<td>1.82</td>
<td>2.57</td>
<td>1.34</td>
<td>1.34</td>
</tr>
<tr>
<td>0.60</td>
<td>0.10</td>
<td>6.00</td>
<td>13.5</td>
<td>8.70</td>
<td>5.02</td>
<td>8.70</td>
<td>2.65</td>
<td>2.65</td>
</tr>
<tr>
<td>0.40</td>
<td>0.10</td>
<td>4.00</td>
<td>6.00</td>
<td>4.85</td>
<td>3.55</td>
<td>4.85</td>
<td>2.26</td>
<td>2.26</td>
</tr>
<tr>
<td>0.20</td>
<td>0.10</td>
<td>2.00</td>
<td>2.25</td>
<td>2.12</td>
<td>1.92</td>
<td>2.12</td>
<td>1.59</td>
<td>1.59</td>
</tr>
<tr>
<td>0.20</td>
<td>0.05</td>
<td>4.00</td>
<td>4.75</td>
<td>4.35</td>
<td>3.73</td>
<td>4.35</td>
<td>2.77</td>
<td>2.77</td>
</tr>
</tbody>
</table>

\( R_A = \text{Risk of developing illness in subpopulation A during time unit.} \)

\( R_B = \text{Risk of developing illness in subpopulation B during time unit.} \)

\( D_A = \text{Mean duration of illness in subpopulation A (time units).} \)

\( D_B = \text{Mean duration of illness in subpopulation B (time units).} \)

NOTICES

**Reminder**

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


**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, Corvus 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 congress@bdu.uva.nl.

**NEW BOOK ANNOUNCEMENTS**

**Health promotion in the Workplace: Alcohol and Drug Abuse**


In developing countries: Sw fr 4-90. Order No 1100833.

**Electromagnetic Fields (300 Hz-300 GHz)**


In developing countries: Sw fr 23-80. Order No 1160137.

**1,3-Dichloropropene, 1,2-Dichloropropene and Mixtures**


In developing countries: Sw fr 21-70. Order No 1160146.

**Methyl Parathion**


In developing countries: Sw fr 19-60. Order No 1160145.

**Correction**

Radiographic abnormalities and mortality in subjects with exposure to crocidolite (1939;50:902-906). During printing fig 2 (p 905) was inadvertently changed. The correct fig 2 is given here:
Prevalence odds ratio v prevalence ratio.

U Strömberg

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