CORRESPONDENCE

Use of the prevalence ratio *v* the prevalence odds ratio as a measure of risk in cross sectional studies

Author's reply

Editor—Our letter pertaining to the proper measure of exposure-response association for cross sectional data[1] elicited comments and elaborations from Stromberg[2] and Axelson and colleagues.[3] Stromberg made several points. Firstly he objected to our calling the measure prevalence rate ratio (PRR) rather than prevalence ratio (PR). We do recognise that the prevalence rate is not a rate but a proportion. Indeed we underscore this very point elsewhere.[4] We called it a "rate" in the letter simply to follow convention as many books still refer to it as a rate. Stromberg asserted that we regard the cross sectional study on an equal footing with the longitudinal study for causal inference. A cursory look at our letter will show that that is not so. Our letter was very explicit that the cross sectional study is clearly inferior to other epidemiological designs for aetiological research. This is what we said: "In aetiological research, especially if the latent period (interval between exposure and occurrence of disease) is protracted and ill defined, a cross sectional study can only be used to assess a statistical association between exposure and a physiological state, leaving causal inference to an appropriate epidemiological design such as a prospective cohort or retrospective case-control that incorporates the time dimension". We are baffled by Stromberg's claim that the "relation between the PR and some aetiologically understandable effect measure may be more difficult to see through". A cursory look at the definition should show that PR is in fact highly intelligible whereas prevalence odds ratio (POR) is virtually incomprehensible.[5] As emphasised by Savitz[6] an effect measure must not only convey the most germane information, but it must also be easy to communicate and to comprehend. As such, the POR has no direct usefulness except as a numerical mimic to other effect measures.[7] In contrast to Stromberg's claim, the logistic regression model can only estimate the odds ratio (by exponentiating the logistic regression coefficient) and its standard error. As we have shown elsewhere[8] the logistical model can be used to obtain model predicted probabilities, and from these probabilities the rate difference and rate ratio can then be determined. But the standard error for these measures is not available. In contrast, Cox's regression gives PR and its standard error.[1] In short we have found nothing in Stromberg's letter that is a valid criticism to detract from the preference of PR over POR, or the preference of Cox's regression over logistic regression, for cross sectional data.

Axelson and colleagues[3] have brought out additional insightful merits of PR over POR, and of Cox's regression over logistic regression for cross sectional data. Their exhortation about the undesirable consequence of the current trend towards an uncritical and indiscriminate use of logistic regression for cross sectional data should be taken seriously.

JAMES LEE
KS CHIA
Department of Community, Occupational, and Family Medicine,
National University of Singapore, NUH,
Lower Kent Ridge Road, Singapore 0511

1 Lee J, Chia KS. Estimation of prevalence rate ratios for cross sectional data: an example in occupational epidemiology. *Br J Ind Med* 1993;50:861–2.
2 Stromberg U. Prevalence odds ratio *v* prevalence ratio. *Occup Environ Med* 1994;51:143–4.
3 Axelson O, Fredriksson M, Ekberg K. Use of the prevalence ratio *v* the prevalence odds ratio as a measure of risk in cross sectional studies. *Occup Environ Med* 1994;51:574.
4 Lee J. Exact confidence interval of the SMR based on prevalence data. *Int J Epidemiol* 1994;23:428–9.
5 Rothman KJ. *Modern epidemiology.* Boston: Little Brown, 1986.
6 Savitz DA. Measurements, estimates and inferences in reporting epidemiologic study results. *Am J Epidemiol* 1992;135:223–4.
7 Lee J. Odds ratio or relative risk for cross-sectional data? *Int J Epidemiol* 1994;23:201–3.
8 Lee J. Regression estimate of the adjusted risk ratio. *Br J Ind Med* 1986;43:356–7.

Airways obstruction, coal mining, and disability

Editor—There are several points of general interest in this paper,[1] but space considerations limit how many can be discussed.

The authors repeatedly refer to the interpretation of chest radiographs by their readers in the context of their use of the International Labour Organisation (ILO) classification of appearances in the chest radiograph in the pneumoconioses. Readers should know that the ILO classification is a descriptive not an interpretative scheme. This is the essence of its use as an epidemiological tool. The use of the ILO scheme for epidemiological purposes requires more than simple consensus reading by several readers. Conditions under which films are read and scored, and the methods of producing a single score for a film in which a number of readers reading independently have produced a range of readings for opacities and profusions, require to be standardised and specified for serious epidemiological study. The possible use of the classification by clinicians to standardise descriptions was suggested early in its development. The ILO rubric, however, has not defined clinical pneumoconiosis, but looks upon the profusions of small radio-opacities in the chest radiograph as a continuum. It seems that coal workers' pneumoconiosis (CWP) was defined by the authors from the consensus clinical interpretations found in the claimants' files, with those readers incapable of reading any of the radiographs as normal being censored. How the readers came to the conclusion that CWP was or was not present is not given.

The text states that: ". . . clinically significant reduction of ventilatory capacity was present when the forced expiratory volume in one second (FEV_1), the forced expiratory volume (FVC), or the ratio of the FEV_1 to the FVC ($FEV_1/FVC\%$) was 60% or less". (Subsequently, table 1 omitted FVC as a criterion of significant impairment, which is consistent with the title which refers to airways obstruction.) If clinical impairment is determined from physiological measurement, what is the appropriate predicted value for workers whose jobs have entailed vigorous physical effort? When fit, studies on such populations would be expected to show a ventilatory function distribution bias to the pluperfect compared with a "standard" population. By the time members of such populations have regressed to 60% of the conventional predicted value, there will have been a large absolute loss of function. (When such people reach 100% of predicted values and have sustained a substantial loss of function, how appropriate is it to consider it not to be of clinical significance?).

Readers might conclude that after the inhalation of coal dust in the absence of progressive massive fibrosis, effects may be found that are of no consequence. The Institute of Occupational Medicine, Edinburgh, mortality study of its population of UK coal miners indicated that one cannot be sanguine in the presence of simple pneumoconiosis.[2]

The concluding paragraph in the text enters the field of political controversy in making the unsupported claim that compensation for misattributed disease under the "Black Lung scheme", contributed to the closure of most of the Appalachian coal mines. When the price of oil rises again, alternative supplies of coal will be available from sources where the fatalism of the miners permits cheap extraction. Concerned Americans refer to this alternative sourcing as exporting diseases.

MORRIS GREENBURG
74 North End Road,
London NW11 7SY.

1 Lapp NL, Morgan WKC, Zaldivar G. Airways obstruction, coal mining, and disability. *Occup Environ Med* 1994;51:234–8.
2 Miller BG, Jacobsen M. Dust exposure, pneumoconiosis, and coalminers' mortality. *Br J Ind Med* 1985;42:723–33.

Editor—Lapp and colleagues[1] suggest that their study of 611 benefit claimants shows that exposure to coal dust is rarely if ever a cause of disabling airflow obstruction. They miss the crucial point that in studying a group of people almost all of whom have been exposed to two causes of a disease and in whom there are no data to quantify exposure, it is not possible to differentiate between the effects of the two causes. Would they, from a different bias, be happy with the conclusion that in the absence of dust exposure smoking is rarely a cause of disabling airflow obstruction in miners?

The evidence that smoking and coaldust exposure have an additive effect in causing emphysema and airflow obstruction has been amply aired in this journal,[2] and the emphysema-dust relation has again recently been clearly shown from Australia.[3] The evidence has been accepted by the British Industrial Injuries Advisory Council, and coalminers with adequate evidence of dust