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 a cross-responsive association for cross sectional data1 elicited comments and elaborations from Stromberg2 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 disagree with the latter’s comment that the letter should 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 Cox’s regression analysis would show that this is not so. Our letter was very explicit that the cross sectional study is clearly inferior to other epidemiological designs for etiological research. This is what we said: “In epidemiological 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 time dimension”. We are baffled by Stromberg’s claim that the “relation between the PR and some aetiologically understandable effect measure may be more directly to see in the graph”. 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 Savitz6 an effect measure must not only convey the most germane information, 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 elsewhere8 the logistic 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 model can determine PR and its standard error.9 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 colleagues1 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.

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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 one and not a disease 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 each individual and professions, 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 profusion of small radio-opacities in the chest radiograph as a continuum. It seems that coal workers’ pneumoconiosis (CWP) workers differ 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 (FEV1), the forced expiratory volume (FVC), or the ratio of the FEV1 to the FVC (FEV1/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 the same as that are of black lung. The Institute of Occupational Medicine, Edinburgh, mortality study of its population of UK coal miners indicated that one cannot equate sanguine in the presence of simple pneumoconiosis.1

The concluding paragraph in the text enters the field of political controversy in making the unsupported claim that pneumoconiosis of 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 and resources where the fatality of the miners permits cheap extraction. Concerned Americans refer to this alternative sourcing as exporting diseases.

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Editor—Lapp and colleagues1 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 coal dust exposure have an additive effect in causing emphysema and airflow obstruction has been amply aired in this journal,1 and the emphysema-dust relation has again recently been clearly shown from Australia.1 The evidence has been accepted by the British Industrial Injuries Advisory Council, and coalminers with adequate evidence of dust exposure from coal are eligible for compensation for work-related lung disease in the UK.2

exposure are now eligible for benefits for impaired lung function. In Lapp and colleagues’ study the important question, unanswerable from their design, is how many of those with airflow obstruction (OA) would not have had this impairment had they not been exposed to coal dust. It is this difficult decision that, in law, is made on the balance of probabilities and where it is necessary to argue from the results of epidemiological studies to the particular cases of sick people.

It is to be noted that 50%, at least, of Lapp and colleagues’ subjects were referred to them by employers’ representatives. It would be unfortunate if their paper were to mislead these people into thinking that it shows anything other than that coalminders who claim benefits usually smoke.

Author’s reply

With reference to Greenberg’s comments concerning the International Labour Office (ILO) classification of chest radiographs for pneumoconiosis, the classification’s usefulness in epidemiological studies of coal workers’ pneumoconiosis depends on the direct relation between the coal content of the lung and the radiological category. A major concern of epidemiologists is the number of false positives and negatives that occur with the use of diagnostic tests. Based on the number of false positives and negatives it is possible to calculate the test’s predictive value; this being the best index of its usefulness. There is little point in quantifying opacities when they are the signs of the inhalation of coal dust! The handbook that accompanies the standard ILO films states “If it is at all probable that all the appearances seen are the result of some other aetiology (ie not dust related) do not classify, but record opinion using appropriate symbols and comments”.

Greenberg comments on the need for a series of independent readings for epidemiological studies. Thirty of the readers in our most recent study have been involved in epidemiological studies of coal workers’ pneumoconiosis (CWP) and other pneumoconioses, some of which were jointly carried out with the National Coal Board (now British Coal). One of us has participated in studies with the originator of the elaboration of the ILO classification, FDK Liddell. We are therefore well aware of his thoughts on the application of the ILO classification in epidemiological investigations. Moreover, Greenberg should be aware of a series of papers published some years ago by our colleague Reger and his coworkers, which examined epidemiological problems encountered in the interpretation of chest x-ray films and appropriate methods for reading progression of the chest x-ray films in diseases related to dust.

We were also asked why we ignore certain readers positive interpretations. This is because they differ so consistently and so greatly from the readings of the United States Public Health Service panel of readers. It is difficult not to conclude that their opinion is influenced by whom they were retained.

Greenberg might also consider furthering his crusade against the inappropriate use of the ILO classification by calling Peter Lilley’s attention to the fact that the Department of Social Security is going to rely on the radiographic presence of CWP in making awards to British coal miners with airways obstruction. This in itself indicates that most workers in the coal mines of the United States are not by means arduous, yet since the miners have been mechanised, an event that took place in the 40s and 50s. Similar studies have been carried out by Haber et al that indicate LOMs despite significant impairment, manage to do their jobs without serious problems. Although disableness depends to some extent on loss of lung function, it is also influenced by the age, sex, and the height of the worker. Thus a 5 ft slender woman of 40 years with normal lung function could not be expected to carry a hundredweight of coal on her back, a 6 ft man aged 26 who has had a lobectomy with residual lung function should be able to manage this without much trouble. The ability to perform a certain task is not related to a specific divergence of lung function from the predicted value, but to the residual lung function that remains.

Greenberg suggests that “we should not be sanguine in the presence of simple CWP”. The diagnosis of CWP and the subsequent mortality in the study he quotes were based on the interpretation of the earliest x-ray films. Many men in the study continued to work for a further 15 to 25 years. What was read as category 1/2 in 1956 became category 2 stage B by 1978.

In reply to Seaton, we accept that emphysema is found more frequently in coal miners with pneumoconiosis than it is in the general population, but it hardly seems necessary to cite recent work from Australia when this point was made 30 to 40 years ago by Gough1 and also in the first edition of a text of which Seaton was a co-editor. What is at issue is whether the diagnosis of emphysema found in non-smoking miners is associated with airways obstruction. The question of how frequently emphysema occurs in non-smoking miners is put in perspective in an Institute of Occupational Medicine monograph that was published during the time Seaton was the Institute’s Director. The text reads, “This suggests that nonsmokers with the highest lung exposure in British collieries have a lower risk of developing centriacinar emphysema than a smoker with minimal dust exposure or a non-smoker without this view, as it seems does Ruckley and coworkers,” that coal miners have slightly more airways obstruction than does a comparable control group, but we maintain that distinguishing airways obstruction in non-smoking coal miners is exceedingly uncommon and can seldom, if ever, be attributed to dust alone in the absence of progressive massive fibrosis.

Seaton poses the question, “How many of those with airflow obstruction would not have been obstructed had they not been exposed to coal dust?” He asked the question slightly differently and asked how many miners with a history of smoking had not smoked, the answer would have been none that we could identify. Thus of the 9076 miners included in the first round of the national coal study who were over 50 years of age, 5403 miners met the Department of Labor’s (DOL) disability criteria whereas 25-3% of a similar non-smoking population qualified.

Seaton states that at least 50% (we wrote in our reply around 50%) of those referred to us by employers’ representatives and although this is true it needs to be pointed out that over 90% of the claimants had previously been referred for disability evaluation to the Department of Labor at one or other of the several medical schools in Appalachia. Referral was not predicated on the fact that they were smokers. If airways obstruction is as common in British coal miners as Seaton suggests, then this is related to the fact that only 13-2% of the population of the Pneumoconiosis Field Research Coal Board Study were lifelong non-smokers whereas 20% of the United States National coal study of miners were lifelong non-smokers.1


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Occup Environ Med 1994 51: 841-842
doi: 10.1136/oem.51.12.841-b