CORRESPONDENCE

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

The letter by Lee and Chia (1993;50: 861-2) is a welcome discussion of the debatable but increasing use of the prevalence odds ratio as a quantified measure of association in cross sectional studies even when the disease is far from rare. In a cross sectional study that involves the entire study population (the study base) of healthy and unhealthy subjects, the perception of a health risk from an exposure is usually founded in the relative occurrence of disease among the exposed and non-exposed subjects. This also means that a health risk may be well appreciated by means of the prevalence ratio. In general, the cumulative incidence ratio or the risk ratio would be the preferred epidemiological measure of risk, although these require a cohort approach rather than cross sectional data.

Given a similar duration of the disease among exposed and non-exposed subjects, the prevalence odds ratio represents the incidence density ratio, which might be used for case-control studies in open (dynamic) populations, even when the disease occurs commonly.1 The requirement for a similar duration of the disease among the exposed and non-exposed subjects for the prevalence odds ratio to be interpreted as an incidence density ratio is not always fulfilled. There has still been little or no consideration of this requirement in the reports of studies based on prevalent cases and non-cases. Given a similar duration of a rare disease, however, the difference between the prevalence odds ratio and the prevalence ratio would be negligible.

The interpretational problems with the prevalence odds ratio in cross sectional studies are perhaps most clearly seen in studies of ergonomic work load and common disorders, such as backache, and neck and shoulder complaints. The duration of such disorders is not unlikely to be influenced by the exposure, but this possibility has been rarely accounted for. Prevalence odds ratios in this context do not seem intelligible for risk even if they indicate associations.

For example, in a meta-analysis of cross sectional studies of ergonomic factors and median nerve entrapment, prevalence odds ratios of about 10 or more were obtained, but disturbed nerve function was as high as 28% in one reference group.² To take another example, high_prevalence odds ratios were reported in a study of osteoarthrosis in the acromioclavicular joint among employees in the construction industry.3 Foremen were taken as the reference group, but they had a high frequency of developed disease, 36.7% on the right side and 23.4% on the left.

The use of the prevalence odds ratio seems to be even more of a problem in the now emerging field of molecular epidemiology. Hence, the prevalence of a certain type of mutation in an oncogene, or in a tumour suppressor gene, might be found to differ between those with and without a particular exposure. The resulting prevalence odds ratio may be found to be considerably increased,4 but the finding is without any clear interpretation in terms of risk. Instead, the proportional aspect as reflected in the prevalence ratio seems to be a more intelligible measure of effect. Even more correct and informative would be to apply a casecontrol design with an adequately selected control group reflecting the exposure frequency in the base population⁵⁶; sometimes different approaches can be found.4

Despite the interpretational problems affecting the prevalence odds ratio, the readily available computers and statistical packages for epidemiological analyses seem to have favoured an interest for computing prevalence odds ratios in cross sectional studies instead of the more correct prevalence ratios. The odds ratio approach seems especially common when there is a need for convenient adjustment for confounding by use of logistic regression.

In cross sectional studies due consideration should not only be given to the fact that the prevalence odds ratio is a poor measure of risk as influenced by differences in duration of disease between exposed and non-exposed subjects. A different pattern of confounding is also present when considering the prevalence ratio compared with the prevalence odds ratio. This means-for example, that the use of the prevalence odds ratio implies confounding even when the study base is unconfounded in terms of prevalence data. Usually the situation is even more complex with some confounding irrespective of which ratio is used, but the result of controlling for confounding would lead to different effects on the estimates of the prevalence ratio and the prevalence odds ratio.

The table is a simple illustration of this, where the various estimates are derived from hypothetical, unconfounded sets of cross sectional data with another determinant of risk also present. This other factor is taken to have an equally strong effect as the determinant of interest (the exposure). The adjustment of the prevalence odds ratio by stratified (Mantel-Haenszel) analysis or logistic regression tends to give a result that is even further away from the prevalence ratio than the crude prevalence odds ratio. The reason for this phenomenon is that the proportion of still unaffected (healthy) subjects gets reduced and so poorly represents the cross sectional study base, especially at higher prevalences and strong effects by the two determinants. In contrast, when considering the prevalence ratio, there is no confounding and consequently no change in the prevalence ratio calculated either crudely, by stratification and the Mantel-Haenszel procedure, or by the Cox regression (suggested by Lee and Chia).

Considering the interpretational difficulties of the prevalence odds ratio, its sensitivity to duration of the disease and the aspects illustrated with regard to confounding, it seems as if the current development towards a more or less uncritical use of logistic regression analyses to obtain an adjusted prevalence odds ratio in analysing cross sectional data is hardly desirable. Instead the prevalence ratio and an analysis by means of a proportional hazards model (Cox regression) to control for confounding seems to be more appropriate, as suggested by Lee and Chia.

OLAV AXELSON MATS FREDRIKSSON KERSTIN EKBERG Department of Occupational and Environmental Medicine, University Hospital, 581 85 Linköping, Sweden

Table 1 Comparisons of prevalence ratios (PRs) and prevalence odds ratios (ORs) from hypothetical cross sectional data at different levels of risk, although equal in terms of the PR for the exposure and another factor. This other factor is taken to occur in 1/3 and 2/3 of the populations. No confounding is present for the PRs but it does occur in the OR calculations; hence, the crude PR equals the Mantel-Haenszel and the Cox PRs.

Background prevalence %	Mantel- Haenszel PR	Cox PR	Crude OR	Mantel- Haenszel OR	Logistic OR
1/3 with other factor:					
5	2.0	2.0	2.2	2.2	2.2
	3.0	3.0	3.7	4.0	4 ·0
	4.0	4.0	6.0	8.5	9.7
10	2.0	2.0	2.4	2.4	2.4
	3.0	3.0	5.0	6.9	7.8
2/3 with other factor:					
5	2.0	2.0	2.2	2.2	2.2
	3.0	3.0	4.1	4.4	4.4
	4.0	4.0	8.5	12.3	13.5
10	2.0	2.0	2.5	2.6	2.6
	3.0	3.0	7.7	11.8	13.6

- Axelson O. Elucidation of some epidemiologic principles. Scand J Work Environ Health 1983;9:231-40.
 State D. W. State and State an
- 2 Stock SR. Workplace ergonomic factors and Stock SR. workplace ergonomic factors and the development of musculoskeletal dis-orders of the neck and upper limbs: a meta-analysis. Am J Ind Med 1991;19:87-107.
 Stenlund B, Goldie I, Hagberg M, Hogstedt C, Marions O. Radiographic osteoarthrosis in the acromioclavicular joint resulting from
- work or exposure to vibration. Br f Ind Med 1992;49:588-93.
 4 Taylor JA, Sandler DP, Bloomfield CD, Shore DL, Ball ED, Neubauer A, et al. ras Oncogene activation and occupational expo-sures in accure myaloid leukaemia ? Natl.
- Oncogene activation and occupational exposures in acute myeloid leukaemia. *J Natl Cancer Inst* 1992;84:1626-32.
 5 Axelson O, Söderkvist P. Characteristics of disease and some exposure considerations. *Appl Occup Environ Hyg* 1991;6:428-35.
 6 Wei Q, Matanoski GM, Farmer ER, Hedayati MA, Grossman I. DNA renair and sains in the s
- MA, Grossman L. DNA repair and aging in basal cell carcinoma: a molecular epidemiol-ogy study. *Proc Natl Acad Sci USA* 1993; 90:1614–8.