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have become obvious with this approach. The exposure-response relation was for active miners. Former miners could be unsuitable, because of a possible disappearance of dysplasia in a proportion of them, thus making the interpretation of the results unclear. In our cohort both smokers and non-smokers showed exposure-response relations, but the group of smokers showed relatively uniform smoking habits (about two thirds of them smoke one pack/day). This could not be expected in every case. Different smoking habits may result in different dysplasia outcomes. Therefore, it is our belief that the non-smokers group gives a better opportunity for the retrospective exposure assessment. In either case, to avoid possible strong differences due to the different way of life, smoking habits,[15] or individual sensitivity of miners in different areas of the world, a preliminary investigation of a group analogous to our group A may be necessary for reliability in such studies.

Conclusion

We conclude that exposure of underground miners to ^{222}Rn progeny results in a significantly increased frequency of squamous cell metaplasia. At the level of notable dysplasia, this frequency follows an exposure-response relation. Sputum cytology could be used for a retrospective assessment of the range of exposures for groups for which this range could not be assessed directly. In the arrangement of such studies the limitations of this approach should be recognised. Further investigations of metaplasia in miners are needed to clarify the exposure-response relation under different conditions.

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2 Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;**294**:687–90.
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significantly related to cumulative exposure to respirable fibres.[3] Skin irritation was related to exposure to both inspirable dust and respirable fibre, but there was an additional independent effect of exposure to fibres.

The changes in lung function are much more strongly related to cumulative exposure to fibres than to exposure to inspirable mass, the effects of inspirable mass become trivial after adjustment for exposure to fibres. Reductions of FEV_1 are confined to smokers, with no effect at all in life long non-smokers. This suggests that the fibres themselves are not directly detrimental to airflow, but promote such effects of cigarette smoke. In summary symptoms related to exposure to both inspirable dust and respirable fibres, and the decrements of FEV_1 seen in smokers are related to the respirable fibre constituent of the exposure.

We thank the European Ceramic Fibre Industries Association (ECFIA) and its scientific committee for supporting this study, and the many staff, employees, and doctors within each plant who provided invaluable assistance with the health and hygiene surveys. We are very grateful to Mr J Dodgson and Dr J Cherrie of the Institute of Occupational Medicine, Edinburgh for their collaboration in undertaking the simultaneous plant hygiene surveys and for their helpful advice. We also thank Dr Alastair Robertson, Dr Birge Berns, and the many others who helped us with the plant surveys both in the United Kingdom and abroad.

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CORRESPONDENCE

Prevalence odds ratio *v* prevalence ratio—some further comments

Editor,—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,[1] Strömberg,[2] Axelson *et al*,[3] and Lee[4] discuss the pros and cons of these two effect measures. I would like to give some further comments on this issue.

Axelson *et al* present hypothetical examples to show that the use of the POR may imply "confounding even when the study base is unconfounded in terms of prevalence data".[3] I think that their description is somewhat misleading. As in their example, consider a dichotomous exposure and another dichotomous factor, F, which both affect the prevalence of the study disease. Assume that the fraction of exposure does not depend on F, so F is not a confounder.[5] Axelson *et al* use hypothetical data, which when stratified on F, produce stratum specific PRs equal to the crude PR and, of course, the adjusted PR as well, whereas the stratum specific PORs differ from the crude PORs and hence the adjusted POR equals a value between those two PORs; this occurs because the exposure specific prevalence ratios with respect to the other factor F coincide. One can also construct an example where the stratum specific PRs differ, whereas the stratum specific PORs are equal; this occurs when the exposure specific PORs for F coincide (table). In that case, the adjusted PR is between the stratum specific PRs, whereas the stratum specific and adjusted PORs are equal, although the crude and adjusted POR may be different. To sum up in other words, these examples show that F may modify the effect of exposure without being a confounder in the conventional meaning; moreover, F may modify the POR and not the PR, and vice versa. Note that, when F does not influence the fraction of exposure, the stratum specific PORs can be equal to each other and still differ from the crude POR (table), whereas this cannot happen when the PR is the effect measure of interest. Effect modification can be examined in the analysis of the data.[5 6]

From an aetiological point of view it is often desirable to estimate effects of exposure on incidence of disease. It is sometimes possible to obtain incidence based effect estimates from cross sectional data. For example, under certain stationarity assumptions, a POR can be converted into an incidence ratio.[5] The association between prevalence and incidence is derived from a complex theory that is based on more or less restrictive assumptions.[7 8] Most commonly, investigators who apply a cross sectional study design focus on exposure effect on prevalence rather than incidence, as such effect can be directly estimated from cross sectional data. If prevalence is the disease measure at issue, one may argue that the PR is easier to interpret than the POR (Axelson *et al*[3]). On the other hand, I do not think that the POR lacks intelligibility (Lee and Chia[1]); instead of reflecting the ratio of two prevalences, it simply reflects the ratio of two prevalence odds. Furthermore, from a statistical point of view, the POR is preferable to the PR (explained later).

Lee and Chia as well as Axelson *et al* apply Cox's proportional hazards model for estimating an adjusted PR.[1 3] To use a statistical model for estimation, it is fundamental to know what type of dependent parameter the model involves. As is well known, the dependent parameter of Cox's proportional hazards model corresponds to intensity (hazard) and the one of the logistic regression model corresponds to probability. Because prevalence is probability and not intensity, Lee and Chia advocate the use of Cox's proportional hazards model by assuming "constant follow up time".[1] They claim that the effect estimate from Cox's model then approximates the relative risk (Lee and Chia use the term rate ratio,[1] whereas Lee[4] uses the term cumulative incidence ratio) by referring to Breslow's paper,[9] which considers censored survival data. Except for the fact that risk as well as prevalence corresponds to probability, their reasoning is confusing: for example, the assumption "constant follow up time" has no clear meaning in a cross sectional study and the relation between prevalence and incidence (incidence corresponds to intensity) is not the same as the one between risk and incidence. In fact, by replacing a log-linear model for the prevalence odds—that is, a logistic model—with a log-linear model for the prevalence, as Lee and Chia propose, the prevalence parameter is not constrained to take values between 0 and 1, but above 0.[6] Therefore, a log-linear model aimed at directly estimating a PR rather than a POR is not satisfactory. As far as I know, there is no useful statistical model for directly estimating a PR with adjustments for several covariates. Such an estimate can be obtained from the logistic model by a straightforward transformation,[6] although further research is needed to provide an appropriate confidence interval.

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Prevalence ratio (PR) and prevalence odds ratio (POR) as effect measures of exposure based on a hypothetical set of cross sectional data. In particular, the table shows the impact of another factor, F, which affects the prevalence of disease, but not the fraction exposed

	Exposed	*Non-exposed*	*PR*	*POR*
F present	500/1000*	250/1000	2·00	3·00
F absent	250/1000	100/1000	2·50	3·00
Total	750/2000	350/2000	2·14	2·83

*(Number of prevalent cases)/(number of people) = the prevalence.

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.
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NOTICES

International symposium on biological monitoring in occupational and environmental health, 11–13 September 1996, Espoo, Finland

The organizer of the Symposium is the Finnish Institute of Occupational Health. Co-sponsors are the International Commission on Occupational Health (ICOH), Scientific Committee on Occupational Toxicology and Scientific Committee on Toxicology of Metals. The Symposium will be a satellite symposium to ICOH Congress in Stockholm, 15–20 September, 1996 (ICOH '96).The topics will include:

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20 Challenge of complex mixtures

For further information contact: Biological Monitoring, c/o Finnish Institute of Occupational Health, Symposium Secretariat, Topeliuksenkatu 41 a A, FIN-00250 Helsinki, Finland. Telephone Int. +358-0-47 471, fax: Int. +358-0-47 47 548 email :s/eh @acuphealth.fi.

Annual Conference of the International Society for Environmental Epidemiology and the International Society for Exposure Analysis. 30 August-1 September 1995. Noordwijkerhout, The Netherlands.

The conference unites people working in environmental epidemiology and exposure assessment to exchange information and synthesise ideas, about the methodology, results and applications of their research. It welcomes epidemiologists, exposure assessors, toxicologists, environmental health officials, and others interested in the field.

The focus of this 7th ISEE/5th ISEA conference will be on methodology to improve the assessment of the public health impact of environmental pollution at the (inter)national and regional level.

Major symposia are foreseen on the following subjects:

- Integrating exposure assessment and epidemiological methods to improve study design in environmental epidemiology and health impact assessment
- Multi-center studies in environmental epidemiology: methodological aspects, and results of a number of recent studies conducted in Europe and elsewhere
- Uses of exposure assessment and environmental epidemiology in public health at the state, regional, and local level.

The programme will feature a number of oral and poster sessions on, among others, the following themes:

- Monitoring and surveillance
- Biological contaminants
- Exposure assessment
- Air pollution
- Environmental equity
- Risk assessment
- Genetic susceptibility
- Molecular epidemiology
- Water quality
- VOC
- Metals
- Multi-center studies
- Adversity of health effects
- Pesticides
- Hazardous wastes
- Motor vehicle emissions
- Chronic diseases
- Reproductive health
- Allergy and other immunological effects
- EMF
- Radon

For any inquiries or assistance, please contact the conference secretariat: Ms Susan Peelen, MSc, Department of Epidemiology and Public Health, University of Wageningen, PO Box 238, 6700 AE Wageningen, The Netherlands. Telephone: +31 8370 84124 Fax: +31 8370 82782 e-mail susan.peelen@medew.hegl.wau.nl.

Continuing Medical Education in Europe: the way forward through European collaboration. London. 30–31 March 1995.

Organised by the Fellowship of Postgraduate Medicine, in association with other bodies with an interest in medical education, this conference brings together the leaders of medical education in Europe. The programme is designed to be comprehensive and cover all specialities. It will explore areas of concern including finance, implementation, assessment, and re-certification. Speakers have been invited from all European Union countries and from the USA, Canada and Australia. There will be ample opportunity for free discussion and small group work. The conference language is English.

For further information please contact: Mrs Jean Coops, Conference Office, Fellowship of Postgraduate Medicine, 12 Chandos Street, London W1M 9DE. Tel: 44 (0) 171 636 6334; Fax: 44 (0) 171 436 2535.

BOOK REVIEW

Veterans and Agent Orange: Health Effects of Herbicides Used in Vietnam. INSTITUTE OF MEDICINE (Pp 832; price $79·95) 1994. Washington: National Academy Press. ISBN 0–309–04887–7.

The effects of modern war extend far beyond the immediate casualties and the obvious health effects of exposure to some of the chemical agents used either in defensive or offensive roles. The possibility of there being long term health effects of the herbicides used in the Vietnam conflict was raised at an early stage and has been the subject of many investigations, both medical and scientific. This extensive volume encompasses a review of the pertinent scientific literature and draws conclusions as to the probability of American and allied troops having been affected by the massive spraying operations used in the defoliation of critical tracts of the Vietnamese forest.

The history of the controversy is outlined and indicates how the concerns about the use of Agent Orange developed to include the toxic contaminant 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), which had been present in appreciable quantities in the herbicide preparations used at that time. There are summaries of the causes and effects of other environmental exposures to TCDD (at Seveso and Times Beach), which themselves resulted in considerable public concern. If this section of the book has a fault, it is that it relies too much on secondary sources, particularly other books that are not well referenced.

In the chapter that describes the military herbicide programme in Vietnam there is a clear reminder that, whatever the public perception, Agent Orange was but one component of a spectrum of preparations used. Purple, blue, pink, green and white each played their part, even although in total they still did not match the volume of Agent Orange that was sprayed. This is reflected throughout the book in that there are sections devoted to each of the compounds in the mixtures, whether it be the TCDD contaminant, 2,4-D, 2,4,5-T, picloram or cacodylic acid. In many instances the sections are small or even non-existent. This reflects not the relative usage but the quantity of information available.

A toxicology chapter describes the studies that have been used to determine what effects should be sought in exposed people. Although thorough and generally accurate, there are errors. The statement that a single dose of TCDD cannot induce porphyria may be true for the rat, but is quite incorrect if applied to mice. It is surprising that the papers that would have contradicted this statement were not found in the detailed literature search described in one of the appendices.

In the epidemiological detection of health effects in a potentially exposed population two main factors have particular importance: the design and methodology of the studies and the assessment of exposure. Each of these are well discussed; the methodology section compares the development of exposure indices for Vietnam veterans with the direct analysis in current body lipid concentrations of TCDD and analogues as a measure of past exposure to the herbicides that contain 2,4,5-T. The conclusion that valid exposure indices may be generated from the available records must remain questionable.

The main part of the book is taken up with a review of the epidemiology: the exposure to herbicides environmentally or occupationally in manufacture or usage, the episodes of exposure to TCDD in the general environment or in factories. This is developed in specific sections that consider the health effects identified as having the most cause for concern: cancer, effects on the reproductive system, neurobehavioural disturbance. The conclusions are developed in each chapter and collected together in the executive summary; unsurprisingly, they are little different from those that have been made for each health effect individually in the scientific literature over the past 20 years. What must be remembered is that the association is with herbicides and not necessarily with any one compound in the mixture.

In conclusion, this is a valuable work on studies of the health effects that may be associated with exposure to the constituents of the herbicides used in Vietnam. It could well be read in conjunction with the recent EPA report on the sources and effects of the dioxin analogues. The reference lists are as up to date as could be expected and, with some notable absences, provide useful points of entry to the original literature.

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