CORRESPONDENCE

Respiratory health effects of opencast coalmining: a cross sectional study of current workers

EDITOR,—We are writing to comment on the manuscript by Love *et al*, on the respiratory health effects associated with opencast mining in the United Kingdom.[1]

Firstly, several years ago, we reported the dangers of surface mining in the United States. Although the general consensus had been that employment as a surface miner was nearly without risk,[2] we identified the very serious risk for aggressive pneumoconiosis in surface drillers and driller helpers.[3] A review of our recent clinical experience has shown that the most severe cases of pneumoconiosis in West Virginia are most often associated with surface mine drilling. We are interested in the prevalence of pneumoconiosis in all groups (primarily the preproduction group), the jobs of the miners with advanced pneumoconiosis, and the relation between workers with pneumoconiosis and exposures in other dusty jobs. Nowhere in this report do the authors tell us the prevalence of pneumoconiosis in the various occupational groups. Without these data, we cannot judge the degree of adverse respiratory risk for the specific jobs in opencast mining. This is relevant to the cases of advanced pneumoconiosis, particularly the two cases of progressive massive fibrosis.

Similarly, this lack of information affects the validity of the authors' recommendations for additional screening for process workers (a term undefined in the text). We think that, because of the way that these data have been presented, the authors have not provided evidence which would justify screening of these workers.

Secondly, in the abstract, the authors note that the risk for pneumoconiosis or decreased lung function was no greater in those opencast miners who had other exposures in the dusty occupations. In view of these data, it remains unclear why the authors did not exclude the 400 men who had worked in previous dusty jobs and focus entirely on workers with opencast mining exposures only. This is especially important as there were twice as many subjects with external dusty exposure as in the preproduction group, the group described as having a considerable number of workers with positive radiographic findings. Frankly, including these 400 miners in the cohort makes it impossible to define the number of miners with pneumoconiosis due to opencast mining with the authors' statistical approach.

The goal of any model building technique in statistics is to find the best fitting, yet biologically reasonable, model to describe the relation between an outcome and a set of explanatory variables. With logistic regression, we cannot separate the health effect of radiographic abnormalities due to either underground mining exposure or opencast mining exposure in the same people. Time spent in opencast mining and time spent in underground mining are both entered into the model as independent variables, even when they occurred in the same subject. Biologically, if a miner had worked as both an underground and opencast miner, it would be impossible to separate the adverse respiratory effects of one type of mining from the other.

Thirdly, table 3 is not helpful without knowledge of the duration of dust exposure or the relation between the development of pneumoconiosis and the history of employment in other dusty jobs (especially employment as an underground miner).

Fourthly, the interpretation of table 6 is unclear. The authors label table 6 as predicting the risk for profusion category 1/0 or greater; however, in the model the authors predict the risks for developing category 0/1 or greater (see the text adjacent to this table). Is this an error, or did the authors use different criteria ?

The authors state that 8% of the workers had pneumoconiosis of category 0/1 or greater (4% category 0/1 and 4% category 1/0 or greater). Yet, the results from the model reported in table 6 shows a 3.6 % prevalence of radiographic films with profusion category 0/1 (or 1/0?) in non-smoking workers and 11.0% for current smokers with zero years of exposure at age 45, and 4.1% positive for profusion category 0/1 (or 1/0?) for non-smokers and 23.4% for smokers at age 55. How can these miners have category 0/1 (or 1/0?) before they had any years of exposure, or do these values only reflect variation in chest radiographic interpretation by the readers ? If so, the amount of variability in the readings (recognised to be false positive) approximates the number described to have disease.

The authors extrapolate the effects of exposure in preproduction for 10 and 20 years into the future, based upon a mean of 6.9 years of exposure (data from table 1). Although the maximal duration of employment was 36 years in preproduction, a mean of 6.9 years implies that few workers had exposures exceeding 10 years. In view of the relatively brief mean duration of exposure and the many apparently false positive radiographs at profusion category 0/1, how accurate is this prediction likely to be?

In summary, this is an interesting study of the respiratory health of opencast miners, but it seems that there are an insufficient number of men in this cohort without exposure to other dusty jobs to determine the risk of employment in the different jobs within this industry. If the data are sufficient to define the risk of employment, we ask that it be presented in a straightforward manner, so that the risk of working in these jobs in the United Kingdom can be evaluated and better support the recommendations made to monitor the opencast miner's respiratory health.

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1 Love RG, Miller BG, Groat SK, *et al*. Respiratory health effects of opencast coalmining: a cross sectional study of current workers. *Occup Environ Health* 1997;**54**:416–23.
2 Fairman RP, O'Brien RJ, Swecker S, *et al*. Respiratory status of surface coalminers in the United States. *Arch Environ Health* 1977;**32**:211–5.
3 Banks DE, Bauer MA, Castellan RN, *et al*. Silicosis in surface coalmine drillers. *Thorax* 1983; **38**:275–8.

Authors' reply—We thank Banks *et al* for their interest in our paper. They make several comments, and we are glad of the opportunity to reply to these.

Banks *et al* suggest that we should present figures representing prevalence in the various jobs in the industry, by which they presumably mean the jobs current at the time of the survey. There are two problems with this approach: men move between jobs; and the contribution to risk of working in a particular job is likely to be related to duration of service in that job. Because of these complications, we sought to characterise risk in relation to full occupational histories taken at survey, a standard epidemiological approach which has proved fruitful in many of our studies. Our stated research objectives, and our presentation of the results,[2] reflected this approach. The detailed exposure data presented in our figure (p 419) and in our table 1 show that measured concentrations could vary widely within occupational groups, but that important contributions to cumulative exposure might be experienced in several of these groups.

The people within our data set varied in age and smoking habits, and had varied durations of exposure in different occupations. Regression methods are standard techniques for analysing such data, because they can both separate the simultaneous effects of different explanatory variables, and measure the degree of any confounding among them (although Banks *et al* seem to question these abilities). We based our conclusions on a careful application of logistic regression modelling, fitting several combinations of variables. In individual people, of course, it is usually difficult or impossible to ascribe the contributions of different factors to overall risk; but we did not attempt this. We confirm that our study was epidemiological, not clinical; and that the film readers were all experienced in their allotted task, the application of the International Labour Organisation classification for the standardised description of radiographic abnormalities. We used the median of their readings of profusion, and attempted to maintain the important distinction between subjects contributing radiographs which were judged to show small opacities, and (being diagnosed as) having pneumoconiosis. Because we did not attempt to diagnose, we did not consider subjects with small opacities but those with little exposure to be false positives: the influence of age and smoking on small opacities are well known.

Banks *et al* express concern that our results may reflect exposures outside the opencast mining industry. We stated[1] that our results showed no relation with time worked in underground mining or other dusty employments, and we have reported elsewhere[2] that exclusion of 198 men who had previously worked as underground miners did not substantially alter the estimates of risk related to opencast working. None of the six men with category 2 small opacities or large opacities had worked at any time as an underground coalminer.[3]

We are confident that our stated findings[1 2] of an association between increased risk of (mostly mild) radiographic abnormalities and time worked in the dustier, preproduction jobs in the industry, after adjustment for smoking habits, are not due to confounding with other occupational risks. Our findings are consistent with the clinical observations and epidemiological film readings of Banks *et al*[3] in drillers, who are included in our preproduction occupational group. We summarised our fitted regression models by showing, in table 6, average risks of showing small opacities $\geqslant$0/1 predicted (although not in the sense of "in the future") by the model for various patterns of age, smoking, and time worked in preproduction occupations. Uncertainty in these predicted averages is measured as standard errors, which are also given in the table. We are grateful to Banks *et al* for spotting the typographic error in the title of table 6, which wrongly implies that the response was ⩾1/0; all the text is correct and consistent in referring to the response as ⩾0/1.

Banks *et al* question our recommendation for screening the men at greatest risk. In fact, our report to the industry[2] made several recommendations: annual dust and quartz monitoring in the dustiest jobs; improved dust control and suppression, particularly at drilling sites and in excavation; static perimeter sampling for airborne dust at selected sites; screening by chest radiography of the more highly exposed employees, every three years in the first instance, until control measures are found to be adequate; follow up study, in due course, to examine the effect of dust control measures on concentrations and on the health of the workforce; and the examination of exposure and health in off site exploratory drilling teams. We thought, and still think, that these recommendations were justified in the light of our findings.

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1 Love RG, Miller BG, Groat SK, Hagen S, Cowie HA, Johnston PP, Hutchison PA, Soutar CA. Respiratory health effects of opencast coalmining: a cross sectional study of current workers. *Occup Environ Med* 1997;**54**:416-423.
2 Love RG, Miller BG, Beattie J, Cowie HA, Groat S, Hagen S *et al. A cross-sectional epidemiological study of the respiratory health and exposure to airborne dust and quartz of current workers in opencast coalmines.* Edinburgh: Institute of Occupational Medicine, 1992. (IOM Report TM/92/03.)
3 Banks DE, Bauer MA, Castellan RM, Lapp NL. Silicosis in surface coalmine drillers. *Thorax* 1983; **38**:275-278.

CORRECTION

Respiratory health effects of opencast coalmining: a cross sectional study of current workers by R G LOVE, B G MILLER, S K GROAT, S HAGEN, H A COWIE, P P JOHNSTON, P A HUTCHISON, C A SOUTAR (1997;**54**:416–23).

The title of table 6 should read "Predicted risks of showing small opacities ⩾0/1 as a function...".

Pulsed electromagnetic fields and cancer

EDITOR,—Savitz, *et al* have further analysed the reported association between lung cancer and 60 Hz magnetic fields and pulsed eletromagnetic fields (PEMF) in electrical utility workers.[1] They found a weak association but raised the possibility that larger associations may have been diluted through misclassification of exposure. They also note that the PEMF measurements were likely to have recorded use of communication devices such as mobile phones and two way radios which emit radio frequency radiations, as well as high frequency transients from the electricity network.

It is important that subanalyses of PEMFs attributed to such radio frequency radiations identify any major differences in the radio systems used by the workers in different industries because of their potentially differing biological effects and so avoid non-differential misclassification. Portable radios operate at differing parts of the spectrum, including 150, 450, and 900 MHz. The wavelengths range from around 4 m to 20 cm. The longer waves couple with the whole body but shorter ones interact more with a local body part such as the head.[2] In turn this could influence development of cancer in different sites, such as leukaemia or brain tumour. The modulations (AM, FM, pulsed) used should also be noted as pulsed frequencies have been found to promote tumours in animals.[3]

The duration of calls is obviously relevant to exposure but the meters used for detecting PEMFs are intended to only detect transient exposures and hence would underestimate exposure to radio frequency radiations. The place of use may also influence exposure. For example communication from a vehicle with an external antenna should cause minimal exposure, but communication from a tower made of metal beams may lead to local field enhancements and increased exposure depending on the power of the radios.

The reported interaction of radio frequency radiations with 3,4-benzopyrene on mouse skin to promote cancer[4] may be relevant to the finding of increased lung cancer in utility workers, some of whom are exposed to occupational carcinogens as well as cigarette smoke. The possibility that radio frequency radiations from radios may act as a cancer promoter could be considered in data analysis of cancer in various sites where differences between industries are found. Adjustment should be made for the types of systems used and the patterns of use by workers.

Given the widespread use of radio frequency radiation communication devices in industry a further detailed analysis of the data would be of much interest.

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1 Savitz D, Dufort V, Armstrong B, *et al.* Lung cancer in relation to employment in the electrical utility industry and exposure to magnetic fields. *Occup Environ Med* 1997;**54**:396–402.
2 *Electromagnetic fields (300 Hz to 300 GHz)*. Geneva: WHO, 1993:76–7. (Environmental Health Criteria 137.)
3 Repacholi M, Basten A, Gebski V, *et al.* Lymphomas in Eu-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields. *Radiat Res* 1997;**147**:631–40.
4 Szmigielski S, Szudzinski A, Pietraszek A, *et al.* Accelerated development of spontaneous and benzopyrene induced skin cancer in mice exposed to 2450 MHz microwave radiation. *Bioelectromagnetics* 1982;**3**:179–91.

BOOK REVIEW

Book review editor: R L Maynard

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Biological Monitoring Methods for Industrial Chemicals, 3rd edition. By RANDALL C BASELT. (Price $129.00; Pp 400) 1997. Ashlona, OH, USA: Bookmasters. ISBN:0-9626523-2-6.

The first edition of this publication appeared in 1980 and since then, there has been much progress in laying down national and international standards for biological monitoring reference values. The format of the third edition remains substantially unchanged. Monographs are presented on over 100 substances to which workers might be exposed and where some form of biological monitoring has been recommended. Each monograph is headed by recent values from German and American authorities for BTV (biological tolerance value), BEI (biological exposure index), EKA (carcinogen exposure equivalent), MAK (maximum workplace concentration), OSHATWA (8 hour time weighted average maximum permissible air concentration defined by the United States Occupational Safety and Health Administration), and TLV (threshold limit value). The half life (usually in blood) is also quoted. Sections follow on occurrence and usage; blood concentrations; metabolism and excretion; toxicity; biological monitoring; and analysis. After the reference list, a few analytical procedures to monitor the concentration of the substance itself in biological fluids, and where appropriate, a biological marker of its effects, are presented in sufficient detail for a laboratory analyst to put them into practice.

Baselt is a past master in producing concise accounts in this format and his more well known book, *Disposition of Toxic Drugs and Chemicals in Man*, has long been a favourite and quick source of reference for clinical and forensic toxicologists. This publication should have the same appeal for occupational physicians and industrial toxicologists and will save hours of ploughing through some of the heavier tomes on industrial hygiene to find the same basic information.

Surprisingly, in view of the fact that Baselt is in essence a laboratory scientist, many of the analytical methods which are cited or described in detail were originally developed over 20 years ago. This probably reflects the tendency for industrial hygiene laboratories to adopt the principle of "if it works don't fix it" and the author is therefore obliged to quote old methods which are in widespread routine use. This is a pity, as numerous analytical advances have been achieved, even for substances which have been monitored for decades and these should have at least received a mention.

This is not designed as a text book and for students of occupational hygiene it can serve only as an adjunct to the more comprehensive volumes available which deal with this specialty. However, as a source of reference for students in related disciplines such as biochemical toxicology it will be quite useful.

The book is not cheap at $129.00, but given that it is likely to be consulted very often, this outlay will be justified.

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