that, to the eye of one reader, small irregular opacities on the chest radiograph may be related to smoking habit. The author calls for further studies of large populations with adequate numbers of older people, using multiple readers.

The article illustrates well a problem facing those who, as is widely the case in the United States, use the International Labour Office (ILO) classification as a diagnostic test, rather than (as it is intended) as an epidemiological tool. The *diagnosis* of pneumoconiosis using, as it does, other clinical information is a very different matter from the *description of radiographic shadows*, which is what the ILO scheme allows. Shadows may have multiple causes, and the studies that Weiss calls for have already shown this. Two such, with which I have had personal involvement, have shown relations between low profusion of small irregular opacities, age, smoking habit and, of course, exposure to dust.[1-3] Not all readers show this relation, which depends critically on conscientious use of the category 0 standard film, thus allowing classification of 0/1 and 1/0 in a high proportion of films of people who turn out to have been elderly, smokers, or both.

In Britain, the diagnosis of pneumoconiosis remains a clinical one, using information from occupational history, examination and investigation of anatomical and physiological abnormalities by *x* ray film, and lung function. The tendency in the United States to equate small irregular shadows with asbestosis can only lead to overdiagnosis of the condition and thus cause unjustified anxiety in many people. It is my impression that this regrettable tendency is now present in medicolegal circles in the United Kingdom and is spreading also to the overinterpretation of computed tomography scans.

ANTHONY SEATON
Professor of Environmental and Occupational Medicine, University Medical School, Foresterhill, Aberdeen AB9 2ZD

1 Seaton A, Louw SJ, Cowie HA. Epidemiologic studies of Scottish oil shale workers: 1. Prevalence of skin disease and pneumoconiosis. *Am J Ind Med* 1986;9:409–21.
2 Dick JA, Jacobsen M, Gauld S, Pern PO. The significance of small irregular opacities in the chest radiographs of British coal miners. In: *Proceedings of the VIth international pneumoconiosis conference, Bochum,* 1983. Geneva: International Labour Office, 1983: 283.
3 Seaton A, Seaton D, Leitch AG. *Crofton and Douglas's respiratory diseases.* 4th ed. Oxford: Blackwell Scientific, 90–1.

A case-control study of lung cancer in a cohort of workers potentially exposed to slag wool fibres

Sir,—The conclusion reached by Wong *et al* (1991;**48**:818–24) that exposure to slag wool fibre did not increase lung cancer risk among a cohort of slag wool workers is supported by results from experimental studies.

For example, no evidence of lung disease related to fibre inhalation was found in hamsters or rats exposed to slag wool fibres in concentrations hundreds of times greater than those found in the workplace.[1] In this study animals exposed to crocidolite did develop lung tumours and statistically significant fibrosis. Also, implantation or injection of slag wool fibres in concentrations thousands of times greater than those found in the workplace into the chest or abdominal cavities of rats did not induce significant numbers of tumours.[2-4]

Results from inhalation studies designed to assess biopersistence of fibres in rat lungs suggested that slag wool fibres are attacked by fluids present in the lungs.[5 6] Slag wool fibres disintegrated and were cleared from the lungs more rapidly than were more durable fibres. These results are consistent with those from analyses of lung tissue samples from deceased slag wool workers.[7] No slag wool fibres were seen in these samples. Perhaps slag wool fibres of dimension classically associated with tumour induction ("Stanton fibres") do not stay in the lung in sufficient quantity or time to induce tumours. Currently TIMA Inc is sponsoring animal inhalation studies at RCC Laboratories in Switzerland to further evaluate the biopersistence of slag wool fibres and other manmade vitreous fibres.

In conclusion, the results reported by Wong *et al* (1991) emphasise the importance of cigarette smoking and detailed exposure assessment data in the analysis and interpretation of occupational epidemiological studies. Although some previous cohort mortality studies suggested a modest increase of lung cancer in workers exposed to slag wool fibres, these studies were inconclusive for a number of reasons.[8 9] The most serious limitations were lack of control for cigarette smoking and the presence of confounders in some of the workplaces studied. Using a case-control study design, Wong *et al* were able to consider these limitations. The next updates of the cohort mortality studies are to include case-control studies of design similar to that of Wong *et al*. These updates are scheduled for completion in early 1994.

ROD P MUSSELMAN
USG Corporation, 101 South Wacker Drive, Chicago, IL 60606-4385, USA

1 Smith DM, Ortiz LW, Archuleta RF, Johnson NF. Long-term health effects in hamsters and rats exposed chronically to man-made vitreous fibers. *Ann Occup Hyg* 1987;**31**:731–54.
2 Stanton MF, Layard M, Tegeris A, *et al*. Carcinogenicity of fibrous glass: pleural response in the rat in relation to fiber dimension. *J Natl Cancer Inst* 1977;**58**:587–603.
3 Wagner JC, Berry GB, Hill RJ, Munday DE, Skidmore JW. Animal experiments with MMM(V)F—effects of inhalation and intrapleural innoculation in rats. In: *Biological effects of man-made mineral fibres (Proceedings of a WHO/IARC conference)*. Vol 2. Copenhagen: World Health Organisation 1984:209–33.
4 Pott F, Ziem U, Reiffer F-J, Huth F, Ernst H, Mohr U. Carcinogenicity studies on fibres, metal compounds, and some other dusts in rats. *Exp Pathol* 1987;**32**:129–52.
5 Hammad YY. Deposition and elimination of MMMF. In: *Biological effects of man-made mineral fibres (Proceedings of a WHO/IARC conference)*. Vol 2. Copenhagen: World Health Organisation 1984:27–59.
6 Hammad YY, Simmons W, Abdel-Kader H, Reynolds C, Weill H. Effect of chemical composition on pulmonary clearance of man-made mineral fibres. *Ann Occup Hyg* 1988;**32**: 769–79.
7 McDonald CC, Case BW, Enterline PE, *et al*. Lung dust analysis in the assessment of US man-made mineral fibre workers. *Ann Occup Hyg* 1990;**34**: 427–41.
8 Marsh GM, Enterline PE, Stone RA, Henderson VL. Mortality update of a cohort of US man-made mineral fiber workers: 1985 follow-up. *J Occup Med* 1990;**32**:594–604.
9 Simonato L, Fletcher AC, Cherrie JW, *et al*. The International Agency for Research on Cancer historical cohort study of MMMF production workers in seven European countries: extension of the follow-up. *Ann Occup Hyg* 1987;**31**:603–23.