Correspondence

of disease was classified as pneumoconiosis instead of silicosis, probably due to a different interpretation when reading the chest x-ray films.

These papers, and the opportunity of a trekking holiday in Ladakh, prompted us to carry out a small study to obtain further information about the environmental risk for silicosis. In particular, the study aimed to characterise dust composition and size.

During our 170 km trek, we took four samples of dust in four villages (Padum, Lingshed, Hanupatta, Lamayuru) located in the Zanskar region, not far from the villages in which previous studies were carried out. Because practical difficulties made collecting samples from house beams impossible, about 2 g of dust were collected from the upper surface of small buildings and put into plastic vials. These buildings were situated along the route in the proximity of or inside villages. The surfaces were at a height ranging from 1 to 1.5 m above the path. Small quantities of rock were also taken. Sedimented dust and previously milled rocks were examined by means of x-ray diffractometry (Siemens Kristalloflex 810-D500/DACO) to determine the quartz content quantitatively and other minerals semi-quantitatively. Dust dimensions and fibre aspect ratios were determined by optical microscopy.

The percentage of quartz in the sedimented dust ranged from 6% to 9% (table), whereas 50% (by weight) of the material collected was of large particle size (>2 mm diameter) and agrees with the hypothesis that if finer material were examined, an amount of quartz greater than that reported would be detected. These values are slightly higher than those measured in previously ground rock samples (quartz content range 5%-7%).

These observations are consistent with the geological nature of the Himalayan range in Ladakh. In the same area, however, a quartz content of up to 42% has been measured in a recent survey.

Dust granulometry was performed on previously sieved dust to remove particles larger than 50 µm. The percentage of particles with a geometrical diameter of between 0.5 and 5 µm ranged from 61% to 89%. Fibrous bodies (aspect ratio 3:1) and fibres of difficult mineralogical classification but of respirable size (length >5 µm and diameter <3 µm) were also detected.

Air samples were obviously not available to us; nevertheless our findings agree with those of Norboo et al.—that is, that quartz, muscovite, and other minerals are present in the environmental dust. Norboo et al. pointed out that "dust . . . included many particles within the range 0.5-5 µm diameter," but no data were given. Our granulometric findings show that the percentage of respirable particles in the sedimented dust reaches relatively high values.

Whether or not there truly are undiagnosed cases of occupational silicosis, as suggested by Valiante and Rosenman's paper, the possibility of environmental silicosis should not be forgotten. Perhaps the 4% of reviewed cases of silicosis, in which no occupational risk was identified, may be ascribed to environmental, non-occupational exposure. We suggest that environmental non-occupational exposure to dust, may represent a potentially important respiratory risk factor for people living in areas where there is a possibility of exposure to silicogenic rock dust.

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3 Saiyed HN, Sharma YK, Sadhu HG, Norboo T, Patel FD, Patel TS, et al.

Analysis of dust samples collected in the villages (figures represent the percentages of quartz and the approximate percentage of other minerals)

<table>
<thead>
<tr>
<th>Villages</th>
<th>Quartz</th>
<th>Muscovite</th>
<th>Kaolinite</th>
<th>Calcite</th>
<th>Feldspar and other minerals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Padum</td>
<td>6</td>
<td>10</td>
<td>70</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Lingshed</td>
<td>7</td>
<td>10</td>
<td>70</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Hanupatta</td>
<td>9</td>
<td>5-10</td>
<td>50</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Lamayuru</td>
<td>7</td>
<td>5-10</td>
<td>70</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

Sir,—The article by Saiyed et al. (1991; 48:825-9) presents shortcomings that we must call to your attention. The diagnosis of "pneumoconiosis" seems to be solely based on radiological appearances classified by the International Labour Office (ILO) system. The ILO classification, in and of itself, is not a diagnostic tool and other differential diagnoses could mimic these radiological findings. Tissue documentation certainly would have been more persuasive. Such materials should be available where the "reported" prevalence of pneumoconiosis approaches epidemic levels in certain population segments.

Furthermore, and equally perplexing is the postulated aetiology of fugitive dust exposure and the paucity of quantitative exposure data. Information regarding the frequency, duration, and severity of dust storms, particle size distributions, and the relationship between soot and the pneumoconioses is clearly lacking.

Also, pulmonary risk factors such as tobacco smoking and other exposures (including occupational) are missing. It is indeed interesting that respiratory symptoms increased in a concomitant fashion with the frequency and extent of radiological classes of pneumoconiosis. Little can be made of this without adequate information on other pulmonary risk factors. Also, pulmonary function tests were performed but no such data were reported.

To the credit of the authors, they realise that further work is necessary to determine the source, concentration, and composition of the causative agent, and the natural history of the disease process. It is hoped that these issues will be pursued vigorously.

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Cigarette smoking and small irregular opacities

Sir,—I was interested to read Weiss's article (1991; 48:841-4), which showed
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. 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.

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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

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.3,4 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.5 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. 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.

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Cigarette smoking and small irregular opacities.

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doi: 10.1136/oem.49.6.453-a

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