Table 1 *Calculated number of mesothelioma cases expected from Wittenoom*

Year of diagnosis	Year of first employment <1940	1940–4	1945–9	1950–4	1955–9	1960–6	Total
<1980	18·3	6·9	9·5	29·1	20·9	5·1	89·8
*1980–85	3	2	5	18	19	9	56
1986–90	2·7	1·5	4·2	24·9	24·7	12·8	70·8
1991–95	) 2·0	1·3	3·1	20·8	34·2	16·7	78·1
1996–00	) 1·0	) 1·0	2·8	15·2	28·5	23·1	71·6
2001–05	) 0·3	) 0·6	) 2·0	13·9	20·9	19·3	57·0
2006–10	)	)	) 1·0	) 10·0	19·0	14·1	44·1
2011–15	)	)	) 0·3	) 6·6	) 17·0	) 10·0	33·6
2016–20	)	)	)	)	) 5·8	) 18·3	24·1
	27·3	13·3	27·9	138·5	190·0	128·4	525·4

*Observed cases based on Australian Mesothelioma Surveillance Program records.
)Estimated distribution of residual cases.

mesothelioma from the Wittenoom crocidolite mine produce a range of estimates dependent on the values of lag time and lung clearance rates. Although published data suggest a minimum lag period of around 20 years there is considerable spread of another 30 years to cover most cases. Also, the assumption of reduced toxicity due to clearance of fibres from the lung is speculative. de Klerk *et al*[1] have estimated future Wittenoom cases using a model based on lag time and exposure variables but their predictions are somewhat higher than Berry's. As the future occurrence of mesothelioma due to past exposure to asbestos is of great interest to government authorities, industry, workers, and the community, the accuracy of these estimates is of importance.

An alternative predictive approach is proposed using the distribution of time lag period since first asbestos exposure derived from information obtained in occupational histories from the Australian mesothelioma statistics.[2] Assuming a similar latency distribution in the Wittenoom cases collected in 1980–5, it is possible to calculate the total number of cases expected from the mine and the number expected in each time period. For example, to calculate the total number of mesotheliomas expected for an employment period 1940–4, use the two observed cases recorded from this period in 1980–5 and the midpoint of employment to yield a latency of 37·5–42·5 years. From the lag data in Ferguson *et al*[2] prepare a graph of the cumulative percentage of cases *v* lag time since first exposure and use this to estimate that these two cases represent 13% (34% less 21%), hence predicting a total of 13·3. Calculation of the expected cases from the 1940–5 employees occurring in the time period 1991–5 (lag of 52·5–47·5 years) gives 11% (88% less 78%) of 13·3 cases or 1·3. Table 1 shows the overall data set.

The uncertainties in such an approach is that it includes environmentally exposed cases and takes a simplified view that there has been a similar distribution of age, the specific occupations, and exposures during the period of operation of the mine and mill, all of which are known to be incorrect.[3,4] Also it assumes compatibility between the Wittenoom group and the whole Australian population. The estimates (71) appear reasonable, however, when compared with the 59 cases reported to the Australian Register in 1986–90. These register records were not used in the calculation as they were not collected with the same rigour as the 1980–5 data and hence cases may have been missed and they are known to contain less detailed information on occupational history.[5]

Table 2 *Comparison of estimates of mesotheliomas*

Year of diagnosis	Latency method estimates	Berry estimates adopted from table 7	de Klerk estimates
1987–90	57	40 - 44	44
1991–95	78	55 - 65	77
1996–00	72	55 - 70	102
2001–05	57	45 - 70	121
2006–10	44	30 - 60	132
2011–15	34	20 - 45	121
2016–20	24	10 - 30	95
	366	265–395	692

Table 2 gives a comparison of the estimates from the various methods.

The proportional latency calculation method predicts a total of 525 mesotheliomas from the 6505 male and 411 female workforce and past residents; 366 cases will occur in the period 1986–2020, numbers will peak in the period 1991–1995, and tail off over the next 25 years. This fits comfortably in the range and distribution of most likely values presented by Berry.

A ROGERS
*National Institute of Occupational Health and Safety,
GPO Box 58,
Sydney, NSW 2001,
Australia*

1 de Klerk N, Armstrong B, Musk B, Hobbs M. Predictions of future cases of asbestos-related disease among former miners and millers of crocidolite in Western Australia. *Med J Aust* 1989;**151**:616–20.
2 Ferguson DA, Berry G, Jelihovsky T, Andreas S, Rogers A, Chung Fung S, Grimwood A, Thompson R. The Australian mesothelioma surveillance program 1979–1985. *Med J Aust* 1987;**147**:166–72.
3 Major G. Asbestos dust exposure. In: Major G, ed. Proceedings of the first Australian pneumoconiosis conference in Sydney, 1968: *Joint Coal Board* 1968:467–74.
4 Armstrong B, de Klerk N, Musk B, Hobbs M. Mortality in miners and millers of crocidolite in Western Australia. *Br J Ind Med* 1988;**45**:5–13.
5 Leigh J, Corvalan C, Grimwood A, Berry G, Ferguson D, Thompson R. The incidence of malignant mesothelioma in Australia 1982–1988. *Am J Ind Med* 1991;**20**:643–55.

Non-occupational pneumoconiosis at high altitude villages in central Ladakh

Sir,—Silicosis is a well established, probably underdiagnosed occupational disease,[1] the importance of which may be underestimated as an environmental disease. Several cases of non-occupational silicosis have recently been reported among people living in the Himalayan range by Naboo *et al*.[2] Another paper[3] (BJIM 1991;**48**:825–9) dealing in greater depth with the radiological characteristics of the disease confirmed the findings of Naboo *et al*,[2] except that the 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.[2,3] 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 1·50 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 semiquantitatively. 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). About 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 42·8% has been measured in a recent survey.[4]

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 $\leqslant 3$ μm) were also detected.

Air samples were obviously not available to us; nevertheless our findings agree with those of Norboo *et al*[2] —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[1] the possibility of environmental silicosis should not be forgotten. Perhaps the 4% of reviewed cases of silicosis, in which no occupational risk was identified[1] 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 silicotigen rock dust.

G FRANCO
Faculty of Medicine, University of Pavia, Italy
A MASSOLA
Industrial Hygiene Laboratory, IRCCS Fondazione Clinica del lavoro, Pavia, Italy

1 Valiante DJ, Rosenman KD. Does silicosis still occur? *JAMA* 1989; **262**:3003–7.
2 Norboo T, Angchuck PT, Kamat SR, Pooley FD, Corrin B, Kerr IH, *et al.* Silicosis in a Himalayan village population: role of environmental dust. *Thorax* 1991;**46**:341–3.
3 Saiyed HN, Sharma YK, Sadhu HG, Norboo T, Patel PD, Patel TS, *et al.* Non-occupational pneumoconiosis at high altitude villages in central Ladakh. *Br J Ind Med* 1991;**48**:825–9.
4 Gaetani M, Casnedi R, Fois E, Garzanti E, Jadoul F, Nicora A, Tintori A. Stratigraphy on the Tethys Himalaya in Zanskar, Ladakh. *Riv It Paleont Strat* 1986;**91**:443–78.

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

Villages	Quartz	Muscovite	Caolinite	Calcite	Feldspar and other minerals
Padum	6	10	10	70	5
Lingshed	7	10	5	70	5
Hanupatta	9	5–10	5–10	50	25
Lamayuru	7	5–10	5–10	70	10

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 relation 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.

M I RANAVAYA
R B REGER
M C BATTIGELLI
Institute of Occupational Health and Safety, West Virginia University, Morgantown, West Virginia, USA

Cigarette smoking and small irregular opacities

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