British Journal of Industrial Medicine 1987;**44**:588–594

Mortality among workers receiving compensation awards for silicosis in Ontario 1940–85

M FINKELSTEIN, G M LISS, F KRAMMER, R A KUSIAK

From the Health Studies Service, Ontario Ministry of Labour, Toronto, Ontario, Canada M7A 1T7

ABSTRACT The mortality experience of 1190 miners and 289 surface industry workers receiving workers' compensation awards for silicosis in Ontario since 1940 has been studied up to mid-1985. Both groups were found to have a significantly increased mortality from lung cancer (miners' SMR: 230; surface workers' SMR: 302) and stomach cancer (miners' SMR: 188; surface workers' SMR: 366). Adjustment for smoking and country of origin did not explain the excesses observed. The lung cancer findings are consistent with observations from silicosis registries in Europe. Possible explanatory factors are discussed.

The mortality experience of workers receiving compensation awards for silicosis has come under study in recent years. Their life expectancy has been found to be reduced, due largely to deaths from tuberculosis and non-malignant respiratory disease. Early reports from silicosis registries in Sweden[1] and Ontario[2] indicating increased mortality from lung cancer among silicotics have been followed by confirmatory studies in Finland[3] and Italy.[4] Goldsmith *et al* suggested that occupational exposure to silica might explain the excess of lung cancer. They hypothesised that silica itself might be a carcinogen, that silica might act as a carrier particle for chemical carcinogens, or that silicosis might be an intermediate pathological state leading to cancer.[5]

The interpretation of occupational data concerning the association between silicosis and cancer is difficult because of confounding by tobacco smoking and the likely presence of diverse carcinogenic agents in the industrial environment. Only the Italian study looked at the question of smoking as a contributory factor.[4] The objective of the present study was to collect additional information relevant to the assessment of the carcinogenicity of silica by: (1) updating the analysis of the silicotic miners studied previously[2]; (2) enrolling a new group of workers awarded compensation for silicosis attributable to exposure in dusty surface industries; and (3) collecting smoking information for this latter group. Foundry workers were excluded from the cohort because of their exposure to suspected organic carcinogens.

Accepted 29 September 1986

The disease of primary interest was lung cancer but in the light of the report by Kurppa and his colleagues of increased rates of stomach cancer among Finnish granite workers[6] this cause of death was to be tested for an association with silicosis as well. In the present study a significant excess mortality was observed for both causes.

Materials and methods

All the workers in the present study received compensation for disability due to silicosis. Claims for silicosis submitted to the Workers' Compensation Board (WCB) are assessed by the Advisory Committee on Occupational Chest Diseases, a panel of university and government physicians that determines, on the basis of radiographic and clinical evidence, whether a diagnosis of silicosis may be made and assesses the degree of disability. A cohort of miners awarded compensation for silicosis between 1940 and 1976 had previously been assembled from the computerised files of the WCB.[2] A second cohort of surface industry silicotics was identified by searching the records of the advisory committee. Individual files of the latter workers were located and examined for details of employment history and smoking habit. Any worker with evidence of malignancy at the time of disability determination was excluded from the cohort. Workers were followed up to 30 June 1985 by the use of WCB records. The miners had previously been followed up until 1978, but on extending the period of follow up it was discovered that the deaths of several dozen miners before 1979 had been missed because they had not been reported to the WCB section maintaining the silicosis registry. To avoid a re-

currence of this error, the vital status of all workers presumed alive at the end of follow up was checked against another source of information, the pension payment files. As a result, it is now believed that the assessment of vital status is accurate.

Death certificates were obtained for 115 (87%) of the new deaths among the miners. Of the remaining 17 deaths, information pertaining to the cause was available in WCB files for 12. As indicated in a necropsy report from British Columbia and a report from Finland, two of these deaths were from lung cancer. For the surface workers, death certificates were obtained for 204 (96%) of 212 deaths. Information was available in WCB files for six of the remaining eight deaths; none was attributed to lung or stomach cancer. For those deaths without official certificates, causes of death derived from WCB files were used for categorisation. Any errors in the statistical analysis due to misclassification of these few deaths are expected to be small.

Causes of death were considered in the following broad groupings (with codes from the eighth revision of the International Classification of Diseases): all malignancies (ICD 140–209); lung cancer (162); gastrointestinal (GI) cancer (150–154); stomach cancer (151); non-malignant respiratory diseases including silicosis (460–519); tuberculosis (010–019); and ischaemic heart disease (410–414). Deaths with cause unknown were assigned solely to the all cause category.

To investigate possible trends in time, workers were grouped into four cohorts based on decade of initial disability award. To assess possible differences related to the exposure environment, a classification was also performed by industry type: mining; ceramics and pottery; granite and quarry; silica brick; and others including sandblasting, silica flour, and abrasives. Miners with experience of uranium mining were identified by comparison with the uranium registry assembled for the Ontario Miners Study.[7]

The mortality experience of the workers with silicosis was compared with that of the general population of Ontario using the person-years method. Age, sex, and time specific Ontario mortality rates were used in combination with the distribution of person-years at risk among the cohorts to determine the "expected" mortality pattern. Tests of statistical significance and calculation of 95% confidence intervals (CI) were performed by assuming a Poisson distribution of observed deaths.

Information about smoking habits was present in the records of 206 (75%) of the 276 men in the surface industry cohort. Data were not available for the remaining 70 because relevant information had not been recorded or because the clinical files of men dying before 1960 had been destroyed. Because quantitative information about cigarette consumption was of uncertain reliability, a dichotomous classification (ever/never smoker) was used. For comparison with the smoking habits of the general population of Ontario, data were used from the Labour Force Survey collected by Statistics Canada (unpublished).

Because it has been reported that death rates from stomach cancer among immigrants to Canada are greater than the rates among Canadian born residents of Ontario,[8] information about birthplace was obtained from work histories or death certificates for the surface industry cohort. These data were available for 272 (99%) of the 276 men. Statistical adjustment of SMRs for the effects of smoking and birth place was done by the method suggested by Axelson.[9]

Results

The original composition of the mining cohorts has already been described.[2] In 1979, on entry to this update of the previous study, there were 21 survivors in the 1940–9 cohort, 38 in the 1950–9 cohort, 118 in the 1960–9 cohort, and 203 in the 1970–5 cohort. Twenty

Table 1 *Description of surface industry cohorts by decade of compensation award and by industry type (men)*

Year of compensation award	*No*	*Person-years of observation*	*Mean age at compensation*	*Mean age at death*
1940–49	38	495	55·8	68·7
1950–59	83	1006	55·8	67·5
1960–69	70	711	59·2	69·1
1970–84	85	599	56·9	66·3
Total	276	2811	57·0	68·0
Industry type				
Ceramics	98	1097	56·2	67·1
Granite and quarry	76	753	60·3	70·7
Silica brick	47	474	59·3	69·8
Other	55	487	51·8	62·4
Total	276	2811	57·0	68·0

Table 2 *Mortality among miners with silicosis*

Cohort		All causes			All malignancies			Lung cancer		
		Obs	Exp	SMR	Obs	Exp	SMR	Obs	Exp	SMR
1940–9	A*	388	171	227	58	32	181	24	7·4	324
	B*	11	10·3	100	3	2·0	150	3	0·5	600
1950–9	A*	235	141	167	38	28	136	17‡	7·5	227
	B*	19	17·2	110	4	3·4	100	2	0·9	222
1960–9	A*	139	87	160	21	18	117	7‡	5·5	127
	B*	41	33·4	123	12	7·0	171	6	2·0	300
1970–5	A*	35	18	194	4	4·2	100	1‡	1·4	100
	B*	37	24·5	151	11	5·6	196	2	1·7	100
Totals		905	502	180†	151	100	151†	62	26·9	230†
Uranium exposed 1960–75	B*	24	13·3	180	7	3·7	190	4	1·3	310

A* = Followed up to end of 1978; B* = followed up 1979–85.
†$p < 0·02$.
‡One man with lung cancer in each cohort had uranium mining experience.

Table 3 *Mortality among surface workers with silicosis*

Cohort	All causes			All malignancies			Lung cancer		
	Obs	Exp	SMR	Obs	Exp	SMR	Obs	Exp	SMR
Men:									
1940–9 (n = 38)	38	17·5	217	4	3·1	129	1	0·4	250
1950–9 (n = 83)	76	32·0	238	11	6·5	169	3	1·7	176
1960–9 (n = 70)	61	25·2	242	7	5·6	125	5	1·7	294
1970–84 (n = 85)	31	16·9	183	9	4·3	209	7	1·5	467
Total (n = 276)	206	91·6	225*	31	19·5	159*	16	5·3	302*
Women	5	1·44	347	3	0·5	600	1	0·05	—

*$p < 0·01$.

Table 4 *Mortality among surface workers with silicosis by industry (all time periods combined; men only)*

Industry	All causes			All malignancies			Lung cancer		
	Obs	Exp	SMR	Obs	Exp	SMR	Obs	Exp	SMR
Silica brick (n = 47)	35	17·6	199	7	3·8	183	2	1·1	183
Ceramics (n = 98)	65	31·6	206	7	7·0	100	6	2·05	293
Granite and quarry (n = 76)	70	30·7	228	10	6·1	164	5	1·39	360
Other (n = 55)	36	11·7	309	7	2·6	266	3	0·77	390
Total (n = 276)	206	91·6	225*	31	19·5	159*	16	5·3	302*

*$p < 0·01$.

three of the 1960–9 group and 112 of the 1970–5 cohort had uranium mining experience.

The male cohort from the surface industries is described by decade of compensation award and by industry type in table 1. In addition, 13 women were followed up for 171 person-years; all but two had worked in ceramics.

MORTALITY EXPERIENCE OF THE MINERS

Table 2 displays the mortality from selected causes for the mining cohorts. The lines labelled "A" present the revised results of follow up to the end of 1978, corrected for loss to follow up and deaths missed by the previous analysis.[2] These additional deaths produced no substantial change in the mortality pattern described earlier.

The lines labelled "B" refer to the additional 6·5 years of follow up from 1 January 1979 to 30 June 1985. Miners who survived to 1979 had, overall, a more favourable experience than that observed in the initial period (A) but mortality was still significantly raised (SMR 126; CI 104–152), primarily attributable

Stomach cancer			*Tuberculosis*		*Respiratory disease*			*Ischaemic heart disease*		
Obs	*Exp*	*SMR*	*Obs*	*Exp*	*Obs*	*Exp*	*SMR*	*Obs*	*Exp*	*SMR*
10	4·3	233	101	1·08	113	9·9	1140	57	72	79
0	0·1	0	0	0·01	5	1·1	455	2	3·7	5
3	2·7	100	30	0·49	70	10·2	686	45	59	76
0	0·2	0	2	0·01	7	1·8	389	2	6·2	32
2	1·6	100	5	0·20	46	7·2	639	6	8·7	69
2	0·4	500	1	0·02	12	3·3	364	4	12·1	33
1	0·3	100	1	0·01	10	1·4	714	9	7·0	129
1	0·3	100	0	0·01	11	2·3	478	6	8·9	67
19	10·1	188†	140	1·83	274	37·2	737†	131	178	74
0	0·2	0	0	0·01	6	0·8	7·5	2	4·8	42

Stomach cancer			*Tuberculosis*		*Respiratory disease*			*Ischaemic heart disease*		
Obs	*Exp*	*SMR*	*Obs*	*Exp*	*Obs*	*Exp*	*SMR*	*Obs*	*Exp*	*SMR*
3	0·47	638	11	0·16	8	0·96	833	8	7·3	110
2	0·70	286	8	0·14	27	2·0	1350	12	13·5	89
2	0·46	435	5	0·06	15	1·9	789	19	10·3	184
0	0·28	0	0	0·02	10	1·2	833	9	6·4	141
7	1·9	366*	24	0·38	60	6·0	984*	48	37·5	128
0	0·02	0	0	0·06	0	0	0	0	0·37	0

Stomach cancer			*Tuberculosis*		*Respiratory disease*			*Ischaemic heart disease*		
Obs	*Exp*	*SMR*	*Obs*	*Exp*	*Obs*	*Exp*	*SMR*	*Obs*	*Exp*	*SMR*
2	0·35	571	5	0·06	8	1·2	667	3	7·2	42
1	0·62	161	9	0·12	15	2·1	714	22	12·8	172
2	0·69	290	7	0·15	24	2·0	1200	16	12·8	125
2	0·25	800	3	0·05	13	0·8	1625	7	4·7	149
7	1·91	366*	24	0·4	60	6·1	984†	48	37·5	128

to deaths from non-malignant respiratory diseases, and cancer. Mortality from lung cancer (13 observed, 5·1 expected, SMR 255; CI 136–436) and stomach cancer (3 observed, 1 expected) were both raised. Combined results from both follow up periods show a significant excess of both lung (SMR 230; CI 178–297) and stomach (SMR 188; CI 120–290) cancers. Deaths from colonic and rectal cancers were fewer than expected so that overall gastrointestinal cancer rates were not raised.

Three of the 49 men dying of lung cancer before 1979 had uranium mining experience; they were estimated to have had cumulative exposures to radon daughter decay products of 10, 29, and 126 working level months. The mortality pattern from 1979 to 1985 of all men with uranium mining experience compensated during 1960–75 is presented separately at the bottom of table 2.

MORTALITY EXPERIENCE OF SURFACE INDUSTRY WORKERS

Table 3 shows the mortality among the surface work-

Table 5 *Smoking habits among surface workers with silicosis*

Compensation cohort	*No of men*	*No with habits known*	*Never smokers*	*Current and former smokers*
1940–49	38	8 (21%)	0	8 (100%)
1950–59	83	53 (64%)	11 (20·8%)	42 (79·2%)
1960–69	70	61 (87%)	7 (11·5%)	54 (88·5%)
1970–84	85	84 (99%)	13 (15·5%)	71 (84·5%)
Totals	276	206 (74·6%)	31 (15%)	175 (85%)

ers for selected causes, by decade of compensation. All cause mortality was consistently about twice that expected, largely arising from excess deaths from tuberculosis and non-malignant respiratory diseases. In addition, a statistically significant excess of deaths due to malignancies ($p < 0·01$) was found. This was accounted for by excess deaths due to lung cancer (SMR 302; CI 173–489) and stomach cancer (SMR 366; CI 147–754). Deaths due to colonic and rectal cancers were the same as expected (3 observed, 2·94 expected). The five deaths occurring among the women in the cohort exceeded expectation (table 3); one death was due to lung cancer in a ceramics worker.

A similar pattern was observed when the mortality experience was examined by industry type (table 4), when a consistent excess of deaths due to lung cancer was seen in each industry.

Information on cigarette smoking was available from WCB records for 206 (75%) of the surface cohort and indicated that 15% were never-smokers (table 5). There was no substantial variation in habit by decade of birth or decade of compensation. Of those alive in 1975, 20% were never smokers. By comparison, Ontario statistics for 1975 indicate that the proportions of never smokers among men aged 45 to 64, and 65 and over were 23% and 33%, respectively (Statistics Canada, unpublished data).

Information about birth place was available for 272 (99%) of the male surface workers and showed that 52% were born outside Canada compared with 33·9% of Ontario residents (men aged 45 and over) in 1971.[10] There were increased proportions in our cohort of workers from countries with high rates of stomach cancer. In particular, 25% were born in the United Kingdom and 10% in Italy compared with 12·2% and 5·6%, respectively, for the male population of Ontario.

Discussion

Workers on the rolls of silicosis registries in six jurisdictions in Canada (this study) and Europe[1 3 4 11 12] have increased rates of lung cancer by comparison with regional or national populations. Although criteria for inclusion in the registries may have differed, one may reasonably conclude that an association exists between certification for silicosis and increased risk of lung cancer. The reasons for this association remain unclear.

When studying lung cancer, the first factor that must be considered is tobacco smoking. Silicosis registries might contain an increased proportion of smokers because blue collar workers may smoke more heavily than the general population, smoking may increase the chance of developing silicosis by interfering with clearance mechanisms, or because smoking related respiratory symptoms may increase the likelihood of certification for silicosis. In the present investigation of silicotic surface workers 15% of those with known smoking habits were reported to be "never smokers" compared with about 30% in the general population of Ontario. In the Veneto registry in Italy only 13% were non-smokers, a proportion believed to be lower than that in the Veneto region.[4] Might the overrepresentation of smokers in these cohorts account for the more than doubled risk of lung cancer?

The appendix shows a calculation in which a lung cancer risk factor of 20 was assumed for ever smokers versus never smokers. This calculation indicates that a 20% increased risk might be expected among the cohort of silicotics because of the difference in smoking prevalence. Blair *et al* compared crude and smoking adjusted lung cancer SMRs for selected occupational groups within a large cohort of United States veterans and found that adjustment for smoking generally resulted in a change of less than 30%.[13] It is concluded that differences in smoking habit alone are unlikely to account for the magnitude of the increased risk of lung cancer observed in the registry studies.

Concurrent exposure to silica might, however, increase the risk from smoking. There is experimental support for this hypothesis. Niemeier[14] and Stenback[15] both treated hamsters with intratracheal instillations of silica, benzo(a)pyrene (BaP), or BaP plus silica. Whereas silica produced no respiratory tumours, combined exposure to BaP and silica produced significantly more tumours than BaP alone. Exposure to silica may have increased the dose of BaP by adsorption on to the surface of the crystals or by

altering its clearance. BaP and other organic carcinogens are present in tobacco smoke and the same effect may occur in people.

In addition to tobacco smoke, workers with silicosis may have been exposed to other carcinogens. Since foundry workers are believed to be exposed to various carcinogens, they were deliberately omitted from this study, but in other studies lung cancer SMRs for foundry workers have not differed substantially from those of silicotics from other industrial settings.[1 3] In the present study silica brick workers may have had some exposure to carcinogens but the lung cancer SMR (based on small numbers) was similar to the average. In general, in registry studies the risk has been similar across occupational groupings so that if the increased risk is not due to smoking, silica, or silicosis, various similarly acting confounding exposures would have to be invoked in explanation.

Goldsmith *et al* proposed that silica itself might be a carcinogen or that the fibrotic process might be an intermediate stage on the way to cancer.[5] Several experiments provide data in support of these hypotheses. Hesterberg *et al* found that silica induced cytogenetic changes and morphological transformation of Syrian hamster embryo cells in culture, a preneoplastic change produced by exposure to a wide variety of physical and chemical carcinogens.[16] They observed that silica was readily taken up by these cells and accumulated in the perinuclear region, suggesting that chromosomal interaction could occur, especially during mitosis when the nuclear membrane disappears.

In animal studies lung tumours were produced in two strains of rats administered silica intratracheally[17 18] and in Fischer 344 rats exposed to silica concentrations of 50 and 12 mg/m^3 in inhalation chambers.[17 19] Lymphomas have also been induced in Wistar rats after intrapleural injection of silica.[20] By contrast with these observations in rats, investigators have been unable to produce silica induced tumours in hamsters. Saffiotti noted that another difference between these species in their response to silica is that rats develop fibrosis but hamsters do not.[21]

Quartz is thus a carcinogen in rats. What is the human evidence? Unfortunately, in most occupations in which there is substantial exposure to silica, concurrent exposure to other agents confounds the interpretation of any silica-cancer association. Miners, for example, may be exposed to radon daughters, fibres, or trace elements such as aresenic, whereas foundry workers may be exposed to polycyclic aromatic hydrocarbons. The quarry and granite industry is the one in which workers are likely to have the least exposure to other agents. Silicotics from this industry in the Ontario, Finnish,[3] and Italian[4] registries have had increased rates of lung cancer. On the other hand, a proportional mortality (PMR) study of Swiss silicotics found no increase in deaths from lung cancer in stone workers.[11]

There have been two PMR studies of granite workers in the United States. Davis *et al* studied mortality in the Vermont granite industry.[22] A small excess (20%) of lung cancer was observed, but there was no apparent dose-response relation. Steenland and Beaumont found a similar excess of lung cancer among members of the Granite Cutters Union.[23] No relation with duration of union membership was found, but men dying of lung cancer were more likely to have silicosis mentioned on the death certificate than men dying of other cancers.

Excess deaths from stomach cancer were observed in both the miners and surface industry workers in the Ontario cohort. There are associations between birth place,[8] tobacco smoking,[24] and stomach cancer. There was an overrepresentation in this cohort of smokers and of individuals from countries with high mortality from stomach cancer, but adjustments for these factors similar to the one shown in the appendix were unable to account for the magnitude of the excess.

An association between stomach cancer and exposure to silica has not been consistently observed. Kurppa *et al* reported an excess of stomach cancer in a group of Finnish quarry workers[6] but did not present a stomach cancer SMR in the Finnish registry study[3] (the gastrointestinal cancer SMR was not raised). Fewer deaths from stomach cancer than expected were found in the Italian registry[4] but stomach cancer mortality was significantly raised among dust exposed workers in Vienna.[12] Excess stomach cancer was observed among gold miners in Ontario[7] but there was a deficit among gold miners in the United States.[25] It is not yet possible to account for these discrepant findings.

In summary, a consistent excess of lung cancer has been found in studies of silicosis registries in Canada and Europe. The reasons for this association are still unclear. All these workers were exposed to silica and developed silicosis but many had concurrent occupational exposures to other agents and most were cigarette smokers. Further study of the health experience of silica exposed populations, preferably in a setting in which dose-response relations can be investigated, would be helpful in evaluating the part that silica plays in this association.

Appendix

ADJUSTMENT OF "EXPECTED" LUNG CANCERS FOR DIFFERENCES IN SMOKING HABIT

The population of surface industry silicotics consisted

of never smokers plus a mixture of current and former smokers. Assume that the lung cancer risk factor for ever smokers compared with never smokers is 20. One may then compare the risks between populations containing 15% (this cohort) and 30% (general population) never smokers respectively, using the method of Axelson[9]

$$I = I_{CF} \times P_{CF} + I_0(1 - P_{CF})$$

where

I = overall incidence (deaths) of lung cancer

I_{CF} = incidence as caused by the confounding factor (smoking)

P_{CF} = proportion of population with the factor in question (smokers)

I_0 = incidence among those without the risk factors (never smokers)

If the effect of the confounding factor is known (here $R = 20$ is chosen) the formula may be rewritten as:

$$I = RI_0 \times P_{CF} + I_0(1 - P_{CF}) .$$

Therefore in the general population

$$I_G = (20)(I_0)(0{\cdot}7) + I_0(0{\cdot}3)$$
$$= 14{\cdot}3\ I_0$$

In the study population

$$I_S = (20)(I_0)(0{\cdot}85) + I_0(0{\cdot}15)$$
$$= 17{\cdot}2\ I_0$$

Therefore the relative risk in the study population due to a greater prevalence of smokers is

$$\frac{I_S}{I_G} = \frac{17{\cdot}2}{14{\cdot}3} = 1{\cdot}2.$$

We thank Ms Lisa Fiorello of the Workers' Compensation Board for help with the follow up of the miners. Ms Brenda Crysler typed this report.

References

1 Westerholm P. Silicosis. Observations on a case register. *Scand J Work Environ Health* 1980;**6** (suppl 2):1–86.

2 Finkelstein MM, Kusiak RA, Suranyi G. Mortality among miners receiving workmen's compensation for silicosis in Ontario: 1940–1975. *J Occup Med* 1982;**24**:663–7.

3 Kurppa K, Gudbergsson H, Hannunkari I, *et al.* Lung cancer among silicotics in Finland. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis, and cancer*. New York: Praeger, 1986:311–9.

4 Zambon P, Simonato L, Mastrangelo G, *et al.* A mortality study of workers compensated for silicosis during 1959 to 1963 in the Veneto region of Italy. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis, and cancer*. New York: Praeger, 1986: 367–4.

5 Goldsmith DF, Guidotti TL, Johnston DR. Does occupational exposure to silica cause lung cancer? *Am J ind Med* 1982;3:423–40.

6 Kurppa K, Koskela RS, Gudbergsson H. Gastrointestinal cancer in workers exposed to quartz. *Lancet* 1982;ii:150.

7 Muller J, Wheeler WC, Gentleman JF, Suranyi G, Kusiak R. Study of mortality of Ontario miners. In: Stocker H, ed. *Occupational radiation safety in mining*. Toronto: Canadian Nuclear Association 1985:335–43.

8 Newman AM, Spengler RF. Cancer mortality among immigrant populations in Ontario, 1969 through 1973. *Can Med Assoc J* 1984;**130**:399–405.

9 Axelson O. Aspects of confounding in occupational health epidemiology. *Scand J Work Environ Health* 1978;**4**:85–8.

10 Statistics Canada. *1971 census of Canada*. Vol 1, pt 4. *Population, catalogue 92–737*. Ottawa, Statistics Canada, 1974.

11 Schuler G, Ruttner JR. Silicosis and lung cancer in Switzerland. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis, and cancer*. New York: Praeger, 1986:357–66.

12 Neuberger M, Kundi M, Westphal G, Grundorfer W. The Viennese dusty worker study. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis and cancer*. New York: Praeger, 1986:415–22.

13 Blair A, Hoar SK, Walrath J. Comparison of crude and smoking-adjusted standardized mortality ratios. *J Occup Med* 1985; **27**:881–4.

14 Niemeier RW, Mulligan LT, Rowland J. Cocarcinogenicity of foundry silica sand in hamsters. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis and cancer*. New York: Praeger, 1986:215–28.

15 Stenback F, Wasenius VM, Rowland J. Alveolar and interstitial changes in silicate-associated lung tumors in Syrian hamsters. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis and cancer*. New York: Praeger, 1986:199–214.

16 Hesterberg TW, Oshimura M, Brody AR, Barrett JC. Asbestos and silica induce morphological transformation of mammalian cells in culture. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis and cancer*. New York: Praeger, 1986:177–90.

17 Holland LM, Wilson JS, Tillery MI, Smith DM. Lung cancer in rats exposed to fibrogenic dusts. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis and cancer*. New York: Praeger, 1986:267–80.

18 Groth DH, Stettler LE, Platek SF, Lal JB, Burg JR. Lung tumors in rats treated with quartz by intratracheal instillation. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis and cancer*. New York: Praeger, 1986:243–54.

19 Dagle GE, Wehner AP, Clark ML, Buschbom RL. Chronic inhalation exposure of rats to quartz. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis and cancer*. New York: Praeger, 1986:255–66.

20 Wagner MMF, Wagner JC. Lymphomas in the Wistar rat after intrapleural inoculation of silica. *JNCI* 1972;**49**:81–91.

21 Saffiotti U. The pathology induced by silica in relation to fibrogenesis and carcinogenesis. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis and cancer*. New York: Praeger, 1986:287–310.

22 Davis LK, Wegman DH, Monson RR, Froines J. Mortality experience of Vermont granite workers. *Am J Ind Med* 1983;**4**:705–23.

23 Steenland K, Beaumont J. A proportionate mortality study of granite cutters. *Am J Ind Med* 1986;**9**:189–201.

24 Surgeon General. *The health consequences of smoking. Cancer*. Rockville: US Department of Health and Human Services, 1982:132–7.

25 Brown DP, Kaplan SD, Zumwalde RD, Kaplowitz M, Archer VE. Retrospective cohort mortality study of underground gold mine workers. In: Goldsmith DF, Winn DM, Shy CM, eds. *Silica, silicosis and cancer*. New York: Praeger, 1986:335–50.