

ORIGINAL ARTICLE

Non-malignant mortality among Norwegian silicon carbide smelter workers

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Occup Environ Med 2002;**59**:345–347

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17 October 2001**Objectives:** To investigate associations between exposures in the silicon carbide (SiC) industry and mortality from non-malignant diseases.**Methods:** Mortality among 2562 men, working in one of three silicon carbide smelters was investigated, giving 52 618 person-years of follow up from 1962 to 1996. Dose-response relations were investigated by internal comparisons using Poisson regression and by stratified standardised mortality ratio (SMR) analyses.**Results:** Mortality from all causes was significantly raised compared with the Norwegian mortalities among men, SMR=1.12, (95% confidence interval (95% CI) 1.05 to 1.20). An excess mortality from asthma, emphysema, and chronic bronchitis combined was found, SMR=2.21 (95% CI 1.61 to 2.95), increasing from 1.05 in the unexposed category to 2.64 (95% CI 1.44 to 4.43) in the upper category of exposure to total dust. The Poisson regression analysis confirmed the results from the stratified SMR analyses, and suggested that smoking did not act as a confounder. No association was found for circulatory mortality.**Conclusions:** There was an increased mortality from asthma, emphysema, and chronic bronchitis combined among SiC workers exposed to dust.

Silicon carbide (SiC) is produced by heating a mixture of quartz and petrol coke to more than 2000°C in open electrical resistance furnaces. Workers in the process departments are exposed to SiC fibres, SiC particulates, crystalline silica, carbon monoxide (CO), and sulphur dioxide (SO₂) as well as small amounts of polycyclic aromatic hydrocarbons (PAHs).

Silicon carbide fibres have shown carcinogenic and fibrogenic properties in animal experiments comparable with or greater than those of asbestos.¹ Contrary to the fibres, angular (non-fibrous) SiC materials have only shown weak or no toxic properties in experimental studies.^{2,3}

In 1941 a case of severe silicosis was reported in a Norwegian smelter. Subsequently, all the production workers were examined for silicosis, and in 1948 Bruusgaard reported that a total of 49 of the 222 workers examined had pneumoconiotic changes.⁴

More recent morbidity studies among SiC workers have also shown pneumoconiotic changes and other respiratory symptoms.^{5,6}

Only one mortality study has earlier been published from this industry.⁷ The study showed an excess mortality of both lung cancer and non-malignant respiratory diseases among Canadian silicon carbide production workers exposed to dust.⁷

As well as the dust problem, SiC production leads to high concentrations of CO in the working atmosphere. This represents a serious acute hazard, with several episodes of CO intoxications. The long term effects of exposure to CO are less certain, but it has been suggested that it is a risk factor for ischaemic heart disease.⁸

Recently a job exposure matrix for three Norwegian silicon carbide plants was established.⁹ Based on this, a dose-response relation was shown between exposure to dust containing SiC fibre and crystalline silica and an increased incidence of lung cancer.⁹ The purpose of the present study was to study non-malignant mortality among SiC smelter workers, using employees' working history, the established job exposure matrix, and data on smoking.

MATERIALS AND METHODS

The present study comprises three SiC plants in Norway; A started operation in 1913; B in 1963; and C in 1965. A job exposure matrix covering all three plants had already been established. Further details of the exposure assessment, levels of exposure, data on smoking, the process, and the smelters are given elsewhere.⁹

Information on each employee was obtained from company records. The company records included 2720 men. Of these, 98 men were found to have died before 1961, and 60 men (3%) were not traceable. This left 2562 men identified by a unique personal identification number to the study.

Cumulative exposure was used as an indicator of individual dose and potential induction and latency periods were investigated by lagging exposure by 20 and 40 years.¹⁰

The follow up of mortality started after 6 months of employment (net employment time) or from 1 January 1962 if the 6 months date of employment was reached before that date. Observation continued until 31 December 1996 or to the time of death or emigration. The cohort contributed 52 618 person-years at risk to the study.

The grouping of causes of death was based on the World Health Organisation, 9th revision of the international classification of diseases (ICD-9), three digit code.

Cause specific mortality was analysed by calculation of the standardised mortality ratio (SMR) where the expected numbers were calculated from the biannual national mortality among men by 5 year age groups. The homogeneity in the SMRs across age and calendar period strata was checked with a χ^2 goodness of fit test in the program package StatXact-4.

Poisson regression was used for the investigation of internal dose-response relations and for the exploration of potential confounding from smoking. Age was included in the models

Abbreviations: SiC, silicon carbide; PAH, polycyclic aromatic hydrocarbons; ICD-9, 9th revision of the international classification of diseases

Table 1 Observed (Obs) and expected (Exp) number of cause specific deaths and standardised mortality ratio (SMR) among 2562 male Norwegian SiC smelter workers in the follow up period 1962–96

Cause of death	ICD-9 codes	Obs	Exp	SMR	95% CI
All causes	1–999	847	753.6	1.1	1.1 to 1.2
Cancer	140–209	204	173.0	1.2	1.0 to 1.4
Circulatory diseases	390–459	376	371.7	1.0	0.9 to 1.1
Ischaemic heart disease	410–414	208	234.9	0.9	0.8 to 1.0
Cerebrovascular disease	431–438	91	71.6	1.3	1.0 to 1.6
Sudden death	798	37	24.3	1.5	1.1 to 2.1
Respiratory diseases	460–519	77	56.6	1.4	1.1 to 1.7
Asthma, chronic bronchitis, emphysema	490–493, 496	45	20.4	2.2	1.6 to 3.0
Pneumoconiosis	501–503, 505	6	0.8	7.9	2.9 to 17.1
Pneumonia	480–483, 485–486, 507	24	30.7	0.8	0.5 to 1.2
Digestive diseases	520–577	17	18.0	0.9	0.5 to 1.4
External causes	E800–999	58	49.1	1.2	0.9 to 1.5

Table 2 Observed (Obs) and expected (Exp) number of deaths from chronic obstructive lung diseases (asthma, chronic bronchitis, and emphysema) and standardised mortality ratio (SMR) by cumulative exposure to total dust (mg/m³.y) and duration of employment

Cumulative exposure to total dust (mg/m ³ .y)	All employees				Employment >3 y			
	Obs	Exp	SMR	95% CI	Obs	Exp	SMR	95% CI
0	3	2.9	1.1	0.2 to 3.1	1	2.2	0.5	0.0 to 2.5
0–14.9	13	5.0	2.6	1.4 to 4.4	2	1.1	1.8	0.2 to 6.6
15–69.9	15	7.2	2.1	1.2 to 3.4	12	5.6	2.1	1.1 to 3.7
>70	14	5.3	2.6	1.4 to 4.4	14	5.3	2.6	1.4 to 4.4

using six age groups (<55, 55–59, 60–64, 65–69, 70–79, ≥80), and period of diagnosis was included using three calendar periods (1962–70, 1971–84, and 1985–96).

Potential confounding by factors related to region of residence was investigated in supplementary internal analyses including indicator variables for plant.

Trend tests were performed by assigning scores from 1 to 4 to the four exposure categories, or by applying the mean cumulative exposure to the exposure categories.

The calculations of SMR and the Poisson regression analyses were performed with the program package “EPICURE”.¹¹

RESULTS

The total number of deaths among SiC workers was significantly increased compared with the expected figures (table 1), SMR 1.12 (95% CI 1.05 to 1.20). An excess mortality from cancer, SMR 1.18 (95% CI, 1.03 to 1.35), and an excess mortality from non-malignant respiratory diseases, SMR 1.36 (95% CI 1.07 to 1.70) was found. Mortality from circulatory disease was close to that expected (SMR 1.01, (95% CI 0.91 to 1.12)), whereas the mortality from cerebrovascular diseases and sudden death were increased. We found no association between cumulative exposure to CO and mortality from ischaemic heart disease, nor between exposure to CO and mortality from cerebrovascular diseases or sudden death (results not shown). A substantial excess mortality from asthma, emphysema, and chronic bronchitis combined was found (SMR 2.21 (95% CI 1.61 to 2.95)). The overall mortality pattern was similar for all the plants.

There were indications of a positive association between cumulative exposure to total dust and mortality from chronic obstructive lung disease, which apparently became clearer when restricting the analyses to workers employed for more than 3 years (table 2). Lagging of exposure did not add to the evidence.

Mortality from obstructive lung disease was further investigated in a Poisson regression analysis (table 3). The analysis showed essentially the same results as the stratified SMR analysis, and suggested that confounding from smoking was probably minor. In an additional internal analysis of chronic obstructive lung disease, we included the contributing causes of death together with the underlying cause. This analysis showed similar results to the precedent analyses (results not shown).

There were also more deaths from pneumoconiosis than expected (SMR 7.9 (95% CI 2.9 to 17)), based on five observed deaths from silicosis and one death due to asbestosis restricted to one of the plants. The five deaths from silicosis were all people employed for more than 25 years in the SiC industry, whereas the single death from asbestosis was a worker employed for less than 1 year.

DISCUSSION

The present study showed an overall increased mortality from asthma, chronic bronchitis, and emphysema among SiC production workers, which increased by increasing cumulative exposure to dust. These findings support the results from a mortality study among 585 SiC workers in Canada which also showed an increased mortality from non-malignant respiratory diseases (SMR 2.2, 95% CI 1.2 to 3.2).⁷

The investigation of mortality from respiratory diseases is problematic due to the possible selection of healthy workers into the workforce, and the selection of susceptible people out of exposed jobs. This bias is difficult to control as it can be related to both duration of work and to levels of exposure, which is the main component of the cumulative exposure index. Such selection may therefore seriously weaken a potential dose-response relation. Furthermore the fact that several workers died before the start of follow up may have

Table 3 Poisson regression analysis of mortality (rate ratio) from asthma, chronic bronchitis, and emphysema combined by smoking habit and cumulative exposure to total dust (mg/m³.y), controlled for age (<65, 65–69, 70–74, 75–79, ≥80) and calendar period (1962–79, 1980–96) (restricted to workers employed for >3 y)

	Deaths (n)	Smoking included		Smoking not included	
		Rate ratio	95% CI	Rate ratio	95% CI
Smoking status:					
Never	3	1.0	Reference	–	–
Former	2	2.0	0.3 to 12.1	–	–
Smoker	22	3.4	1.0 to 11.5	–	–
Unknown	2	1.6	0.3 to 9.8	–	–
Exposure to total dust (mg/m ³ .y):					
0	1	1.0	Reference	1.0	Reference
0.1–14.9	2	3.0	0.3 to 33.4	3.7	0.3 to 40.9
15–69.9	12	4.5	0.6 to 34.4	4.5	0.6 to 35.0
≥70	14	5.1	0.7 to 38.9	5.5	0.7 to 42.1
Evaluation of linear trend:					
Scores 1,2,3,4			p Value		p Value
Mean dose*		0.10	0.07	0.16	0.08

*Mean dose; arithmetic mean cumulative exposure of total dust.

resulted in a left censoring of the data. For instance, it was known that 17 people with a confirmed diagnosis of silicosis died before 1961.

In the present study, neither mortality from ischaemic heart diseases nor mortality from cerebrovascular diseases was associated with agents in the work environment. However, also for circulatory diseases the selection of healthy people into employment and survival of the fittest in employment may lead to bias, distorting a possible relation.

High correlation between the various exposures to dust made it difficult to separate potential effects posed by the different types of exposures. It is, however, likely that several of the agents in the work environment have been of importance for our findings. The deaths from pneumoconiosis are likely to have been caused by exposure to crystalline silica and SiC fibres. The observed increased mortality from asthma, chronic bronchitis, and emphysema is probably related to the high overall exposures to dust and possibly exposure to SO₂.

Despite being based on a substantial number of measurements, the estimation of exposure was inevitably subject to uncertainty.⁹ As this is a cohort study and the estimation of exposure can be assumed to be unrelated to disease, differential misclassification of exposure is unlikely.

Smoking is a major risk factor for the diseases of interest, but the results did not seem to have been seriously confounded by smoking. The effect estimates in the internal subanalyses were only slightly affected after the inclusion and adjustment for smoking.

As mortality varies in different parts of Norway, our use of national rates may have biased the overall SMR. The potential bias introduced with national rates has, however, probably not affected the dose-response analyses as the additional internal analyses including indicator variables for plant did not lead to essential changes in the effect estimates.

In conclusion we found an increased mortality from obstructive lung diseases (asthma, emphysema, and chronic bronchitis) among workers exposed to dust in the Norwegian SiC industry. No association was found for circulatory mortality.

ACKNOWLEDGEMENTS

This work was part of a project supported by grants from the Work Environment Fund of the Confederation of Norwegian Business and Industry. We acknowledge the support from Ole Tormod Fure at the Safety, Health, and Environment Secretariat for the Norwegian smelters. We thank Jan Ivar Martinsen for valuable help in programming and data analyses and Geir Helland-Hansen for the collection and handling of data. We are grateful to Tor Enger for his support.

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