Non-malignant mortality among Norwegian silicon carbide smelter workers

P Romundstad, A Andersen, T Haldorsen

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.

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

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.

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.

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 χ² 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 as a confounder.

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

<table>
<thead>
<tr>
<th>Cause of death ICD-9 codes</th>
<th>Obs</th>
<th>Exp</th>
<th>SMR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>All causes 1-999</td>
<td>847</td>
<td>753.6</td>
<td>1.1</td>
<td>1.1 to 1.2</td>
</tr>
<tr>
<td>Cancer 140-209</td>
<td>204</td>
<td>173.0</td>
<td>1.2</td>
<td>1.0 to 1.4</td>
</tr>
<tr>
<td>Circulatory diseases 390-459</td>
<td>376</td>
<td>377.7</td>
<td>1.0</td>
<td>0.9 to 1.1</td>
</tr>
<tr>
<td>Ischaemic heart disease 410-414</td>
<td>208</td>
<td>234.9</td>
<td>0.9</td>
<td>0.8 to 1.0</td>
</tr>
<tr>
<td>Cerebrovascular disease 431-438</td>
<td>91</td>
<td>71.6</td>
<td>1.3</td>
<td>1.0 to 1.6</td>
</tr>
<tr>
<td>Sudden death 798</td>
<td>37</td>
<td>24.3</td>
<td>1.5</td>
<td>1.1 to 2.1</td>
</tr>
<tr>
<td>Respiratory diseases 460-519</td>
<td>77</td>
<td>56.6</td>
<td>1.3</td>
<td>1.1 to 1.7</td>
</tr>
<tr>
<td>Asthma, chronic bronchitis, emphysema 490-493, 496</td>
<td>45</td>
<td>20.4</td>
<td>2.2</td>
<td>1.6 to 3.0</td>
</tr>
<tr>
<td>Pneumoconiosis 501-503, 505</td>
<td>6</td>
<td>8.0</td>
<td>0.79</td>
<td>2.9 to 17.1</td>
</tr>
<tr>
<td>Pneumonia 480-483, 485-486, 507</td>
<td>24</td>
<td>30.7</td>
<td>0.8</td>
<td>0.5 to 1.2</td>
</tr>
<tr>
<td>Digestive diseases 520-577</td>
<td>17</td>
<td>18.0</td>
<td>0.9</td>
<td>0.5 to 1.4</td>
</tr>
<tr>
<td>External causes E800-999</td>
<td>58</td>
<td>49.1</td>
<td>1.2</td>
<td>0.9 to 1.5</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Cumulative exposure to total dust (mg/m³.y)</th>
<th>All employees</th>
<th>Employment &gt; 3 y</th>
</tr>
</thead>
<tbody>
<tr>
<td>All employees</td>
<td>Obs</td>
<td>Exp</td>
</tr>
<tr>
<td>0</td>
<td>1</td>
<td>2.2</td>
</tr>
<tr>
<td>0–14.9</td>
<td>13</td>
<td>5.0</td>
</tr>
<tr>
<td>15–69.9</td>
<td>15</td>
<td>7.2</td>
</tr>
<tr>
<td>&gt;70</td>
<td>14</td>
<td>5.3</td>
</tr>
</tbody>
</table>

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. 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). There was an indication of a positive association between cumulative exposure to 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.

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”.  

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.
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.

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.

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