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Pulmonary impairment in workers exposed to silicon carbide

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Abstract

Two hundred and sixty seven workers employed in the manufacture of silicon carbide (SiC) were examined to determine the effects of exposure to contaminants (SiC, quartz, and SO_2) in the workplace on pulmonary function. No exposure concentrations exceeded the current permissible limits. Ten subjects (3·7%) showed rounded opacities (profusion $\geqslant$ 1/0). Two subjects employed only in the final stages of the production process and not exposed to crystalline silica showed opacities (profusion q1/0 and q2/1) on *x* ray film suggesting a role of SiC in the genesis of interstitial lung disease. Chest abnormalities on *x* ray film were correlated with cumulative exposure to dust and pulmonary function was affected by cumulative dust exposure, profusion of opacities, and smoking. It is concluded that the current standards do not provide adequate protection against pneumoconiosis and chronic pulmonary disease in this industry.

In recent years several epidemiological and clinical investigations have suggested that workers employed in silicon carbide (SiC) manufacture may present with specific occupational lung disorders when exposed to contaminants not exceeding current standards.[1–3]

This study was conducted to determine the pulmonary effects of exposure to contaminants (SiC, quartz, SO_2) developed during the production of SiC abrasive (carborundum).

Material and methods

SILICON CARBIDE PROCESS

The manufacturing process consists of running an electric current through a graphite electrode that lies in a mixture of petroleum coke, high purity crystalline silica, and sawdust. The plant investigated used Acheson furnaces, which heated the mixture to 2300°C. Silica reacts with the carbon in the coke and produces SiC and CO.

The production process includes: (1) mixture of raw materials; (2) charge of furnaces; (3) firing; (4) cooling; (5) disassembling of furnaces. The lumps of SiC are then broken with pneumatically powered chisels. The final products are crushed, screened into several sizes, and stored before shipping out to users.

Six four-furnace batteries were in the plant: while one furnace was firing, the others within each battery were at the cooling or disassembling stage. The exposure to contaminants was variable with time; no one worked on the furnace during the heating cycle, but many worked nearby. The heating cycle was performed at night and during the weekend to reduce the power cost.

Workers employed in the manufacture of SiC may be exposed to several contaminants including silica and SiC dust, SO_2 resulting from impurities of the coke, and CO which burns at the surface of the furnaces. Hydrocarbon fumes are also released during pyrolysis of the petroleum coke.

EXPOSURE EVALUATION

Exposures were measured by personal sampling and sample collections were grouped by job category. A total of 135 samples were collected, 120 to measure respirable dusts and 15 to measure exposure to SO_2. Respirable particulate dust was collected on a weighed filter of porosity 0·8 μm made of acetate and cellulose nitrate; the filters were conditioned, before and after sampling, at 100°C for two hours to eliminate absorbed humidity and weighed on a precision scale to seven decimal places. Each filter was pretreated in a Lippmann cyclone to remove non-respirable particles. Quartz and crystobalite were measured by absorption in hydrogen peroxide and titration of the resulting sulphuric acid solution according to the method of the National Institute for Occupational Safety and Health.[4] Air was drawn at 0·8 l/min through impingers containing hydrogen peroxide.

Individual cumulative exposures to respirable

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Table 1 Concentrations (mg/m³) of respirable dusts, quartz, and crystobalite by job category

Job category	No of samples	Respirable dust		Quartz (range)	Crystobalite (range)
		Geometric mean	Range		
Mixer	12	0·85	0·14–2·97	0–0·037	0–0·023
Furnace	39	0·34	0·11–1·25	0–0·003	0–0·010
Screen 1	13	0·78	0·16–2·95	absent	0–0·018
Crusher	14	0·30	0·13–0·86	absent	0–0·012
Grinding	7	0·50	0·14–1·50	absent	0–0·008
Screen 2	31	1·00	0·10–7·82	absent	absent
Storage	4	0·44	0·30–0·63	absent	absent

dusts and SO_2 were calculated as suggested by Peters *et al*[1]; the product of the time spent in each job and its mean exposure concentration was expressed as mg/m³ × years for cumulative dust and ppm × years for cumulative SO_2).

SUBJECTS

We examined 141 subjects at work in the factory during February 1988. Attempts were made to contact 164 workers employed in the past in SiC production for at least two years between 1983 and 1987, to act as controls; twelve of these subjects were dead and 26 refused to participate, leaving a final control group of 126 ex-workers.

HEALTH EFFECTS

The investigation was carried out during five consecutive days in February 1988. Relevant information on health and smoking habits was obtained from the questionnaire of the European Community of Coal and Steel that deals with chronic bronchitis and emphysema.[5] This was administered by three trained interviewers who also completed forms describing the subjects' past exposure in dusty industries, as well as present and past jobs in the production of SiC.

Tests of pulmonary function were performed with a computerised spirometer in the first aid room of the factory. Forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were measured with the subject in a seated position wearing a nose clip. The maximum value was taken from three satisfactory tracings; values were adjusted for body temperature, pressure, and saturation. The predicted values used were those quoted by the European Community of Coal and Steel.[6]

Standard posteroanterior chest radiographs were taken according to International Labour Office (ILO) standards.[7] *x* Ray films were read by two independent physicians and classified by the ILO international classification of radiographs for pneumoconiosis[7] without knowledge of age, exposure, or other information.

STATISTICAL ANALYSIS

The FVC and FEV_1 were presented as a percentage of the predicted values to adjust for differences in age and height; these values were then grouped according to several variables—namely, smoking, particulate exposure, exposure to SO_2, and profusion of radiographic opacities. Non-parametric one way analysis of variance (Kruskal-Wallis test[8]) was performed to assess the significance of group differences. In tables 4–8 the p values generated from the Kruskal-Wallis test are reported.

A binary (0, 1) dependent variable was created in which 1 and 0 indicates *x* ray film profusion categories higher or equal to 0/1 and 0 respectively; a stepwise logistic regression[8] was then fitted to assess the effect of the independent variables: age, smoking, years worked, particulate exposure, and exposure to SO_2 on radiographic profusion. A multiple linear regression[8] was used to find the best subset of predictor variables for FVC and FEV_1 among these independent variables.

Results

EXPOSURES TO AIRBORNE DUSTS AND SO_2

Table 1 shows the concentrations of respirable dusts. Average exposures were all below the existing standards for permissible exposure; in some cases high values in the screening, mixing, and selection areas were found. The concentrations of quartz and crystobalite were always low. The geometric mean concentration of SO_2 was 0·05 ppm, with a maximum concentration of 0·4 ppm in the furnace.

SUBJECTS

The final study group consisted of 267 subjects (141 workers and 126 ex-workers). Table 2 reports the average age, smoking habits, years worked, and

Table 2 Age, smoking and exposure data for 267 SiC production workers

Age (y)	45·9 (12·7)
Never smokers (%)	38·8
Current smokers (%)	40·1
Ex-smokers (%)	21·0
Years worked	15·2 (8·9)
Cumulative dust exposure (mg/m³ × years)	9·7 (12·1)
Cumulative SO_2 exposure (ppm × years)	0·6 (1·3)

Standard deviation in parentheses.

Table 3 Frequency of radiographic abnormalities in workers (n = 141) and ex workers (n = 126)

Profusion	Workers No (%)	Ex-workers No (%)	Total No (%)
None	130 (92·2)	110 (87·3)	240 (89·9)
0/1	8 (5·7)	9 (7·1)	17 (6·4)
1/0	2 (1·4)	1 (0·8)	3 (0·1)
≥1/1	1 (0·7)	6 (4·8)	7 (2·6)

Table 4 Average cumulative particulate exposure (mg/m³ × years) by radiographic profusion

Profusion	No of cases	Particulate exposure geometric mean (SD)
None	240	8·8 (11·1)
0/1	17	13·7 (13·9)
1/0	3	29·8 (30·8)
1/1	3	20·4 (15·5)
>1/1	4	25·3 (20·1)

$p < 0·03$.

Table 5 Effect of smoking on pulmonary function

	Smokers (% predicted) n = 163	Never smokers (% predicted) n = 104	p Value
FVC	94·0	97·1	<0·08 NS
FEV_1	96·4	104·2	<0·0002

cumulative exposure to dust and SO_2. Most (92·9%) of the ex-worker group were distributed in the age classes over 50 years. Duration of exposure of more than 20 years was found in 54·0% of ex-workers and 23·4% of workers.

The percentage of smokers was similar (41·1% and 38·9%) but more ex-smokers were in the ex-worker group (25·4%) then in the worker group (17·1%). Smokers and ex-smokers were considered together in the analyses.

RADIOGRAPHIC FINDINGS

Table 3 reports the profusion of radiographic opacities in worker and ex-worker groups. Ten subjects (three workers and seven ex-workers) had average profusions ≥1/0 and seven (one worker and six ex-workers) ≥1/1. Among subjects with profusion ≥1/1, three ex-workers had 1/1, two 1/2, and one 2/1 profusion. One subject at work had a 2/2 profusion. Linear opacities (profusion 1/0) were found in one case only (worker group).

The duration of exposure in workers with opacities profusion ≥1/1 was 19 (SD 7·8) years (range 8–29).

The average cumulative exposure to respirable particulates increased significantly ($p < 0·03$) with profusion (table 4). No relation between years at work and cumulative exposure to SO_2 was found.

Table 5 shows the effect of smoking habits on pulmonary function (FVC and FEV_1). The values are presented as a percentage of those predicted. Significant differences ($p < 0·0002$) between smokers and never smokers were found for FEV_1 measures.

Tables 6 and 7 present the degrees of pulmonary function related to cumulative exposure. Cumulative exposure to respirable particulates affected FVC and FEV_1 significantly but only in smokers ($p < 0·0015$ and $p < 0·0027$ respectively). No significant effect of cumulative exposure to SO_2 was shown for either smokers or never smokers. The influence of the

Table 6 Effect of cumulative particulate exposure (mg/m³ × years) on percentage predicted pulmonary function by smoking

Particulate exposure	Smokers No	Smokers FVC%	Smokers FEV_1%	Never smokers No	Never smokers FVC%	Never smokers FEV_1%
≤3·6	75	96·7	98·8	50	97·0	104·0
3·7–7·4	20	98·5	102·6	13	103·7	112·5
7·5–14·9	30	94·3	98·2	22	97·5	104·3
≥15	38	86·1	86·9	19	92·3	99·0
p Value		<0·0015	<0·0027		NS	NS

Table 7 Effect of cumulative exposure to SO_2 (ppm × years) on percentage predicted pulmonary function by smoking

SO_2 exposure	Smokers No	Smokers FVC%	Smokers FEV_1%	Never-smokers No	Never-smokers FVC%	Never-smokers FEV_1%
≤0·37	8	93·6	97·5	7	98·1	102·5
0·38–0·74	7	90·5	92·4	6	99·9	105·4
0·75–2·2	34	98·1	98·9	21	96·9	104·0
>2·2	114	93·0	95·8	70	96·8	104·3
p Value		NS	NS		NS	NS

Table 8 Percentage predicted pulmonary function by profusion of radiographic opacities and smoking

Profusion	Smokers No	Smokers FVC%	Smokers FEV_1%	Never smokers No	Never smokers FVC%	Never smokers FEV_1%	Total No	Total FVC%	Total FEV_1%
None	141	95·7	98·4	99	97·3	104·3	240	96·8	100·8
0/1	15	87·1	87·1	2	101·7	115·5	17	89·5	90·5
1/0	2	88·8	83·1	1	75·6	86·9	3	84·4	84·4
≥1/1	5	72·8	73·1	2	87·1	98·1	7	76·9	80·2
p Value		<0·0029	<0·0017		NS	NS		<0·001	<0·001

Table 9 Significant independent variables identified by logistic regression and multiple linear regression

Dependent variable	*Independent variable*	*Improvement χ^2*	*T-stat*	*p Value*
		Logistic regression		
Profusion	Particulate exposure	6·232	—	<0·013
		Multiple linear regression		
% Predicted FVC	Particulate exposure	—	−3·03	<0·003
	Profusion	—	−3·04	<0·003
% Predicted FEV_1	Particulate exposure	—	−3·56	<0·0001
	Profusion	—	−2·90	<0·004
	Smoking	—	−2·22	<0·027

duration of exposure on pulmonary function was not significant.

Table 8 shows parameters of pulmonary function broken down by radiographic profusion in smokers and never smokers. A significant negative trend between both FVC and FEV_1 and profusion of opacities was found in the total population ($p < 0·001$ in both cases) and in the smokers ($p < 0·0029$ and $p < 0·0017$ respectively) but not in never smokers alone.

Table 9 summarises the results of multiple regression to determine the major effects of independent variables on pulmonary function and profusion of opacities. The profusion of opacities related significantly to cumulative exposure to dust. Both FVC and FEV_1 had a significantly negative relation with cumulative exposure to dust and profusion of opacities; FEV_1 was also associated with smoking habit.

Discussion

Silicon carbide is generally considered inert in human lungs.[9] Research conducted with laboratory animals supports this view; when exposed to a high concentration of SiC dust they failed to develop fibrosis.[10] Nevertheless, abnormal chest radiographs among the workers exposed to SiC have been described.[11–13] In more recent years investigations in the carborundum industry have documented an excess of radiographic abnormalities (compatible with pneumoconiosis) and a significant reduction of pulmonary functions. These were related either to cumulative exposure to dust and SO_2 or to smoking habits[1] in conditions in which the exposure concentrations were substantially below the threshold limit value.[14] Finally, Osterman *et al* found a statistically significant loss of pulmonary function related to duration at work in production of SiC, which was independent of smoking habits.[3]

The pneumoconiosis in workers involved with SiC production has generally been attributed to crystalline silica used in the process. Yet Funahashi *et al*[2] conducted a study using *x* ray diffraction analysis of open lung biopsies on two SiC production workers with diffuse reticulonodular densities on radiographs. The authors identified six different silicon carbides but not significant amounts of quartz. They interpreted these findings as suggesting that some silicon carbides are more fibrogenic than others. Recent findings, however, raised the possibility that mineral dusts that are inert when in a particulate form, may have biological activity when they occur in a fibrous form.[15 16] Masse *et al*[17] and Durand *et al*;[18] have documented lung lesions sufficiently distinct from those of silicosis and asbestosis to allow recognition as a separate entity.

In the present study, conducted on a population with a mean duration of exposure of 19·0 (SD 7·8) years (range 8–29), we have been able to show a lower prevalence (2·6%) of pneumoconiosis (profusion $\geq 1/1$) than that (14%) reported by Peters *et al*[1] The profusion of opacities was related to cumulative exposure to dust. Two subjects (profusion q 1/0 and q 2/1) were employed only in the final stages (screening and storage) of the production process and therefore they were not exposed to crystalline silica. The cumulative exposure to dust also determined impairments of pulmonary function, particularly in smokers. No effect of cumulative exposure to SO_2 was shown. A significant negative trend between both FVC and FEV_1 and profusion of opacities was found only in smokers.

These results confirm the findings from other epidemiological studies.[1] It can therefore be suggested that the differences in prevalence of pneumoconiosis is probably related to lower concentrations of total and respirable dust and crystalline silica measured in the SiC factory.[14]

In conclusion, our data show the presence of a respiratory risk in the manufacture of SiC at exposures below current permissible limits. The finding of two cases of pneumoconiosis in workers not exposed to silica strongly suggests a possible role for SiC in the genesis of interstitial lung disease.

This leads us to consider, in agreement with others,[1 15] that the current permissible limits for exposure to the mixture of contaminants in SiC production are too high to protect the health of exposed workers.

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