Work related decrement in pulmonary function in silicon carbide production workers

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ABSTRACT The relation between pulmonary function, cigarette smoking, and exposure to mixed respirable dust containing silicon carbide (SiC), hydrocarbons, and small quantities of quartz, cristobalite, and graphite was evaluated in 156 SiC production workers using linear regression models on the difference between measured and predicted FEV₁ and FVC. Workers had an average of 16 (range 2-41) years of employment and 9.5 (range 0.6-39.7) mg-year/m³ cumulative respirable dust exposure; average dust exposure while employed was 0.63 (range 0.18-1.42) mg/m³. Occasional, low level (≤ 1.5 ppm) sulphur dioxide (SO₂) exposure also occurred. Significant decrements in FEV₁ (8.2 ml; $p < 0.03$) and FVC (9.4 ml; $p < 0.01$) were related to each year of employment for the entire group. Never smokers lost 17.8 ml ($p < 0.02$) of FEV₁ and 17.0 ($p < 0.05$) of FVC a year, whereas corresponding decrements of 9.1 ml ($p = 0.12$) in FEV₁ and 14.4 ml ($p < 0.02$) in FVC were found in current smokers. Similar losses in FEV₁ and FVC were related to each mg-year/m³ of cumulative dust exposure for 138 workers with complete exposure information; these findings, however, were generally not significant owing to the smaller cohort and greater variability in this exposure measure. Never smokers had large decrements in FEV₁ (40.7 ml; $p < 0.02$) and FVC (32.9 ml; $p = 0.08$) per mg-year/m³ of cumulative dust exposure and non-significant decrements were found in current smokers (FEV₁: -7.1 ml; FVC: -11.7 ml). A non-significant decrement in lung function was also related to average dust exposure while employed. No changes were associated with SO₂ exposure or an SO₂ dust interaction. These findings suggest that employment in SiC production is associated with an excessive decrement in pulmonary function and that current permissible exposure limits for dusts occurring in this industry may not adequately protect workers from developing chronic pulmonary disease.

Silicon carbide (SiC, Carborundum) is a widely used abrasive material manufactured by heating high purity crystalline silica, petroleum coke, and sawdust at 2200°C for 36 hours by direct electric current passed through a graphite conductor. Heating causes the reduction of silica by carbon in the coke to form SiC and large quantities of carbon monoxide. Sulphur, an impurity in the coke (2-4%), is released as sulphur dioxide gas (SO₂). Various hydrocarbons are also released during pyrolysis of the petroleum coke which is similar to the destructive distillation of coal. Workers employed in manufacturing SiC may therefore be exposed to several contaminants

including dusts of silica, SiC, graphite, polycyclic hydrocarbons, and gases of SO₂ and carbon monoxide.

Despite widespread use and known toxic properties of some of these substances, there are few published studies concerning the respiratory effects of airborne exposures in this industry. Bruusgaard described radiographic densities on chest x ray films and a restrictive pattern of pulmonary function loss in 12 of 32 workers from a SiC production facility in Norway. Two workers were disabled by second stage pneumoconiosis and pronounced reductions in vital capacity. More recently, Gauthier *et al* evaluated 68 Quebec SiC production workers who were referred to workmen's compensation for respiratory complaints. They found a highly significant relation between

duration of exposure and radiographic changes (nodular and linear opacities; pleural thickening), although lung function changes were limited to smokers. Finally, in 1984 Peters *et al* described small round and irregular opacities with an average perfusion reading of 1/1 or greater in 24 of 171 workers from a Quebec SiC production plant.³ Lifetime exposure to respirable dust was closely related to the prevalence of pulmonary opacities in smokers and non-smokers. Smokers had statistically significant reductions in FEV₁ and FVC related to duration of employment and cumulative exposure to respirable dust, as well as losses in FEV₁ associated with cumulative SO₂ exposure. Smaller, non-significant decrements were found in non-smokers. Unfortunately, adjustments could not be made for the effects of cumulative smoking on lung function because this information was unavailable.

The present study is an extension of the work of Peters *et al*.³ On this occasion we obtained new measurements of pulmonary function and exposure to airborne emissions and collected additional information on respiratory symptoms, smoking, and work history. The present paper explores the relation between exposure and effects on lung function. Our findings on respiratory symptoms are presented in a companion article.⁴

Methods

SUBJECTS

From union membership lists we identified 177 SiC production workers with at least two years experience who were employed at the plant some time between 1977 and 1982. Five workers refused to participate, three retired and two laid off workers were unavailable, and two were excused because of long term disability, thus leaving a final study group of 165 men or 93% of the study population.

HEALTH EFFECTS

Relevant information on health and smoking habits was obtained from a French translation⁵ of the American Thoracic Society (ATS) respiratory disease questionnaire⁶ administered by one of three trained interviewers. Workers also completed forms describing past employment, employment in other dusty trades, and employment in SiC production. Occasional missing information was obtained by telephone interview about one month later.

Pulmonary function was evaluated following ATS guidelines.⁶ Five acceptable maximal expiratory efforts were performed in a seated position, without noseclips, using an Eagle spirometer system (Warren E Collins, MA) with computerised measurement and printout of FVC, FEV at various time intervals, flow

rates at different lung volumes, MMFR, and peak flow, corrected to BTPS. Height was measured to the nearest centimetre in stockinged feet at the time of lung function testing. Observations on 157 workers were obtained in December 1982 after a six month plant closure; the remaining eight workers were evaluated four months later.

AIRBORNE EXPOSURES

Estimates of job specific eight hour, time weighted average (TWA) exposures to respirable dust were calculated from 124 personal samples obtained in 1980, and have been published elsewhere.⁷ An additional 113 personal samples were obtained in 1983 to test the stability of these estimates over time and to evaluate new control measures introduced by the company. Job specific SO₂ exposure estimates were obtained in 1980 from 15 personal samples and a series of stationary samples from the major work areas.⁷ Ten additional stationary samples were obtained in 1983 from the production area, the only area in which exposure to SO₂ occurred. Spot samples of SO₂ were also obtained by detector tube throughout the working week. The new SO₂ samples were measured simultaneously by impinger with trichloromercurate solution and by active treated filter downstream from a particulate filter.

Exposures for each of the 22 job categories were estimated by calculating the geometric mean of individual (arithmetic) mean exposures measured for each job title. Because exposures for several job titles were identical, data were collapsed into 10 overall job exposure categories. Company records indicated that important technical changes were introduced to the plant in 1962 and 1980. To adjust for these improvements we used 1983 dust exposure measures for the period 1980–3, 1980 measures for 1962–80, and 1980 measures for the pre-1962 period with the assumption that, at that time, exposures for furnace workers were probably similar to those of payloaders. Sulphur dioxide concentrations were fairly constant over time.

For 145 workers with complete company records of individual, within plant job histories, cumulative exposures to respirable dust and SO₂ were calculated from the product of the time spent in each job category and its mean exposure level, summed over the 10 categories and expressed in units of concentration time (mg-year/m³ for cumulative dust, and ppm-year for cumulative SO₂). Each worker's average exposure to dust (mg/m³) or SO₂ (ppm) was obtained by dividing his cumulative exposure by duration of employment at the plant.

DATA ANALYSIS

Questionnaire, lung function, and exposure data were

coded, entered into an IBM 360 computer, and checked extensively for completeness and accuracy. Descriptive statistics, correlation matrices, analysis of variance, and multiple linear regressions were performed with a standard statistical package (Statistical Analysis System Inc, Cary NC, 1982). Partial F tests were used to measure the statistical significance of individual coefficients in the regression models and two tailed Student's *t* test was used to compare various descriptive results.⁸

Pulmonary function tests (FEV₁, FVC, and FEV₁/FVC ratio) were evaluated by multiple linear regression techniques,⁸ using the prediction equations of Dockery *et al* to adjust for age and height.⁹ For FEV₁ and FVC, the difference between actual and predicted values for the average of the best three of five measurements were regressed on various combinations of independent variables including smoking and workplace exposures; these models converted coefficients of independent variables into volume units. FEV₁ and FVC expressed as per cent of the predicted value were examined similarly. The ratio of FEV₁ to FVC was evaluated by regressing the best value for each worker on age, smoking, and exposure measures. Step up and step down procedures⁸ were used to determine the best regression models in terms of simplicity, analysis of residuals, statistical significance, and variance explained. Pulmonary function was not evaluated relative to each component of respirable dust (alpha quartz, cristobalite, graphite, SiC, hydrocarbons) because dust composition was available for only a limited number of samples.

Results

SUBJECTS

The study group consisted of 165 white French-Canadian men, most (152) of whom had spent their entire lives in or around the community where the plant is located. Nine workers aged under 25 were excluded from the remaining analysis because the prediction equations of Dockery *et al* were developed for white adults aged 25 to 74.⁹ Table 1 gives the average age, height, pack-years of cigarette smoking, and years of employment in SiC manufacture for the remaining 156 workers. The oldest worker studied was

69. Twenty per cent of the study group had never smoked cigarettes, 55% currently smoked, and 25% were ex-smokers. Other tobacco products including pipes, cigars, and cigarettos were used infrequently and generally by cigarette smokers, although four non-cigarette smokers had smoked small quantities, but did not inhale. The study group had spent an average of 16 years (range 2-41) in silicon carbide manufacture. Current smokers were younger and had spent significantly fewer years in the industry than either never or ex-smokers ($p < 0.02$).

EXPOSURE

Geometric means for eight hour TWA exposures to respirable dust and SO₂ were low (table 2). Dust concentrations in 1980 and 1983 were generally similar except in job categories where exposure control measures had been installed recently. Exposures to SO₂ estimated by personal sampling techniques in 1980 corresponded closely to area samples obtained in 1983.

For 138 workers with complete company work histories (job assignments, dates, and periods of absence) mean cumulative dust exposure was 9.5 mg-years/m³ (range 0.63-39.7) and average dust exposure was 0.63 ± 0.26 mg/m³ over the entire period of employment (figure). Exposure to SO₂ was low (average 0.12 ppm) and highly skewed; 75% of the workforce had less than 2.5 ppm-years of cumulative exposure and no individual had an average exposure while employed of greater than one ppm.

Respirable dust was composed principally of carbonaceous matter and silicon carbide, in addition to small amounts of quartz and cristobalite (table 3). Exposures to each of these constituents were well below generally accepted exposure limits. Dust composition in the furnace area was generally similar among job assignments. Although quartz exposures appeared to be greater for payload operator than for other furnace area tasks, these estimates were derived from only two samples and should be interpreted cautiously. Dust exposure to carbosellectors differed considerably from exposures elsewhere in the plant, with SiC being the largest constituent (44.4%). Crusher operators were exposed to dust similar in composition to the furnace area.

Table 1 Age, height, cigarette smoking, and years of employment for 156 silicon carbide workers, according to smoking habit

	No	Age (y)	Height (cm)	Pack-years of smoking	Years of employment
Never smokers	32	43.8 ± 14.7	168.6 ± 5.5	—	18.8 ± 12.5
Current smokers	86	41.3 ± 12.4	169.3 ± 6.3	24.2 ± 17.7	14.1 ± 10.6
Ex-smokers	38	44.4 ± 13.7	170.5 ± 5.4	24.6 ± 21.4	18.2 ± 11.8
Total	156	42.5 ± 13.2	169.5 ± 5.9	19.4 ± 19.5	16.1 ± 11.5

Values reported are mean ± SD.

Table 2 Sample size and geometric means for eight hour, time weighted average exposures to respirable dust (mg/m^3) and SO_2 (ppm) by job category in 1980 and 1983

Job category	Respirable dust				SO_2			
	1980*		1983		1980*		1983	
	No	Mean	No	Mean	No	Mean	No	Mean
Outside area:								
Labourer	2	0.65	3	0.61				
Payloader	2	1.66		—				
Preparation area:								
Coke	4	0.48	5	0.24				
Sand and sawdust	2	0.21		—				
Mixer	5	1.01	7	0.44				
5th floor		—	2	0.48				
Acheson furnace area:								
Cranement†	20	0.42	10	0.30	3	1.5		
Loader	4	0.59	3	0.60		1.0		1.1
Labourer	14	0.38	6	0.61	5	1.0		1.1
Electrode cleaner	5	0.44	5	0.46		1.0	10	1.1
Asst elect cleaner	8	0.17	3	0.52		1.0		1.1
Payloader†	13	1.46	10	0.35	2	1.2		1.1
Old mix operator	7	0.85	3	1.12	2	0.2		
Carbosclector	24	0.72	26	0.57	3	0.2		
Maintenance:								
General	2	0.11	10	0.45		0.1‡		
Millwright	1	0.21	4	0.77				
Mechanic	2	0.28	2	0.13				
Electrician	3	0.20	8	0.26				
Product area:								
Crusher	1	0.45	3	0.45				
Other:								
Vehicle mechanic	2	0.44		—				
Welder	3	1.35		—				
Superintendent		—	3	0.42				

*Smith *et al* with permission from the *British Journal of Industrial Medicine* 1984;41:100-8.⁷

†Job categories for which control measures were installed between 1980 and 1983.

‡Estimated from area samples.

Note: Permissible exposure limits (8 hour TWA) in Quebec are $5 \text{ mg}/\text{m}^3$ for inert respirable dust and 5 ppm for SO_2 . Respirable dust and SO_2 exposures for 145 silicon carbide workers.

Table 3 Eight hour TWA geometric mean exposures for constituents of respirable dust in SiC production and composition (in per cent)*

Job category	No of samples	Respirable dust (mg/m ³)	Carbonaceous matter (mg/m ³) (%)	Quartz (µg/m ³) (%)	Cristobolite (µg/m ³) (%)	SiC µg/m ³ (%)
Acheson furnace area:						
Loader	4	0.32	0.19 (59.4)	ND	ND	29 (9.0)
Payloader	2	0.63	0.44 (69.8)	86 (13.6)	6 (1.0)	88 (14.0)
Labourer	13	0.49	0.32 (65.3)	12 (2.4)	5 (1.0)	54 (11.0)
Old mix operator	4	0.72	0.44 (61.1)	7 (1.0)	14 (1.9)	99 (13.8)
Carbosclector	22	0.95	0.37 (38.9)	9 (0.9)	20 (2.1)	422 (44.4)
Product area:						
Crusher operator	4	0.43	0.27 (62.8)	ND	4 (0.9)	82 (19.1)

*Dufresne *et al* with permission from the American Industrial Hygiene Association Journal 1987;48:160-6.¹⁰

ND = Not detected.

Note: Permissible exposure limits (8 hour TWA) in Quebec are 5 mg/m³ for respirable dust, including SiC and carbonaceous matter, 100 µg/m³ for quartz, and 50 µg/m³ for cristobolite.Table 4 FEV₁ and FVC (expressed as % of predicted) and their ratio (in %) for 156 silicon carbide workers by cigarette smoking habit

	No	FEV ₁	FVC	FEV ₁ /FVC
Never smokers	32	99.4 ± 15.5	99.2 ± 14.6	80.6 ± 6.9
Current smokers	86	92.9 ± 15.8***	95.8 ± 12.9**	78.5 ± 8.4
Ex-smokers	38	96.3 ± 12.8	100.4 ± 7.4	77.5 ± 8.7
Total	156	95.1 ± 15.1***	97.6 ± 12.3*	78.7 ± 8.1

Values are mean ± SD.

Predicted values for FEV₁ and FVC were derived from the equations of Dockery *et al*.⁹Two tailed Student's *t* test differs from 100% predicted: **p* < 0.05; ***p* < 0.01; ****p* < 0.001.

PULMONARY FUNCTION

FEV₁ and FVC were respectively 95.1% and 97.6% of predicted for the entire group and the ratio FEV₁/FVC was 78.7% (table 4). Current smokers had significantly less than predicted FEV₁ and FVC and a non-significant reduction in FEV₁ was present for ex-smokers. Although the ratio of FEV₁/FVC tended to be lower in those who had smoked cigarettes, no significant differences were found.

Most time related variables, including age, years of employment, and cumulative exposures to dust and SO₂ were highly intercorrelated (table 5). Less correla-

tion occurred with pack-years of smoking, however.

To adjust for the effects of cigarette smoking on lung function, we examined several measures of smoking habit, including cumulative smoking (pack-years), duration of smoking, quantity smoked a day, and smoking categorical variables. Of these, pack-years and smoking duration explained the largest amount of variance in our regression models. Pack-years was considered superior, however, because it also accounted for quantity smoked a day and was less correlated to other time related variables. A significant decrement in FEV₁ related to pack-years (table 6) was found in current smokers (-7.3 ml per pack-year, *p* < 0.05) and a smaller decrement was found for ex-smokers (-2.6 ml per pack-year, *p* > 0.10).

After adjusting for age, height, and cigarette smoking by simultaneously regressing pack-years and exposure on the difference between measured and predicted pulmonary function, we found a statistically significant loss of 8.2 ml of FEV₁ and 9.4 ml of FVC per year of employment in SiC manufacture for the entire study group (table 7). This decrement was greatest in never smokers (-17.8 ml a year for FEV₁, *p* < 0.02 and -17.0 ml a year for FVC *p* < 0.05). Current smokers also showed significant work related FVC decrement (-14.4 ml a year, *p* < 0.02) as well as

Table 5 Correlation matrix for age, pack-years of cigarettes smoked, years of employment, and various measures of exposure for 156 silicon carbide workers

Age	Pack-years	Years of employment	Cumulative dust (mg-year/m ³)	Average dust (mg/m ³)	Cumulative SO ₂ (ppm-year)	Average SO ₂ (ppm)	
1	0.42§	0.84§	0.67§	0.12	0.48§	0.20	Age
1	1	0.29†	0.32§	0.08	0.22†	0.04	Pack-years
		1	0.81§	0.21*	0.60§	0.30†	Years of employment
			1	0.66§	0.76§	0.50§	Cumulative dust
				1	0.41§	0.44§	Average dust
					1	0.83§	Cumulative SO ₂
						1	Average SO ₂

One pack-year is equivalent to 20 cigarettes smoked a day throughout one year.

Coefficient (r) differs from zero: *0.01 ≤ *p* < 0.05; †0.001 ≤ *p* < 0.01; ‡0.001 ≤ *p* < 0.001; §*p* < 0.0001.

Pulmonary function decline in SiC workers

Table 6 Regression coefficients (ml) per pack-year of cigarettes smoked with respect to changes in FEV₁ and FVC, adjusted for age, height, and years of employment

	No	FEV ₁	FVC
Current smokers	86	-7.3 ± 3.4*	-2.3 ± 3.5
Ex-smokers	38	-2.6 ± 4.2	-0.7 ± 5.9

Values reported (mean ± SEE) are the coefficients of the pack-years term calculated from the regression equation. Δ PFT = $b_0 + b_1$ (pack-years) + b_2 (years worked). Δ PFT (either Δ FEV₁ or Δ FVC) is the measured PFT less the predicted PFT derived from the prediction equations of Dockery *et al.*⁹ p Values were obtained from the partial F tests on the pack-year coefficient. *Differs from zero, $p < 0.05$.

a non-significant decrement in FEV₁. Ex-smokers had a non-significant loss of pulmonary function related to duration of employment.

Cumulative dust exposure was associated with losses in FEV₁ and FVC similar in magnitude to findings related to duration of exposure but which were not statistically significant in the entire study group. For never smokers, a large and significant loss in FEV₁ was related to cumulative dust exposure (-40.7 ml per mg-year/m³, $p < 0.02$), as was an important and nearly statistically significant decline of FVC (-32.9 ml per mg-year/m³, $p = 0.08$). Cumulative dust related losses in pulmonary function for current smokers were similar to findings related to duration of exposure but were not statistically significant. Ex-smokers showed non-significant improvements in pulmonary function related to cumulative dust exposure. Small, non-significant declines in FEV₁ and FVC were associated with average dust exposure while employed; however, these changes were inconsistent across smoking categories.

Table 7 Regression coefficients and standard errors for changes in FEV₁ (ml), FVC (ml), and their ratio (%) associated with various measures of exposure and adjusted for age, height, and pack-years of cigarettes smoked

	Years of employment	Cumulative dust (mg-year/m ³)	Average dust (mg/m ³)	Cumulative SO ₂ (ppm-year)	Average SO ₂ (ppm)
Never smokers	No	32	28	28	28
	FEV ₁	-17.8 ± 7.0†	-40.7 ± 15.0†	-52.7 ± 35.8	-166.8 ± 625.7
	FVC	-17.0 ± 8.3†	-32.9 ± 18.0*	-43.1 ± 41.3	-229.3 ± 706.1
	Ratio	-0.05 ± 0.04	-0.19 ± 0.21	-0.38 ± 0.34	-0.02 ± 0.05
Current smokers	No	86	75	75	75
	FEV ₁	-9.1 ± 5.6	-7.1 ± 7.8	-19.8 ± 20.2	545.4 ± 536.6
	FVC	-14.4 ± 5.9†	-11.7 ± 8.0	-8.2 ± 21.2	278.6 ± 562.1
	Ratio	0.02 ± 0.15	0.16 ± 0.14	0.51 ± 0.33	0.22 ± 0.18
Ex-smokers	No	38	35	35	35
	FEV ₁	-5.0 ± 7.6	15.0 ± 11.1	4.3 ± 33.1	-494.3 ± 691.4
	FVC	-0.7 ± 5.9	11.7 ± 9.4	20.9 ± 27.8	129.7 ± 589.3
	Ratio	0.28 ± 0.17	0.10 ± 0.19	-0.59 ± 0.52	-0.18 ± 0.10*
Total	No	156	138	138	138
	FEV ₁	-8.2 ± 3.6†	-7.5 ± 9.4	-0.2 ± 15.2	51.9 ± 34.2
	FVC	-9.4 ± 3.7§	-9.5 ± 6.0	-3.2 ± 15.8	7.1 ± 355.8
	Ratio	0.05 ± 0.09	0.11 ± 0.09	0.16 ± 0.22	0.16 ± 0.22

For FEV₁ and FVC, values reported are coefficients of the exposure term from the regression equation Δ PFT = $b_0 + b_1$ (pack-years) + b_2 (exposure). Δ PFT (either Δ FEV₁ or Δ FVC) is the measured PFT less the predicted PFT obtained from the equations of Dockery *et al.*⁹ For FEV₁/FVC ratio, reported values are coefficients of the exposure term from the equation FEV₁/FVC ratio = $b_0 + b_1$ (age) + b_2 (pack-years) + b_3 (exposure). p Values were obtained from the partial F-tests on the exposure coefficients. Differs from zero: *0.05 < $p < 0.10$; †0.02 < $p < 0.05$; ‡0.01 < $p < 0.02$; § $p < 0.01$.

Table 8 Regression coefficients (ml) associated with years of employment with respect to changes in FEV₁ and FVC, adjusted for age, height, and pack-years of cigarettes smoked, and stratified by years of employment

Years of employment	No	FEV ₁	FVC
0-20	103	-12.0 ± 32.1	-8.8 ± 13.0
> 20	53	-9.7 ± 11.9	-7.2 ± 12.1

Values reported (mean ± SEE) are the coefficients of years worked obtained from the regression equation Δ PFT = $b_0 + b_1$ (pack-years) + b_2 (years worked). Δ PFT (either Δ FEV₁ or Δ FVC) is the measured PFT less the predicted PFT obtained from the prediction equations of Dockery *et al.*⁹

Lung function changes associated with cumulative and average SO₂ exposure were not statistically significant. Although never and current smokers showed non-significant cumulative SO₂ related lung function decline, this relation disappeared when dust and SO₂ were included simultaneously in regression models. Moreover, no significant SO₂ dust interaction effects were found.

To verify whether the exposure related effects found in this study group were due to exposures occurring before 1962 when technological improvements were introduced to the plant, we compared workers having more than 20 years experience with those having fewer (table 8). Both groups showed similar FEV₁ and FVC changes per year of employment in the industry.

Sixty eight members of our study group had previously worked in one or more dusty trades, including 10 former miners or quarrymen, nine former forestry workers, 33 textile workers, four shipyard workers, 17 in the construction trades, and 13 in assorted other industries. When FEV₁ and FVC for

these workers were compared with findings for the remaining 88, only minor non-significant differences were found. We also examined pulmonary function in workers formerly employed in each of these dusty trades. Only miners and quarrymen had mean values for FEV₁ and FVC significantly lower than the group average, largely resulting from two workers with poor lung function who had more than 30 years experience in SiC manufacture and only two years as miners or quarry workers. After adjusting for cigarette smoking, no trends were found for FEV₁ or FVC relative to duration of work in other dusty trades.

Discussion

In this study we observed a statistically significant, restrictive pattern of pulmonary function loss related to duration of work at this SiC production factory. These changes were particularly evident in never smokers and current smokers indicating an effect independent of smoking habit. Significant changes in pulmonary function were not present in ex-smokers, however, possibly due to the decision to stop smoking. Speizer *et al* found in a study of more than 6000 Americans from six different cities that pulmonary function improved after smoking stopped, an effect which was not adjusted in our linear regression models.¹¹

Work related loss of pulmonary function appeared to be principally due to cumulative exposure to respirable dust. Never smokers had large, cumulative dust related lung function losses, and current smokers had decrements similar to those found relative to work duration. These findings were generally not statistically significant, however, possibly because of the smaller number of workers for whom cumulative dust exposure could be quantified, and greater statistical variance in the measure.

Low level cumulative and average SO₂ exposures were not related to changes in lung function and no effects from a dust-SO₂ interaction were observed.

Historical exposure estimates, which were derived from current measurements and adjusted for known technological changes at the plant, appeared to be reasonable. For most job categories, measurements obtained in 1980 and 1983 were similar suggesting that, in the absence of major technological changes, current exposure measures reflect those of the past. In addition, lung function changes occurring in workers hired before and after partial mechanisation in 1962 were similar, suggesting that both groups were exposed to similar levels of toxic dust.

Dust composition varied among job categories with carboselectors, in particular, exposed to a greater proportion of SiC and correspondingly less carbonaceous matter than other job categories. Such

differences in dust composition may have misclassified the relevant toxic portion of the respirable dust, leading to an underestimate of the true exposure-effect relation. This relation may have been further underestimated if workers with poor pulmonary health brought on by work related exposures had left the industry before our investigation, thereby excluding themselves from this study.

Although other investigators have associated respirable dust exposure in SiC manufacture with radiographic evidence of pneumoconiosis and a restrictive pattern of lung function loss,¹⁻³ it is unclear why these changes occur. Generally, SiC has been considered inert with little or no fibrogenic potential.^{12,36} This position is supported by early studies of animals exposed to high concentrations which found little or no pulmonary fibrosis.¹³⁻¹⁵ Such studies can only be regarded as inconclusive, however, because the latent period for fibrosis may be longer than the lifespan of the animals. On the other hand, early studies of workers manufacturing grindstones from SiC and aluminium oxide documented several cases of pulmonary fibrosis,¹⁶⁻¹⁹ and a recent proportional mortality study of a grindstone manufacturing plant showed a twofold excess of death from non-malignant respiratory disease.²⁰ Unfortunately, these studies were not able to distinguish between the effects of aluminium oxide and SiC, although one author claimed that two workers with second stage pneumoconiosis were exposed to SiC dust only.¹⁹

Recently, Funahashi *et al* reported on two workers who had been exposed to SiC dust only for 14 and 20 years in a factory manufacturing refractory bricks.²¹ Both complained of dyspnoea, had bilateral diffuse reticulonodular densities on chest x ray examination, and showed a restrictive pattern of lung function impairment with FVC measurements of 83% and 76% of predicted. An open lung biopsy of the first worker found large amounts of black material in the fibrosed alveoli which was shown by x ray diffraction to be almost exclusively SiC.

Bye *et al* have analysed dust samples collected from three Norwegian silicon carbide production facilities.²² Using various techniques including scanning and transmission electron microscopy, energy dispersive x ray spectrometry, and electron diffraction they characterised fibres of alpha silicon carbide, 80% of which were less than 0.5 µm in diameter and greater than 5 µm in length, in concentrations up to five fibres per cubic centimeter of air. The toxic potential of these previously unknown SiC fibres may be similar to other fibrogenic dusts.

Although SiC was a major component of the respirable dust at the plant we studied, the dust also contained large amounts of carbonaceous matter and small quantities of quartz, cristobalite, and graphite.

Silica, in the form of quartz or cristobalite, is a well known cause of pulmonary fibrosis in man.¹² An extensive review²³ and subsequent research²⁴ suggest that the current limit of 100 µg/m³ should be reduced by at least half to ensure a safe working environment. Because exposures at our study plant were generally at or below this recommended level, we think that silica is unlikely to be responsible by itself for the lung function changes we describe. Graphite is a rare but documented cause of pneumoconiosis.²⁵ Exposure at our study plant was low and it is therefore, like silica, unlikely to be the sole agent responsible for our findings. The respirable carbonaceous materials in emissions from SiC production are qualitatively similar to those found in the coke industry. Such exposures have been associated with chronic bronchitis and a pattern of lung function decline that is obstructive rather than restrictive.^{26, 27}

Occupational exposure to low levels of SO₂ has been associated with pulmonary function decrement in copper smelter workers^{28, 29} but the evidence is conflicting.³⁰⁻³² Smith *et al* found important pulmonary function impairment related to SO₂ exposure in one group of smelter workers,²⁹ but a re-evaluation of the same group, after compulsory respirator use was imposed, found no effect.³² This suggests that SO₂ effects, if present, were transient in nature and dissipated when exposure was reduced, an observation that concurs with experimental evidence.³³ No changes in FEV₁ or FVC have been found in other occupationally exposed groups such as pulp workers^{34, 35} and corn refiners.³⁶ We found no SO₂ related effects on lung function in SiC production workers, unexposed during the preceding six months, which suggests that if transient effects occurred, they were reversible in a short time. Similarly, we found no evidence to support theories of a synergistic effect between particulate matter and SO₂ on lung function.³³

In conclusion, our findings suggest that a restrictive pattern of lung function loss is associated with duration of work and cumulative exposure to respirable dust in the SiC production industry. These findings suggest that long term exposure to respirable dust in this industry may affect the lung parenchyma leading to fibrosis and a loss of functioning lung units, a process consistent with the development of pneumoconiosis. Moreover, these changes occur at low levels of exposure, well below current permissible limits. Although the "active" ingredient in this dust is unknown, a review of published reports suggests that the silicon carbide portion may play an important part, possibly due to the recently described, fibrous nature of some SiC crystals. The small amounts of crystalline silica and graphite present in the dust may be contributing factors but appear unlikely, by themselves, to explain the observed changes. Current

permissible limits for exposure to the mixture of contaminants in SiC production do not appear to protect the health of SiC production workers.

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