Respiratory symptoms associated with low level sulphur dioxide exposure in silicon carbide production workers

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ABSTRACT Relations between pulmonary symptoms and exposure to respirable dust and sulphur dioxide (SO₃) were evaluated for 145 silicon carbide (SiC) production workers with an average of 13.9 (range 3-41) years of experience in this industry. Eight hour time weighted average exposures to SO₂ were 1.5 ppm or less with momentary peaks up to 4 ppm. Cumulative SO₂ exposure averaged 1.94 (range 0.02-19.5) ppm-years. Low level respirable dust exposures also occurred (0.63 ± 0.26 mg/m³). After adjusting for age and current smoking status in multiple logistic regression models, highly significant, positive, dose dependent relations were found between cumulative and average exposure to SO₂, and symptoms of usual and chronic phlegm, usual and chronic wheeze, and mild exertional dyspnoea. Mild and moderate dyspnoea were also associated with most recent exposure to SO₂. Cough was not associated with SO₂. No pulmonary symptoms were associated with exposure to respirable dust nor were any symptoms attributable to an interaction between dust and SO₂. Cigarette smoking was strongly associated with cough, phlegm, and wheezing, but not dyspnoea. A greater than additive (synergistic) effect between smoking and exposure to SO₂ was present for most symptoms. These findings suggest that long term, variable exposure to SO_2 at 1.5 ppm or less was associated with significantly raised rates of phlegm, wheezing, and mild dyspnoea in SiC production workers, and that current threshold limits for SO₂ may not adequately protect workers in this industry.

Silicon carbide (SiC) production workers are exposed to an array of respirable contaminants with known or suspected human toxicity. These include: crystalline silica used as a primary reagent; furnace emissions containing polycyclic aromatic hydrocarbons (PAH), sulphur dioxide (SO₂), and carbon monoxide; graphite used as an electric conductor; and large volumes of dust containing SiC, hydrocarbons, quartz, and cristobalite which is produced when workers crush blocks of newly formed SiC with hand held pneumatic tools. Estimated exposures to all these contaminants have been published elsewhere.¹⁻³

Although several investigators have described radiographic densities³⁻⁶ and a restrictive pattern of pulmonary function loss³⁻⁵ consistent with the development of pneumoconiosis among these workers, pulmonary symptoms in SiC workers have not previously been evaluated. In this paper we present

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findings on the relation between pulmonary symptoms and exposures in SiC production.

Methods

SUBJECTS

From union membership lists we identified 177 SiC workers with at least two years experience who had been employed some time between 1977 and 1982. Five workers refused to participate, three retired and two laid off workers were unavailable, and two disabled workers were excluded. Exposure records were not available for 20 workers, leaving a final study group of 145 men.

HEALTH EFFECTS

Information on respiratory symptoms, illnesses, relevant medical history, and smoking habits was obtained through the administration by one of three trained interviewers of a French translation⁷ of the American Thoracic Society (ATS) respiratory disease questionnaire.8 Workers also completed forms describing past employment in other industries, the period worked in SiC manufacture, and specific questions about employment in 15 dusty trades. A semiquantitative estimate of cough frequency was obtained by administering a cough questionnaire developed by Field.9 Workers were also asked to grade their subjective feeling of dyspnoea on a linear scale.¹⁰ A physical examination of the respiratory and cardiovascular systems was performed by one of us (JWO), without knowledge of exposure history, pulmonary function, or responses to questionnaire. The physical examination included an evaluation of productive cough on request,¹¹ during which each worker was asked to cough deeply, and presence or absence of loose phlegm was ascertained according to the sound produced. Pulmonary function tests were performed according to ATS guidelines⁸ and are reported elsewhere.³

AIRBORNE EXPOSURES

Job specific exposure measurements of airborne contaminants in this SiC production factory were obtained in 1980 and 1983, and are reported elsewhere.¹³ Company records included job titles, dates, and time off for each worker's entire period of employment at the factory. From this information we could construct cumulative exposure indices for respirable dust and SO₂, calculated from the product of time spent at each job assignment and the job specific exposure level, summed over all job categories that the worker had occupied, and measured 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/m^3) or SO₂ (ppm) was obtained by dividing his cumulative exposure by the duration of his employment. We also obtained the job specific SO₂ exposure level (ppm) for the most recent position held; this measure was used in place of current SO₂ exposure because the plant had been closed during the six months preceding our study.

DATA ANALYSIS

Questionnaire, lung function, and exposure data were coded and entered into an IBM 360 computer, and were extensively checked to ensure completeness and accuracy. Descriptive statistics, correlation matrices, analysis of variance, multiple logistic and linear regressions, and corresponding tests of significance were performed with a standard statistical package (Statistical Analysis System Inc, Cary, NC, 1982). Chisquared statistics were used to measure the statistical significance of individual coefficients in the logistic regression models. Two tailed Student's *t* test and the
 Table 1
 Age, smoking, and exposure data for 145 silicon carbide workers

Age (v)	39.6 ± 11.8
Never smokers (%)	21.4
Ex-smokers (%)	24.8
Current smokers (%)	53-8
Pack-vears*	24.3 ± 18.6
Years worked	13.9 ± 9.6
Cumulative dust exposure (mg-year/m ³)	9.5 ± 7.7
Average dust exposure (mg/m ³)	0.63 ± 0.26
Cumulative SO, exposure (ppm-years)	1.94 ± 2.91
Average SO, exposure (ppm)	0.12 ± 0.12
Most recent SO ₂ exposure (ppm)	0.26 ± 0.47

Values are mean \pm SD.

*Excluding non-smokers. One pack-year is equivalent to one pack of 20 cigarettes smoked a day throughout one year.

Mantel-Haenszel chi-squared statistic were used to compare various descriptive results.¹² To control for confounding by age and smoking habit, as well as to differentiate between the effects of work duration, dust, and exposure to SO_2 , logistic regression models were used to evaluate most of our symptoms data. Step up and step down procedures were used to determine the best regression models in terms of simplicity, analysis of residuals, statistical significance, and variance explained.¹²

Results

The final study group consisted of 145 white French-Canadian men with an average age of 39.6 (range 21-65) years and 14 years (range 3-41) experience in the industry (table 1). Thirty one workers had never smoked cigarettes; ex-smokers and current smokers had smoked similar amounts. Other tobacco products

 Table 2 Definitions of respiratory symptoms obtained by questionnaire

Symptom	Definition
Usual cough	Positive response to "Do you usually have a cough?"
Usual phlegm	Positive response to "Do you usually bring up phlegm from your chest (not from back of your nose)?"
Chronic phlegm	Positive response to "Do you bring up phlegm like this on most days for three consecutive months or more during the year?" and a response of more than two years to "For how many years have you had this phlegm?"
Wheezing most days or nights	Positive response to "Does your chest ever sound wheezy or whistling? Most days or nights?"
Chronic wheezing	Positive response to wheezing most days or nights and a response of more than two years to "For how many years has this been present?"
Dyspnoea while hurrying	Positive response to "Are you troubled by shortness of breath when hurrying on the level or walking up a slight hill?"
Dyspnoea keeping up with peers	Positive response to "Do you have to walk slower than people of your own age on the level because of breathlessness?"

	Never smokers (n = 31)	Ex-smokers (n = 36)	Current smokers (n = 78)	Entire cohort $(n = 145)$
Usual cough	9.7	13.9	53.8	34.5
Usual phlegm	19.3	33.3	47.4	37.9
Chronic phlegm	16-1	19-4	32.1	25.5
Wheezing on most days/nights	16-1	13.9	42.3	30.0
Chronic wheezing	16-1	11-1	37.2	26.2
Dysphoea when hurrying	45.2	41.7	52.6	48 ·3
Dyspnoea keeping up with peers	12.9	H-i	16.7	14.5

Table 3 Prevalence (%) of respiratory symptoms by current smoking status for 145 silicon carbide workers

were used infrequently and generally by cigarette smokers.

EXPOSURE

The workforce had a mean cumulative dust exposure of 9.5 mg-years/m³ (range 0.6-39.7) and was exposed on average to 0.63 mg/m³ of dust while employed (table 1). Exposure to SO₂ was low; personal and area samples indicated that furnace workers were exposed to levels of 1.0-1.5 ppm (eight hour TWA), while carboselectors, preparation, maintenance, and other workers were exposed to 0.2 ppm or less. Occasional instantaneous peaks up to 4 ppm were measured in the furnace area by indicator tubes. No worker had a mean SO₂ exposure of more than 1 ppm while employed. Cumulative exposure to SO₂ was also low, averaging less than 2 ppm-years (range 0.02-19.5). Most recent exposure to SO₂ averaged 0.26 ppm (range 0-1.5) with 20% of the workforce exposed to one ppm or more (eight hour TWA).

SMOKING

The prevalence of the seven symptoms defined in table 2 was analysed by smoking habit for the entire study

group (table 3). Other symptoms obtained from the ATS questionnaire but not included in table 2 occurred infrequently (severe dyspnoea, wheezing attacks, chest colds) or gave similar results to the symptoms presented (other questions pertaining to cough and phlegm). With the exception of "usual phlegm," where a twofold, non-significant difference was present, the prevalence of symptoms was similar among never and ex-smokers; never and ex-smokers were therefore considered together as non-smokers in subsequent analyses. All symptoms occurred more frequently in current smokers.

After adjusting for age and occupational exposures by multiple logistic regression, current smoking habit was found to be strongly related to cough, phlegm, and wheeze, but only slightly related to mild dyspnoea (tables 4–6). Cigarettes smoked a day showed a similar but weaker association, whereas variables which included past cigarette smoking (years smoked, packyears) were poorly related to symptoms. In order to maximally control potential confounding from cigarettes, the current smoking/non-smoking categorical variable was retained for regression models evaluating relations between pulmonary symptoms and occupational exposures.

Table 4 Odds ratios from multiple logistic regression models for various symptoms on categories of cumulative SO_2 exposure, current smoking, and age

	Cumulative sulphur dioxide exposure category (ppm-years)					
	$\frac{0-0.25}{(n=35)}$	>0.25-1.00 (n = 47)	> 1.00-3.00 (n = 37)	$ > 3 \cdot 0 (n = 26) $	Current smokers (n = 78)	Age
Usual cough	1.00	1.44 (0.46- 4.48)	2.36 (0.71- 7.81)	1.73 (0.40- 7.63)	12·18 (4·62-31·5)	$\mathbf{p} = 0 \cdot 06$
Usual phlegm	1.00	1.26 (0.45- 3.51)	2.85* (0.93- 8.78)	5·47† (1·45–20·6)	2.65 (1.26- 5.58)	NS
Chronic phlegm	1.00	(0.43 - 0.01) (0.42 - 0.43)	2.94* (0.84-10.3)	(1.18 ± 20.0) 11.8 \pm (2.58 - 52.9)	2.40+ (1.03- 5.47)	NS
Wheezing on most days or nights	1.00	3.46† (1.02-11.6)	4.18†	8.17 (5.51-36.9)	5·31§ (2·24-12·6)	NS
Chronic wheezing	1.00	4·79† (1·15–19·9)	7.57; (1.72-33.0)	12.3 (2.28-65.0)	4·88§ (2·01-11·9)	NS
Dyspnoea while hurrying	1.00	3.16^{+} (1.12- 8.92)	2.07 (0.65- 6.50)	(8.58; (2.03-35.9))	2.09* (0.98- 4.45)	$\mathbf{p} = 0 \cdot 03$
Dyspnoea keeping up with peers	1.00	1.65 (0.28- 9.65)	1·87 (0·32–10·9)	3·46 (0·55–23·6)	1·83 (0·66- 5·03)	p = 0.08

Odds ratios for SO₂ exposure are relative to workers exposed to 0-0.25 ppm-years after adjusting for smoking and age. Ranges of values in parentheses indicate 95% CI for the odds ratio.

p Values were obtained from the chi-squared statistic for each odds ratio. * $0.05 \le p < 0.10$; $\pm 0.01 \le p < 0.05$; $\pm 0.001 \le p < 0.01$; p < 0.001. 631

	Average sulphur dioxide exposure category (ppm)					
	0-0.05 (n = 57)	>0.05-0.10 (n = 38)	>0.10-0.20 (n = 24)	> 0.20 (n = 26)	Current smokers (n = 78)	Age
Usual cough	1.00	0.81 (0.28-2.31)	1·90 (0·62- 5·85)	1·25 (0·40- 3·91)	10·3§ (3·91–26·7)	p = 0.01
Usual phlegm	1.00	1·12 (0·45-2·84)	3.49^{\dagger} (1.23-9.86)	2.54*	2.20^{+} (1.05-4.64)	NS
Chronic phlegm	1.00	0.67 (0.21-2.15)	2.61* (0.89- 7.67)	3.25^{\dagger} (1.13-9.38)	1.89 (0.82-4.37)	NS
Wheezing on most days or nights	1.00	1·52 (0·53-4·30)	2.48* (0.83- 7.44)	3·78† (1·26–11·3)	(0.02 + 57) 4.48§ (1.87-10.7)	NS
Chronic wheezing	1.00	2.64*	4·39† (1·35–14·2)	5·41‡ (1:67–17:6)	4.13	NS
Dyspnoea while hurrying	1.00	1.40 (0.58-3.39)	1.78 (0.63- 5.03)	3.00†	1.67 (0.80-3.51)	p = 0.0006
Dyspnoea keeping up with peers	1.00	0·44 (0·08–2·37)	2·15 (0·57- 8·11)	2·28 (0·64- 8·09)	1·46 (0·50–4·22)	p = 0.01

Table 5 Odds ratios from multiple logistic regression models for various symptoms on categories of average SO_2 exposure while employed, current smoking, and age

Odds ratios for SO₂ exposure are relative to workers exposed to an average of 0–0.05 ppm throughout their entire period of employment after adjusting for smoking and age. Range of values in parentheses indicate 95% CI for the odds ratio. p Values were obtained from the chi-squared statistic for each odds ratio.

squared statistic for each odds ratio. * $0.05 \le p < 0.10; \dagger 0.01 \le p < 0.05; \ddagger 0.001 \le p < 0.01; \$ p < 0.001.$

SULPHUR DIOXIDE

A strong, positive, and approximately linear, dose dependent relation was found between several symptoms and both cumulative (table 4) and average (table 5) SO₂ exposure while employed. Workers in the highest exposure categories had significantly increased rates of usual phlegm. This association was stronger for the more stringently defined symptom of chronic phlegm. Usual phlegm appeared to be principally related to exposure duration (p < 0.02), rather than exposure level (p > 0.10); this relation, however, was reversed for chronic phlegm (exposure level: p < 0.001, exposure duration: p > 0.10).

A strong association was present between exposure to SO_2 and usual wheeze, which also became greater for the more stringently defined symptom of chronic wheeze. Wheezing was primarily associated with exposure level (p < 0.04) and was not associated with duration of exposure. Mild dyspnoea (while hurrying) was found to increase with exposure to SO_2 , and significant odds ratios occurred in the highest SO_2 exposure categories. Moderate dyspnoea (keeping up with peers) followed a similar and generally linear trend but was not statistically significant. More severe dyspnoea occurred too infrequently to be analysed. Mild dyspnoea was associated with exposure level (p < 0.05) but not work duration. Age, however, was the most important factor related to dyspnoea.

Dysphoea was also evaluated on a linear scale proposed by Gandevia.¹⁰ After adjusting for age and current smoking status by multiple regression, each ppm of average SO₂ exposure was associated with a 20% reduction in self perceived respiratory function (p = 0.06).

Cough was not associated with exposure to SO_2 but was associated primarily with current smoking habit and to a lesser extent with age. Similar results were

 Table 6
 Odds ratios from multiple logistic regression models for various symptoms on categories of most recent SO₂ exposure, current smoking, and age

	Recent sulphur dioxide exposure category				
	0-1 ppm (n = 115)	≥ 1 ppm (n = 30)	Current smokers (n = 78)	Age	
Usual cough	1.00	1.00 (0.38- 2.65)	11.6§ (4.43-30.3)	p = 0.005	
Usual phlegm	1.00	0.88 (0.37- 2.67)	2·581 (1·27- 5·26)	NS	
Chronic phlegm	1.00	1.58 (0.65– 3.83)	2·16* (0·98– 4·77)	NS	
Wheezing on most days or nights	1.00	1.79 (0.73- 4.40)	4.486 (1.95-10.3)	p = 0.08	
Chronic wheezing	1.00	1.89 (0.76- 4.72)	4.181 (1.76- 9.90)	p = 0.05	
Dysphoea while hurrying	1.00	2.06* (0.87- 4.89)	1.67 (0.83- 3.39)	p = 0.0001	
Dyspnoea keeping up with peers	1.00	3.58† (1.22-10.5)	1.68 (0.61- 4.66)	p = 0.002	

Odds ratios for SO₂ exposures are relative to workers with most recent exposures of less than 1 ppm after adjusting for smoking and age. Ranges of values in parentheses indicate 95% CI for the odds ratio. p Values were obtained from the chi-squared statistic for each odds ratio. $*0.05 \le p < 0.10$; $†0.01 \le p < 0.05$; $‡0.001 \le p < 0.01$; \$p < 0.001.

SO₂ associated respiratory symptoms in SiC workers

Table 7 Prevalence of various symptoms (%) classified by current smoking habit and average exposure to SO₂ while employed for 145 silicon carbide workers

Symptom	Average SO ₂ exposure level (ppm)	Non and ex-smokers	Current smokers
Usual cough	00-1	12.2	43.5
	>0.1	11-1	68·8
Usual phlegm	0-0-1	24.5	32.6
1 0	>0.1	33-3	68 ∙8
Chronic phlegm	0-0.1	14.3	19.6
1 8	>0.1	27.8	50·0
Wheezing most days or	0-0-1	10.2	32.6
nights	>0.1	27.8	56.3
Chronic wheezing	0-0-1	8.2	26.1
chi	>0.1	27.8	53-1
Dysphoea while	0-0-1	36.7	43.5
hurrying	>0.1	61-1	65.6
Dysphoea keeping up	0-0-1	8.1	8.7
with peers	>0.1	22·2	28.1

Non-smokers and ex-smokers were respectively 31 and 36; 78 workers currently smoked, and 95 workers were exposed, on average, to less than 0.1 ppm SO₂ while employed.

obtained from the semiquantitative cough scale developed by Field.⁸ Productive cough on request was related to smoking (OR = 3.8; p < 0.001) but not to SO₂ exposure.

Plant closure prevented the study group from working during the six months preceding our health evaluation, so we could not assess symptoms with respect to current exposure to SO_2 . Instead, we analysed symptoms and job specific SO_2 exposure levels for the most recent position held (table 6). The 30 workers exposed to 1 ppm or more of SO_2 on their last day at work had slightly increased odds ratios for most symptoms, although significant results were found only for dyspnoea keeping up with peers.

RESPIRABLE DUST

Unlike SO₂, cumulative and average exposures to respirable dust rarely contributed to the logistic regression models used to evaluate pulmonary symptoms in this population of SiC workers. When analysed independently of SO2, cumulative dust exposure was significantly associated with chronic wheeze in the 10-20 mg-years/m³ exposure category (OR = 3.45; p < 0.05), and non-significant odds ratios greater than one were present for most symptom-exposure categories. A slight, non-significant association also was present between average dust exposure and symptoms of usual phlegm, wheeze, and dyspnoea keeping up with peers. In regression models which included dust and SO₂ exposure variables simultaneously, however, dust had no effect and all dust symptom associations were small and non-significant. The strong, highly significant SO₂ symptom associations remained and were nearly identical to values obtained from regression models which did not include a dust variable. No evidence of a dust SO_2 interaction effect was present.

SULPHUR DIOXIDE AND SMOKING SYNERGISM A greater than additive (synergistic) effect between smoking and exposure to SO_2 was present for most of the symptoms we evaluated, including cough, phlegm, and wheezing on most days or nights, but not for dyspnoea (table 7). Results were similar whether SO_2 was measured by cumulative exposure or by average exposure while employed.

Discussion

Silicon carbide production workers are exposed to several airborne emissions that are thought to damage the respiratory system. Evidence suggests that exposure to mixed dust containing SiC and small amounts of crystalline silica is responsible for the work related abnormalities found in the chest radiographs and pulmonary function tests of these workers.³⁶¹³⁻¹⁷ Our study suggests, on the other hand, that excess work related respiratory symptoms are associated primarily with the low level SO₂ exposures encountered in this industry.

Several symptoms, including phlegm (mucous hypersecretion), wheeze, and mild exertional dyspnoea showed strong, highly significant, dose dependent relations to SO₂ measured cumulatively, or by average SO₂ exposure while employed. Exposure to SO₂ on the last day of employment preceding six months of non-exposure was generally unrelated to pulmonary symptoms; however, misclassification caused by frequent job changes made this measure a poor estimate of true exposure.

The low levels of average SO₂ exposure while employed which were associated with pulmonary symptoms should not be confused with daily time weighted average exposure. Average SO₂ exposure while employed was a time weighted average of all exposures to SO_2 occurring throughout the entire duration of each worker's employment; as such, it was useful as an exposure index to evaluate epidemiological relations in an industry with frequent changes in job assignments. Included in its calculation, however, were long periods when SO₂ exposure was low or absent, making the measure numerically very small. Daily exposure to SO₂ primarily occurred to 20% of the workforce assigned to the furnace area and overhead cranes where levels of 1.0 to 1.5 ppm (eight hour TWA) were recorded; other workers were exposed to 0.2 ppm or less.

The linear exposure response relations we found for these pulmonary symptoms are consistent with the well known direct irritant effects of SO2, and support findings from studies of other industrial workers. Kehoe et al in their classic paper found haemoptysis, chest pain, epistaxis, hacking cough, prolongation of colds, morning cough, nasal irritation, and increased expectoration in refrigerator workers regularly exposed to 30 ppm of SO₂ throughout the day.¹⁸ More recently Skalpe reported a significantly higher frequency of cough, expectoration, and exertional dyspnoea in Norwegian pulp workers when compared with their paper worker counterparts.¹⁹ Their exposures, which averaged about 12 ppm (range 2-36 ppm), were probably overestimated by today's standard because they were calculated from area samples collected over short periods and not weighted for the entire working day. Ferris et al conducted a similar investigation in northern New Hampshire where the most recent exposures to SO, among pulp workers were about 2 ppm as calculated from area samples weighted for the working day.²⁰ Although differences were not statistically significant, pulp workers, especially those who smoked, had higher rates of chronic bronchitis and other non-specific respiratory illnesses when compared with workers in an adjacent paper plant.

Several studies of copper smelter workers also found a greater prevalence of respiratory symptoms associated with low level exposure to SO₂. Smith et al reported that workers exposed from 1.0 to 2.4 ppm SO₂ measured by eight hour TWA personal samples had raised rates of chronic cough and phlegm when compared with workers whose exposures were less than 1.0 ppm, although these findings were not statistically significant.²¹ Archer and Gillam evaluated a much larger cohort, comparing smelter workers with copper miners.²² They found highly significant excesses of several symptoms including morning cough, chronic phlegm, mild exertional dyspnoea, and chest tightness on returning to work. An often cited "negative" study also showed increased rates of a combination of symptoms defined by the authors as chronic obstructive lung disease among high exposed current smokers when compared with low exposed smoking controls.23

Unlike the variable exposures occurring in pulp mills, copper smelters, and SiC production operations SO₂ emissions in the corn refining industry tend to be low and fluctuate little over the workday. In this industry Fabbri *et al* found significantly increased wheezing in 57 workers exposed to SO₂ levels from 0.6 to 3.2 ppm when compared with 49 unexposed coworkers.²⁴ Similarly, Greaves *et al* described highly significant rates of cough, phlegm, and wheeze in workers exposed to SO₂ levels of 3 ppm or greater measured by personal samples and averaged over an eight hour day.²⁵ Episodes of wheezing were more frequent at exposures as low as 0.5 ppm.

In addition to studies of industrial workers, a large body of experimental evidence shows that low level SO₂ exposure, even below 1.0 ppm, may significantly affect airway reactivity and induce transient airway narrowing leading to respiratory symptoms and a temporary decline in pulmonary function.²⁶⁻²⁸ These effects are greater in asthmatics but occur in normal individuals as well.²⁹

In our study pulmonary symptoms were not associated with exposure to respirable dust, despite the presence in the dust of hydrocarbon particles shown by others to be associated with symptoms of chronic bronchitis.^{30 31}

Cigarette smoking was significantly associated with most of the pulmonary symptoms we evaluated and a strong synergistic effect between exposure to SO₂ and cigarette smoking was present. Similar synergy with cigarette smoking has been found in other workers exposed to SO₂,²⁵ cotton dust,^{22,33} and grain dust.^{34,35}

In conclusion, our study found that low level exposure to SO₂ in SiC production workers was associated with significantly increased rates of several pulmonary symptoms, including chronic phlegm, wheezing, and mild dyspnoea. Exposures to SO₂ primarily occurred to the 20% of the workforce employed in the furnace area and overhead cranes where fluctuating levels of SO₂ were measured averaging 1.0 to 1.5 ppm over an eight hour day, and irregular instantaneous peaks up to 4 ppm were found. In SiC production the current permissible exposure limit for SO₂ of 5 ppm does not appear to provide adequate protection from the effects of SO₂; rather, the level of 0.5 ppm proposed by NIOSH would be more appropriate for this industry.³⁶

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SO₂ associated respiratory symptoms in SiC workers

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