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Diurnal variation in peak expiratory flow rate among polyvinylchloride compounding workers

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Abstract

The diurnal variation in peak expiratory flow rate (PEFR) was studied in 24 mixers and 24 non-mixers in three polyvinylchloride (PVC) compounding plants and 24 non-PVC controls from a marine police workshop. The three groups (all men) were matched for age, race, and smoking. The mean respirable dust concentration (essentially PVC dust) was 1·6 mg/m³ for mixers and 0·4 mg/m³ for non-mixers. The mean diurnal variation in PEFR of the mixers was 6·5%. This was significantly higher than the 4·8% for non-mixers and 4·3% for the non-PVC controls. Six mixers had a diurnal variation of more than 15% on at least one day compared with none among the other two groups. Twenty nine per cent of mixers complained of wheezing compared with 4% of non-mixers and none among non-PVC workers. These differences were significant. Forced expiratory volume in one second (FEV_1) for the mixers was 10% below the predicted values whereas that of non-PVC workers was 2% below predicted values. The study indicates a significant acute airway constriction from occupational exposure to PVC dust.

A case of occupational asthma due to unheated polyvinylchloride (PVC) resin dust has been reported recently.[1] Both obstructive and restrictive ventilatory impairment and a high prevalence of wheezing complaints have been reported among PVC fabrication workers.[2,3] Abnormalities of lung function have also been reported in surveys of other workers exposed to PVC dust.[4,5]

We studied the diurnal variation in the peak expiratory flow rate (PEFR) in a group of PVC compounding workers and in a control group. Our aim was to determine whether PVC compounding workers have increased diurnal variation in PEFR suggesting exposure to a potential bronchoactive agent.

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Materials and Methods

MANUFACTURING PROCESS

The study was conducted in three PVC compounding factories. Polyvinylchloride pellets were produced by mixing together PVC resin powder with other additives such as plasticisers (for example di-octylphthalate), stabilisers (for example, lead sulphate), fillers (for example, calcium carbonate), and pigments. Azodicarbonamide (a blowing agent) was not used in the three factories. The bulk of the raw material was the PVC resin powder constituting more than 90% by weight of the mixture. The mixture was then blended and heated up to 170°C and extruded as pellets.

The most visibly dusty job was that of the mixers who had to open bags of dry powdered materials and tip them into hoppers. This was carried out on raised platforms (about 8–10 m high). Temperatures in the hoppers were around 135°C, the result of frictional heat from blending. The hoppers were equipped with local exhaust ventilation.

Heating and extrusion took place at the floor level. The extruded pellets were collected and packed by the packers who were exposed to a relatively low level of dust. Other less exposed workers were forklift drivers, storemen, mechanics, fitters, electricians, material testers, cleaners etc.

STUDY POPULATION

A total of 72 male workers were studied, consisting of 24 mixers, 24 low exposure non-mixers, and 24 non-PVC controls. All mixers in the three factories were invited to participate in the study. The 24 mixers represent 80% of all mixers. The low exposure PVC workers and the non-PVC controls were matched with the mixers for age (±five years), race, and smoking state. The low exposure PVC workers were for example, forklift drivers, storemen, maintenance staff, and material testers from the same three PVC factories. The non-PVC controls were mechanics and maintenance staff from a marine police workshop with no exposure to PVC dust or any known asthma inducing agents.

PEFR RECORDING

Each participant was given a mini-Wright peak flow meter and instructed in its correct use. He was asked to perform three blows after maximal inspiration on each occasion and to record the results on a form. The highest of the three readings was taken. Six daily recordings (every three hours) during the waking hours were made for one week (six working days and one day off). Recording started on a Monday and were made both at the workplace and at home.

The diurnal variation in PEFR was calculated as the difference between the highest and lowest PEFR values as a percentage of the highest PEFR on each day. For each worker the mean diurnal variation for the one week period was calculated.

PULMONARY FUNCTION

Forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) were performed on a dry wedge spirometer (Vitalograph) on Monday mornings when workers began their shifts. The spirometer was calibrated before use. The best FEV_1 and best FVC were taken from three technically satisfactory forced expiratory manoeuvres where the best two recordings were within 5% of each other. All values were corrected to body temperature and pressure saturation. Height (to the nearest centimetre) was measured without shoes. Predicted normal values for FEV_1 and FVC were calculated based on regression equations developed by Zee[6] for local Chinese and Malay men.

RESPIRATORY QUESTIONNAIRE

Each subject was interviewed by a trained field investigator using a structured questionnaire. Data were obtained on pulmonary symptoms, personal biodata, detailed work history including previous employment, past medical history, atopy, and smoking habits.

ENVIRONMENTAL ASSESSMENT

A total of 45 personal breathing zone samples of respirable PVC dust were collected over two to four hours on cellulose ester membrane filters of 37 mm diameter and 8·0 μm pore size using SKC personal dust sampling equipment at flow rates of 2·0 l/minute. Twenty one samples were taken from mixers and 24 samples from less exposed workers (non-mixers).

A total of eight static samples of air were collected for analysis of hydrogen chloride (HCl) and vinyl chloride monomer (VCM) (four each). Sampling was taken from four points: one near the mixer (on the platform), two near the extruder (floor level), and one outside the factory (ambient air). Sampling duration was three hours. For HCl sampling, air was drawn through an impinger at 1 l/min. Air was drawn through a charcoal tube at 80 ml/min for VCM sampling. Analysis was by liquid chromatography for HCl and by gas chromatography for VCM.

STATISTICAL ANALYSIS

Statistical methods to compare exposed workers and controls were analysis of variance (F test), t test for independent quantitative variables, χ^2 test, and Fisher's exact test.

Results

ENVIRONMENTAL ASSESSMENT

Respirable dust concentrations for mixers (21 samples) ranged from 0·2 to 2·9 mg/m^3 with a mean of 1·6 mg/m^3. Respirable dust concentration for non-mixers (24 samples) ranged from 0·1 to 1·0 mg/m^3 with a mean of 0·4 mg/m^3.

Hydrogen chloride and VCM were not detected in any of the eight samples taken (detection limit = 0·03 mg/m^3 for both HCl and VCM).

STUDY POPULATION

Among the 72 subjects, 33·3% were Malays and the rest Chinese. Fifty four per cent were smokers (including 17% ex-smokers). Tables 1–3 summarise personal data, prevalence of respiratory symptoms, and results of lung function tests and PEFR monitoring of these subjects. Non-PVC subjects were taller than those in the other groups. Although they

Table 1 Characteristics of study population

	High exposure mixers (n = 24)	*Low exposure non-mixers (n = 24)*	*Non-PVC workers (n = 24)*
Age	38·0 (8·4)	36·8 (8·1)	36·3 (8·1)
Exposure duration to PVC (y)	11·1 (5·7)	13·0 (5·8)	—
Height (cm)	165·8 (5·8)	166·0 (7·0)	170·5 (5·7)*
Cigarette-years	150·1 (258·0)	165·2 (208·7)	186·7 (281·1)
FEV_1 (l)	2·8 (0·5)	3·0 (0·6)	3·3 (0·5)
FVC (l)	3·2 (0·6)	3·4 (0·7)	3·7 (0·6)

Data are given as mean (SD).
*p < 0·05 (ANOVA).

Table 2 Prevalence of symptoms

	High exposure mixers (n = 24)	*Low exposure non-mixers (n = 24)*	*Non-PVC workers (n = 24)*
Cough	4 (16·7)	2 (8·3)	2 (8·3)
Phlegm	6 (25·0)	4 (16·7)	1 (4·2)
Rhinitis	5 (20·8)	7 (29·2)	5 (20·8)
Eye irritation	4 (16·7)	1 (4·2)	1 (4·2)
Breathlessness	3 (12·5)	2 (8·3)	1 (4·2)
Wheeze	7 (29·2)*	1 (4·2)	0 (0)

Data are given as number (%) with positive symptoms.
*p = 0·002 (χ^2 test); p = 0·005 compared with non-PVC controls (Fisher's test); p = 0·02 compared with low exposure non-mixers (Fisher's test).

Table 3 Results of lung function and PEFR

	High exposure mixers (n = 24)	*Low exposure non-mixers (n = 24)*	*Non-PVC workers (n = 24)*
% Predicted FEV_1	90·4 (10·8)*	94·2 (13·7)	97·6 (10·9)
% Predicted FVC	90·9 (12·5)	93·4 (12·6)	96·9 (10·9)
FEV_1/FVC (%)	86·5 (6·1)	87·1 (4·6)	87·4 (5·6)
DV PEFR (%)	6·5 (4·2)**	4·8 (2·3)	4·3 (2·3)

Data are given as mean (SD).
*p = 0·03 compared with non-PVC controls (*t* test).
**p = 0·03 compared with non-PVC controls (*t* test); p = 0·05 compared with low exposure non-mixers (*t* test).

smoked more, this was not statistically significant (table 1). More mixers complained of wheeze than did other subjects. No significant difference was found in the prevalence of other symptoms (table 2). The FEV_1 of the mixers was 10% below the predicted values whereas that of the non-PVC subjects was 2% below the predicted values (p = 0·03). The mean diurnal variation in PEFR of the mixers of 6·5% was higher than either of the other control groups (table 3). Six mixers had a diurnal variation of greater than 15% on at least one day compared with none among the other groups (p = 0·01, Fisher's test). The highest diurnal variation in a day was 23·9% in a mixer.

Discussion

Diurnal variation has been studied in workers exposed to grain dust[7] and tobacco dust.[8] It has been shown that age and smoking were significantly correlated in a positive fashion with diurnal variation in PEFR. The mixers in our study had a higher diurnal variation in their PEFR compared with both low exposure and non-PVC controls matched for age, smoking state, and race.

The diurnal variation in PEFR among PVC workers has not been investigated in any previous study. The median diurnal variation in PEFR of grain elevator workers was 5·9%.[7] This is slightly higher than the median diurnal variation in PEFR of 4·8% for PVC mixers. The method for calculating diurnal variation in PEFR, however, for the grain elevator workers was different and resulted in a higher diurnal variation. The diurnal variation in PEFR was calculated as the difference between the highest and lowest PEFR values as a percentage of the mean PEFR on each day. In our case we expressed the difference as a percentage of the maximum PEFR on each day. Furthermore, the grain elevator workers were older (median age = 44 years) and had higher prevalence of smoking (81% were smokers).

The mean diurnal variation in PEFR in a group of tobacco workers was 14·4% and for their controls, it was 9·5%.[8] This is much higher than that seen in our subjects although method of calculating diurnal variation in PEFR was similar to ours. Their subjects were mainly women, however, and older (mean age = 43 years). The prevalence of smoking was 43·8%. Also only four PEFR recordings were taken each day.

The higher prevalence of wheezing complaints and reduced FEV_1 (below predicted values) among mixers compared with non-PVC controls provide further evidence that mixers in the PVC compounding industry may be exposed to a bronchoactive agent. We did not detect any overt cases of occupational asthma. This is however, not unexpected in a cross sectional study since we are likely to be studying a survivor population. We have recently completed a similar study of polyurethane foam operators exposed to toluene diisocyanate (TDI) (not yet published). No overt cases of occupational asthma were detected in this group of workers exposed to a known asthma inducing agent, TDI.

Possible asthma inducing agents in the PVC compounding industry include PVC dust, additives, and PVC decomposition products (for example HCl, VCM). Among the additives, only azodicarbonamide is known to cause asthma but azodicarbonamide was not used in the three plants surveyed. So far dioctylphthalate (DOP) and other phthalate esters have not been identified as asthma inducing.[10] Furthermore, the DOP was in liquid form. Other additives such as stabilisers (for example, lead, barium, cadmium, and zinc salts) are not likely to cause asthma. Polyvinylchloride is thermally stable at temperatures below 225°C.[11] Above 225°C, PVC will degrade, releasing first HCl and then, above 300°C, carbon monoxide, carbon dioxide, benzene, and VCM. Above 600°C small amounts of phosgene and chlorine are formed. Under normal operating conditions, temperatures do not exceed 170°C. At the mixing station, temperatures do not exceed 135°C. Hydrogen chloride and VCM were not detected in the vicinity of the mixer or the extruder.

The bulk of the dust is PVC. That unheated PVC dust can induce asthma has been shown by a positive challenge test.[1] Our study provides further evidence of a significant effect of variable acute airway constriction from exposure to PVC dust.

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Examples of common forms of references are:

1 International Steering Committee of Medical Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1979;1:532–5.
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Pulmonary effects of polyvinyl chloride dust exposure on compounding workers

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NG TP, LEE HS, LOW YM, PHOON WH, NG YL. Pulmonary effects of polyvinyl chloride dust exposure on compounding workers. *Scand J Work Environ Health* 1991;17:53—9. Spirometry, chest radiography, environmental measurements, and a questionnaire on respiratory symptoms were used to evaluate the effects of exposure to polyvinyl chloride (PVC) dust on 171 Chinese and Malay PVC compounding workers in comparison with an unexposed reference group. Workers with high cumulative PVC dust exposure had a lower forced expiratory volume in 1 s and forced vital capacity, and a higher prevalence of radiological profusion of small opacities. Wheezing or chest tightness was also significantly more frequent in this group. Unlike previous studies, the PVC compounding workers in this study were exposed to only negligible amounts, if any, of vinyl chloride monomer or thermal degradation products of PVC such as hydrogen chloride, phosgene, or chlorine. The conclusion was drawn that a low grade of pneumoconiosis and a small degree of lung function impairment is associated with PVC dust exposure. Reversible airways obstruction is also likely and warrants further investigation.

Key terms: radiological profusion, spirometry, symptoms.

In 1970, Szende et al (1) first described radiological and histopathological evidence of a moderately diffuse fibrosis with granulomatous reaction in a worker exposed to PVC dust. Since then, several epidemiologic studies have investigated the pulmonary effects of occupational exposure to vinyl chloride monomer (VCM) and polyvinyl chloride (PVC). These studies have shown an increased prevalence of radiological, lung function, or subjective abnormalities (2—9). It would appear, however, that, in many of the earlier studies, control of possible biases or confounding was less than adequate because of the lack of appropriate reference groups, failure to take into account the influences of age or cigarette smoking properly, or the absence of standardized procedures in radiographic reading such as the use of standard radiographs of the International Labour Office (ILO) or multiple readers blind to the exposure status of the subjects. Several studies have also provided conflicting evidence for lung function impairment (4, 6, 7).

It is also difficult to be certain that the pulmonary abnormalities reported in these studies were entirely due to exposure to PVC dust alone, since combined exposures to VCM and other air contaminants were known to be present. Most previous studies have investigated only vinyl chloride polymerization workers, who were exposed to both PVC dust and VCM (2—6). VCM has been reported to cause pulmonary fibrosis in animals (10), and PVC dust exposure among workers has been shown to induce less severe respiratory abnormalities than combined exposure to PVC and VCM (3).

PVC fabrication workers have also been studied (7—9), but these included other process workers, such as in calendering, laminating, or extruding, with potential exposure to thermal degradation products, solvents, and possibly additives known to cause acute bronchial reaction.

No previous investigation has studied a group of workers exposed almost exclusively to PVC dust. We have conducted a study of one such group, namely, PVC compounding workers, and present the results of the radiological, lung function, and symptoms evaluation in this report.

Materials and methods

Manufacturing processes

The study was conducted in three factories which produced PVC pellets which were then supplied to user industries for fabrication into other PVC products. The manufacturing processes were similar in all the factories, and no other processes were in operation except in one factory, where the pellets were fabricated into PVC rails in a separate building 100 m away. The bulk of the raw material, constituting more than 90 % by weight, was PVC resin powder, which was compounded with other chemicals such as plasticizers, stabilizers, and pigments in semiautomated processes at temperatures not exceeding 170°C. The most visibly dusty jobs in the factories were performed by

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mixers, who opened bags of the dry powdered materials and tipped them into hoppers. Other workers with less PVC dust exposure were employed as packers/weighers, forklift drivers, storagemen, machine operators, fitters, electricians, material testers, cleaners, and so forth (table 1).

Environmental assessment

Current personal exposure to respirable PVC dust was measured for 10 job titles in each factory. A total of 56 personal samples of respirable PVC dust from the breathing zone of the workers was collected over sampling periods of about 4 h at the same time of the day on cellulose ester membrane filters with a diameter of 37 mm and a pore size of 8.0 μm. Personal dust sampling equipment (SKC Inc) was used at a flow rate of 2.0 l/min. The job-specific exposures are summarized in table 1.

Table 1. Dust concentrations in the three PVC factories by job title.

Job title	Number of samples	Mean (mg/m³)	Range (mg/m³)
Mixers	21	1.55	0.21—2.90
Foremen	3	0.42	0.38—0.70
Cleaner	1	0.72	
Packers	9	0.28	0.16—0.58
Forklift operators	6	0.45	0.10—1.02
Storagemen	3	0.30	0.07—0.53
Machine operators	2	0.47	0.37—0.58
Mechanical/electrical fitters	3	0.44	0.09—0.85
Material testing workers	4	0.20	0.12—0.31
Screwrailing workers	3	0.33	0.06—0.81

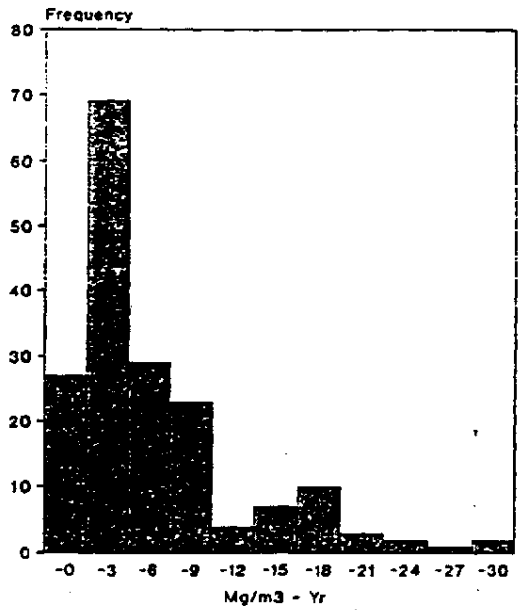

Figure 1. Distribution of cumulative dust exposure.

Study populations

All of the eligible 171 Chinese and Malay male workers aged 21 years and above and currently employed in production and maintenance work in these factories were studied. Since almost all of the workers were exposed to some degree of PVC dust, an external comparison group of unexposed workers was also studied at the same time. These were Chinese and Malay male workers, aged 21 years and above, with equivalent job titles such as drivers, mechanical workshop workers, electricians, and plumbers in three granite quarries (27 workers, 100 % response) and in one hospital (21 workers, 87 % response). The comparison workers in the quarries were exposed to low levels of silica (average 0.15 mg/m³ respirable granite dust).

A structured questionnaire was administered by trained field investigators to each worker, who provided his personal data, a detailed work history (including previous employment in other dusty occupations), information on previous medical history of tuberculosis, hypertensive heart diseases and so forth, smoking habits, and responses to the British Medical Research Council questionnaire on chronic bronchitis, as well as to additional questions on wheezing or chest tightness and atopy.

Spirometric testing was performed on a dry-wedged bellows spirometer (Vitalograph[5]), which was regularly calibrated before use, on Monday mornings when workers began their shifts after a weekend off. Each subject performed maximal forced expiratory maneuvers in the standing position without a nose clip, and the best of three acceptable curves was used in deriving the forced vital capacity (FVC), forced expiratory volume in 1 s ($FEV_{1.0}$), and the FEV % [$(100 \times FEV_{1.0})$/FVC]. All of the values were corrected to body temperature and pressure saturation (BTPS). Height (to the nearest centimeter) and weight (to the nearest gram) was measured, the subject being without shoes and in light summer clothing.

Chest radiography was performed on the entire group of mixers (who were the most highly exposed to PVC dust) and subsamples of exposed nonmixers and unexposed referents. Since the mixers were a generally older group, the exposed nonmixers included other production and maintenance workers except those in the PVC railing and screw departments, who were mostly young workers. The external comparison group included only workers in the quarry, who were also comparable in age to the mixers. Postero-anterior radiographs of the chest were taken on high-quality large-sized film and were then read independently and in random order by three readers, who were not aware of the exposure status of the subjects to which the radiographs belonged. The profusion of rounded and irregular small opacities was read, with reference to standard films, and according to the ILO International Standard Classification of Radiographs of Pneumoconiosis of 1980. There was reasonably good reading agreement between the observers (appendix).

Analyses

Since the observed respiratory effects were almost certainly the results of cumulative PVC dust exposure, which is a function of exposure intensity and duration, a cumulative dust exposure index was estimated for each PVC-exposed worker on the basis of information provided on the duration of employment for every job ever held in the factories. The current factory- and job-specific dust concentration was assigned to each job held and multiplied by the duration in years and calculated as follows:

$$C = \Sigma K_j \cdot T_j,$$

where C = the cumulative exposure index, K = the mean dust concentration (mg/m^3), T = exposure duration (years), and j = job category. It was recognized, however, that current measurements could not be assumed to reflect the previous dust exposure intensities accurately. Hence, the cumulative indices were only used to categorize workers into high and low cumulative exposure groups. This classification was done with the use of an arbitrary level of 10 mg/m^3-years to separate those with high cumulative exposure from those with low (or average) exposure. Typically, those with a cumulative index higher than 10 mg/m^3-years would represent a mixer exposed to an average PVC dust concentration of 1.0 mg/m^3 for 10 or more years. The inclusion of, say, a packer typically exposed to 0.33 mg/m^3 for 30 or more years was also allowed for. The distribution of the cumulative PVC dust exposure indices is shown in figure 1. The unexposed group was assigned a nil cumulative exposure to PVC dust.

Comparisons of the lung function parameters between the exposed groups were performed by multiple regression techniques using general linear regression modeling procedures from the SAS (statistical analysis system) programs. The $FEV_{1.0}$, FVC, and FEV % were adjusted for age, height, cigarette-years, and race, entered as covariates in the models. The lung function parameters of the exposed groups were compared with those of the unexposed group with significance testing by an analysis of covariance. In addition, the lung function parameters of all the groups were compared with external population norms with the use of prediction equations for Singapore Chinese and Malay males (11, 12).

Radiological opacities were defined categorically as a profusion of small opacities of 0/1 or greater, 1/0 or greater, or 1/1 or greater, as read by two or more of the three readers. Separate analyses of the prevalence of radiological opacities for each reader were also performed. Stratified analyses and the Cochran-Mantel-Haenszel test were used when the prevalences of radiological opacities were compared, and also when the prevalences of symptoms were compared by exposure groups with control for the influence of age (<45 years, ≥45 years) and smoking status (nonsmoker and ever smoker).

Thirteen workers with significant dust exposure from other previous occupations, one worker with previous pulmonary tuberculosis, one worker with hypertensive heart disease, and one worker with current respiratory tract infection were excluded from the analysis.

Results

Table 2 shows the mean age, height, and the proportions of each ethnic group and smokers by exposure group and table 3 the exposure characteristics of the groups. Multiple regression modeling showed both the $FEV_{1.0}$ and FVC to be significantly related, in expected fashions, to age ($P<0.001$), height ($P<0.0001$), and cigarette-years ($P<0.01$). Race was not a significant independent variable after adjustment for smoking in the model. $FEV_{1.0}$ and FVC, after adjustment for the effects of these influencing variables, were both lower in the PVC-exposed groups (table 4). The percentage of predicted $FEV_{1.0}$ and FVC in the unexposed group indicates that their lung volumes were comparable to the external population norms, whereas both $FEV_{1.0}$ and FVC were about 4 % lower in the low-exposure group and 7 % lower in the high-exposure group.

The prevalence of respiratory symptoms among the exposure groups, after control for age and smoking, did not show any statistically significant differences except for frequencies of wheezing or chest tightness, which was greatest in the high-exposure group (table 5). Radiological small opacities, as read by at least two readers, with profusion 0/1 and greater, age and smoking being controlled for, were significantly more frequent in the exposed groups in comparison with the

Table 2. Physical and smoking characteristics of the subjects by cumulative exposure group.

Group	Age years		Height (cm)		Race				Smoking							
					Chinese		Malay		Non-smoker		Ex-smoker		Current smoker		Cigarette-years	
	Mean	SD	Mean	SD	N	%	N	%	N	%	N	%	N	%	Mean	SD
Unexposed (N = 48)	36.0	12.7	167.1	5.6	39	81	9	19	16	33	4	8	28	58	239	377
Low exposure (N = 121)[a]	35.2	10.1	167.1	6.2	102	84	19	16	63	52	6	5	52	43	105	162
High exposure (N = 34)[b]	41.9	8.77	165.4	5.3	24	71	10	29	10	29	9	26	15	44	317	404

[a] < 10 mgm³-years.
[b] ≥ 10 mgm³-years.

Table 3. Exposure characteristics of the subjects by cumulative exposure group. (PVC = polyvinyl chloride)

Group	PVC cumulative exposure		Years exposed to PVC		Job			
					Mixers		Exposed nonmixers	
	Mean	SD	Mean	SD	N	%	N	%
Unexposed	—	.	—	.	..	..	..	..
Low exposure (< 10 mg/m³-years)	2.9	2.6	7.5	6.2	9	7.4	112	92.6
High exposure (≥ 10 mg/m³-years)	18.0	5.5	14.7	4.2	30	88.2	4	11.8

Table 4. Lung functions by exposure group. [$FEV_{1.0}$ = forced expiratory volume in 1 s, FVC = forced vital capacity, FEV% = (100 · $FEV_{1.0}$/FVC]

Group	$FEV_{1.0}$ (l)[a]		FVC (l)[a]		$FEV_{1.0}$ (% of predicted)[b]		FVC (% of predicted)[b]		FEV%[b]	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Unexposed	3.13	0.41	3.64	0.48	99.3	12.2	99.4	11.8	86.3	6.2
Low exposure (< 10 mg/m³-years)	3.03	0.44	3.47	0.53	96.4	12.3	95.1	11.9	87.4	6.2
High exposure (≥ 10 mg/m³-years)	2.93	0.41**	3.39	0.47**	92.7	12.4**	92.3	12.0**	86.5	6.3

[a] Adjusted for age, height, race, cigarette-years.
[b] Adjusted for cigarette-years.
* $P<0.05$, ** $P<0.01$ (analysis of covariance).

Table 5. Respiratory symptom prevalence by exposure group.

Symptom	Unexposed		Low exposure (< 10 mg/m³-years)		High exposure (≥ 10 mg/m³-years)	
	N	%	N	%	N	%
Cough						
Morning	5	10.2	11	9.1	6	17.6
Day or night	8	16.3	7	5.8	5	14.7
Three months in year	3	6.1	6	5.0	4	11.8
Phlegm						
Morning	8	16.3	22	18.2	8	23.5
Day or night	9	18.4	13	10.7	6	17.6
Three months in year	8	16.3	14	11.6	6	17.6
Periods of cough and phlegm	3	6.1	1	0.8	—	0.0
Breathlessness	10	20.8	22	18.2	6	17.6
Wheezing/chest tightness	2	4.1	10	8.3	8	23.5

* $P<0.01$ (Cochran-Mantel-Haenszel test of general association controlling for age and cigarette smoking).

unexposed group (table 6). Stratified analyses showed that age, but not smoking, was significantly associated with radiological small opacities with profusion ≥0/1 (at least two readers).

Altogether 17 persons had small opacities of profusion 1/0 and greater in their radiographs, as read by at least two readers. Four persons had radiological profusion 1/1, and one had 1/2 profusion. No one in the unexposed group had radiological profusion greater than 1/0. There was a twofold higher prevalence of radiological opacities of profusion 1/0 or greater in the high-exposure group as compared with the low-exposure group. Radiological small opacities of profusion 1/1 and greater were seen only in the high-exposure group.

Similar results were obtained when the radiographic readings for each reader were used in the analyses. The predominant types of opacities were irregular although the readers differed in their interpretation, particularly with respect to the shape but not the size of the opacities (which were all read as <0.5 mm in diameter). Reader one read mostly irregular opacities, reader two read more irregular than rounded opacities, and reader three read more rounded than irregular opacities (table 7).

Discussion

In a cross-sectional study, the reference group is the most appropriately chosen from within the same fac-

Table 6. Prevalence of radiological opacities by reader and exposure group.

Parameters	Unexposed[a] (N = 27)		Low exposure[b] (N = 81)		High exposure[c] (N = 29)	
	N	%	N	%	N	%
Reader 1						
Profusion ≥ 0/1	7	25.9	49	60.5	21	72.4*
Profusion ≥ 1/0	1	3.7	13	16.0	8	27.6*
Profusion ≥ 1/1	1	3.7	—	0.0	4	13.8*
Reader 2						
Profusion ≥ 0/1	4	14.8	38	46.9	16	55.2*
Profusion ≥ 1/0	—	0.0	12	14.8	8	27.6*
Profusion ≥ 1/0	—	0.0	—	0.0	4	13.8**
Reader 3						
Profusion ≥ 0/1	4	14.8	32	39.5	13	44.8*
Profusion ≥ 1/0	—	0.0	14	17.3	7	24.1*
Profusion ≥ 1/1	—	0.0	8	9.9	6	20.1**
Two or more readers						
Profusion ≥ 0/1	4	14.8	37	45.7	16	55.2*
Profusion ≥ 1/0	—	0.0	10	12.3	7	24.1*
Profusion ≥ 1/1	—	0.0	—	0.0	5	17.2**

[a] Mean age of the group 41.1 (SD 12.4) years with seven (26 %) nonsmokers, three (11 %) ex-smokers, and 17 (63 %) current smokers.
[b] Low exposure = < 10 mg/m³-years; mean age of the group 35.2 (SD 10.3) years with 60 (50 %) nonsmokers, seven (6 %) ex-smokers, and 53 (44 %) current smokers.
[c] High exposure = ≥ 10 mg/m³-years; mean age of the group 41.0 (SD 9.1) years with nine (26 %) nonsmokers, nine (26 %) ex-smokers, and 16 (47 %) current smokers.
* $P \leq 0.05$, ** $P < 0.01$ (Cochran-Mantel-Haenszel test of general association, controlling for age and smoking status).

Table 7. Distribution of radiological types by reader and exposure group.

	Unexposed		Low exposure (< 10 mg/m³-years)		High exposure (≥ 10 mg/m³-years)	
	Rounded	Irregular	Rounded	Irregular	Rounded	Irregular
Reader 1						
Profusion ≥ 0/1	3	4	—	49	—	21
Profusion ≥ 1/0	—	1	—	13	—	8
Profusion ≥ 1/1	—	1	—	—	—	4
Reader						
Profusion ≥ 0/1	3	1	11	27	8	8
Profusion ≥ 1/0	—	—	4	8	5	3
Profusion ≥ 1/1	—	—	—	—	3	1
Reader 3						
Profusion ≥ 0/1	3	1	24	8	9	4
Profusion ≥ 1/0	—	—	10	4	5	2
Profusion ≥ 1/1	—	—	5	3	4	2

tory if it is certain that it is indeed unexposed. Our environmental assessment clearly indicated a secondary exposure in the ambient air for almost all of the workers in these factories; and this secondary exposure was likely in itself to produce some degree of pulmonary effects. It was therefore necessary to choose an external comparison group, which we believed was appropriate for the study since it was an occupational counterpart in other industrial undertakings. The validity of this group was well supported externally by the good approximation of their lung functions to predicted norms from other independent population samples.

The previous dust levels in the factories were not known, but there were reasons to suppose that they were probably higher than current levels. Since only current dust levels were known, it should be emphasized that the estimates of cumulative PVC dust exposure were not based on actual dust exposure levels. The data in this study were, therefore, used only for categorizing broad groups of PVC-exposed subjects and not for deriving quantitative exposure-response relationships.

Exogenous sources of VCM and thermal decomposition products may be ruled out as the cause of the observed pulmonary effects since VCM exposure from residues in the PVC powder was negligible and irritant and toxic gases like hydrochloric acid, VCM, phosgene, and chlorine are only formed at temperatures in excess of 225°C. We also made measurements

at the mixer and extruder, and hydrochloric acid and VCM were not detectable. However, it is more difficult to exclude the possibility that the radiological opacities could also have resulted from additives containing radio-dense calcium and barium compounds in the mixture, although only small quantities of these substances were used.

The radiological readings were all done by physicians experienced in the assessment of radiographs for pneumoconiosis, and standard guidelines were used under controlled conditions so as to eliminate subjective bias and produce unequivocal case identification. Under the circumstances, the effects of aging and cigarette smoking were unlikely to produce radiological opacities more profuse than category 1/0. There should therefore be little if any doubt that at least radiological profusion 1/1 in five workers indeed represented PVC pneumoconiosis.

Both diffuse micronodular and linear-reticular opacities have been described in case reports of PVC pneumoconiosis, characterized histopathologically as pulmonary fibrosis associated with granulomatous lesions (1, 13). Similar histological changes were observed in experimental studies with guinea pigs and rats (14, 15). As in previous epidemiologic studies (3, 5, 7), the present study indicated that the radiological lesions arising from PVC dust exposure were of limited profusions with a predominance of irregular small opacities. The reported prevalence of radiological lesions varied greatly among the studies. It is, however, difficult to make valid comparisons among the studies because of differences in procedures and criteria for radiological case definition, although in two of the studies, ILO standard films and radiological classification were employed for reading by a minimum of two or three readers (5, 7). Furthermore, the prevalence of radiological small opacities may be expected to vary with the level of PVC dust exposure, but such measurements were only performed in one study (6). In the study by Soutar et al (6), the estimated dust index of 14 mg/m^3-years appeared to be comparable to that of the high-exposure group in our study, but in both studies, past levels of PVC dust exposure was unknown.

Previous reports of an association between PVC exposure and a restrictive type of lung function impairment (2, 3, 6) was supported by the findings of the present study. The average lung function loss was modest, but, given individual variation in responses to PVC exposure, a few persons in any exposed population may be expected to experience clinically significant lung function impairment. PVC exposure was also found to be associated with an increased prevalence of dyspnea in the study by Soutar et al (6). No such association was demonstrated in our study, but it should be noted that, whereas the study by Soutar et al included past workers, we could only study current workers who might probably be overrepresented by those whose functional impairment was not severe enough to prevent them from continuing with their rent employment. We did, however, find an increa prevalence of wheezing or chest tightness, and findings have not been reported in previous stud Apparently, responses to these symptoms were n elicited in previous studies. The possibility of re ble airway obstruction, however, corroborates wit results of a recent study showing across-shift FE changes in PVC fabrication workers (9), and a re report of bronchial asthma from inhalational pr cation tests with PVC dust (16), and should there be further investigated.

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Appendix

Agreement between the readers of the radiographs

Reader 2	Reader 1					
	0/—	0/0	0/1	1/0	1/1	1/2
0/—	—	8	—	—	—	—
0/0	—	48	21	2	—	—
0/1	—	4	24	9	1	—
1/0	—	—	10	5	1	—
1/1	—	—	—	1	2	—
1/2	—	—	—	—	—	1

Kendall's tau-B 0.637 (SE 0.041)

Reader 3	Reader 2					
	0/—	0/0	0/1	1/0	1/1	1/2
0/—	—	—	—	—	—	—
0/0	8	63	14	3	—	—
0/1	—	7	18	2	—	—
1/0	—	1	4	2	—	
1/1	—	—	2	8	3	—
1/2	—	—	—	—	—	1

Kendall's tau-B 0.632 (SE 0.054)

Reader 3	Reader 1					
	0/—	0/0	0/1	1/0	1/1	1/2
0/—	—	—	—	—	—	—
0/0	—	55	20	3	—	—
0/1	—	5	17	5	1	—
1/0	—	—	5	2	0	—
1/1	—	—	3	7	3	—
1/2	—	—	—	—	—	1

Kendall's tau-B 0.574 (SE 0.054)

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HYDRO POLYMERS
Vinyls Division

Your ref. | Your letter of

Our ref. | Date

HMC/PAM | 16 April 1991

Mr R T Gottesman
Executive Director
The Vinyl Institute
Wayne Interch. Plaza II
155 Route 46 West
Wayne, NJ 07470
U S A

Dear Roy,

PVC DUST

I draw your attention to the attached paper, together with the handwritten note from our own Medical Advisor.

You will be advised of the results of a critique of the paper.

Yours sincerely,

H M Clayton
Senior Environmental Advisor

BFG21391

22531008

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Internal Memorandum

To Mr. Harold Clayton.

From Dr. I.M.M. Bagnall.

Date 8/4/91

Sorry for handwritten Memo but I have no typist.

Re: CIA. VCM. HWG. meeting 28/3/91

Your paper: PVC Dust - Swedish Criteria Document.

The Committee agree with you that they know of no published work which would support the statements on pages 19, 20, 22.

Attached is a further little gem from Singapore Polymers which Geoff Paddle does not feel is very significant. We are in the process of obtaining a quality critique.

Yours

2253109

BFG21392