Respiratory Survey of Workers in a Pulp and Paper Mill in Powell River, British Columbia[1-3]

MOIRA CHAN-YEUNG, ROBERT WONG, LONIA MACLEAN, FELISA TAN, ELAINE DORKEN, MICHAEL SCHULZER, RONALD DENNIS, and STEFAN GRZYBOWSKI

SUMMARY

A respiratory survey was carried out in 1,932 workers in the pulp and paper mill in Powell River, British Columbia. The survey included a medical-occupational questionnaire, spirometry, chest radiographs, and environmental monitoring. The results obtained in 1,826 white male workers were analyzed.

The workers were divided into 6 groups according to the type of exposure at work: subjects in group 1 were mainly exposed to gases, vapors, and chemicals in the kraft mill; group 2, to wood dust; group 3, to paper dust; group 4, to CO; group 5, to all kinds of air contaminants; group 6 (control subjects), to much lower levels of various air contaminants at work. We were unable to demonstrate an increased prevalence of respiratory symptoms and pulmonary function abnormalities among workers exposed to gases and chemicals in the kraft mill. However, we found that workers exposed to wood dust had slightly but significantly lower pulmonary function compared to other groups.

Although cigarette smoking had a significant deleterious effect on pulmonary function, as expected, alcohol consumption also affected pulmonary function. Among nonsmokers, those who admitted to having more than 3 drinks per day had significantly lower pulmonary function than those who had no drinks or fewer than 3 drinks per day during the previous week. Among current smokers and ex-smokers, the effect of alcohol consumption on pulmonary function was not significant.

The concentrations of sulfur dioxide, hydrogen sulfide, and chlorine encountered in Powell River were low, well below the currently accepted threshold limit values, and this may account for the negative findings in this study. Similar studies should be carried out in other pulp and paper mills in British Columbia to establish whether the currently accepted threshold limit values for these gases are adequate.

Introduction

Pulp and paper production is one of the major industries in British Columbia. There are 21 pulp and paper mills in this province, with 13,000 employees; all except one are kraft mills. In the process of pulp production by the kraft process, wood chips are first introduced into large digesters and "cooked" under increased temperature and pressure with sodium sulfide, hydroxide, sulfite, and carbonate. In the final process, the pulp is washed and bleached with chlorine or chlorine dioxide. Workers involved in these proceses are exposed to sulfur dioxide (SO_2), hydrogen sulfide (H_2S), mercaptans, chlorine, chlorine dioxide, and wood dust.

In the paper making, the pulp is resuspended in water, and various additives and fillers are added to the mixture. The types of additives and fillers vary with the type of paper product to be manufactured. The slurry of the mixture is then poured out on a moving screen that allows the water to drain off, leaving behind a mat of the blend that is then run around steam-heated colanders to dry

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[1] From the Department of Medicine, University of British Columbia, Vancouver General Hospital, and the Industrial Hygiene Department, Workers' Compensation Board of British Columbia, Canada.

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[3] Requests for reprints: Dr. Moira Chan-Yeung, Department of Medicine, Vancouver General Hospital, Vancouver, B.C., Canada.

into paper products. Workers in the paper-making process are exposed mostly to paper dust.

Although there is considerable literature on the acute effects of chlorine, chlorine dioxide, and SO_2 on the lungs (1–5), comparatively little has been reported on the effects of long-term exposure, and the results are often conflicting. Ferris and co-workers (6) studied 147 workers in a pulp mill with possible significant exposure to the noxious gases and 124 workers in a paper mill where there was no such exposure. No significant differences were found in respiratory symptoms or in simple tests of ventilatory function between the 2 groups. Both working populations together had a lower prevalence of respiratory disease than did the male population of Berlin, N. H. However, in the study by Skalpe (7), significantly higher frequencies of cough, expectoration, and dyspnea on exertion were found among 54 workers in pulp mills compared to 56 workers in paper mills. The average maximal expiratory flow was also significantly lower in the group exposed to SO_2. In a longitudinal study of workers with long-term exposure to SO_2 in a smelter, Smith and associates (8) found that exposure to SO_2 was associated with an increase in respiratory symptoms and excessive loss of forced expiratory volume in one second (FEV_1) during 1 yr. Very little is known about the relationship between respiratory disease and measured environmental levels of air contaminants in the pulp mills.

An epidemiologic health survey and an industrial hygiene survey were carried out by the Occupational Lung Disease Unit, Department of Medicine, University of British Columbia, together with the Industrial Hygiene Department of the Workers' Compensation Board of British Columbia among the employees of the pulp and paper mill in Powell River, B.C. between October 1978 and April 1979. In this report we present the prevalences of respiratory symptoms and pulmonary function abnormalities.

Methods

Materials. The pulp and paper mill in Powell River has not only a kraft mill and a paper mill, but also a sawmill. The total available work force was 2,090 persons. Of these, 1,932 took part in the survey, for a participation rate of 92.4%. There were 60 women, but they were excluded from the analysis because they comprised only a relatively small fraction of the work force. The 40 nonwhite workers were also excluded, because in our previous study (9) we found that the predicted values of pulmonary function for white subjects were not appropriate for nonwhite subjects. The results obtained in 1,826 white male workers were analyzed.

The workers were divided into 6 groups according to the exposure in their current job; the classification was based not only on the job title, but also on the results of industrial hygiene data. Group 1 (vapors) included 219 workers exposed to gases and fumes; these were workers in the kraft mill and maintenance staff permanently assigned there. Group 2 (wood) included 315 workers exposed to wood dust; they were of workers in the sawmill and maintenance staff permanently assigned to the area. Group 3 (paper) included 278 workers who were exposed mostly to paper dust and occasionally to some chemicals; these were workers in the paper mill and its maintenance staff. Group 4 (CO) included 190 workers who were exposed mostly to CO from automobile exhaust or who were truck drivers. Group 5 (mixed) included 324 maintenance workers who were exposed to all kinds of air contaminants in the mill. Group 6 (control group) included 496 workers who were not exposed to any significant air contaminants in the mill; most of these men worked in the office or the log pond.

Health Studies. The health study carried out at the plant site by the research team included 4 types of study.

(*1*) Questionnaire. The questionnaire was filled in by trained interviewers. It contained questions on individual characteristics such as age, height, weight, sex, race, lifestyle (smoking and alcohol), detailed work history in the mill, and work history before employment in the mill. The medical part of the questionnaire included questions obtained from the British Medical Research Council standard questionnaire on chronic bronchitis (10) and questions related to symptoms arising as a result of acute exposure to gases and fumes. Past medical history, and personal and family history of atopy were also included. The questionnaire took 10 to 15 min to complete.

(*2*) Physical examination. A limited physical examination was carried out, including measurement of height, weight, and blood pressure; examination for clubbing of fingers; and auscultation of the chest for rales and rhonchi.

(*3*) Pulmonary function tests. Spirometric measurements were obtained for all workers using a 13.5-L Collins spirometer. At least 5 readings were obtained with the worker seated and wearing a noseclip. The highest FEV_1 and forced vital capacity (FVC) were obtained from all the spirograms. Forced expiratory flow during the middle half of the FVC ($FEF_{25-75\%}$) was obtained from the spirogram with the highest sum of FEV_1 and FVC. Predicted normal values for each measurement were obtained from the reports of Cotes and co-workers (11), Goldman and Becklake (12), and Leuallen and Fowler (13). The results were corrected to BTPS.

(*4*) Chest radiographs. Posteroanterior chest radiographs (14 × 17 inches) were obtained and were read independently by 3 observers.

Fifteen milliliters of blood were also obtained from each worker for measurement of hemoglobin, hematocrit, leukocyte count, liver function tests, and renal function tests. Urinalysis was also performed. The results of these tests will be reported in a separate communication.

Environmental Studies. Detailed environmental monitoring was carried out by the Industrial Hygiene Department of the Workers' Compensation Board of British Columbia at the time of the health survey. Emphasis was placed on obtaining personal sample results; area samples were used only for background information. Workers from all departments and various jobs were monitored for levels of air contaminants including SO_2, H_2S, chlorine, methyl mercaptan, penta/tetrachlorophenol, CO, and total concentration of wood dust and paper dust whenever appropriate.

Personal sampling for SO_2, H_2S, chlorine, and CO was carried out using long-term gas-detector tubes. Each lot of detector tubes was calibrated in the laboratory, and only those within the accuracy limits of ± 25 % were used in this study. The sampling duration was 6 to 7 h.

Methyl mercaptan sampling was accomplished by following 13 different workers from all parts of the kraft mill during the full course of various normal 8-h work shifts. Grab samples were taken in the breathing zone at 15-min intervals during the work shifts using short-term detector tubes that had previously been calibrated to ± 25 % accuracy. Altogether, 221 samples were obtained for 13 workers.

Penta/tetrachlorophenol personal samples were collected on Tenax absorption tubes with subsequent laboratory analysis by gas chromatography. Due to limitations of the sampling method, durations of only 30 to 90 min were possible.

Personal sampling for the total amount of airborne particulate matter was performed with preweighed polyvinyl chloride filters worn for the entire work shift, resulting in 6- to 8-h sample duration. Gravimetric analysis was then used to determine concentrations of particles.

Additional airborne sampling for various other contaminants such as metal fumes, solvent vapors, mercury vapor, aldehydes, and hydrazine was also conducted to group the workers according to exposure accurately. Biologic monitoring for lead and penta/tetrachlorophenol was carried out on a limited number of workers.

Analysis of data. Analysis of variance and covariance, and multidimensional contingency table analysis (14, 15) were used to examine the relation of respiratory symptoms and pulmonary function abnormalities to age, duration of employment, work exposure, smoking and alcohol intake, and their interaction.

Results

Demographic data. The characteristics of the workers in the 6 exposure groups are shown in table 1. The mean age of all workers was 41.8 ± 12.3 yr. The mean duration of employment in the plant was 14.9 ± 10.3 yr, whereas the mean duration in present job was 12.4 ± 8.9 yr; 21.8 % of the workers were nonsmokers, 35.7 % were ex-smokers, and 42.5 % were current smokers. The average daily alcohol consumption during the 7 days before the interview was calculated for each

TABLE 1

CHARACTERISTICS OF THE WHITE MALE WORK FORCE, BY EXPOSURE TO AIR CONTAMINANTS

	Exposure Group						
Characteristic	1 (Vapors)	2 (Wood)	3 (Paper)	4 (CO)	5 (Mixed)	6 (Control)	Total
No. of subjects	219	319	278	190	324	496	1,826
Age,* yr	43.2 ± 10.8	39.1 ± 12.3	41.4 ± 11.5	45.2 ± 10.5	44.7 ± 11.2	39.7 ± 13.8	41.8 ± 12.3
Height,* cm	177.0 ± 6.8	176.3 ± 7.1	177.1 ± 6.3	175.7 ± 7.0	176.8 ± 6.4	176.6 ± 7.2	176.4 ± 6.9
Duration of employment,* yr	13.9 ± 9.3	12.6 ± 9.0	17.6 ± 10.0	18.7 ± 8.6	17.4 ± 11.3	12.3 ± 10.4	14.9 ± 10.3
Duration of present exposure,* yr	11.9 ± 8.6	11.8 ± 8.9	16.6 ± 9.9	15.5 ± 8.0	13.4 ± 10.6	10.3 ± 9.6	12.9 ± 8.9
Smoking Status,* no. and (%)							
Nonsmokers	37 (16.9)	56 (17.6)	53 (19.1)	33 (17.4)	83 (25.6)	136 (27.4)	398 (21.8)
Ex-smokers	89 (40.6)	95 (29.8)	105 (37.8)	85 (44.7)	131 (40.4)	147 (29.6)	652 (35.7)
Current smokers	93 (42.5)	168 (52.7)	120 (43.2)	72 (37.9)	110 (34.0)	213 (42.9)	776 (42.5)
Alcohol intake,*† no. and (%)							
0 drink/day	37 (17.1)	92 (29.0)	72 (26.1)	47 (24.7)	68 (21.3)	94 (19.4)	410 (22.6)
< 1 drink/day	78 (35.9)	107 (33.8)	78 (28.3)	70 (36.8)	112 (35.0)	161 (32.8)	606 (33.5)
1–3 drinks/day	84 (38.7)	86 (27.1)	92 (33.3)	57 (30.0)	103 (32.2)	168 (34.2)	590 (32.6)
> 3 drinks/day	18 (8.3)	32 (10.1)	34 (12.3)	16 (8.4)	37 (11.6)	68 (13.8)	205 (11.3)

* Differences between the groups were significant at $p < 0.01$ by analysis of variance or chi square analysis.
† Alcohol intake per day was calculated from the total number of drinks during the 7 days before the day of interview divided by 7. One drink = 1 bottle of beer or 1 glass of wine or 1 ounce of hard liquor.

TABLE 2

PREVALENCE OF CHEST SYMPTOMS, BY EXPOSURE GROUP AND SMOKING STATUS

Chest Symptoms, by Smoking Status	Exposure Group						Total	p Value*
	1 (Vapors)	2 (Wood)	3 (Paper)	4 (CO)	5 (Mixed)	6 (Control)		
Nonsmokers, no.	37	55	53	33	83	136	397	
Cough,† %	5.4	5.5	9.4	3.0	4.8	4.4	5.3	0.79
Phlegm,† %	10.8	3.6	5.7	3.0	8.4	4.4	5.8	0.52
Wheeze,‡ %	8.1	5.4	9.6	3.0	9.6	8.2	7.8	0.83
Dyspnea,§ %	8.1	7.1	9.4	6.1	7.2	5.1	6.8	0.93
Ex-smokers, no.	88	94	105	84	131	145	647	
Cough,† %	5.7	5.3	7.6	4.8	5.3	4.8	5.6	0.95
Phlegm,† %	10.1	13.7	7.6	4.8	8.5	6.1	8.3	0.27
Wheeze,‡ %	7.9	12.6	10.5	3.5	4.6	8.8	8.0	0.15
Dyspnea,§ %	14.6	10.5	12.4	14.1	12.3	16.3	13.5	0.83
Current smokers, no.	93	166	119	72	110	212	772	
Cough,† %	30.1	33.7	37.0	29.2	32.7	34.4	33.4	0.87
Phlegm,† %	30.4	27.3	24.8	18.3	25.9	30.3	27.1	0.44
Wheeze,‡ %	24.7	25.1	26.7	12.5	26.4	26.9	24.8	0.24
Dyspnea,§ %	16.1	17.9	12.5	15.5	17.3	19.2	16.9	0.74

* p Values refer to differences in prevalence of symptoms between different exposure groups, by chi square analysis.
† On most days (or nights) for as much as 3 months of the year.
‡ Without a cold.
§ While hurrying on level ground or walking up a slight hill.

worker; 22.6 % of the workers claimed to have had no drinks, 33.5 % admitted to having less than 1 drink per day, 32.6 % had more than one but less than 3 drinks per day, and 11.3 % had more than 3 drinks per day. There were significant differences between exposure groups in the mean age, height, duration of employment, and smoking habit, and alcohol intake.

Prevalence of chest symptoms. The prevalence of chest symptoms (cough, phlegm, wheeze, and dyspnea) by exposure group and smoking habits are shown in table 2. Among nonsmokers, the prevalence of all chest symptoms was relatively low (cough, 5.3 %; phlegm, 5.8 %; wheeze, 7.8 %; dyspnea, 6.8 %). The prevalence of all chest symptoms was significantly higher ($p < 0.01$) among current smokers than nonsmokers. Cough was present in 33.4 %; phlegm, 27.1 %; wheeze, 24.8 %; and dyspnea, 16.9 % of all workers.

There was no significant difference in the prevalence of any of the chest symptoms in any exposure group compared to the control group.

The prevalence of chest symptoms by age and smoking habit is shown in figure 1. It can be seen that age had no influence on the prevalence of cough and phlegm among nonsmokers, but had a significant effect among current smokers. Although age had no influence on the prevalence of wheeze, it was significantly correlated with the prevalence of dyspnea, irrespective of smoking habits. The effect of alcohol intake on the prevalence of various chest symptoms was also analyzed; no significant correlation was found.

Prevalence of past illnesses. There was no significant difference in the prevalence of past illnesses such as asthma, hay fever, heart diseases, bronchitis, and pulmonary tuberculosis in various exposure groups. The proportion of workers who gave a positive history of skin rash was significantly higher among workers in groups 1 (vapor) and 3 (paper) compared to other groups. Past history of pneumonia and pleurisy was significantly higher among workers in groups 1 (vapor), 3 (paper), and 5 (mixed) than in the control group.

Prevalence of positive physical signs. In the limited physical examination, the prevalences of rales,

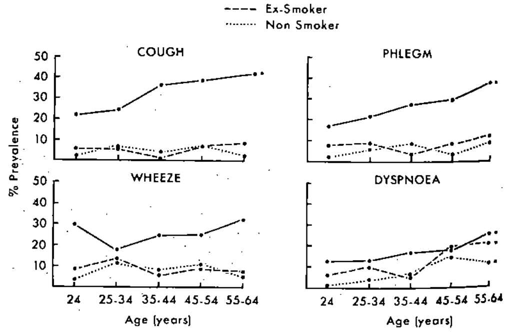

Fig. 1. Prevalence of chest symptoms in different age groups according to smoking habits.

rhonchi, and clubbing of fingers were, respectively, 0.3 %, 2.4 %, and 0.8 %. There were no significant differences between various exposure groups.

Pulmonary function tests. The mean spirometric measurements, expressed as percentages of predicted values, are shown for each exposure group in table 3. By analysis of variance, pulmonary function (FEV_1, FVC, and $FEF_{25-75\%}$) was significantly lower in current smokers than in ex-smokers and nonsmokers, irrespective of the exposure group. Among nonsmokers, group 1 (vapors) workers had the highest mean FEV_1 (110.2 ± 12.8 % predicted); group 2 (wood) workers had the lowest value (104.6 ± 12.4 % predicted). Among ex-smokers and current smokers, workers in the control group had the highest mean FEV_1 (105.2 ± 13.6 and 101.9 ± 14.2 % predicted, respectively). Group 2 (wood) workers again had the lowest value compared to other groups (102.6 ± 17.5 and 98.6 ± 14.2 % predicted, respectively). However, the differences between the groups were not significant in any of the smoking categories when pulmonary function expressed as per cent predicted was used in the statistical analysis. The mean FVC and $FEF_{25-75\%}$ in various exposure groups were within normal limits, and again, there were no significant differences between the groups. Among nonsmokers, the mean $FEF_{25-75\%}$ was lowest among group 2 (wood) workers; however, among ex-smokers and current smokers, the mean $FEF_{25-75\%}$ was lowest among group 5 (mixed) workers.

The effect of alcohol intake on pulmonary function tests was analyzed using analysis of variance (table 4). Among nonsmokers, there was a significant negative correlation between alcohol intake and pulmonary function. Workers who had more than 3 drinks per day had significantly ($p = 0.05$) lower FEV_1 and FVC than those who had had no alcohol intake during the previous 7 days. Among ex-smokers, workers who had less than 1 drink per day had significantly higher FEV_1 and FVC compared to those who had higher daily alcohol intake. There was no obvious effect of alcohol intake on pulmonary function among current smokers.

The effects on pulmonary function of age, height, work exposure, duration of employment, smoking habit, alcohol intake, and their interaction were analyzed using analysis of covariance. In this analysis, the absolute values of FEV_1, FVC, and $FEF_{25-75\%}$ were used. The results are shown in table 5: age, age^2, height, and smoking were significantly correlated with pulmonary function measurements. With this analysis, we found that the mean FEV_1 and FVC of the group 2 (wood) workers were significantly lower than the mean FEV_1 and FVC of the workers of the control group. However, the mean FEV_1, FVC, and $FEF_{25-75\%}$ of workers in other exposure

TABLE 3

MEAN PULMONARY FUNCTION VALUES, BY EXPOSURE GROUP AND SMOKING STATUS

	Exposure Group						
	1 (Vapors)	2 (Wood)	3 (Paper)	4 (CO)	5 (Mixed)	6 (Control)	Total
Nonsmokers, no.	37	56	53	33	83	136	398
FEV_1, % pred.	110.2 ± 12.8	104.6 ± 12.9	106.7 ± 12.2	106.0 ± 12.9	106.5 ± 14.7	107.3 ± 13.0	106.9 ± 13.2
FVC, % pred.	107.6 ± 11.6	104.0 ± 12.6	105.6 ± 11.0	106.5 ± 16.7	104.6 ± 12.9	107.4 ± 13.1	106.0 ± 12.9
FEF_{25-75}%, % pred.	107.4 ± 22.3	100.7 ± 22.5	103.4 ± 24.0	109.0 ± 27.3	105.4 ± 24.8	101.2 ± 26.6	103.5 ± 25.0
Ex-smokers, no.	89	95	105	85	131	147	652
FEV_1, % pred.	104.9 ± 15.8	102.6 ± 17.5	103.6 ± 14.7	104.9 ± 13.5	104.8 ± 14.9	105.2 ± 13.6	104.4 ± 14.9
FVC, % pred.	106.6 ± 14.6	103.7 ± 15.6	105.0 ± 11.8	105.8 ± 12.4	106.4 ± 13.3	104.8 ± 11.9	105.4 ± 13.2
FEF_{25-75}%, % pred.	95.0 ± 31.5	97.8 ± 31.5	97.5 ± 27.2	97.1 ± 27.8	95.0 ± 28.2	101.3 ± 29.9	97.5 ± 29.3
Current smokers, no.	93	168	120	72	110	213	772
FEV_1, % pred.	100.7 ± 18.2	98.6 ± 14.2	99.1 ± 13.2	99.9 ± 19.7	98.6 ± 14.6	101.9 ± 14.2	99.9 ± 15.2
FVC, % pred.	102.8 ± 14.0	101.8 ± 12.5	102.9 ± 11.5	102.0 ± 16.5	102.8 ± 11.8	104.8 ± 13.3	103.1 ± 13.1
FEF_{25-75}%, % pred.	90.0 ± 32.4	88.1 ± 26.1	86.6 ± 25.9	90.4 ± 32.7	83.4 ± 27.9	91.1 ± 27.5	88.5 ± 28.2
All Subjects, no.	219	319	278	190	324	496	1,826
FEV_1, % pred.	104.0 ± 16.7	100.8 ± 15.2	102.2 ± 13.9	103.2 ± 16.2	103.1 ± 15.1	104.3 ± 13.9	103.0 ± 15.0
FVC, % pred.	105.2 ± 13.9	102.8 ± 13.5	104.2 ± 11.5	104.5 ± 14.9	104.7 ± 12.8	105.5 ± 12.9	104.5 ± 13.1
FEF_{25-75}%, % pred.	95.0 ± 31.0	93.2 ± 27.7	93.9 ± 26.9	96.7 ± 30.2	93.7 ± 28.5	96.9 ± 28.4	95.0 ± 28.6

Definitions of abbreviations: FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity; $FEF_{25-75\%}$ = forced expiratory flow during the middle half of the FVC.

TABLE 4

PULMONARY FUNCTION TESTS ACCORDING TO ALCOHOL INTAKE AND SMOKING HABIT

	Alcohol Intake, no. of drinks/day				
	0	1	1–3	+3	p Value*
Nonsmokers, no.	99	159	104	35	
FEV$_1$, % pred.	107.1 ± 14.1	108.2 ± 13.3	106.1 ± 13.1	101.6 ± 8.6	0.05
FVC, % pred.	105.9 ± 14.1	108.1 ± 12.6	104.3 ± 13.3	101.9 ± 7.6	0.02
FEF$_{25-75}$%, % pred.	105.0 ± 26.7	103.6 ± 25.9	104.1 ± 23.1	96.2 ± 20.8	0.32
Ex-smokers, no.	142	224	225	57	
FEV$_1$, % pred.	103.4 ± 14.9	107.2 ± 14.2	102.8 ± 15	102.7 ± 15.4	0.008
FVC, % pred.	104.3 ± 14.2	107.9 ± 13.2	103.8 ± 12.3	104.6 ± 13.0	0.006
FEF$_{25-75}$%, % pred.	96.4 ± 27.8	100.9 ± 28.1	95.7 ± 30.2	95.5 ± 33.2	0.231
Current smokers, no.	169	223	261	113	
FEV$_1$, % pred.	99.8 ± 15.9	99.6 ± 15.3	100.0 ± 15.4	99.8 ± 13.9	0.99
FVC, % pred.	102.1 ± 14.1	103.1 ± 12.7	103.3 ± 13.0	103.0 ± 12.3	0.81
FEF$_{25-75}$%, % pred.	90.1 ± 27.5	87.7 ± 28.9	88.5 ± 28.5	86.5 ± 27.5	0.75

* p Value refers to differences in pulmonary function between groups of workers with different alcohol habits by analysis of variance.

groups were not different from those of the control group. The duration of employment had no effect on pulmonary function, but the effect of alcohol was again demonstrated in this analysis. There was no potentiation between age and work exposure, smoking and alcohol intake, or work exposure and alcohol intake, but there was significant potentiation between age and smoking on pulmonary function, as expected.

Chest radiographs. Chest radiographs were obtained at a separate time from the interview. As a result, the percentage of participation was lower (77 to 90 % in different exposure groups). Most chest radiographs were normal. On the average, 5 to 10 % of the workers in each group had abnormal findings. Most of the radiographic changes were related to, or were probably caused by, previous pulmonary tuberculosis (healed primary complex, apical scarring, calcified foci, and previous thoracic surgery for tuberculosis). Diffuse shadows occurred in only 2 workers; one worker had a previous history of mining, and the other had a previous history of exposure to asbestos. Of the 8 workers with parenchymal lesions, one had active tuberculosis; one, resolving pneumonitis; one, old tuberculoma; 3, linear atelectasis; 2, ill-defined densities, the nature of which has yet to be investigated. There were no significant differences in the distribution of these abnormalities among the various exposure groups.

Industrial hygiene data. The summary of industrial hygiene data is shown in table 6. Workers in group 1 were exposed to time-weighted average concentrations of < 0.05 to 2.0 ppm of SO_2, < 0.1 to 0.2 ppm of H_2S, and < 0.05 to 0.1 ppm of chlorine. The stated exposures to methyl mercaptan (< 0.05 to 32 ppm) were based on short-term breathing zone measurements, which cannot be

TABLE 5

RESULTS OF ANALYSIS OF COVARIANCE ON THE EFFECTS OF VARIOUS FACTORS ON PULMONARY FUNCTION

	FEV$_1$		FVC		FEF$_{25-75}$%	
Variable	Slope Coefficient	p Value	Slope Coefficient	p Value	Slope Coefficient	p Value
Age	−6.061	< 0.0001	+7.520	< 0.0001	−7.736	< 0.0001
Age^2	−0.348	< 0.0001	−0.470	< 0.0001	−0.564	< 0.001
Height	+42.02	< 0.0001	+62.75	< 0.0001	+22.14	< 0.001
Duration of employment	+2.33	2.82	+1.53	0.526	+5.68	0.156
Smoking†		< 0.0001		< 0.001		< 0.0001
Alcohol intake†		0.021		0.002		0.150
Work exposure†		0.016		0.033		0.335
Age and smoke interaction		< 0.0001		0.006		0.002

† Slope coefficients were not obtained for these variables because they were categorical. Significance was not found for the following interactions: work exposure and smoke, smoke and alcohol intake, work exposure and alcohol intake, age and work exposure.

TABLE 6

SUMMARY OF INDUSTRIAL HYGIENE DATA (PERSONAL SAMPLE)

Group	Air Contaminant	Mean	Range	No. of Samples
1 (Vapors)	Total particulate matter, mg/m³	0.8	< 0.1–3.5	26
	Sulfur dioxide, ppm	0.3	< 0.05–2.0	52
	Chlorine, ppm	< 0.05	< 0.05–0.1	23
	Hydrogen sulfide, ppm	0.05	< 0.05–0.2	46
	Methyl mercaptan, ppm	Not applicable	< 0.05–32	13
	CO, ppm	8.3	< 1–15	7
2 (Wood)	Total particulate matter, mg/m³	0.5	< 0.1–2.7	71
	Pentachlorophenol, mg/m³	0.009	< 0.001–0.118	30
	Tetrachlorophenol, mg/m³	0.027	< 0.001–0.08	31
3 (Paper)	Total particulate matter, mg/m³	1.6	< 0.1–9.8	56
4 (CO)	Total particulate matter, mg/m³	0.2	< 0.1–0.6	14
	CO, ppm	22.1	< 1–74	77
5 (Mixed)	Total particulate matter, mg/m³	1.7	< 0.1–7.3	31
	Sulfur dioxide, ppm	0.1	< 0.05–0.9	6
	Chlorine, ppm	< 0.05	< 0.05–0.1	6
	Hydrogen sulfide, ppm	0.06	< 0.05–0.2	6
6 (Control)	Total particulate matter, mg/m³	0.4	< 0.1–2.0	56
	Sulfur dioxide, ppm	0.1	< 0.05–0.7	20
	Hydrogen sulfide, ppm	< 0.05	< 0.05–0.1	8
	CO, ppm	1.9	< 1–10	29

The currently accepted threshold limit value (16) for total particulate matter (nuisance dust) is 10 mg/m³; sulfur dioxide, 5 ppm; hydrogen sulfide, 10 ppm; chlorine, 1 ppm; methyl mercaptan, 10 ppm; CO, 50 ppm; pentachlorophenol, 0.5 mg/m³.

used for accurate calculations of time-weighted averages. However, using these short-term results, an approximate time-weighted average exposure for group 1 workers was 1 to 2 ppm of methyl mercaptan. Group 2 (wood) workers were exposed to < 0.1 to 2.7 mg/m³ of total airborne particulate matter. Essentially all of this particulate matter was wood dust from fir, hemlock, balsam, and spruce species. Very small amounts (< 1 % of the total) of wood processed in the wood room was western red cedar. Group 2 (wood) workers were also exposed to low concentrations of the antistain chemicals pentachlorophenol (< 0.001 to 0.118 mg/m³) and tetrachlorophenol (< 0.001 to 0.08 mg/m³). Relatively low biologic results confirmed these low airborne exposures, with a urine pentachlorophenol concentration of 28 to 154 μg/L and a urine tetrachlorophenol concentration of 43 to 175 μg/L. Group 3 (paper) workers were exposed to total airborne particulate paper dust concentrations of < 0.1 to 9.8 mg/m³. It was probable that trace amounts of slimicide, fungicide, defoamer, and dye were contained in this dust as well; however, their concentrations were not quantified. Group 4 (CO) workers were exposed to a time-weighted average concentration of < 1 to 74 ppm of CO. Exposures to other air contaminants such as nitrogen oxides and aldehydes were extremely low. Group 5 (mixed) workers, such as maintenance personnel who toured all areas of the mill, were exposed to all types and concentrations of air contaminants. It was impossible to assign a reliable exposure index to them. Group 6 (control) workers were exposed to lower levels of various air contaminants than were other groups.

Discussion

In this study we were unable to demonstrate that workers in the kraft mill in Powell River who were exposed to gases and chemicals had higher prevalences of chest symptoms and pulmonary function abnormalities than did the control subjects who were not exposed to any gases or chemicals. Our findings were similar to those of Ferris and coworkers (6), but at variance to those of Skalpe (7) and Smith and associates (8). It is very likely that the differences between the results of these various studies are due to the different levels of exposure to SO_2 and other contaminants. Workers in the kraft mill in Powell River were exposed to low levels of SO_2 (mean, 0.3 ppm; range, < 0.05 to 2 ppm) and chlorine (mean, < 0.05 ppm; range, < 0.05 to 0.1 ppm). In studies in which adverse effects on the respiratory system were demonstrated, the workers were exposed to much higher concentrations of SO_2 (2 to 36 ppm). Smith and coworkers (8) demonstrated that exposure to 1 to 2.5 ppm of SO_2 was associated with excessive yearly loss of FEV_1 and an increase in respiratory symptoms. Moreover, some of the workers in this smelter were exposed to very high levels of SO_2 from leaks for short periods of time. It is possible that this intermittent brief, heavy exposure could be an important factor in causing pulmonary impairment.

Workers exposed to wood dust had somewhat lower pulmonary function values compared to the control group after adjustments had been made for differences in age, height, duration of employment, and smoking habits. These workers were exposed to all kinds of wood dust. The concentration of wood dust in the sawmill ranged from < 0.1 to 2.7 mg/m³. We compared the pulmonary function findings of these workers with those of other sawmill workers who were not exposed to red cedar in British Columbia and were unable to show any significant differences in any of the parameters. The study of the sawmill workers was carried out by the same research team using the same water spirometer to measure pulmonary function (16). It appears that wood dust, or perhaps the chemicals used as preservatives for the wood, may have some deleterious effect on pulmonary function. The results of this study and the previous one (16) suggest that the currently accepted threshold limit value (TLV) for nonallergenic wood dust should be lower than 5 mg/m³ (17). The actual level should be established by further studies.

Workers exposed to paper dust did not have a higher prevalence of respiratory symptoms or pulmonary function abnormalities compared to the control group. The concentrations of paper dust encountered there ranged from < 0.1 to 9.8 mg/m³. These findings suggest that paper dust can be regarded as nuisance dust, and that the currently accepted TLV of 10 mg/m³ for nuisance dust (17) appears to be adequate. Similarly, workers exposed mostly to CO because of their job location in the garage, warehouse, and so on did not appear to have a higher prevalence of respiratory abnormalities compared to the control group. Carbon monoxide was present in relatively high concentration (> 50 ppm) in warehouse and storage areas. These workers, however, showed somewhat higher hemoglobin concentrations; these findings will be reported separately.

This study, as many other studies, demonstrated that cigarette smoking is harmful to the respiratory system; smokers have more respiratory symptoms and lower pulmonary function values than do nonsmokers. We were unable to demonstrate any synergistic effect between work exposure and cigarette smoking on pulmonary function. The effect of alcohol on pulmonary function has been less well studied. We found that among nonsmokers, pulmonary function tests were significantly lower among workers with daily alcohol intake of more than 3 drinks per day than among those who had no alcohol intake during the previous week; these differences were not evident in smokers in our study. Our finding in this respect was different from those reported by Cohen and associates (18), who were unable to demonstrate any influence of alcohol consumption on airway obstruction. The reason for the lower pulmonary function among nonsmoking heavy drinkers is unknown. One wonders whether it could be due to passive inhalation of cigarette smoke while these men were at the bar.

Excess in death rate from cancers of the small intestine and cancers of the lymphatic and hematopoietic tissue had been reported by Milham (19) among pulp and paper mill workers; excess mortality from lung cancer was not documented in his studies. More thorough investigation should be directed to possible carcinogenic effects of exposures encountered in pulp and paper industry.

In conclusion, we were unable to demonstrate an increased prevalence of respiratory symptoms and pulmonary function abnormalities among the pulp and paper mill workers in Powell River in this prevalence survey. However, it is important to note that the level of air contaminants, such as SO_2, H_2S, and chlorine encountered in this mill, was very low. Similar studies should be carried out in other pulp and paper mills in other parts of British Columbia to establish whether the currently accepted TLVs for SO_2, H_2S, chlorine, and methyl mercaptan (17) are adequate.

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References

1. Chasis H, Zapp JA, Bannon JH, Whittenberger JL,

Helm J, Doheny JL, MacLeod CD. Chlorine accident in Brooklyn. Occup Med 1947; 4:152–9.

2. Kowitz TA, Reba RC, Parker RT, Spicer WS. Effects of chlorine gas upon respiratory function. Arch Environ Health 1967; 14:545–58.
3. Weill H, George R, Schwarz M, Ziskind M. Late evaluation of pulmonary function after acute exposure to chlorine gas. Am Rev Respir Dis 1969; 99:374–9.
4. Anderson I, Lundqvist GR, Jensen PL, Proctor DF. Human response to controlled levels of sulphur dioxide. Arch Environ Health 1974; 23:31–9.
5. Lawther PJ, MacFarlene AJ, Waller RE, Brooks AGF. Pulmonary function and sulphur dioxide: some preliminary findings. Environ Res 1975; 10:355–67.
6. Ferris BG, Burgess WA, Worcester J. Prevalence of chronic respiratory disease in a pulp mill and a paper mill in the United States. Br J Ind Med 1967; 24:26–37.
7. Skalpe IO. Long-term effects of sulphur dioxide exposure in pulp mills. Br J Ind Med 1964; 21:69–73.
8. Smith TJ, Peters JM, Reading JC, Castle CH. Pulmonary impairment from chronic exposure to sulphur dioxide in a smelter. Am Rev Respir Dis 1977; 116:31–9.
9. Corey PN, Ashley MJ, Chan-Yeung M. Racial differences in lung function: search for proportional relationships. J Occup Med 1979; 21:395–8.
10. Medical Research Council's Committee on the Etiology of Chronic Bronchitis: Standardized questionnaires on respiratory symptoms. Br Med J 1960; 2:1665–8.
11. Cotes JE, Rossiter CE, Higgins ITT, Gibson JC. Average normal values for the forced expiratory volume in white caucasian males. Br Med J 1966; 1:1016–19.
12. Goldman HL, Becklake MR. Respiratory function tests: normal values at median altitudes and the prediction of normal results. Am Rev Tuberc 1959; 79:457–65.
13. Leuallen EC, Fowler WS. Maximum mid-expiratory flow. Am Rev Tuberc 1955; 72:783–97.
14. Snedecor GW, Cochran WG. Statistical methods, 6th ed. Ames, Iowa: The Iowa Univerity Press, 1967:419–46.
15. Bishop YMM, Fienberg SE, Holland PW. Discrete multivariate analysis: theory and practice, 1st ed. Cambridge, Mass.: MIT Press, 1975:57–122.
16. Chan-Yeung M, Ashley MJ, Corey P, Willson G, Dorken E, Grzybowski S. A respiratory survey of cedar mill workers. I. Prevalence of symptoms and pulmonary function abnormalities. J Occup Med 1978; 20:323–7.
17. Threshold limit values for chemical substances in woodroom air. Adopted by American Conference of Governmental Industrial Hygienists, 1979.
18. Cohen BH, Celentano DD, Chase GA, et al. Alcohol consumption and airway obstruction. Am Rev Respir Dis 1980; 121:205–16.
19. Milham S. Neoplasia in the wood and pulp industry. Ann NY Acad Sci 1976; 271:294–300.