PLAINTIFF'S EXHIBIT WRG-1085

Respiratory Illness in the Construction Trades

I. The Significance of Asbestos-Associated Pleural Disease among Sheet Metal Workers

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To assess the rate of roentgenographic and lung function abnormalities in asbestos-exposed construction workers, the authors studied 314 white male members of a local sheet metal workers union. Health outcomes were assessed by questionnaire, simple spirometry, and chest roentgenography; data were collected and interpreted following guidelines of the American Thoracic Society and the International Labor Office. Analyses of union records showed the tested population to be representative of all those eligible for testing. Pleural abnormalities were common, increasing to a prevalence of approximately 70% in workers with more than 30 years employment. Roentgenographic evidence of pleural disease was significantly correlated with decreased forced vital capacity ($p = 0.027$) after controlling for the potential confounding effects of age, height, cigarette consumption history, and employment duration. In contrast, forced expiratory volume in 1s showed a stronger association with amount smoked ($p = 0.022$) than with pleural abnormality ($p = 0.316$). Logistic regression analyses showed that cigarettes act to increase the effect of asbestos in causing pleural disease among exposed workers. Cigarettes, in the absence of significant asbestos exposure, do not appear to cause pleural disease. The authors conclude that construction workers, such as those described herein, have a considerably increased rate of pleural disease, which has functional significance in view of the correlations noted with forced vital capacity measurements. Therefore, pleural disease in asbestos-exposed workers is not only an indicator of exposure but also an indicator of early impairment of pulmonary function.

Many studies of the pulmonary effects of inhaling asbestos fibers have demonstrated significant impairment of pulmonary function in individuals with roentgenographic evidence of interstitial fibrosis.[1] In many individuals exposed to asbestos, pleural disease is noted also and is characterized by bilateral fibrotic plaques or diffuse thickening along the chest walls and diaphragm. In population surveys, pleural disease is observed more commonly than parenchymal lung disease (i.e., pulmonary asbestosis). Despite the relatively high frequency of pleural disease, the significance of this manifestation of asbestos exposure is debated. Recent epidemiologic studies have evaluated the effects of asbestos inhalation among workers whose exposures to asbestos were less than those of insulation and asbestos manufacturing workers.[2-4] Often, pleural abnormalities, without parenchymal disease, were identified in workers exposed to relatively low levels of asbestos.

The largest group of asbestos-exposed workers in the United States are construction workers. Recent estimates indicate that 7,500,000 construction workers have received significant asbestos exposure in the past.[5] Workers in these trades are exposed to asbestos directly, through their own work activities, and indirectly by inhaling airborne asbestos fibers generated by others working nearby on construction sites. The extent of asbestos-related disease in Swedish construction workers has been evaluated and pleural changes were reported in 62% of those without radiological evidence of pulmonary fibrosis.[6] In other occupational settings, the presence of pleural plaques in asbestos-exposed workers has correlated well with the duration and intensity of exposure to asbestos.[7-11]

Asbestos-induced pleural plaques usually do not interfere with lung function directly. Moreover, progression of

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diffuse pleural thickening or pleural plaques to mesothelioma has not been clearly documented, and, therefore, asbestos-related pleural disease is not regarded as a premalignant lesion.[1,7,8] However, the risk of developing bronchial carcinoma and mesothelioma is higher among asbestos-exposed workers with pleural disease than among controls with no evidence of pleural abnormalities.[12-14] Workers with pleural disease also have a higher frequency of chronic bronchitis and an increased lung closing volume.[9] As such, pleural abnormalities may serve both as an indicator of asbestos exposure[10] and as a marker for increased risk of developing pulmonary malignancy or nonmalignant pulmonary disease.[8,10]

The interaction of cigarette smoking and asbestos exposure in the development of lung cancer is significant and well documented.[15,16] Less certain is the relationship of smoking and asbestos in the development of pleural abnormalities. Smoking does not cause pleural disease in the absence of asbestos exposure. Two studies of asbestos-exposed workers have concluded that smoking is significantly associated with an increase in the prevalence of pleural plaques and pleural fibrosis,[8,17] while a third found no such association.[4] It has been hypothesized that smoking facilitates the initial formation of pleural abnormalities.[8] In addition, individuals with diffuse pleural fibrosis have been noted to have smoked a greater amount than workers with hyaline and/or calcified plaques, the latter group having smoking histories comparable with those of workers with normal x-ray films.[18] One report suggests that cigarette smoking may be more important than asbestos exposure in the development of pleural disease.[18]

The current study was performed to evaluate the prevalence of pleural and parenchymal abnormalities in a group of asbestos-exposed construction workers from the sheet metal trade. The relationship of cigarette smoking and asbestos exposure to the presence of pleural disease was explored. In addition, pulmonary function was compared between sheet metal workers with and without pleural disease.

Subjects and Methods

Subjects — In January and May, 1981, Local No. 17 of the Sheet Metal Worker's International Association sponsored medical evaluations for its members. All active and retired union members belonging to the union Health and Welfare Plan (N = 1,413) were notified of the screening by mail and encouraged to participate. Due to scheduling constraints, examinations were limited to the first 314 workers to make appointments.

Characterization of Exposure to Asbestos — Sheet metal workers in the building trades fabricate and install metal ducts for purposes of ventilating commercial and residential buildings. Asbestos was often used in the past to insulate these ducts. During their installation, workers manually removed asbestos fireproofing sprayed onto the steel girders where the ducts were being hung. In addition, sheet metal workers were often in areas where asbestos was disseminated into the general work environment from adjacent activities. In the past, sheet metal workers have also been exposed to asbestos during the installation and removal of boilers, and while performing smoke breaching.

Environmental sampling results indicate that sheet metal workers have been exposed to variable concentrations of asbestos. In one study, sheet metal workers received exposures ranging from 0.1 to 0.2 fibers/ml when applying asbestos paper.[19] Where asbestos exposure resulted from adjacent activities (e.g., spraying of insulation materials), personal air sampling recorded levels as high as 1.61 fibers/ml for sheet metal workers, compared with 80 fibers/ml in the spray operator's breathing zone.[20] Mean fiber concentrations of 8.9 fibers/ml were recorded in areas where workers were tearing out old materials to reinsulate with new products.[21] Since direct exposure measurement was not performed in this study, years of employment as a sheet metal worker was used as the exposure variable, including time spent as an apprentice in the trade and any employment while a member of another construction union where similar levels of exposure to asbestos likely occurred.

Assessment of Health Effects — All participants completed a modified American Thoracic Society[22] questionnaire and occupational history developed specifically for this study. Pulmonary function tests were recorded on a Collins' Stead-Wells survey spirometer while the subjects were seated. Nose clips were not employed. A minimum of five trials was required and acceptability was judged by the criteria set forth by the American Thoracic Society.[22] Utilizing the equations of Knudsen et al,[23] percent predicted forced vital capacity (%FVC) and forced expiratory volume in 1 s (%FEV_1) were calculated using the largest acceptable measurement of FVC and FEV_1. As specified in the American Thoracic Society guidelines,[22] the FVC and FEV_1 value may come from different trials. For the analysis, a participant was considered to have abnormal pulmonary function test results if his %FVC or %FEV_1 was below 80% of the predicted value. All films (posteroanterior projection only) were interpreted by a certified "B" reader (R.E.G.) who used the *ILO/UC 1971 Classification of Radiographs of Pneumoconioses*[24] and who had no knowledge of the clinical status or exposure history of the individuals.

A worker was considered to have evidence of a pleural abnormality if he met one or more of the following three roentgenographic conditions[24]:

1. Bilateral pleural thickening of the chest walls and/or diaphragm of extent 1, 2, or 3 (circumscribed or, rarely, diffuse).
2. Bilateral pleural calcification of the chest walls and/or diaphragm of extent 1 or 2.
3. Bilateral pleural plaques (uncalcified).

Workers with parenchymal opacities were also characterized as having roentgenographic abnormalities, while individuals with unilateral pleural thickening, calcification, or plaques only were classified as having normal x-ray films.

Characterization of Smoking History — Individuals were classified as having never smoked ("nonsmokers") if they had smoked less than 20 packs of cigarettes in a lifetime. We classified the remaining workers as "current smokers" if they had smoked cigarettes during the month preceding their physical examination. Therefore, "ex-smokers" consisted of individuals smoking more than 20 packs in a lifetime and stopping more than one month prior to medical testing. The average number of packs smoked per day was also calculated.

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Statistics — Student's t test and one- and three-way analyses of variance were used to compare mean demographic and pulmonary function values.[45] Chi square statistics were calculated to test the significance of overall differences among prevalence rates when the latter were stratified by one variable.[46] In the event that significant differences were identified, prevalence rates were partitioned into two groups for intergroup and intragroup χ^2 analyses. The hypothesis that pleural disease prevalence rates increased monotonically with length of employment was tested using an analysis for linear trend in proportions.[46,47] The Mantel extension test was used to investigate the overall association between exposure (length of employment) and effect (prevalence of pleural disease) while stratifying to control for smoking.[48,49] Logistic regression analyses yielded odds ratios and 95% confidence intervals (CI) for the effects of smoking and length of employment (independent covariates) on the presence of pleural disease (dichotomous dependent variable).[50]

All analysis of variance calculations were generated by the General Linear Model procedure in the Statistical Analysis System program package (SAS GLM).[51] Logistic regression models were tested using the LOGIST procedure developed by the Duke Medical Center.[52] The Mantel extension tests and analyses of linear trends were generated using programs specifically designed for the Hewlett-Packard 67.[53]

Results

Population Characteristics — There were 1,413 union members eligible for our evaluations, 314 of whom participated. Induction dates into the union were available for 312 (99.4%) of the study subjects and 1,087 (98.9%) of the eligible nonparticipants. To determine whether those evaluated were representative of the entire union membership, all eligible union members were categorized by years since joining the local union and compared with the study subjects. The distribution of local union tenure was similar for those screened and for the overall union membership (Table 1). Furthermore, the mean age of 47 years in the study participants was similar to that of the entire union membership (48 years). Thus, the workers who were examined in the study appeared to be representative of all potential participants.

A significant number of individuals had been employed as sheet metal workers prior to joining Union Local No.17 in Boston. Therefore, our questionnaire obtained information on the total duration of employment as a sheet metal worker and this variable was used to estimate cumulative asbestos exposure. Since this information was unavailable for union members not participating in our survey, comparability of the participants and nonparticipants could be assessed only by comparing tenure in the local union (Table 1).

Table 1 — Population Composition of Eligible Union Members and Study Members (Alone) According to Years Since Initiation in Local Union

Years Since Initiation	No (%) Eligible for Study	No. (%) Screened
1-10	382 (27.3)	78 (25.0)
11-20	393 (28.1)	100 (32.1)
21-30	334 (23.9)	89 (28.5)
31 +	290 (20.7)	45 (14.4)
Total	**1,399 (100)**	**312 (100)**

Fifteen workers were excluded from data analysis: those without x-ray reports (n = 2), uninterpretable x-ray films (five), unsatisfactory pulmonary function data (four), and unavailable information regarding smoking habits (four). The following analyses are based on the residual 299 individuals.

All workers evaluated were white males ranging in age from 27 to 76 years. Age was positively correlated with length of employment ($r = .853$) as a sheet metal worker. Twenty-three percent had never smoked; 33% were current smokers, and 44% were ex-cigarette smokers. The proportion of current smokers was higher among sheet metal workers employed for less than 21 years than for those who had worked longer (Table 2). The proportion of ex-smokers increased with increasing duration of employment. Thus, ex-smokers were older and had worked longer than current smokers and nonsmokers. However, there was no significant difference in the average amount smoked between ex-smokers and current smokers. The mean age and length of employment of nonsmokers was significantly lower than the same measures in workers who had smoked an average of 2.5 to 3.0 packs/day ($p < .05$).

Roentgenographic Abnormalities — One hundred fifty-two workers (51%) had one or more pleural abnormalities. One hundred twenty-nine had bilateral pleural thickening, either alone (31), with calcified plaques (four), uncalcified plaques (89), or parenchymal opacities (five). Of the remaining 23 workers with pleural thickening, 21 had gross, bilateral uncalcified plaques, either alone (20) or with large opacities (one). One worker had bilateral calcified plaques (alone), while another had bilateral pleural calcification and parenchymal opacities. Six roentgeno-

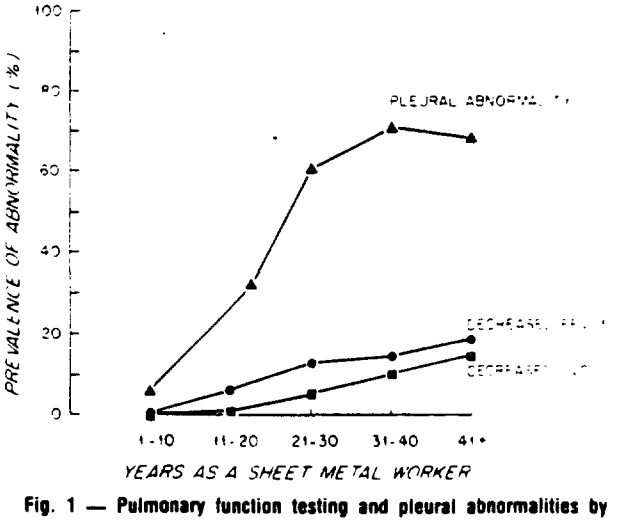


Fig. 1 — Pulmonary function testing and pleural abnormalities by duration of exposure.

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Table 2 — Age and Smoking Status in All Study Participants According to Length of Employment

	Years as a Sheet Metal Worker					
	1-10	11-20	21-30	31-40	40+	Total
No. of workers	16	100	99	62	22	299
Mean age, yr	33.6 ± 7.1	37.0 ± 6.3	48 8 ± 8.6	56.9 ± 4.6	64.3 ± 4.3	46.9 ± 11.5
Smoking, %						
Current smoker	50.0	43.0	26.3	27.4	27.3	33.4
Ex-smoker	25.0	31.0	49.5	58 1	45.5	43.5
Nonsmoker	25.0	26.0	24.2	14.5	27.2	23.1

graphic abnormalities were limited to parenchymal opacities in the absence of any pleural disease. Among workers with diffuse bilateral pleural thickening, 35 (27.1%) abnormalities were of grade 2 extent.

Workers with normal x-ray films were younger and had been in the sheet metal trade for shorter periods than individuals with pleural and/or parenchymal abnormalities. Among those with bilateral pleural thickening (in the absence of calcification or parenchymal changes), workers with thickening of grade 2 were older and more often smokers (ex-smokers or current smokers). As expected, those with pleural calcifications were older.

The prevalence of pleural abnormalities increased with duration of employment as a sheet metal worker (Fig. 1). Prevalence rates ranged from 6.3% in workers with 10 or fewer years in the trade to approximately 70% in individuals with more than 30 years of experience. The χ^2 statistic for the overall differences in pleural disease prevalence rates was significant ($\chi^2=43.4$, $p<.001$). The hypothesis of a monotonic (linear) trend, however, was rejected. To isolate the sources of significant differences, prevalence rates were partitioned into two employment strata (group A: 1 through 20 years; group B: >20 years), with overall prevalence rates being calculated for each group. The prevalence for group A (0.43) was significantly lower than that for group B (0.65) ($\chi^2=17.65$, $p<.05$). In contrast, individual differences within each group were not significant (group A: $\chi^2=3.65$, $p>.5$; group B: $\chi^2=0.05$, $p>.05$).

Pulmonary Function and Asbestos Exposure — The prevalence of abnormal pulmonary function measurements was also associated with the length of employment. Among workers with fewer than 11 years of employment in the trade, all pulmonary function test results were above 80% of that predicted based on age, height, race, and sex. However, with increasing employment, the percentage of workers with decreased %FVC and %FEV, (i.e., <80% of predicted) increased up to 13.6% and 18.2%, respectively, in workers with more than 30 years employment (Fig. 1). Statistical analyses of the differences in prevalence rates were not performed since the number of workers with abnormal pulmonary function measurements in each stratum was small.

Pleural Abnormalities, Asbestos Exposure, and Smoking History — The overall association between length of employment and pleural abnormalities was highly significant ($\chi^2_{mant}=5.73$, $p<.0001$) while controlling for smoking category (Table 3). The marginal rate of pleural abnormalities in ex-smokers (57.7%) was higher than that observed in current smokers (45.0%) and nonsmokers (46.4%). This finding may be explained, in part, by the observation that ex-smokers were older and had worked longer than current smokers and nonsmokers. A clear association of years of asbestos exposure and prevalence of pleural disease was also noted while controlling for average amount smoked during lifetime ($\chi^2_{mant}=5.71$, $p<.01$). Within individual employment strata, the prevalence of pleural abnormalities appeared to increase in relation to average amount smoked (Fig. 2).

To evaluate further the associations between smoking, asbestos exposure, and pleural disease prevalence, logistic regression techniques were employed in which pleural disease was treated as the dichotomous dependent variable. In a preliminary analysis of all workers, the model of "best fit" including length of employment as the only significant independent covariate. Dummy variables representing smoking group (ex-smoker, current smoker, or nonsmoker) did not make a significant contribution. For workers with 10 years of employment in the trade, this model yielded a relative odds of having pleural disease equal to 1.91 (95% CI, 1.52 to 2.40) compared with individuals with one year of experience. The relative odds for workers with 20 and 30 years of experience (compared with one year) were 3.91 (95% CI, 2.42 to 6.33) and 8.02 (95% CI, 3.85 to 16.7), respectively.

To determine whether amount smoked was a more significant predictor of pleural disease than smoking group, a second analysis was limited to smokers. The model of best fit in this case included the average amount smoked and length of employment as significant covariates. Con-

Table 3 — Prevalence of Pleural Abnormalities in Study Participants by Length of Employment and Smoking Status

	Years as a Sheet Metal Worker			
	1-20	21-30	31+	Total
Smoking status, %				
Never smoked	26.7	54.2	73.3	46.4
Current smoker	27.5	57.7	69.6	45.0
Ex-smoker	31.4	65.3	69.6	57.7

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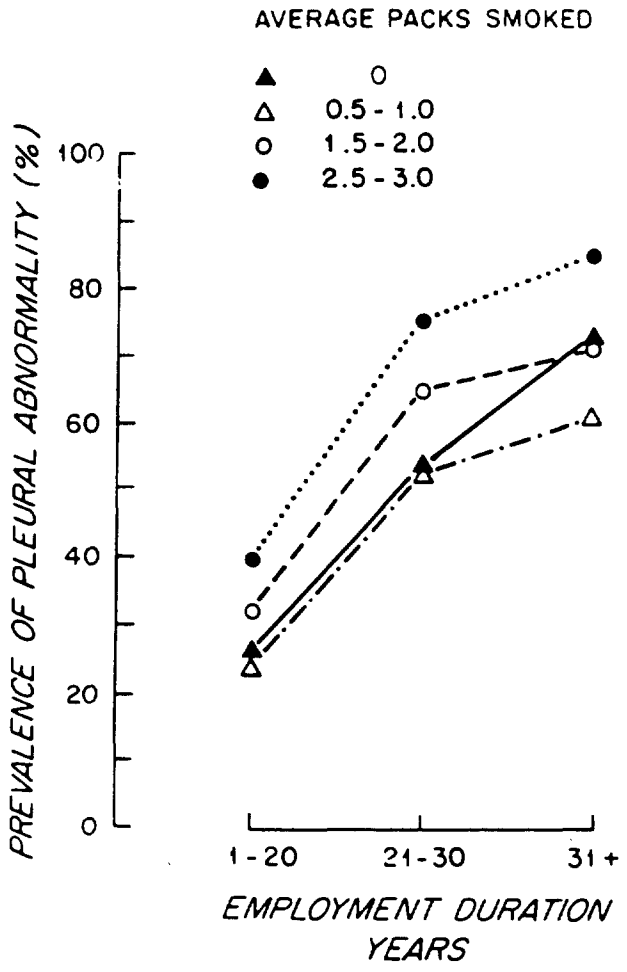


Fig. 2 — Rate of pleural disease by amount smoked and duration of exposure.

trolling for the effects of average amount smoked, this model yielded a relative odds of pleural disease of 1.84 (95% CI, 1.42 to 2.38) for workers with 10 years of experience compared with those with only one year in the trade. Workers with 20 and 30 years of experience had relative odds of 3.61 (95% CI, 2.09 to 6.25) and 7.10 (95% CI, 3.08 to 16.38), respectively, compared with individuals with one year of experience. Controlling for the effects of length of employment, the odds of having pleural disease among smokers of 1.0 pack/day relative to smokers of 0.5 pack/day was 1.31 (95% CI, 1.06 to 1.62). For workers who smoked an average of 2.5 packs/day, the odds of having pleural disease (relative to smokers of 0.5 pack/day) was 2.93 (95% CI, 1.25 to 6.86).

Pleural Abnormalities and Pulmonary Impairment — After excluding from analysis the 12 individuals with small irregular opacities on x-ray films, FVC was lower in individuals with pleural abnormalities than in workers having a normal x-ray film (99.5% of predicted *v* 103.2%) ($p<.05$). After stratifying by the average amount smoked and the length of employment, this relationship was still significant (Table 4). An analysis of variance showed significant contributions from variables indicating pleural abnormality ($p=.027$) and average packs smoked ($p=.057$), while employment duration was relatively unimportant ($p=.667$). The %FVC also appeared to be decreased in workers who smoked greater amounts per day. When this analysis was limited to a comparison of workers with normal x-ray films and those with bilateral pleural thickening of grade 1, similar results were obtained.

Percent FEV_1 was also lower in workers with pleural abnormalities (97.1% *v* 100.7% in individuals with normal chest x-ray films); however, this difference was not statistically significant (Table 5). After controlling for the average amount smoked and the length of employment, smoking was found to be the most important variable in explaining the observed trends in $\%FEV_1$. Analysis of variance showed that average packs smoked was the most important variable ($p=.002$) in identifying sources of variability in the FEV_1, while employment duration ($p=.079$) and presence of pleural abnormalities ($p=.316$) were considerably less important.

For both %FVC and $\%FEV_1$, all possible analysis of variance models that tested interactions of explanatory variables were evaluated and none were found to be significant. A slight, nonsignificant trend was noted between the extent and width of pleural thickening and the average pulmonary function measurements. Workers with greater than stage A1 pleural thickening had slightly lower %FVC and $\%FEV_1$ than normal persons or those with A1 degree of pleural disease.

Discussion

This investigation revealed a high rate (51%) of pleural abnormalities in construction workers with moderate asbestos exposure. Only 12 workers (4%) were found to have parenchymal opacities, six of whom also had pleural abnormalities. The prevalence of pleural abnormalities in this population increased with length of employment as a sheet metal worker, prevalence rates being significantly higher in workers with more than 20 years of employment in the trade. In addition to asbestos exposure history, cigarette smoking appeared to have an effect on the rate of occurrence of pleural disease. In a logistic regression analysis of smokers, both the average amount smoked and the length of employment were significant determinants of the rates of pleural disease. FVC was reduced on average in individuals with pleural abnormalities, after controlling for age, height, amount smoked, and length of employment. In contrast, FEV_1 was not reduced in those with pleural disease, but was influenced significantly by smoking history.

These results support the view that pleural disease is the most prevalent roentgenographic abnormality in workers with moderate asbestos exposure. The highest prevalence rates of pleural disease occurred in individuals with the longest periods of employment. It is uncertain whether this was a result of cumulative dose to asbestos or a latency effect, and the present data cannot distinguish between these two possibilities.

Smoking was not a prerequisite of pleural disease development, because pleural abnormalities were identified in nonsmokers. Since the development of pleural disease in smokers without asbestos exposure has not been documented by other reports, the authors' findings of similar

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Table 4 — Differences in Mean %FVC by Length of Employment, Amount Smoked, and Pleural Disease*

Employment Duration, yr	Average No. of Packs Smoked	Pleural Abnormalities: Present %FVC (Mean ± SD)	Pleural Abnormalities: Absent %FVC (Mean ± SD)
1-20	Nonsmoker	102.8 ± 14.5	107.3 ± 13.5
	0.5-1.0	97.3 ± 10.1	101.9 ± 8.3
	1.5-2.0	96.1 ± 10.6	102.6 ± 11.2
	2.5-3.0	94.2 ± 11.9	99.8 ± 0.9
21-30	Nonsmoker	104.8 ± 17.4	110.3 ± 13.7
	0.5-1.0	100.4 ± 8.3	97.0 ± 17.7
	1.5-2.0	99.0 ± 12.0	103.3 ± 18.1
	2.5-3.0	98.4 ± 12.3	100.4 ± 15.5
31+	Nonsmoker	99.0 ± 12.8	104.8 ± 6.3
	0.5-1.0	99.5 ± 14.9	103.2 ± 21.4
	1.5-2.0	99.3 ± 19.1	99.1 ± 19.9
	2.5-3.0	99.1 ± 18.1	101.2 ± 12.3

* Workers with parenchymal or large opacities (N = 12) were excluded from this analysis

Table 5 — Differences in Mean %FEV_1 by Length of Employment, Amount Smoked, and Pleural Disease*

Employment Duration, yr	Average No. of Packs Smoked	Pleural Abnormalities: Present %FEV_1 (Mean ± SD)	Pleural Abnormalities: Absent %FEV_1 (Mean ± SD)
1-20	Nonsmoker	104.2 ± 16.4	108.2 ± 16.8
	0.5-1.0	97.6 ± 9.7	102.9 ± 11.0
	1.5-2.0	92.1 ± 10.7	100.7 ± 15.9
	2.5-3.0	92.2 ± 11.9	98.4 ± 12.7
21-30	Nonsmoker	107.3 ± 23.1	110.1 ± 14.3
	0.5-1.0	98.1 ± 14.3	92.1 ± 24.5
	1.5-2.0	93.8 ± 14.7	92.1 ± 18.9
	2.5-3.0	96.3 ± 13.0	98.2 ± 17.2
31+	Nonsmoker	96.6 ± 17.2	109.6 ± 11.7
	0.5-1.0	98.3 ± 22.1	89.6 ± 36.2
	1.5-2.0	95.1 ± 18.1	93.1 ± 20.2
	2.5-3.0	94.3 ± 16.6	99.9 ± 5.23

* Workers with parenchymal or large opacities (N = 12) were excluded from this analysis

prevalence rates in workers with more than 30 years of employment, regardless of smoking group, suggest that smoking may increase the rate of development of pleural abnormalities in asbestos-exposed workers. In this sense, smoking is an effect modifier rather than a confounder of the association between asbestos exposure (employment) and pleural disease development. From these analyses, assessing the role of smoking on pleural disease, a better explanation of the data is provided by estimating amount smoked (heavily influenced by smoking duration) rather than by simply using smoking category, expressed as current smoker, ex-smoker, or never smoked.

A reduction in the %FVC in individuals with pleural disease suggests that the presence of pleural thickening on chest x-ray films may serve as an indicator of early restrictive pulmonary disease not diagnosable by characteristic opacities on chest roentgenography. This marker was not limited to the more advanced forms of pleural abnormalities since the relationship between %FVC and the presence of diffuse pleural thickening of grade 1 was also highly significant.

The composition of this study population was somewhat different from that of the entire union membership, with fewer individuals having worked for less than 11 or for greater than 30 years since initiation. However, the authors do not believe that selection bias was operating in this study because they would not expect workers to have any knowledge of existing pleural disease prior to the evaluations. Pleural disease does not manifest itself by causing chest pain or other pulmonary symptoms. Hence, workers would not volunteer because of a perceived disease condition. In addition, extensive occupational his-

tories were compiled during the examinations to validate the length of employment in the trade. We reviewed union records to identify all eligible union members and the number of years since each worker's initiation. Those participating in our study were closely representative of the entire union membership (Table 1).

The findings of the present study agree with those of many other studies where pleural disease has been significantly correlated with the length of employment in a trade involving asbestos exposure.[6] Hedenstierna et al[6] did not find an association between %FVC and pleural disease, although they did observe a significant relationship between closing volume of the lung and pleural disease. Unlike Weiss and co-workers'[18] evaluation of asbestos workers, the authors did not find a strong association between smoking group and pleural disease. However, there does appear to be an association between the average amount smoked and the rate of development of pleural disease in asbestos-exposed individuals.

The high rate of pleural disease in this population of moderately exposed asbestos workers indicates a surprisingly high extent of asbestos-related disease in a group of workers with exposures comparable with exposures in many trades in the construction industry.[5] This study, along with others described above, points to an alarmingly high rate of disease among this large working population. Since pleural disease, as a marker of asbestos exposure, points to the occurrence of more severe conditions, particularly asbestos-related pulmonary disease and malignancy, future surveillance of populations with similar exposure is warranted. More importantly, prevention of further exposure to asbestos in construction workers is essential to prevent the occurrence or the exacerbation of disease in the future.

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