Loss of Lung Function Among Sheet Metal Workers: Ten-Year Study

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One hundred and twenty-two sheet metal workers in New England were examined over a 10-year interval for loss of pulmonary function and the development of asbestosis or asbestos-related pleural fibrosis. Regression models using the generalized estimating equation (GEE) approach were created to investigate the relationship between exposure and pulmonary function after adjusting for smoking status, age, height, and asbestos-related x-ray changes. A history of shipyard work was a significant contributor to the loss of forced vital capacity (FVC). Among smokers, loss in forced expiratory volume at 1 sec (FEV$_1$) also had a significant relationship to prior shipyard work. There was a borderline significant relationship between percentage predicted FEV$_1$ and cumulative years of asbestos exposure in smokers, as well as years-since-initial-exposure in never-smokers. This study supports previous findings of obstructive airway changes in asbestos-exposed workers and identifies shipboard work as an important predictor of loss in pulmonary function even years after shipyard exposure to asbestos has ceased. Am. J. Ind. Med. 32:460–466, 1997.   © 1997 Wiley-Liss, Inc.

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INTRODUCTION

In studies of asbestos exposed workers, more rapid development of restrictive or interstitial lung disease has repeatedly been associated with cumulative asbestos expo-

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sure [Blanc et al., 1988], the development of opacities on chest x-ray [Kilburn and Warshaw, 1990b; Kouris et al., 1991; Miller et al., 1992; Rom, 1992; Rosenstock et al., 1988], smoking [Blanc et al., 1988; Kilburn et al., 1986; Lilis et al., 1986, 1991], and, to variable degrees, with the extent of pleural plaques or thickening on chest xray [Baker et al., 1985; Garcia-Closas and Christiani 1995; Kouris et al., 1991; Lilis et al., 1992; Miller et al., 1994; Schwartz et al., 1990, 1994; Rom, 1992]. Obstructive pulmonary deficits have been the subject of recent investigations into nonmalignant conditions initiated or exacerbated by occupational exposure to asbestos [Blanc et al., 1988; Hunting et al., 1993; Kilburn et al., 1986, 1990; Kouris et al., 1991; Nakadate 1995; Rom, 1992; Rosenstock et al., 1988; Siracusa et al., 1984]. Some investigators have found evidence that lung opacities [Kilburn and Warshaw, 1990a; Rom, 1992], pleural plaques [Garcia-Closas and Christiani, 1995; Rom, 1992; Rosenstock et al., 1988; Schwartz et al., 1990], year since first exposure to asbestos [Blanc et al., 1988] and diffuse pleural thickening [Kouris et al., 1991; Rom, 1992] may indicate a more rapid progression of obstructive disease in these workers [Kilburn and Warshaw,

1990b and c]. Obstructive changes have been documented among nonsmokers exposed to asbestos [Blanc et al., 1988; Kilburn et al., 1985; Mohsenifar et al., 1986; Rom, 1992] . Longitudinal investigations of exposed workers have sought to confirm reports from case-control and cross-sectional studies on the contributions of smoking, exposure intensity and exposure duration to the progression of restrictive and now, obstructive lung diseases. Other than one large cohort of asbestos insulators studied on multiple occasions by Selikoff and colleagues [Lerman et al., 1988; Lilis et al., 1986, 1991, 1992; Miller et al., 1992, 1994], the mean length of follow-up for larger longitudinal studies has been in the ranges of 30 months [Rom, 1992], 2 years [Schwartz et al., 1994], 4 years [Ohlson et al., 1985], and one study of 7 years [Siracusa et al., 1984].

While numerous reports are also available on the morbidity and mortality among heavily exposed workers [insulators, miners, textile workers], cohorts of less-intensively exposed workers have only recently been investigated [Hunting et al., 1993; Lilis et al., 1992; Welch et al., 1994].

The goal of this study was to identify indicators for the loss of lung function in workers exposed directly and indirectly to asbestos (sheet metal workers). Potential determinants examined included: x-ray findings, smoking history, occupational history, initial asbestos exposure time and exposure duration. The extended follow-up period (ten years), standardized health assessment tools, and appropriate analytical methods for longitudinal data were employed to enhance the usefulness of this investigation.

MATERIALS AND METHODS

Study Population and Data Collection

Medical records were reviewed for 125 New England sheet metal workers who had attended two voluntary union screenings approximately 10 years apart [1981–1982 and 1991]. These screenings were conducted at the Massachusetts Respiratory Hospital in Braintree, Massachusetts, and the 1991 cross-sectional findings were evaluated as part of the previously reported National Sheet Metal Worker Asbestos Disease Screening Program [Welch et al., 1991]. All active and retired members belonging to the union Health and Welfare Plan (n = 1,413) were invited for the initial screening. Three hundred and thirty-one workers were evaluated during the 1981–1982 examination. Thirty-eight percent (125 workers) of these workers returned for the second screening one decade later. Two workers of the 125 had incomplete records and were excluded from this study. Clinical exclusion criteria included malignancy or other serious medical condition. As a result, one worker with congestive heart failure was excluded from the

analysis, leaving 122 participants eligible for later-period analysis.

During both examinations, workers completed a medical and occupational history questionnaire, underwent simple spirometry, a physical examination, and a postero-anterior and lateral chest x-ray. Occupational history and duties were recorded in detail for type and duration, including exposure to other occupational pulmonary hazards and the history of shipboard work of any duration. Cigarette smoking was recorded as pack-years (packs per day $\times$ years smoked) in ex-smokers and current smokers. Spirometry was performed using an 8-liter Eagle II survey spirometer (Collins, Braintree MA) in accordance with the existing American Thoracic Society recommendations at the time of screening [ATS, 1987]. Original test results were reviewed and recorded for this investigation in accordance with the latest recommendations [ATS, 1987]. Prediction equations for forced vital capacity (FVC) and forced expiratory volume at 1 sec (FVC_1) were obtained from Knudson et al. [1976].

Radiographs were interpreted at the time of the screening by both a certified NIOSH B-reader according to ILO classifications [ILO 1980] and later by the examining board-certified occupational physician who was blinded to exposure status. Only 1991 radiographic data were used in this study. Any discrepancies on x-ray interpretation were reviewed by an experienced and unblinded third reader (D.C).

Statistical Analysis

The primary objective of this investigation was to identify the determinants of lung function changes in sheet metal workers exposed to asbestos. The generalized estimating equations (GEE) approach [Liang and Zeger, 1986; Zeger and Liang, 1986] was used for the analysis of pulmonary function outcome variables including the primary outcomes of FEV_1, and FVC, and the secondary outcomes of percentage of predicted FEV_1, percentage of predicted FVC, the ratio FEV_1/FVC and the presence of obstructive deficit (yes or no). The presence of obstructive deficit was defined using a common clinical consolidation of FEV_1 and FEV_1/FVC (FEV_1 percentage of predicted <80% or FEV1/FVC <70%). First, an analysis was performed for a full model using the whole study population and all independent variables of interest including age and height (if the outcome was other than a percentage of predicted value already incorporating age and height), indicator variables for smoking status, pack-years of smoking, year of initial asbestos exposure, total years of asbestos exposure, history of ship work and an indicator variable for asbestos-related disease. To obtain a more parsimonious model, backward elimination of predictors was performed for FEV_1 and FVC, while always keeping age and height in the model. In

addition, subgroup analyses were conducted for FEV_1 by smoking status and by presence of asbestos-related pleural disease (ARPD). A separate regression analysis using an integrated pleural index variable [Lilis et al., 1992] was also performed. The analyses of raw FEV_1 and FVC, which control for the effects of age and height through the modeling procedure, were considered more relevant than the analyses of percentage of predicted FEV_1 and FVC, which are derived through age and height prediction equations that may not remove the possible confounding effects of these variables in the analysis.

Analysis of possibly non-Gaussian longitudinal data, including dichotomous longitudinal data, is difficult partly because few models are available for the joint distribution of the repeated observations for a subject. The GEE approach avoids the need for multivariate distributions by only assuming a functional form for the marginal distribution at each time point, but it also takes correlation across time points into account to increase efficiency. Using this methodology it is only necessary to specify the relationship between the mean and the variance and a hypothesized ''working'' correlation structure. The resulting estimating equations have consistent solutions even when the time dependence is misspecified. In addition, robust estimators of the variances and covariances of the estimated parameters are used. The specified relationship between the mean and the variance determines the ''link'' used in a GEE analysis. The identity link was used for all continuous outcomes while the logit link was used for the one dichotomous outcome. An unstructured working covariance function was used for all analyses due to the small number of variance parameters that would need to be estimated (one variance at each of two time points, and one covariance between time points).

RESULTS

On initial examination, study subjects were, on average, older than nonparticipants, with longer work experience (Table I). Clinical measures of both groups were similar with the exception of a greater number of mild restrictive changes and advanced obstructive changes in the nonparticipants. Smoking history could not be closely compared because of the lack of recorded smoking status among several nonparticipants, although it appeared that there were a greater number of current smokers among nonparticipants.

All workers in the study population were white males ranging in age from 31 to 73 years of age at the time of the first screening examination (1981–1982). The year of their initial employment as sheet metal workers ranged from 1924 to 1971 (Table II). At the time of the second screening examination, their total years of exposure to asbestos (at any level) ranged from 16 to 55 years. Thirty-five percent of workers had practiced their trade aboard ship. Nearly all workers had exposure to welding fumes and fiberglass dust.

TABLE I. Comparison Between 125 Twice-Examined and 206 Once-Examined Sheet Metal Workers

	Study population		Nonparticipants			
	Mean	SD	Mean	SD	t	P
Age	49.2	10.0	45.7	12.2	2.70	<0.01
Years in trade	27.3	9.6	23.7	10.2	3.18	<0.01
	n	%	n	%	χ^2	P
Smoking						
Never-smoker	29	23.2	27	13.1	a	—
Ex-smoker	58	46.4	56	27.2		—
Current smoker	36	28.8	74	35.9	a	—
Unknown	2	1.6	49	23.8		
Total	125		206			
X-ray						
Pleural abnormality	63	50.4	119	57.8	1.70	0.19
Parenchymal abnormality	6	4.8	10	4.9	.001	0.98
Pulmonary function						
Obstructive changes						
Mild[b]	21	17.1	29	14.1	0.45	0.50
Moderate[c]	4	3.3	9	4.4	0.26	0.56
Severe[d]	1	0.8	6	2.9	1.65	0.20
Total (mild–severe)	26	20.8	44	21.3	0.01	0.90
Restrictive changes						
Mild[e]	6	4.9	24	11.7	11.56	<0.01
Moderate[f]	1	0.8	5	2.4	1.15	0.28
Severe[g]	0	0	0	0	—	—
					5.77	0.02

[a]Unknowns prohibit statistical comparison.
[b]65% <FEV_1 percent of predicted <80% or 60% <FEV_1/FVC <70%.
[c]45% <FEV_1 percent of predicted <65% or 41% <FEV_1/FVC <60%.
[d]FEV_1 percent of predicted <45% or FEV_1/FVC <41%.
[e]65% <FVC percent of predicted <80%.
[f]45% <FVC percent of predicted <65%.
[g]FVC percent of predicted <45%.

Fifty-eight percent of attendees were ex-smokers, and 18% were current smokers at the time of the second examination.

Seventy-four percent of workers did not meet common clinical criteria for airflow limitation or restriction and were considered to have no impairment (Table III). In a regression analysis, the presence of an obstructive deficit was more likely in those with increased pack-years of smoking, as expected.

Parenchymal changes were seen in only 6 of 122 workers and no workers had diffuse pleural thickening on chest x-ray. Forty percent of workers had bilateral circumscribed pleural fibrosis [plaques] (Table IV). Mean pulmonary function changes by smoking subgroup are given in Table V.

TABLE II. Exposure History of 123 Sheet Metal Workers

	Mean	SD	Range
Asbestos exposure			
Year of initial exposure	1952		1924–1971
Duration of exposure			
Before 1981–82 examination	27.3	9.6	10–48 yr
Before 1991 examination	35.0	8.6	16–55 yr

	n	(%)
Asbestos exposure in a parent	3	2.5
Shipyard work	44	35.8
Other occupational exposures		
Welding fumes	120	98.3
Fiberglass	113	92.6
Silica/rock dust	13	10.6
Wood dust	12	10.0
Coal dust	2	1.6
Beryllium	1	0.8
Smoking status		
Never-smoker (0 pack-years)	29	23.6
Ex-smoker [mean = 34.7 pack-years (SD = 24.8)]	72	58.5
Current-smoker [mean = 38.6 pack-years (SD = 24.7)]	22	17.9

TABLE III. Respiratory Impairment in Study Population Follow-up Examination of 122 Sheet Metal Workers[a]

	n	%
No impairment	90	74.0
Mild[b]	27	22.0
Moderate[c]	4	3.2
Severe[d]	1	.8

[a]One subject with congested heart failure excluded.
[b]65% ≤FVC percent of predicted <80%, 65% ≤FEV$_1$ percent of predicted <80%, or 60% <FEV$_1$/FVC <70%.
[c]45% ≤FVC percent of predicted <65%, 45% ≤FEV$_1$ percent of predicted <65%, or 41% <FEV$_1$/FVC <60%.
[d]FVC percent of predicted ≤45%, FEV$_1$ percent of predicted ≤45%, or FEV$_1$/FVC <41%.

In the saturated regression model with FVC as an outcome (Table VI), age, height, and status of a current smoker were significant predictors of lower FVC. Using backward elimination for the best fit, age, height, being an ex-smoker, being a current smoker and shipyard work remained in the model. Workers with a history of shipyard work had lower FVC values. In models using Knudson's [Knudson et al., 1976] equations for percentage predicted FVC, exposure and pleural plaques as predictors were not statistically significant variables.

TABLE IV. Radiographic Abnormalities at Second Survey of 122 Sheet Metal Workers[a]

	n	%
None	56	45.9
Asbestos-related pleural plaques	60	49.2
Diffuse pleural thickening	0	0
Parenchymal changes	6	4.8
ILO[b] perfusion 1/1	5	4.0
ILO perfusion 1/0	1	0.8

[a]One subject with congestive heart failure was excluded.
[b]ILO - International Labor Organization Classification.

TABLE V. Mean Pulmonary Function Changes by Smoking Status (10-year) in Sheet Metal Workers Examined Twice

	FEV$_1$ change (l)	SD	FVC change (l)	SD
Never-smoker	−0.407	0.446	−0.435	0.342
Ex-smoker	−0.355	0.416	−0.402	0.368
Current smoker	−0.434	0.335	−0.351	0.379
All	−0.381	0.409	−0.401	0.362

TABLE VI. Multivariate Models for Determinants of Changes in FVC in Sheet Metal Workers Examined Twice

	Saturated model		Best-fit model	
Variable	Coefficient (SE)	P	Coefficient (SE)	P
Age (yr)	−0.041 (0.006)	<0.001	−0.038 (0.003)	<0.001
Height (cm)	0.098 (0.022)	<0.001	0.098 (0.022)	<0.001
Ex-smoker	−0.210 (0.132)	0.11	−0.253 (0.117)	0.03
Current smoker	−0.344 (0.135)	0.01	−0.375 (0.122)	<0.01
Cigarette pack-years	−0.002 (0.002)	0.45		
Year of initial asbestos exposure	0.005 (0.005)	0.38		
Total years of asbestos exposure	0.006 (0.006)	0.32		
Shipwork (1 = yes)	−0.179 (0.102)	0.08	−0.205 (0.103)	0.05
Asbestos-related pleural plaque (1 = yes)	−0.084 (0.062)	0.17		

The subgroup of current smokers had significant relationships between FEV$_1$, age and ship work (Table VII). Workers who smoked and had a history of ship work had lower mean FEV$_1$. The percentage predicted FEV$_1$ in

TABLE VII. Multivariate Models for Determinants of Changes in FEV$_1$ Among Smokers and Never-Smokers in Follow-up Study of Sheet Metal Workers

Variable	Current smokers (FEV$_1$) Coefficient (SE)	P	Current smokers (% predicted FEV$_1$)[a] Coefficient (SE)	P	Never-smokers (FEV$_1$) Coefficient (SE)	P	Never-smokers (% predicted FEV$_1$)[a] Coefficient (SE)	P
Age (yr)	−0.037 (0.008)	<0.001			−0.063 (0.010)	<0.001		
Height (cm)	−0.005 (0.052)	0.93			0.045 (0.053)	0.40		
Year of initial asbestos exposure	−0.004 (0.013)	0.79	−0.192 (0.394)	0.63	0.008 (0.017)	0.64	0.844 (0.450)	0.06
Total years of asbestos exposure	−0.007 (0.012)	0.57	−0.486 (0.262)	0.06	0.030 (0.010)	<0.01	0.252 (0.239)	0.29
Shipyard (1 = yes)	−0.502 (0.188)	<0.01	−9.413 (5.882)	0.11	−0.103 (0.212)	0.63	−2.550 (6.690)	0.70
Asbestos-related pleural disease	−0.004 (0.140)	0.98	−1.743 (4.506)	0.70	−0.039 (0.105)	0.71	3.959 (4.390)	0.37

[a]Knudson's prediction equations using age and height [Knudson et al., 1976].

smokers had a borderline-significant relationship with total years of asbestos exposure; workers who smoked and had longer exposures had a lower percentage predicted FEV$_1$. In never-smokers, year of initial exposure had borderline significance in its relationship to percentage predicted FEV$_1$; those never-smokers with earlier exposure had a decreased percentage predicted FEV$_1$. Age and total years of asbestos exposure were related to FEV$_1$ in never-smokers; however, total years of exposure had a positive coefficient. Exposure and pleural plaques were not significant predictors in the ex-smoker subgroup. FEV$_1$ in the population as a whole (including all smoking subgroups) had the expected relationships to age, height, and smoking but showed an unexpectedly positive coefficient (of borderline statistical significance) for total years of asbestos exposure. On backward elimination of variables, years of exposure becomes nonsignificant and drops from the model for FEV$_1$.

Subgroup analysis by presence or absence of pleural disease and a separate analysis using the continuous variable for an integrated pleural score [Lilis et al., 1992] gave no significant results among the occupational exposure variables.

DISCUSSION

This study demonstrates that shipyard work is a significant risk factor for increased pulmonary function loss over 10 years among sheet metal workers exposed to asbestos. Sheet metal workers with a history of shipyard work sustained a significantly accelerated loss of FVC, and the smokers among this group also showed relatively larger decrements in FEV$_1$. Similarly, smokers with more total years of asbestos exposure had accelerated loss of FEV$_1$. While shipyard work did not predict decreased FEV$_1$ in the never-smokers, the data suggest that never-smokers with

more remote exposure to asbestos also had greater loss of FEV$_1$.

It is generally accepted that shipyard work involves high level exposures to asbestos (when present) and other toxic materials (e.g., welding, metal grinding and polishing, sandblasting, painting and other surface coating) [Kilburn and Warshaw, 1990a]. One limitation to our study is that our findings could result from the confounding effects of other occupational exposures in this environment, although some may be expected to cause a greater decline in flow rates and FEV$_1$ than FVC.

This study adds to the evidence of an interaction between smoking and asbestos exposure for nonmalignant lung disease. It has been hypothesized that smoking may act as a risk factor for progressive fibrosis [Blanc, 1988; Schwartz et al., 1994] and that it may also act synergistically with asbestos for obstructive changes [Blanc, 1988; Miller et al., 1993]. It should be noted that mild pulmonary fibrosis has also been associated with obstructive changes in the absence of smoking [Nakadate, 1995; Ohlson et al., 1985; Siracusa et al., 1984]. In our analysis, we found evidence that shipyard work and total duration of exposure to asbestos is associated with accelerated declines in FEV$_1$ among smokers while controlling for asbestos-related radiographic changes. Smoking appeared to be additive in its effect on loss of lung function. Moreover, we found that cumulative exposure to asbestos in smokers had a borderline significant relationship with 10-year decline in FEV$_1$.

Among nonsmokers, a borderline significant relationship between year-of-initial exposure to asbestos and percentage predicted FEV$_1$ was found.

We explored, but failed to find, a relationship between pleural plaque and loss of pulmonary function in this population. Similarly, when sheet metal workers were

compared to more heavily exposed insulation workers in one study [Lilis et al., 1992], the relationship between pleural index and FVC in sheet metal workers was not as convincing as in the insulators.

The major limitation of this study was the low proportion of workers who returned for the second screening examination, an expected consequence of the extended follow-up period. When compared (Table I), the study subjects initially appeared older and healthier than nonparticipants. If self-selection led to the return of predominantly more (or less) healthy screening participants who also had a differential history of asbestos exposure, the results would be biased. However, our population (49% with pleural disease and 5% with parenchymal disease) was found to have mid-range disease rates as compared with previous reports of asbestos-related disease prevalence in New England sheet metal workers with more than 30 years of seniority (70% with pleural disease and 4% with parenchymal abnormalities)[Baker et al., 1985], and when compared with the national sheet metal worker population (13% with pleural disease and 6% with parenchymal disease)[Welch et al., 1991]. Estimated annual loss of lung function in our study population was averaged at 38 ml/year for FEV_1 and 40 ml/year for FVC, consistent with working populations with low to moderate asbestos exposure [Rom, 1992].

It is possible that a healthy worker effect [Monson, 1990] could account for the absence of a clear relationship between duration of exposure and disease outcome. Bias could be introduced if ill workers with early x-ray evidence and symptoms were more likely to leave work, go on compensation, or pursue intensive medical evaluations, thereby no longer having interest in screening examinations. The relationships between pleural changes and pulmonary function loss would therefore be biased toward the null in this study. This could account for the absence of significant x-ray predictors of pulmonary function loss.

In summary, this long-term follow-up of sheet metal workers provides evidence that past shipyard work, as a likely surrogate for asbestos exposure intensity, has the most significant impact on 10 year loss of lung function among the easily categorized exposure variables. Evidence is also offered, similar to that found among more heavily exposed occupations, that asbestos-exposed workers have accelerated obstructive changes in relation to basic measures of exposure. This is particularly evident in long-term smokers.

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