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Occupational exposure to dust and lung disease among sheet metal workers

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Abstract

A previous large medical survey of active and retired sheet metal workers with 20 or more years in the trade indicated an unexpectedly high prevalence of obstructive pulmonary disease among both smokers and non-smokers. This study utilised interviews with a cross section of the previously surveyed group to explore occupational risk factors for lung disease. Four hundred and seven workers were selected from the previously surveyed group on the basis of their potential for exposure to fibreglass and asbestos. Selection was independent of health state, and excluded welders. A detailed history of occupational exposure was obtained by telephone interview for 333 of these workers. Exposure data were analysed in relation to previously collected data on chronic bronchitis, obstructive lung disease, and personal characteristics. Assessment of the effects of exposure to fibreglass as distinct from the effects of exposure to asbestos has been difficult in previous studies of construction workers. The experienced workers studied here have performed a diversity of jobs involving exposure to many different types of materials, and this enabled exposure to each dust to be evaluated separately. The risk of chronic bronchitis increased sharply by pack-years of cigarettes smoked; current smokers had a double risk compared with those who had never smoked or had stopped smoking. The occurrence of chronic bronchitis also increased with increasing duration of exposure to asbestos. Workers with a history of high intensity exposure to fibreglass had a more than doubled risk of chronic bronchitis. Obstructive lung disease, defined by results of pulmonary function tests at the medical survey, was also related to both smoking and occupational risk factors. Number of pack years smoked was the strongest predictor of obstructive lung disease. Duration of direct and indirect exposure to welding fume was also a positive predictor of obstructive lung disease. Duration of exposure to asbestos was significantly associated with obstructive lung disease but the dose-response relation was inconsistent, especially for those with higher pack-years of smoking exposure. Exposure to fibreglass was not a risk factor for obstructive lung disease.

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The Sheet Metal Occupational Health Institute (SMOHI) was formed in 1985 by the Sheet Metal Workers International Association (SMWIA) and the Sheet Metal and Air Conditioning National Association to study the health hazards of the sheet metal industry. The Institute invited SMWIA members who were first employed as sheet metal workers at least 20 years earlier to participate in a survey and medical examination provided for them at a convenient time and place. A total of 12 454 (47%) United States and Canadian sheet metal workers were examined out of 26 329 invited. Methods and results of this study have been previously described.[1 2]

A substantial prevalence of obstructive lung disease was seen among both smokers and non-smokers in the surveyed population. In an analysis of 8 288 male United States sheet metal workers who worked only in the building trades and who had complete screening data, the prevalence of chronic bronchitis was 22·8% among smokers and 11·7% among non-smokers. Pulmonary function tests found 17·6% of the smokers and 3% of the non-smokers to have obstructive disease.[1] A substantial proportion of participants were also found by radiography to have parenchymal or pleural disease.

Respiratory hazards identified with sheet metal work include exposure to asbestos, welding fumes, and man made mineral fibres, primarily fibreglass. Although the craft of sheet metal work does not itself use asbestos, sheet metal workers in construction

were, for many years, exposed to asbestos while working in areas that were being sprayed with asbestos for fireproofing, working on beams fireproofed with asbestos, and removing asbestos insulated metal ventilation systems. Before 1973, when this application was banned, over half of the high rise buildings constructed in the United States used a sprayed inorganic fibre as fire proofing.[3] Concentrations as high as 100 fibres/ml were measured in the spray zone during application of asbestos, and as high as 4 fibres/ml 30 minutes after spraying stopped.[3] Balzer *et al*[4] reported in 1968 that insulators experienced concentrations ranging from 0·1–61·6 fibres/ml for various tasks including application and tearing out. Sheet metal workers whom we interviewed reported performing similar tasks to those monitored among insulators. Currently, because of stringent regulations on its use, exposure to asbestos in the sheet metal trade occurs only during removal work. During current work with asbestos, appropriate protective measures are generally taken; such protection was used much less often in the past.

Man made mineral fibres, primarily fibreglass, are used in many sheet metal applications. In particular, sheet metal workers fabricating ducts for ventilation systems line metal ducts with fibreglass in the shop before installation on the job site. Some ventilation ducts are made primarily of fibreglass rather than sheet metal. Also, fibreglass is removed and replaced in existing duct systems. Exposures of sheet metal workers working in fabrication shops with duct liner or duct wrap have been reported to range from 0·11–1·6 fibres/ml.[5] Fowler *et al*[6] measured somewhat higher exposures (0·51–2·34 fibres/ml) among insulators who were wrapping ducts with fibrous glass insulation, a task that sheet metal workers sometimes perform. Esmen *et al*[7] found lower exposures, ranging from 0·008–0·068 fibres/ml, among workers who were fabricating or installing ductboard ventilation systems in well ventilated environments. The same study reported that insulators applying pipe covering, blanket insulation, and wrap around insulation had exposures ranging from 0·012–0·39 fibres/ml. These monitoring results for fibreglass users can be contrasted with ranges of 0·001–0·074 fibres/ml in 10 plants manufacturing glassfibre products of greater than 1 μ nominal diameter.[8] (One other plant manufactured finer glass fibres and had much higher fibre concentrations.)

It is generally difficult to identify construction workers with exposure to fibreglass who have not also been exposed to asbestos. Engholm *et al*[9 10] found that man made mineral fibres and asbestos exposures were correlated, but with careful exposure assessment in their large population they were able to separately assess the respiratory effects of these exposures. The importance of distinguishing such exposures has recently been underscored by Kilburn and Warshaw.[11] These authors found (in an industrial setting) that most workers had both exposures, and that their respiratory effects could not be independently attributed.

Only a few studies have suggested a positive relation between exposure to man made mineral fibres and chronic bronchitis[9 12] or cough and phlegm.[13] Two other studies showed impairment of lung function among exposed workers, but not impairment defined as obstructive disease.[14 15] A few studies also linked exposure to asbestos with chronic bronchitis,[16 17] and exposure to asbestos has also been associated with small airways disease.[18 19]

Methods

STUDY POPULATIONS

We identified 19 SMWIA locals—predominantly in the south east sun belt states or on the west coast of the United States—that reported relatively high use of fibreglass. Using data from the previous medical screening, and applying certain selection criteria, 407 eligible sheet metal workers were selected from these locals. Selected workers had (1) participated in the medical screening; (2) reported working in the sheet metal shop for at least 70% of their working career, or reported doing removal (ripout) for at least 40% of their working career; and (3) not welded for more than 20% of their working careers. These criteria, based on presumed potential for exposures, were chosen to obtain a study population with a range of exposures to fibreglass and asbestos that would not necessarily be correlated. The influence of welding exposure, a known risk factor for bronchitis,[20] was reduced by excluding workers who often welded.

Current contact information was obtained from local business managers or from the Sheet Metal Workers' National Pension Fund. Where contact information was wrong or unavailable, phone books and directory assistance were used to locate study population members.

DATA COLLECTION

The questionnaire was pilot tested with 15 sheet metal workers outside of this study population, and was revised before data collection. Four trained interviewers assisted with the data collection, which began in May 1990 and ended in April 1991. Before telephone contact by interviewers, eligible sheet metal workers received a mailed packet that contained a letter of introduction from the SMWIA president, an informational letter from investigators, and an example questionnaire that indicated material to be covered in the telephone interview. Packets were mailed out in batches, and interviewers attempted to complete interviews within two months of mail contact. Informed consent was obtained at the beginning of each 20–40 minute interview; the protocol had previously been approved by the

university's committee on human research. In some cases it was necessary to recontact participants to clarify responses.

OCCUPATIONAL EXPOSURE DATA

The questionnaire was developed to collect detailed information on the fibreglass and asbestos materials with which a person had worked for at least three months in his career. Fabrication or installation of fibreglass ductboard, and installation or removal of asbestos insulation are examples of tasks and materials that were included. We also asked about exposures that occurred indirectly when the sheet metal worker worked in close proximity to other workers, for instance, in a shop within 10 feet of a fibreglass lining department or within 20 feet of workers removing fibreglass or asbestos (table 1 in the appendix has details).

Each worker's sheet metal experience was divided into an early half and a recent half. The end of a worker's career was considered to be the year a worker left sheet metal work or the year of that subject's screening exam, whichever was earlier. The percentage time that each participant spent working with or around each fibreglass and asbestos material was then determined for the early half and for the recent half of his working career, as was the percentage time spent welding or working in the vicinity of welding fumes. Interviewers also collected summary information on jobs held outside the sheet metal trade. Each job was rated according to whether it involved probable exposures to known lung toxicants such as asbestos, metal dust, or metal fumes.

The numbers of years each subject worked as a sheet metal worker during the early and recent career halves were adjusted to indicate full time equivalent years, using data on whether the participant had worked full time or part time during each decade as a sheet metal worker. The adjusted number of years working in exposed jobs outside sheet metal work was also calculated for each subject.

Cumulative exposure models were developed to indicate the adjusted number of years of high, medium, and low intensity exposure to fibreglass, and the adjusted number of years of high and medium intensity exposure to asbestos. Adjusted years of welding and exposures outside sheet metal work were also calculated. Adjusted years reflects the number of years that a subject worked with specified materials/tasks, adjusted for the percentage of his work time that he said he was exposed. The appendix describes the methods used to assign exposure levels and to calculate cumulative exposure. An example is also presented that shows the method for calculating cumulative exposures.

Summary variables among those exposure measures evaluated were total adjusted years of sheet metal work; adjusted years of exposure to materials/tasks involving high, medium, and low level exposure to fibreglass; adjusted years of exposure to materials/tasks involving high and medium level asbestos exposure (no task was rated as low exposure); adjusted years of exposure to welding fumes; and adjusted years of exposure to lung toxicants outside sheet metal work.

DEMOGRAPHIC AND MEDICAL DATA

The medical examination conducted in 1986–90 consisted of completion of a self administered occupational and medical questionnaire; physical examination; spirometry performed according to American Thoracic Society (ATS) guidelines[21]; posteroanterior and lateral chest *x* ray film interpreted using the International Labour Office (ILO) classification for pneumoconiosis;[22] and stool guaiac for occult blood.[1]

Medical examinations for the 407 prospective participants in the interview study were performed at 15 different clinics. More than 90% of the study population was examined in 1987, and no one was examined after 1988. After data collection and coding by the clinics, data were centrally key punched. Data on pulmonary function were checked for consistency with the ATS standards; results lying outside of the recommended limits of variability were retained in the analysis as recommended by Eisen *et al*.[23]

Predicted values and confidence intervals for spirometric examinations, based on age, sex, and height were derived from the predicted equations of Crapo *et al*,[24] whose 95% confidence intervals (95% CIs) were also employed in characterising individual results of pulmonary function tests into the categories: *normal lung function*: forced vital capacity (FVC), and forced expiratory volume in one second (FEV_1) within the 95% CI; *obstructive disease*: an FEV_1 less than the lower bounds of the 95% CI with an FEV_1/FVC ratio less than the lower bounds of the 95% CI; *restrictive disease*: an FVC less than the lower bounds of the 95% CI, with an FEV_1/FVC ratio within the 95% CI; *small airways disease*: a subset of those with normal lung function, where FEV_1 and FVC were within the 95% CI, and FEF_{25-75} was less than the lower value of the 95% CI.

Parenchymal disease was defined as the presence of profusion of 1/0 or greater on chest *x* ray film reading. A participant was considered to have pleural disease if there were any notations of positive findings on sections 3A–D of the NIOSH/ILO coding form.

Other data collected on the questionnaire included age; years worked in the sheet metal trade; year started working in the sheet metal trade; work state; smoking state; and average packs of cigarettes smoked each year. Also, the average percentage times spent working in four broad areas of sheet metal work were ascertained—namely, shop work, welding, jobsite installation, and ripout.

STATISTICAL METHODS

Differences between the distributions of participant and non-participant characteristics were evaluated for categorical variables by the Mantel-Haenszel χ^2 test. For continuous variables, Student's t test was used to evaluate the statistical significance of differences in means. Pearson correlation coefficients were evaluated to determine the degree of correlation between continuous variables.

For factors such as age, cigarette pack-years, and duration of occupational exposures, regression analyses were run first with categorical dummy variables to determine whether it was appropriate to model these factors as continuous variables.

Multiple logistic regression models were constructed to determine whether the occupational exposure, smoking, or demographic variables were predictive factors for chronic bronchitis and obstructive lung disease. Models were built by adding one variable at a time to the model, strongest predictors first, until the difference in twice the log likelihood between successive models was no longer statistically significant at $p < 0{\cdot}10$. A separate set of models was developed for each outcome. SAS, version 5·1 was used to perform the regression analyses. Odds ratios and 95% CIs are presented.

Results

PARTICIPATION

Table 1 describes interview participation among the study population. Of the 367 subjects contacted, 333 (90·7%) completed an interview. We were unable to contact 40 (9·8%) of the 407 persons in the study population; nearly half of this uncontacted group was deceased.

Table 2 describes baseline characteristics of participants and non-participants. These data were collected at the time of the medical screening about three years before the interview. The non-participants were older and had spent more time working on the jobsite and doing removal jobs (which general involve more dust exposure). More non-participants had stopped smoking. They also had a higher prevalence of abnormalities of lung function (obstructive disease or restrictive disease) and abnormalities on radiography (parenchymal or pleural disease). The prevalence of chronic bronchitis and small airways disease did not differ by participation state.

Table 1 Participation by study population in study of sheet metal workers by interview

	No	*(%)*
Persons meeting selection criteria	407	(100·0)
Contacted, completed interview*	333	(81·8)
Contacted, refused interview	34	(8·4)
Unable to contact	40	(9·8)
Reasons:		
Deceased	18	
Unable to trace	8	
Traced; couldn't contact by phone	5	
Ill, memory loss, etc	5	
Declined to local union	3	
Outside United States	1	

*333 of 367 persons contacted (90·7%) completed an interview.

EXPOSURE

This cross section of experienced workers performed various jobs involving exposure to many different types of fibreglass and asbestos materials. Table 3 presents summary data on occupational exposures reported by the study population. Participants had worked in the trade for a median of 34 adjusted years. (This differs from typical years worked in that it is adjusted downward for part time work). Of this time, participants had median exposures to fibreglass (all levels) of about 12 adjusted years and median exposures to asbestos (all levels) of about five adjusted years. The population as a whole had little welding exposure because those who reported during the previous medical survey that they welded more than 20% of their working time were excluded from this study. None the less, 23% (73/322) of the participants had cumulative welding exposures of five or more adjusted years.

Our original intention was to determine associations between lung disease and the years of exposure to fibreglass and asbestos at each exposure level, but some of the reported exposures were too correlated to permit this. The Pearson correlation coefficient for adjusted years of medium and high level exposure to asbestos was 0·70; therefore, duration of all asbestos exposures was instead analysed for its effect on the outcomes. Years of low and medium exposures to fibreglass were highly correlated with each other but not with high exposure to fibreglass. The analysis of exposures to fibreglass therefore included all exposures combined as well as high exposure to fibreglass separately. Because there was so little spread in the adjusted years of high exposure to fibreglass, an indicator variable ($\geqslant 1$ year high exposure, v none) was employed in the multivariate analysis. The correlation coefficient (r) between total years of exposure to fibreglass and total years of exposure to asbestos was 0·48.

RISK FACTORS FOR CHRONIC BRONCHITIS

Chronic bronchitis was diagnosed symptomatically at the medical survey—on average, three years before this interview study. Both unadjusted and multivariate analyses showed that smoking sharply increased the risk of chronic bronchitis and that exposures to fibreglass and asbestos were also associated with mildly increased risk. Table 4 presents the regression results for 309 participants with complete data on all variables considered.

Table 2 Characteristics of participants and non-participants in interview study of sheet metal workers at time of medical screening*

Characteristic	Participants (n = 333) %† or mean (SD)	Non-participants (n = 74) %† or mean (SD)	p Value for difference
Age (y)	57·6 (8·5)	61·5 (9·2)	0·0005
Years worked sheet metal‡	34·2 (6·7)	34·9 (7·9)	0·43
Latency:§			0·11
< 30 years	20	12	
⩾30 years	80	88	
Average % time:			
In shop	73·3 (26·3)	63·7 (35·4)	0·03
Doing welding	3·6 (5·2)	2·5 (4·2)	0·05
On jobsite	15·3 (13·9)	20·4 (19·9)	0·04
Doing ripout	8·1 (15·6)	15·7 (23·5)	0·01
Ever worked in shipyard	17	12	0·32
Work state:‖			0·03
Working	45	35	
Retired	30	46	
Unemployed	5	8	
Disabled	2	0	
Unknown	17	11	
Smoking state:			0·37
Current	23	16	
Former	52	59	
Never	25	24	
Pack-years‡‡ (for current/former smokers only)¶	22·6 (22·2)	27·0 (25·6)	0·13
% With:			
Chronic bronchitis	15	15	0·92
Restrictive/obstructive disease**	20	30	0·07
Small airways disease††	10	8	0·71
x Ray film abnormality	28	43	0·01

*Non-participant category includes those who refused an interview as well as those who could not be contacted.
†Percentage may not sum to 100 due to rounding.
‡Five missing; §two missing; ‖18 missing; ¶four missing; **13 missing; ††59 missing.
‡‡Pack-years = average packs of cigarettes/day × number of years smoked.

Multiple logistic regression odds ratios (ORs) and 95% CIs (in parentheses) for risk factors for chronic bronchitis were: 1–59 pack-years *v* zero pack-years, OR = 5·2 (1·2–23·2); ⩾60 pack-years *v* 0 pack-years, OR = 10·5 (1·9–59·5); current smoker, OR = 2·1 (1·0–4·5); and a history of high level exposure to fibreglass, OR = 2·3 (1·1–4·9). Years of exposure to asbestos was modelled as a continuous variable and produced an OR of 1·04 for each one year increase of exposure, which may be extrapolated to OR = 1·5 (1·1–2·1) for a 10 year increase in exposure to asbestos. It should be borne in mind, however, that these one year and 10 year increase estimates were both derived from the same coefficient and were therefore not independent.

Table 3 Summary of occupational exposures reported by study participants (n = 333)

Exposure	Adjusted years: Range (min-max)	Adjusted years: IQ range† (25%–75%)	Adjusted years: Median
Work in sheet metal trade	8–51	29–39	34
Fibreglass*			
Low level	0–22	2– 9	5
Medium level	0–36	2–11	5
High level	0– 7	0– 1	0
All fibreglass	0–55	5–20	12
Asbestos*			
Medium level	0–34	2– 8	4
High level	0–24	0– 4	1
All asbestos	0–52	2–12	5
Welding*			
All welding	0–21	0– 4	2
Non-sheet metal work exposures*	0–22	0– 2	0

*Although 333 participants were interviewed, only 315 provided complete and reliable data on fibreglass and asbestos exposures; 322 provided complete and reliable data on welding exposure and jobs held outside of sheet metal work.
†Interquartile range represents the middle 50% of the exposure distribution for each variable. These observations fall between the 25th and the 75th percentile.

RISK FACTORS FOR OBSTRUCTIVE LUNG DISEASE

Obstructive lung disease was determined by pulmonary function test results from the previous medical survey. Cigarette pack-years and adjusted years of exposure to welding were shown in multiple logistic regression analyses to be associated with the occurrence of obstructive lung disease. Including adjusted years of asbestos exposure significantly improved the fit of the model; the associations were, however, inconsistent. Table 5 shows that the results for pack-years were similar to those seen for chronic bronchitis. The ORs for 1–29, 30–59, and ⩾60 pack-years respectively *v* zero pack-years were: 3·0, 6·3, and 17·2; 95% CIs for 30–59 and ⩾60 pack-years excluded unity.

Exposure to asbestos was a statistically significant predictor of obstructive disease, but the dose-response relation was inconsistent. Compared with those with no exposure to asbestos, persons with up

*Table 4 Risk factors for chronic bronchitis: results of multiple logistic regression analysis**

Variable	*OR (95% CI)*
Intercept†	0·014 (0·003–0·060)
Pack-years of cigarettes	
None	1·00
1–59 pack-years	5·21 (1·17–23·20)
⩾60 pack-years	10·48 (1·85–59·52)
Smoking state:	
Never/former	1·00
Current	2·12 (1·00–4·50)
Adjusted years of asbestos exposure, all levels:	
Increase of one year	1·04 (1·01–1·07)
Increase of 10 years‡	1·51 (1·08–2·10)
Level of fibreglass exposure:	
None, low, or medium exposure only	1·00
Ever had high level exposure	2·28 (1·07–4·86)

*No = 309, 44 with chronic bronchitis; model r = 0·347.
†Odds ratio for intercept equals the adjusted rate of disease for those with values equal to zero for all variables in model.
‡Years of asbestos exposure was modelled as a continuous variable, so the estimate for an increase of 10 years is not independent of the estimate for an increase of one year of exposure. Categorical analyses indicated that years of exposure to asbestos was appropriately modelled as a continuous variable.

*Table 5 Risk factors for obstructive lung disease: results of multiple logistic regression analysis**

Variable	*OR*	*(95% CI)*
Intercept†	0·03	(0·006– 0·13)
Pack-years of cigarettes:		
None	1·00	
1–29 pack-years	3·01	(0·76 –11·93)
30–59 pack-years	6·28	(1·62 –24·41)
⩾60 pack-years	17·21	(3·57 –82·91)
Adjusted years of asbestos exposure:		
None	1·00	
>0–<10 years	0·19	(0·06 – 0·62)
10–<20 years	0·41	(0·12 – 1·44)
⩾20 years	1·36	(0·43 – 4·28)
Adjusted years of welding exposure:		
None	1·00	
1–3 years	2·86	(0·95 – 8·58)
⩾4 years	4·09	(1·27 –13·22)

*No = 301, 37 with obstructive lung disease; model r = 0·323.
†Odds ratio for intercept equals the adjusted rate of disease for those with values equal to zero for all variables in model.

to 10 adjusted years of exposure or with 10 to 20 years of exposure had decreased risks of obstructive lung disease, with ORs of 0·2 and 0·4 respectively. The OR for subjects with 20 or more years of exposure to asbestos was 1·4, not significantly different from the reference category. These data are difficult to explain and indicate that those without exposure to asbestos were not a low risk reference group.

Exposure to welding was also associated with obstructive lung disease. The OR was 2·9 (95% CI just including unity) for persons with one to three years of exposure, and 4·1 (95% CI 1·3–13·2) for those with four or more adjusted years of welding exposure.

Notably, current smoking state did not have an important effect on obstructive lung disease risk in the same way that it did for chronic bronchitis. Also, exposure to fibreglass was not associated with obstructive disease.

As results of the multiple logistic regression indicated a higher risk of obstructive disease among persons with no exposure to asbestos compared with those with up to 20 years of exposure, we carried out further analyses to attempt an explanation. Table 6 describes the prevalence of obstructive lung disease cross classified by adjusted years of asbestos exposure and cigarette pack-years. For the group consisting of those who had never smoked and those with less than 30 pack-years of cigarette exposure, the prevalence of obstructive lung disease increased with duration of exposure to asbestos. Only 14 (7·4%) of 189 subjects in this group had obstructive disease, so these estimates are based on small numbers. A logistic regression analysis of obstructive disease among the less than 30 pack-years subgroup, adjusting for other potential risk factors (results not presented), gave increased risks for increasing exposure to asbestos similar to those seen in table 6.

The prevalence of obstructive disease was nearly three times higher among those who had 30 or more pack-years of cigarette exposure (23/112 = 20·5%). A steady increase in prevalence of obstructive disease was not, however, seen in relation to duration of exposure to asbestos for these high pack-year subjects. This was because the small subgroup with no exposure to asbestos had a noticeably high prevalence of obstructive disease (5/12 = 41·7%). The five with obstructive disease were somewhat younger than the rest of the population with 30 or more pack-years of smoking, and worked mostly in sheet metal shops as opposed to on construction sites. Further exploratory analysis, however, including logistic regression analysis, did not produce clues as to why this subgroup had such a high prevalence of obstructive disease. The numbers with obstructive disease at each level of exposure to asbestos are small, so these results should not be given undue emphasis.

RISK FACTORS FOR SMALL AIRWAYS DISEASE

Because other researchers have found small airways disease to be associated with exposure to asbestos (see discussion), we carried out a logistic regression analysis to evaluate risk factors for small airways disease. Data for FEF_{25-75} were missing, and consequently small airways disease state was unknown, for many participants. This analysis included only 247 subjects, 28 of whom had small airways disease. None of the occupational exposure, demographic, or smoking variables were significantly associated with the presence of small airways disease. Both pack-years smoked and current smoking were positive predictors at $0{\cdot}10 < p < 0{\cdot}05$.

Table 6 Prevalence of obstructive lung disease by duration of asbestos exposure and cigarette pack-years

	Prevalence of obstructive lung disease			
	0–29 pack-years		*≥30 pack-years*	
Adjusted years of asbestos exposure	*%*	*(No)*	*%*	*(No)*
None	5·1	(2/39)	41·7	(5/12)
>0–<10 years	3·3	(3/90)	10·3	(6/58)
10–<20 years	12·1	(4/33)	16·0	(4/25)
≥20 years	18·5	(5/27)	47·1	(8/17)
Total	7·4	(14/189)	20·5	(23/112)

Discussion

This study evaluated the effect of occupational exposures on the prevalence of lung disease among sheet metal workers. Medical data had been previously collected. A detailed assessment by questionnaire of both direct and indirect lifetime exposures to fibreglass and asbestos materials provided data for estimating years of exposure adjusted for the proportion of time spent working with the materials. Welding exposures were also ascertained for their potentially confounding role.

For chronic bronchitis, exposure to asbestos and high intensity exposure to fibreglass were positive predictors of disease. Exposure to fibreglass was not, on the other hand, found to be a risk factor for obstructive lung disease, and exposure to asbestos had inconsistent effects. Exposures to welding were positively associated with obstructive lung disease but not with chronic bronchitis. Previous studies, reviewed by Sjogren[25] have found a stronger association of welding with chronic bronchitis. Smoking had a powerful effect on both lung disease outcomes, consistent with the results of other studies.[26]

A few other studies have found increased prevalence of chronic bronchitis or related symptoms in relation to exposure to man made mineral fibres. Moulin *et al*[13] gave a respiratory health questionnaire to 2024 workers in five French man made mineral fibre production plants. The results were presented separately for the largest glasswool plant (plant A) where the 1041 workers had been employed longer than workers in the other four plants. Exposures to fibre dust were probably also higher in this plant, although comparative industrial hygiene data were not presented. A strong association was found in plant A (but not other plants) between the prevalence of phlegm and ever having worked in jobs with exposure to man made mineral fibres. Odds ratios ranged from 3·9–6·0 ($p = 0·01$) for those with exposure to glasswool; there was no trend in the ORs with increasing duration of employment in exposed jobs. Odds ratios for cough were also somewhat increased (ORs ranged from 1·3–1·9, $p = 0·17$, no trend with increasing duration). These ORs were adjusted for smoking and age, which were also positive predictors for both cough and phlegm.

A large study by Engholm and von Schmalensee[9] found an increased rate of chronic bronchitis among 135 000 Swedish construction workers exposed to fibrous glass. Data were collected by self administered questionnaires between 1971 and 1974 from construction workers presumably representing a variety of trades. Respiratory symptoms, smoking, and duration of exposure to asbestos and man made mineral fibres were ascertained. Among never smokers the prevalence of chronic bronchitis was associated with the duration of exposure to mineral fibre, both among those with and without exposure to asbestos. There was some indication among never smokers older than 50 that exposure to asbestos also increased the risk of chronic bronchitis. Standardised prevalence ratios were calculated, adjusting for age and exposure to asbestos, and using those with no man made mineral fibre exposure as a reference group. Among never smokers the ratio for less than three years of exposure to man made mineral fibre was 231; for three or more years of exposure the ratio was 439. Among former smokers, the standardised prevalence ratios were 142 and 209 respectively for less than three and three or more years of exposure. Among current smokers, exposure to man made mineral fibre was not associated with chronic bronchitis. In interpreting these results and those of a later study carried out by the same authors,[10] questions were raised regarding the accuracy of self reported data on dust exposure, and the high degree of correlation between exposure to asbestos and to man made mineral fibres.

Sanden and Jarvholm[27] found that shipyard workers exposed to man made mineral fibres had a slightly increased frequency of cough with phlegm (rate ratio = 1·3, $p < 0·01$), adjusted for smoking but not for exposure to asbestos. No differences in pulmonary function were found between exposed and non-exposed groups.

Maggioni and others[12] studied 467 glass wool workers and found that the incidence of chronic and dysplastic pharyngolaryngitis were increased among workers with at least five years working in departments with high levels of glass wool exposure. With regard to lower respiratory symptoms, an analysis carried out among a subset of the population showed

the incidence of chronic bronchitis to increase with increasing duration of exposure among both smokers and non-smokers. Differences in pulmonary function between exposure groups were not seen.

Previous studies have not indicated an association between obstructive lung disease and exposure to fibreglass, although two studies have found non-specific impairment of pulmonary function. Hill *et al*[14] reported a significantly increased prevalence of non-specific lung disease among 340 current and former glass wool factory employees aged 55–74. Impairment of lung function was more severe among workers with high exposure to fibre, but was not related to duration of exposure. In a small study of non-smoking, non-asbestos exposed sheet metal workers, Sixt *et al*[15] found that workers exposed to fibreglass had greater elastic recoil than non-exposed referents. The study, however, found no evidence of small airway dysfunction or restrictive or obstructive lung disease.

Some other studies of populations exposed to man made mineral fibres have produced negative or equivocal results for bronchitic symptoms or abnormalities of pulmonary function. These studies have been recently reviewed by the International Agency for Research on Cancer[28] and by the World Health Organisation.[29]

Several earlier studies have reported an increased incidence of chronic bronchitis in populations exposed to asbestos. Hedinestierna *et al*[16] reported that 20% of asbestos workers with pleural plaques had chronic bronchitis compared with 5% in other workers; the pleural plaques served as a marker of exposure in this study. Engholm *et al*[9] found an association between exposure to asbestos and chronic bronchitis among non-smoking construction workers, as already discussed. Copes *et al*[17] reported that risk factors for chronic bronchitis in asbestos miners include early exposure and duration of exposure to asbestos.

Although we did not find such an association in this study, asbestos has been linked in many studies to abnormalities of airflow in the small airways. Studies on the pathology of the lungs of asbestos workers have shown a thickening of the walls of the membranous and respiratory bronchioles, as part of a peribronchiolar alveolitis;[18 19] this same lesion is seen in animals exposed to asbestos.[30 31] Reduction in midflow has been reported in human populations exposed to asbestos[32–34] and Hjortsberg *et al* reported an increase in the volume of trapped gas, an indicator of small airway dysfunction, in a study of asbestos exposed railroad workers with pleural plaques.[35]

Reduction in flows in the larger airways, manifest as a reduction in the FEV_1 and FEV_1/FVC ratio, has not been consistently associated with exposure to asbestos. Kilburn and Warshaw[34] reported a reduction in FEV_1, FEV_1/FVC ratio, and an increase in both total lung volume (TLV) and the ratio of residual volume to TLV in non-smoking construction and shipyard workers exposed to asbestos. Oliver *et al* reported that pleural plaques were not associated with obstructive disease when smoking is accounted for in a logistic regression model,[36] but the duration of employment was a significant predictor for obstructive lung disease. Hjortsberg *et al* found an increase in the FEV_1/FVC ratio in non-smoking asbestos workers, consistent with restrictive disease alone.[37]

The effects of exposure to asbestos on the prevalence of obstructive lung disease in this study seemed to differ by smoking exposure. A stratified analysis indicated that exposure to asbestos may have a dose-related effect among never smokers and subjects with fewer than 30 pack-years of smoking. The effects among heavier smokers, however, were inconsistent and not indicative of a causal relation. Indeed, the effect of heavy smoking may overwhelm any effect of exposure to asbestos. The results of this study suggest that future studies should look separately at non-smokers in assessing whether asbestos increases the risk of obstructive disease. Given the high prevalence of current or former smokers in occupational groups exposed to asbestos, it is essential that the relation between asbestos exposure and chronic bronchitis or obstructive disease be examined either in a large group of non-smokers, or by accounting for smoking in a logistic regression model. The data presented here suggest that findings that have been attributed to smoking in the past may in part be due to exposure to asbestos.

High intensity exposure to fibreglass was associated with chronic bronchitis in this study; neither the adjusted years of lower intensity exposures nor the total adjusted years of exposure to fibreglass were identified as risk factors for chronic bronchitis. It should be noted that fibreglass removal was the only task for which exposure to fibreglass was rated as high. Because tasks with lower intensity of exposure were not associated with lung disease in this study, the results indicate that control of exposure to fibreglass dust to low levels may prevent the development of non-malignant lung disease in working populations. The importance of detailed exposure assessment in future research must be emphasised; assessment of task specific exposures is especially important for epidemiological studies of construction workers.

Appendix

CUMULATIVE EXPOSURE MODEL

This appendix describes the methods used to assign exposure levels and to calculate cumulative exposure. An example of the method for calculating cumulative exposures is presented.

Appendix table 1 Fibreglass and asbestos materials and tasks, with assigned exposure categories

Exposure category	*Material or task**
Fibreglass (FG):	
High	Ripout involving any FG material (direct)
Medium	FG ducts or ductboard (direct)
	FG sound lining (direct)
	FG duct wrap or insulation (direct)
	FG ripout (indirect)
Low	Moulded FG pipe (direct)
	FG ductboard or FG lining (indirect)†
Asbestos (ASB):	
High	Ripout involving ASB (direct)
	Other shipyard ASB exposures (direct)
	Ripout involving ASB (indirect)
Medium	Transite pipe (direct)
	Spray on ASB insulation (direct)
	Other ASB materials (insulation, vibration isolators, gaskets, ASB tape) (direct)
	Pipe covering, ASB spraying, transite pipe, other ASB (indirect)†

*Direct refers to tasks in which the subject was working directly with the material. Indirect refers to tasks in which the subject was working within a specified distance of another worker who was using the material.
†For indirect exposure tasks within the low level FG category and the medium level ASB category, only the task with the highest percentage time was summed. This was to avoid double counting indirect exposures that may have occurred simultaneously.

Appendix table 2 Illustration of method used to calculate cumulative exposures

	Hypothetical questionnaire data			
Material/task (exposure category)	*% Time early half*	*(No of years)**	*% Time recent half*	*(No of years)**
FG ripout (high FG)	10	(20)	5	(20)
Install FG ducts (medium FG)	10	(20)	30	(20)
FG ripout—indirect (medium FG)	5	(20)	5	(20)
ASB ripout (high ASB)	20	(20)	10	(20)
Spray on ASB insulation (medium ASB)	5	(20)	0	(20)
Welding fume exposure (welding)	0	(20)	5	(20)
No exposure	50	(20)	45	(20)
Calculated cumulative years of exposure				
Fibreglass:				
Adjusted years high FG = (10% × 20 y) + (5% × 20 y)	= 3 years			
Adjusted years medium FG = (15% × 20 y) + (35% × 20 y)	= 10 years			
Adjusted years total FG (any exposure level)	= 13 years			
Asbestos:				
Adjusted years high ASB = (20% × 20 y) + (10% × 20 y)	= 6 years			
Adjusted years medium ASB = (5% × 20 y) + (0% × 20 y)	= 1 year			
Adjusted years total ASB (any exposure level)	= 7 years			
Welding:				
Adjusted years welding = (0% × 20 y) + (5% × 20 y)	= 1 year			

*Assume that this person worked full time for his 40 year career. If he had worked part time, the adjusted years would have been reduced proportionately.

The intent was to assess the effect of exposure intensity as well as the effect of adjusted years of exposure to fibreglass and asbestos. Because we did not have quantitative data on amount of dust generated during specific types of sheet metal tasks, an ordinal ranking scheme was developed to indicate the amount of exposure for each task using a specific material. Each type of fibreglass and asbestos task/material was assigned an exposure of high, medium, or low. Appendix table 1 describes the fibreglass and asbestos exposure categories to which the various sheet metal work materials and tasks were assigned. Exposure categories were determined based on the average of independent ratings by six industrial hygienists. Eight categories of exposure to fibreglass and eight categories of exposure to asbestos were rated as high, medium, or low intensity. In all cases, at least three out of six raters concurred on the rating. For five out of eight fibreglass categories, and four out of eight asbestos categories, at least four out of six raters gave the same rating.

Cumulative exposure models were developed to indicate the adjusted number of years of high, medium, and low intensity exposure to fibreglass, and the adjusted number of years of high and medium intensity exposure to asbestos. Adjusted years of welding and non-sheet metal work exposures were also calculated. Adjusted years reflects the numbers of years that a person worked with specified materials or tasks, adjusted for the percentage of his work time that he said he was exposed.

For exposure to fibreglass and asbestos, the percentage times were summed for tasks/materials within each exposure level, separately for the early

half and the recent half of the career, and then totalled to represent exposure over the entire sheet metal career; years of exposure were also adjusted for whether the subject performed sheet metal work full time or part time. According to the cumulative exposure model, one adjusted year of exposure to a material/task is equivalent to a person exposed to that material/task for 100% of his time, full time for a year.

An example is given in appendix table 2, which presents one person's hypothetical percentage time responses with cumulative years of exposure calculated. The table shows that during this subject's 40 year career in sheet metal work, he had 13 adjusted years of total fibreglass exposure (three years high level, 10 years medium level), seven adjusted years of total asbestos exposure (six years high level, one year medium level), and one adjusted year of welding exposure.

Analysis of the exposure data showed that the adjusted years of medium and low exposure to fibreglass, and of high and medium exposure to asbestos, were too highly correlated to obtain separate risk estimates. Thus in the data analysis, total adjusted years of exposure to asbestos and total adjusted years of exposure to fibreglass were the principal exposure measures evaluated. We were, however, able to separately evaluate the effect of high intensity fibreglass exposure, as it was not highly correlated with the adjusted years of lower level exposures to fibreglass.

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