PROGRESSIVE MASSIVE FIBROSIS AND THE INFLUENCE OF BODY SHAPE

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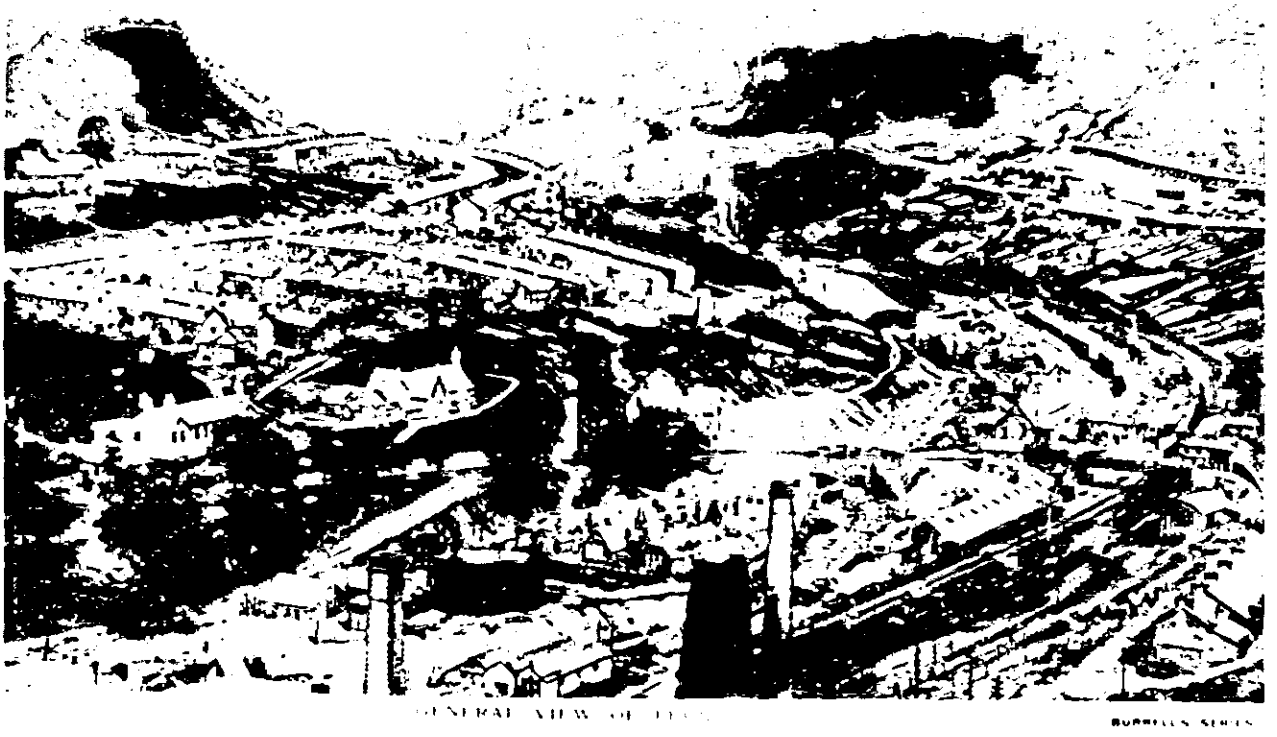
ABSTRACT

New evidence is presented of the increasing risk of attack with reduced body weight. Unlike the recently reported case control study by British Coal[1] there is only alight evidence of an association with Body Mass Index. For the first time it has been possible to use logistic regression models to assess the influence of individual-identifying information such as age, height, and weight in analysis of these prospective data. This has been achieved by employing enhanced computational methods to process the multi-punched records of Cochrane.[2]

Investigation of the cross-sectional data from the twenty-year follow-up of the men of the Rhondda Fach in South Wales[3] provides strong evidence of a lower mean weight of those with Progressive Massive Fibrosis (PMF) category A than in those unaffected. These findings were consistent across categories 2 and 3 background Simple Pneumoconiosis and nearly every age-group.

Little support was found for the hypothesis that pulmonary tuberculosis has a role in the development of PMF.

There is clear evidence that higher grades of Simple Pneumoconiosis are strongly associated with an increased probability of developing PMF.



INTRODUCTION

To facilitate study of factors relating to the development of Progressive Massive Fibrosis, in 1950 the Pneumoconiosis Research Unit of the British Medical Research Council initiated a survey of the smaller of the two Rhondda Valleys in mid Wales—the Rhondda Fach.[4] This total population epidemiological and intervention study was designed to investigate the importance of exogenous tuberculosis infection in the occurrence of PMF. Through extensive hospitalization, the environment was made as tuberculosis-free as possible. The adjacent Aberdare valley was used as an untreated reference.

At the time of initial survey, the eight towns of the Rhondda Fach were grouped around four collieries, which provided employment for most of the men. A view of the valley is given above. The total population was approximately 19,000; pneumoconiosis was commonplace amongst the numerous miners and ex-miners.

RESULTS FROM THE TWENTY-YEAR FOLLOW-UP

All Miners and Ex-miners

Comparison of mean Heights for all men alive at the start of study indicates little difference between radiographic categories 0, 1+2+3, and A+BC. Marked differences exist between the mean Weights of the PMF (A+BC) and SP groups (Figure 1).

Miners and Ex-miners with Category 2, 2A, and 3,3A

The men with PMF category A were sub-divided on the basis of their background SP category. The youngest age-group was omitted since few of these men have PMF. The oldest age-group was omitted to reduce the confounding influence of advanced age. Mean Weights of men with PMF category A and those of corresponding SP groups free from PMF at the time of the first survey are presented in Figure 2; all groups are large (> 20), except for the oldest 3A's. For nearly every age-group and within each background SP category, the mean Weights for those with category A are much lower than those for men without category A (Figure 2). The between-group differences in mean Height are small.

COMPARATIVE STUDY

As a test of the hypothesis that men who were initially free from Progressive Massive Fibrosis and who subsequently developed it (i.e. were attacked) do not differ appreciably in their physical characteristics from those not attacked, having allowed for their background category of simple pneumoconiosis, the prospective eight-year incidence of PMF in miners and ex-miners of the Rhondda Fach was compared with that of miners in the Aberdare valley.[2] The radiographs were read in chronological order independent of any other knowledge about the individual; the average of the radiographic categories of simple pneumoconiosis at time of survey and follow-up was used. This is henceforth referred to as "simple pneumoconiosis (SP) category."

The Data

Of the 1853 men originally seen in the surveys, 1226 men had complete data: 581 in the Rhondda Fach, and 645 in the Aberdare valley. A binary indicator of PMF status at time of follow-up was established—not attacked: 0, attacked: 1.

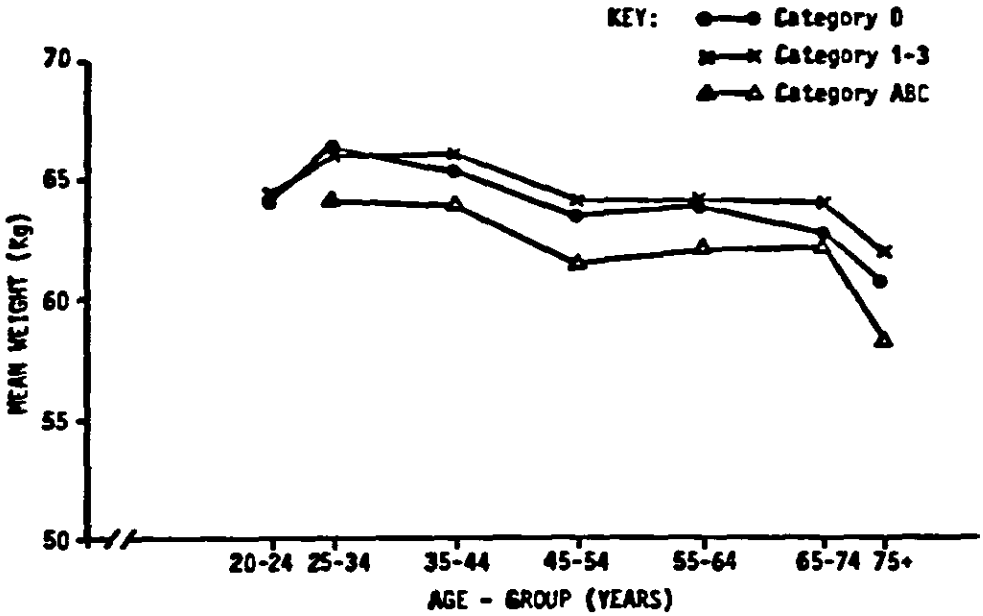


Figure 1. Mean weights by age-group for total initial Rhondda Fach population of miners and ex-miners.

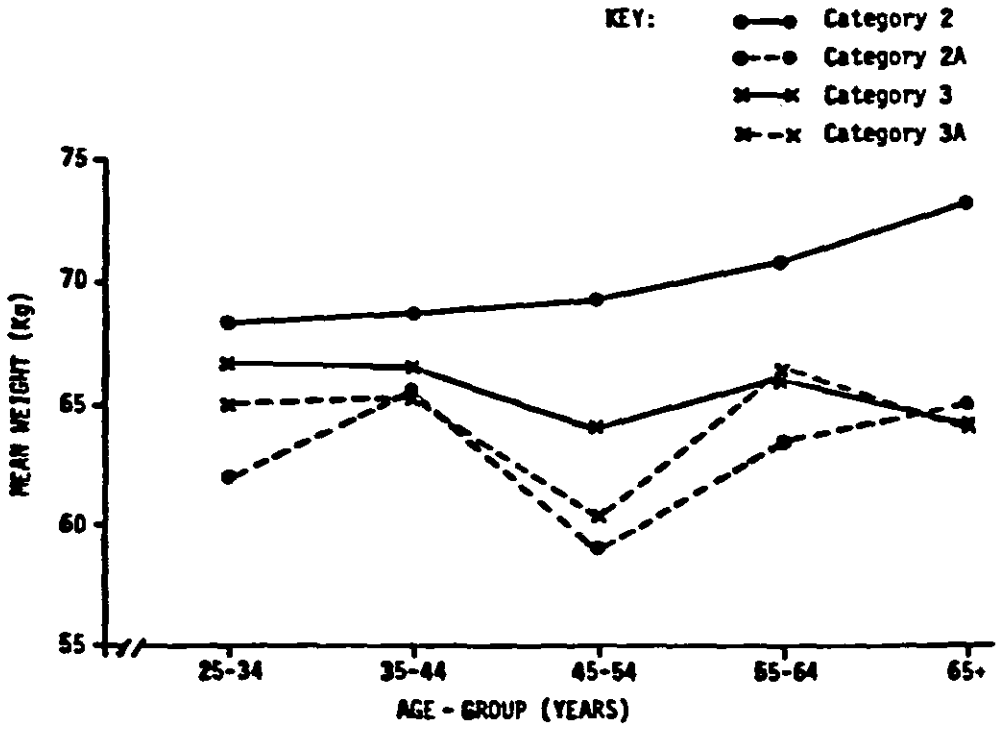


Figure 2. Mean weights by age-group for miners and ex-miners with category A PMF and those without: for background SP categories 2, 3

Simple Pneumoconiosis category was noted on a five-point scale: 1, 1.5, 2, 2.5, 3. The arithmetic mean of chest width at full inspiration and at full expiration was denoted Mean Chest Diameter (MCD). The Body Mass Index (BMI = Weight/Height^2) of Quetelet,[6] and the index of Body Type, i.e. Height^2/(10 * MCD) due to Rees and Eysenck[7] were calculated. Age at time of survey was grouped: 1 = 25–34, 2 = 35–44, 3 = 45–54, 4 = 55–64, 5 = 65+ years.

Preliminary Results

Table I displays the summary description of these data. The percentage and number attacked for each Valley, together with the number at risk, are shown in Table II.

Due to the similarity of corresponding cell entries for Weight and BMI amongst those attacked and those not attacked for each valley, tables were combined across the Valleys. Those attacked have consistently lower mean Weight than those who did not develop PMF (Table III); little such difference is found for the Body Mass Indices (Table IV).

Tabulation of percentage attacked by Age-group and Grouped Weight for SP category 2.5 suggested that the group of men weighing 60-69 Kg are at particular risk of attack by PMF. However, interpretation poses problems since there are a large number of cells which are either empty or are based on small numbers.

Logistic Regression Analysis

To further investigate the role of anthropometric indices in the probability of attack by PMF, logistic linear models were used to analyse the data.[8] The outcome variable being a binary indicator of attack by PMF, regression coefficients with a positive sign indicate variables associated with increased probability of attack, and conversely for a negative sign. Variables Age, SP category, and Valley were included as factors (dummy variables).

Model development. A variety of models were fitted in the development of a parsimonious model that adequately described the data. Models were compared by referring the change in Deviance, as terms are added or removed, to tables of Chi-squared. The Deviances do not have an "absolute" meaning, only differences between them may be safely treated as Chi-squared variables.[9] The importance of individual regressors was considered both in terms of the size of their contribution to the regression equation and their atandard Normal Deviate (SND).

To provide a baseline for assessing subsequent models, a null model (ϕ) containing only the Grand Mean (%GM) was fitted.

Main effect and first order interaction terms were added to form model I. The regression estimates for these and the other models fitted are summarized in Table V.

Table I

PMF Attack Study: Ranges, Means and Standard Deviations of Explanatory Variables

VARIABLE	VALLEY			
	Rhondda Fach	Aberdare	Rhondda Fach & Aberdare	
	Mean (SD)	Mean (SD)	Range	Mean (SD)
Valley	0	1	0 - 1	0.526
Proportion Attacked	0.189	0.127	-	0.157
SP category	2.419 (0.445)	1.869 (0.652)	1 - 3	2.130 (0.627)
Age group	2.336 (1.089)	2.386 (1.033)	1 - 5	2.362 (1.060)
Height (M)	1.679 (0.061)	1.679 (0.067)	1.29 - 1.94	1.679 (0.064)
Weight (Kg)	65.06 (8.94)	64.03 (9.16)	44.40 - 107.0	64.52 (9.07)
Body Mass Index	23.07 (2.87)	22.68 (2.66)	16.73 - 44.42	22.87 (2.76)
Mean Chest Diameter (cm)	27.30 (2.09)	27.40 (2.00)	20.0 - 38.5	27.35 (2.05)
Body Type	103.8 (9.2)	103.4 (9.2)	61.63 - 141.12	103.56 (9.20)

During model development, it was clear that SP terms were dominant, with their regression coefficients increasing monotonically with increasing category of SP. The contributions made by the Age terms were small and non-significant. There appeared to be little contribution made by the Valley term, either directly or through associated interaction terms.

Little evidence was found to reject the null hypothesis of a true value of zero for the coefficients for MCD, Body Type, or BMI (QI). Coefficients for Height, though consistently negative suggesting reduced probability of attack amongst taller men never attained anywhere near a conventional level of significance ($P > 0.2$). The contribution made by the Weight term was consistently negative, with generally strong evidence to reject the null hypothesis of a true value of zero. Thus indicating reduced probability of attack amongst heavier men.

Additional support for inclusion of the Weight term was provided by further models which omitted this term (VIII), included a Height term (IX), or replaced the Weight term by the BMI (X). The results from model IX indicated a true effect of Weight rather than an effect of overall 'size'. Little improvement in fit resulted from the adoption of any of these models.

Many other models were assessed. In particular, SP. Weight interaction terms were included but none of these models showed any useful improvement in fit.

Predictive Value of Model VII

Using the inverse logistic transform and three categories of Weight: Low (50 Kg), Medium (65 Kg), and High (80 Kg), the percentage probabilities of a miner being attacked by PMF were evaluated as a function of SP category. The estimates are shown in Table VI.

Table VII was formed by applying these predicted percentage attacks to the numbers at risk.

Although changes in Deviance indicate that the explanatory ability of the model VII incorporating Weight and SP is much greater than that of model VII using SP alone, further investigations indicate that the main contribution of Weight to the predictive ability of the model is due to its smoothing-out of perturbations in the predicted numbers corresponding to SP category 2. Simple pneumoconiosis is the dominant factor in the attack process.

Table II

Percentage and Number Attacked by PMF, and Number at Risk for Each Valley by Age-group and SP Category

KEY: in each cell) figures) arranged as) Percent attacked / Number attacked / Number at risk

Age Group	Rhondda Fach					Aberdare					Total				
	Category of Simple Pneumoconiosis					Category of Simple Pneumoconiosis					Category of Simple Pneumoconiosis				
	1	1.5	2	2.5	3	1	1.5	2	2.5	3	1	1.5	2	2.5	3
20 - 34	-	(0.0)	14.6	13.5	29.3	0.0	4.5	9.1	19.4	35.3	0.0	3.9	12.2	15.7	31.0
	0	0	6	7	12	0	2	3	6	6	0	2	9	13	18
	0	7	41	52	41	12	44	33	31	17	12	51	74	83	58
35 - 44	-	(0.0)	13.2	15.4	30.2	0.0	11.0	18.0	5.9	35.3	0.0	11.3	25.4	19.7	43.7
	0	0	9	12	19	0	8	9	2	12	0	8	18	14	31
	0	6	68	78	63	46	73	50	34	34	46	79	118	112	97
45 - 54	-	(0.0)	17.8	21.7	24.4	0.0	7.7	3.2	26.9	43.5	0.0	6.5	11.8	23.6	31.3
	0	0	8	10	10	0	4	1	7	10	0	4	9	17	20
	0	9	45	46	41	39	52	31	26	23	39	61	76	72	64
55 - 64	-	(0.0)	17.9	38.5	16.7	0.0	5.6	20.0	45.5	(11.1)	0.0	4.5	18.8	41.7	14.3
	0	0	5	5	2	0	1	4	5	1	0	1	9	10	3
	0	4	28	13	12	27	18	20	11	9	27	22	48	24	21
65 +	-	(0.0)	26.7	(0.0)	(25.0)	(0.0)	(20.0)	(0.0)	(20.0)	(0.0)	(0.0)	(12.5)	23.5	10.0	(20.0)
	0	0	4	0	1	0	1	0	1	0	0	1	4	1	1
	0	3	15	5	4	2	5	2	5	1	2	8	17	10	5

where ( ) indicates percentage based on number at risk < 10.

Table III

Weights (Kg) of Men—Combined Valleys

SP Category	Total Group Mean (SD) Range	Not attacked No.	Not attacked Mean	Attacked No.	Attacked Mean	No. at Risk
1	64.94 (10.02) 46.70 - 97.10	126	64.94	0	-	126
1.5	64.32 (9.46) 45.80 - 107.00	205	64.44	16	62.84	221
2	64.93 (9.35) 47.20 - 98.40	284	65.06	49	64.53	333
2.5	64.76 (9.14) 44.40 - 106.10	246	65.50	55	61.47	301
3	63.55 (7.59) 46.70 - 87.10	172	64.09	73	62.26	245

Linear regression analyses for men attacked by PMF provide some slight evidence of a trend of decreasing average Weight with increasing average category of SP. By contrast, the evidence for the Body Mass Index is somewhat stronger.

DISCUSSION

Strong evidence of the importance of weight in the aetiology of PMF category A is provided by the cross-sectional data shown in Figures 1, 2. However, by their very nature, it is not possible to make inferences about the prognostic value of SP or Weight in determining the probability of a man being attacked by PMF.

There is a strong influence of SP category on the probability of attack by PMF throughout all the models considered. The (independent) influence of reduced Weight on the probability of attack is also obvious. There was however no evidence of B relation between probability of attack and Age. This is similar to the finding of Cochrane.[2] The logiatic regression analyses provide strong evidence of increasing risk of attack with reduced weight. There is only slight evidence of an influence of the Body Mass Index.

The consistent but slight trend of decreasing Weight (BMI) with increasing Lung Dust content as found in the final regression analyses supports the idea that lighter men are preferentially selected for attack.

Model VII provided the best parsimonious description of the data; it was able to predict well the number of men attacked.

In terms of the hypothesis of Cochrane et al[4] that pulmonary tuberculosis has a role in the development of PMF, the consistently small and non-significant coefficient for Valley and the corresponding terms for interaction with SP suggest little support for this idea. Cochrane[2] noted that despite the efforts of the field experiment, there may have been no real difference in the level infection in the two valleys. However, his further work comparing the attack rate during different periods following the initial survey was regarded as undermining the 'tuberculosis hypothesis'.

CONCLUSION

Further study of the prognostic value of these indices, in particularly weight, would be useful. In view of the great variability of the attack rate of PMF in different coal-fields, consideration pf datasets based on other types (ranks) of coal would be informative.

If the importance of weight as a predictive factor in attack by PMF can be validated, the use of a weight-monitoring procedure might lead to further improvements in the health of miners.

Table IV

Body Mass Indices of Men—Combined Valleys

SP Category	Total Group Mean (SD) Range	Not attacked No.	Not attacked Mean	Attacked No.	Attacked Mean	No. at Risk
1	23.26 (3.00) 17.80 - 34.39	126	23.26	0	-	126
1.5	22.80 (2.86) 17.37 - 36.60	205	22.85	16	22.23	221
2	23.01 (2.89) 17.32 - 33.86	284	23.00	49	23.05	333
2.5	22.91 (2.89) 16.73 - 44.42	246	22.94	55	22.80	301
3	22.48 (2.11) 17.88 - 30.32	172	22.68	73	22.02	245

Table V

Summary of Models

Regression coefficient
Standard error
SND

Model	Grand Mean	Val1sq(2)	Age(2)	Age(3)	Age(4)	Age(5)	Staple Pronouncements category 1.5	2	2.5	3	Height	Weight	Mean Chest Diameter	Body Type	Quetelet Index	Val(2) (SP = 1.5)	Val(2) (SP = 2)	Val(2) (SP = 2.5)	Val(2) Height	Val(2) Weight	Variance
0	-1.670 0.01838																				1868 on 1225 df
1	-4.626 5.111 -0.88	-0.3832 0.517 -0.83	0.1958 0.2188 0.53	0.09698 0.2616 0.38	0.2297 0.3045 0.75	0.1736 0.4711 0.37	0.2358 0.897 0.83	2.192 3.878 1.85	2.322 3.878 1.88	2.883 3.889 2.84	-1.916 2.857 -0.83	-0.02855 0.01784 -1.71	0.887736 0.85274 0.15			5.968 0.819 0.76	-0.5444 0.4392 -1.26	-0.1768 0.4294 -0.41	0.5131 3.834 0.28	-0.8862231 0.02449 -0.25	959.6 on 1268 df
II	-0.319 0.823	0.3293 0.2986 1.13	0.08821 0.2177 0.85	0.1811 0.2356 0.77	0.3482 0.2866 1.15	0.2325 0.4622 0.58	0.3528 0.831 0.84	2.209 3.929 1.86	2.412 3.928 1.88	2.983 3.911 2.84				-73.82 89.19 -0.82		5.852 0.161 0.72	-0.6231 0.4355 -1.43	-0.2012 0.4243 -0.47			968.3 on 1212 df
III	-0.368 0.571	0.2635 0.2929 0.90					0.2747 0.928 0.83	2.195 3.873 1.86	2.388 3.873 1.88	2.868 3.864 2.84	-1.981 1.511 -1.26	-0.02198 0.01436 -1.26	0.884188 0.85261 0.88			5.936 0.856 0.76	-0.5661 0.4372 -1.29	-0.1835 0.4262 -0.43			968.3 on 1214 df
IV	-6.838 6.876	0.3822 0.2913 1.88					0.2565 9.825 0.83	2.208 3.918 1.86	2.318 3.918 1.87	2.873 3.881 2.82			-0.85614 0.84857 -1.28		-0.81882 0.83798 -0.56	5.982 0.146 0.72	-0.6021 0.4355 -1.38	-0.1886 0.4244 -0.43			967.4 on 1215 df
V	-6.962 3.836	0.2598 0.2923 0.89					0.2157 0.944 0.82	2.172 3.879 1.85	2.286 3.879 1.88	2.837 3.878 2.83		-0.02915 0.01813 -2.88				5.922 0.871 0.73	-0.5738 0.4385 -1.31	-0.1954 0.4248 -0.38			961.8 on 1216 df
VI	-6.756 3.928	0.08186 0.1777 0.47					5.998 3.871 1.55	6.845 3.861 1.77	7.185 3.861 1.86	7.724 3.867 2.88		-0.02933 0.01891 -2.91									968.3 on 1219 df
VII	-6.844 3.911						5.979 3.876 1.54	6.797 3.864 1.76	7.853 3.864 1.83	7.878 3.863 1.98		-0.02988 0.01895 -2.97									968.5 on 1220 df
VIII	-6.566 3.946						6.015 3.926 1.53	6.888 3.919 1.76	7.868 3.919 1.88	7.986 3.916 1.99											977.8 on 1221 df
IX	-6.948 4.439						5.999 3.863 1.55	6.815 3.857 1.77	7.872 3.857 1.83	7.898 3.857 2.88	-1.855 1.589 -1.23	-0.02288 0.01171 -1.88									967.8 on 1218 df
X	-7.496 3.972						5.998 3.917 1.53	6.798 3.986 1.76	7.849 3.986 1.88	7.874 3.986 1.96					-0.86613 0.83232 -1.83						975.7 on 1220 df

Table VI

The Percentage Probability of Attack Using Model VII

Category of SP	Category of Weight			% Predicted on Overall Weight
	Low	Medium	High	
1	0.03	0.02	0.01	0.02
1.5	10.4	6.9	4.5	7.2
2	20.8	14.4	9.7	14.7
2.5	25.3	17.8	12.2	18.3
3	38.6	28.7	20.5	29.8

Table VII

The Number of Attacks Predicted by Model VII

Category of SP	Number of men at risk	Number of attacks	
		Observed	Predicted
1	126	0	0.024
1.5	221	16	15.991
2	331	49	49.006
2.5	301	55	55.000
3	245	73	73.002

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SUGGESTIONS FOR REVISION OF THE ILO CRITERIA FOR ABESTOSIS BASED ON SCREENING 10,000 WORKERS

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ABSTRACT

In a screening program from 1985-87, 4,572 American shipyard and construction workers were present and were questioned about abnormalities while posteroanterior (PA) and lateral chest radiographs were interpreted for asbestosis using ILO International Classification of Radiographs of Pneumoconiosis 1980. Technically, unsatisfactory PA films were of 3 types: 1) underexposed due usually to body build or obesity, 2) overexposed due to body build or loss of lung substance due to cigarette smoking (CS) and 3) underinflated with diaphragms above the 9th intercostal space. A repeat PA film was needed in 7% of workers to have 99% films of technical quality 1. The profusion of irregular opacities in underexposed and/or underinflated films may appear to be 1/0 while ideal films on the same subject are 0/0 or 0/1. Overexposed films because of reduced lung parenchyma in smokers may be read as 0/0 when they are 1/0 or 1/1. Irregular opacities should be defined as: 1) distinct from vascular shadows and breast shadows, frequently accompanied by septal lines and present in 2 or more zones. Obliteration of one costophrenic angle with diffuse thickening is a frequent sequalae of pleural effusions from asbestosis which occur in 1–2% of subjects. Pleural fibrosis may subsequently extend into lung. Otherwise, unilateral pleural disease is often due to trauma, pleurisy, fractured ribs or infection with effusion.

INTRODUCTION

The ILO classification for chest radiographs[1] has provided a thoughtful and rational method for quantifying pneumoconiosis. Some critics are disturbed by its success which has broadened its purpose, which is to provide internationally agreed criteria for description of radiographs for epidemiological studies. Clearly it has been adopted for clinical diagnosis of asbestosis, coalworkers pneumoconiosis and silicosis in individual patients, for epidemiological studies of occupationally exposed and bystander populations,[2] hospitalized patients[3] and for compensation and litigation. Inter observer variation remains a problem. Intra observer variation has been steadily reduced. Some users have by oversight or deliberately misinterpreted the scale by leaving out the diagnostic boundry of 1/0 profusion of irregular opacities for asbestosis.[4] Others fault the classification for less than perfect correlation with pathology particularly the microscopic recognition of fibrosis.[5] On the whole, its defenders exceed its critics although no one would argue seriously that it could not be improved.

With improvements in mind 30 scientists convened in mid May in Athens to share concerns about the approaching decade birthday of the 1980 revision of a classification scheme which originated in 1916. The workshop was sponsored by the Collegium Ramazzini, an international organization, which is dedicated to the observational and descriptive principles of Bernardo Ramazzini, the father of occupational health. Irving J. Selikoff of Mount Sinai Medical School in New York, President of the Collegium, convened the meeting and described the evolution of a classification for the major dust diseases: asbestosis, coalworkers pneumoconiosis and silicosis. The initial classification, developed about 1916, was adopted by the First International Conference of Experts on Pneumoconiosis in Johannesburg under ILO auspices. It was perfected at the 3rd International Conference in Sidney in 1950. The so-called Geneva classification was adopted there in 1958 with revisions and additions made in 1968. In 1971 the UICC scheme was integrated with the ILO. The 1980 ILO revision provided a new set of 22 standard radiographs selected as mid-category profusion of small opacities to facilitate the classification of radiographs for epidemiology and disease progress.

METHODS AND RESULTS

Currently over 21.5% of workers screened for asbestosis are categorized as 1/0 or 1/1, only 5.1% are 1/2 or greater while 73.4% are negative, Table I. Because the critical border is 0/1 to 1/0 major effort was directed at obtaining ideal films, minimizing confounding by over or under exposure and competing shadows contributed by breast, pectoral girdle muscle and fat is important. Under inflation of lungs so that diaphragms are not below the 9th intercostal space can contribute

Table I

Frequency of Asbestosis (ILO Profusion Categories) in 4572 Boilermakers and Pipefitters in the United States 1986–1988

ILO :	Freq.	Percent	Cum.%	AGE	LATENCY
0/0 :	2956	64.65	64.65	50.8 ± 11.9	25.4 ± 10.9
0/1 :	399	8.73	73.38	56.6 ± 10.5	29.3 ± 11.2
1/0 :	487	10.65	84.03	59.4 ± 9.8	30.9 ± 10.2
1/1 :	498	10.89	94.93	61.8 ± 9.4	32.9 ± 9.4
1/2 :	94	2.06	96.98	63.5 ± 9.9	33.1 ± 9.9
2/1 :	53	1.16	98.14	65.9 ± 10.7	31.1 ± 12.5
2/2 :	61	1.33	99.48	66.3 ± 9.5	34.6 ± 11.0
2/3 :	14	0.31	99.78	64.7 ± 12.4	28.9 ± 12.1
3/1 :	4	0.09	99.87	59.0 ± 3.5	33.3 ± 12.1
3/2 :	3	0.07	99.93	66.7 ± 13.3	30.0 ± 7.9
3/3 :	3	0.07	100.00	67.0 ± 16.0	30.7 ± 15.0
	4572	100.00		mean 54.1 ± 12.2	mean 27.5 ± 11.1

to a spurious impression 1/0. Such hypo-inflation occurs in 12% of PA radiographs unless technicians instruct subjects to breathe out and take in their "absolutely biggest possible breath." When such instructions are insisted upon only 2.1% of radiographs have diaphragms not below the 9th intercostal space.

DISCUSSION

The instructions for readers should be improved to reduce ambiguity and more emphasis should be given to production of new standard chest radiographs which are of film quality 1, ideal for ILO interpretation rather than marginal films. There are serious questions as to whether films below quality 1 should or even can be interpreted? It may be best to make the decision as to pneumoconiosis the summary, not as it is now, the readers initial decision. Can a single radiograph be considered adequate for detecting and quantifying pleural as well as parenchymal asbestosis. Adding a lateral chest radiograph would contribute dorsal and ventral pleural mapping, survey the area behind the heart and assist in classifying emphysema (see below) by showing the anteroposterior slope of the diaphragms, at a small increase in cost. Optional density of chest structures especially the lung fields and full inflation of the lungs as judged by diaphram descent to at least below the right 9th intercostal space are essential.

How can the precision of the basic decision between a positive and negative radiograph, especially for asbestosis, the 0/1 1/0 decision be improved. The concrete addition to this decision making would be employment of "boundry films," films which define the division between steps of classification. It would be a testable strategy. A detailed protocol for developing a set of single or paired radiographs on both sides of boundry (0, 1, 2 and 3) has been described for the classification by Michael Jacobson of Edinburgh's Institute of Occupational Medicine. This would add 3 or 6 films to the standard 18 which define the mid categories. "The hypothesis is that it would be easier to classify films within categories that are defined by lower and upper limits of profusion of small opacities, than it is to match appearances defined as typical of a particular category." Ideal boundry films would represent an even 50:50 split of interpretation on both sides by a large number of experienced readers. The practicality of this strategy could be tested by determining if it reduced inter-reader variability, intra-reader variability and correlated with dust exposure.

Dr. G.K. Sluis-Cramer (South Africa) has questioned whether the size of opacities in asbestosis s/s vs s/t or t/t predict population differences for non radiograph measures particularly for pulmonary function. If not, is it important to record size? There is a further question as to whether large irregular opacities, u/u, are ever seen in asbestosis.

Considerable discussion concerned the asbestos pleural dilemma. Would more detailed and comprehensive description be useful in the ILO reading or is the 3 fold separation into diffuse pleural, plaques and calcification sufficient. Clearly analysis using the present scale vs an experimental extended

scale of one or two large populations correlating pulmonary functional impairment, clinical findings and these pleural changes would answer this question. At least two large cohorts of asbestos exposed workers have been studied in the United States by Selikoff (New York) and by Kilburn (Los Angeles) which could be analyzed to answer such questions. Perhaps of greatest importance is whether to include obliteration of both costophrenic angles plus diffuse thickening as a most advanced pleural category. Should diffuse pleural thickening be divided into upper, mid and lower zones plus costophrenic angles? Further research on large collected cohorts of asbestos exposed workers as proposed would answer this question as well as establishing the predictive value of pleural asbestos disease. The ILO classification has already been employed to describe the radiologic progression of asbestosis.[6] Similarly it should be quite simple to settle the film quality issue: as to which quality produces unacceptable variability in interpretation and thus determine whether films below quality 1 can be interpretated without unreasonable variability.

The listing of nonpneumoconiotic observations, the "other disease" category, is useful but needs updating to incorporate current findings. Perhaps an intuitive or logical organization would help rather than the present alphabetical array. In addition to bullae and definite emphysema, step wise incremental emphysema criteria to include low flat diaphragms on PA film, low flat diaphragms on the lateral film, wide (beyond 2.5 cm) retrosternal clear zone, and radiolucency or hypovascular zones[7] which has already been field tested in cotton textile workers with byssinosis are suggested.[8] Other useful additions would include 1) dilated aorta, 2) surgical clips, 3) staples and sutures, and 4) bronchiectasis. Calcified primary complex could be added to tuberculosis.

Technical advances such as computerized tomography greatly increase the cost of radiographic diagnosis with a modest yield epidemiologically. Nevertheless high resolution (extended scale) CT scans may be well justified in individual cases, for example, in differentiating pseudotumors or linear pleuroparenchymal infiltrations. Even computerized interpretation should be explored because it has the potential to eliminate intra-reader and inter-reader variability.

Research should be directed to expand the range and or test the predictive capacity of the ILO classification for chest X-rays. Time will help sort out these needs. We need to consider ways to extend the usefulness of this venerable classification scheme. It is time to examine the issues which need to be faced before 1991, the proposed revision date.

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PULMONARY FUNCTIONAL IMPAIRMENT FROM YEARS OF ARC WELDING

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ABSTRACT

Construction welders without shipyard exposure had vital capacity and flows measured by spirometry, chest radiographs classified for asbestosis using ILO criteria and were administered an occupational and respiratory questionnaire. Data on workers with asbestosis (ILO profusion 1/0 or greater) was deleted from analysis. The 226 men without asbestosis were 45 years old, 69.2 inches tall and had welded for 21.3 years (means). Expressed as mean percent predicted after adjusting for height, age and years of smoking mean FVC was 101%, FEV_1 was 98.7%, FEF_{25-75} was 94.0%, FEF_{75-85} was 91.8% and TGV was 104.2%. The 43 nonsmokers had similar reductions in flows. The regression coefficient for years of welding for FVC was −.0031, for FEV_1 was −.0035, for midflow (FEF_{25-75}) was −.0080, all significant ($p < 0.05$), and for FEF_{75-85} was −.0056 not significant. Calculated from regression equations 40 years of welding would reduce FVC to 95.2%, FEV_1 to 92.2%, midflow to 79.2% and flow at low lung volume to 81.3%. Chronic exposure to welding gases and fumes reduces flows in small airways in welders without asbestosis or shipyard exposures.

INTRODUCTION

Metal welding generates ozone, nitrogen oxides and metal oxide aerosols which cause respiratory tract irritation, cough and excessive phlegm and impair pulmonary function in welders.[1] Arc welders in shipyards have reductions in vital capacity and FEV_1[2–6] which have been considered permanent. Because many studies were done when 80 to 90% of the welders were also cigarette smokers, the degree of interdependence of effects of welding exposure and smoking is unclear. It follows that the chronic functional impairment in those welders who had never smoked is also poorly defined. Also earlier studies ignored the effects of asbestosis on pulmonary function.[7,8] Thus, this study of 226 welders, 43 of whom had never smoked, explored the chronic effects of welding on pulmonary function and interactions with cigarette smoking. We examined 226 welders, employed largely at construction sites and power plants, who had never worked in shipyards and had no evidence of asbestosis on chest radiographs. Pulmonary functions were expressed as percentage of predicted based on individual comparisons corrected for height, age and for cigarette smoking[9] to assess the contribution to impairment of years of welding.

METHODS

226 male welders from two midwestern locals of the International Brotherhood of Boilermakers, Iron Shipbuilders, Blacksmiths, Forgers and Helpers were studied during 1987 for asbestosis by questionnaires for occupational exposures and respiratory disease and for symptoms, a chest physical examination, posteroanterior and lateral chest radiographs and pulmonary function measurements. Informed consent was obtained after the nature of all procedures was fully explained to each welder. The questionnaire was adapted from DLD-78[10] as used in the study of the Michigan population[11] and inquired about chronic bronchitis, asthma, wheezing and shortness of breath as well as past respiratory and cardiovascular disease symptoms and illnesses. It was administered by trained interviewers. The mobility of welders doing construction jobs, variation in composition of base metal and rod, type of welding, and the uniqueness of each site for their lifetime made exposure measurement impossible. As an exposure surrogate, years of welding was used in analyses.

Spirometry was measured on Saturday or Sunday, at least 16 hours after a workshift on Ohio Rolling Seal spirometers which were repeatedly calibrated during the study with a 3 L syringe. Spirometry was repeated until 2 agreed within 5% with subjects standing, wearing a noseclip, and followed American Thoracic Society[12] criteria including that the origin of FEV, was established by back extrapolation. The best curve was digitized and FVC, FEV_1, FEF_{25-75}, and FEF_{75-85} and for each individual value as percentage predicted was adjusted for height, age and years of cigarette smoking. Values were compared to the Michigan male population sample in smoking specific groups.[9] Posteroanterior and lateral chest radiographs were made at full inflation. They were examined for evidence of asbestosis of lung or pleura using ILO classification criteria.[13] The 65 welders with signs of asbestosis of the 291 originally studied

were not analyzed further.

Statistical testing was performed in a Hewlett-Packard 9816 computer using the Hewlett-Packard library of statistical programs including calculation of confidence intervals, regression analysis and equation development. Analysis of variance using an SAS statistical program in an IBM computer was used to compare function values for caucasian male welders to the entire sample of Michigan men, not just those selected as normals for modelling pulmonary function.[9] What this means is that we restored to the group used for modelling "normal" predictive pulmonary function all subjects who had been excluded because of clinical abnormalities. Thus, the comparison group included all adult males studied in Michigan. A P value of < 0.05 was used to demonstrate significance.

RESULTS

The 226 electric arc welders were 45.0 years old, and 175.8 cm. tall (mean values) and they had welded an average of 21.3 years, Table I. Only 19.0% had never smoked cigarettes. Nearly 20% had chronic bronchitis diagnosed by phlegm production for 3 months per year for at least two years and 11.3% had a history of asthma. Nonsmokers prevalence of chronic bronchitis was 23% and of asthma was 13%; thus they were as symptomatic as smokers for these disorders. Pulmonary functions for the 226 men are presented as group means and percentages of predicted, adjusted for effects of height, age and for years of cigarette smoking,[9] Table I. There were no years of smoking adjustment for FVC, for FEV_1 it was -0.0094 for years of smoking for log FEF_{25-75} it was -0.0052 years of smoking and for log FEF_{75-85} it was 0.0112 years of smoking. The mean vital capacity of the group was 101.0%, FEV_1 was 98.7%, FEF_{25-75} was 94.0%, FEF_{75-85} was 91.8% and TGV was 104.2%. Although nonsmokers had slightly better function than the whole group, after adjustment for smoking only the difference in FEF_{75-85} was statistically significant, Table I.

To test for chronic effects of welding the critical analysis was to correlate years of welding, as the independent variable with the percentage of predicted values for FVC, FEV_1, FEF_{25-75}, and FEF_{75-85}, as dependent variables by regression analysis, Table II. This was done after each individual observation of pulmonary function was adjusted for height, age and for years of cigarette smoking. For the 226 welders the regression coefficients for years welding were -0.0031 for FVC, Figure 1a; -0.0035 for FEV_1, Figure 1b; -0.0080 for FEF_{25-75}, Figure 1c; and -.0058 for FEF_{75-85}, Figure 1d. All were significant ($P < 0.05$) except for FEF_{75-85}. Calculating the effects of 40 years of welding on percent predicted using the regression equations for all welders reduced FVC to 95.2%, FEV_1 to 92.2%, FEF_{25-75} to 79.2% and FEF_{75-85} to 81.3%, Table II. The 43 nonsmokers had larger regression coefficients for years of welding on mid and terminal flows. Thus for FEF_{25-75} the regression coefficient was -.0095 and it was -.0171 for FEF_{75-85}. Both were significant ($P < 0.05$). The smaller coefficients for FVC of -.0013 and for FEV_1 of -.0036 were not significant. (Figure 2) Thus, in nonsmokers the significant effects of duration of welding were limited to small airways. Perhaps the implications of these relationships for subjects who weld but have never smoked are best shown by calculation of the reduction in function expected from 40 years of welding using regression equations. Thus without exposure to cigarette smoke 40 years of welding would not reduce vital capacity but FEV_1 would fall to 93.3% of predicted. Midflow would fall to 77.6% of predicted and terminal flow would drop to 62.0%.

DISCUSSION

Chronic exposure to arc welding gases and fumes reduced flows including FEV_1. Such airway obstruction moderately impairs function of welders after 20 to 40 years. Cigarette smoking welders showed additional adverse effects which exceed standard adjustments for duration of smoking.[9] Thus, welders who smoked showed more than the sum of functional impairments from welding and from cigarette smoking. Initially, welding fumes and gases reduce flows in small airways as seen unequivocally in the 43 nonsmokers. When cigarette smoking is added vital capacity and FEV_1 are also reduced to an extent beyond the standard adjustment for smoking. If the effects of the cigarette smoke and welding smoke aerosols were equal and additive then 40 years of welding alone would resemble 20 years of welding in cigarette smokers. That the 40 year calculated effect of welding alone is to reduce flows including FEV_1 but not to reduce FVC suggests that the effects of welding and cigarette smoking are similar. Predictive regression equations for pulmonary function in these welders account stepwise for height, age and duration of cigarette smoking (years) by multiple linear regression, and isolate the effect of welding exposure. Horizontal lines would reflect decrements from occupational exposure to welding. These men all had some exposure to asbestos in insulating materials, gloves and blankets. Excluding those with signs of pulmonary asbestosis is a major step in removing its effects and goes beyond earlier studies of welders[2-6] but does not guarantee the absence of subtle effects.[7]

The additional decrement which occurred in smokers appears to be best explained by the synergism between the effects of particles, gases and chemicals adsorbed on particles in two complex aerosols.[14] One, cigarette smoke, is an exceedingly complex mixture of tobacco distillates and combustion products characterized as particles of complex hydrocarbons or tar containing over 2,500 chemical species and a mixture of gases including carbon monoxide, nitrogen oxides and aldehydes.[15] Welding fumes are oxides of metals with additives from flux, rod coating and surface treatments and gases, largely ozone and nitrogen oxides.[16] The particles from both cigarette combustion[17] and the welding arc[18] are poorly digestible by pulmonary macrophages, damage lining cells of distal airways and connective tissue during phagocytosis and disposal.[18] Thus, cigarette smoke, and welding of asbestos produce goblet cell metaplasia and mucous obstruction[19] in distal terminal bronchioles.[7] This is followed by peribronchiolar cuffs of cells and fibroblast proliferation, collagen production, and scarring with luminal narrowing and obstruction of terminal and respiratory bronchioles.[19,20,21] Functional loss of small airways by anatomic obstruction and obliteration removes respiratory units from ventilation and eventually reduces vital capacity. Although one might speculate that the welding particles would also stimulate digestion of lung at the alveolar level to destroy alveolar walls

Table I

Pulmonary Functions, Means (m), Standard Deviations (sd), and Percentage Predicted in 226 Midwestern Welders without Evidence of Asbestosis

	ALL WELDERS m	sd	NONSMOKING WELDERS m	sd
Number	226		43	
Age - years	45.0	± 10.4	44.9	± 10.7
Ht - cm.	175.8	± 6.5	175.5	± 8.9
Welding years	21.3	± 10.0	20.5	± 10.4
Smoking years	21.3	± 13.9	0	
Cig/day	25.1	± 18.9	0	
Asbestos exp. yrs.	22.6	± 10.8	19.9	± 9.8
Ch. Bronchitis %	19.9		23.3	
Asthma history %	11.3		13.2	
FVC L.	4.93	± .87	5.03	± .89
% pred.	101.0	± 14.6	103.9	± 15.6
FEV_1 L.	3.77	± .82	3.98	± .79
% pred.	98.7	± 16.6	100.4	± 15.8
FEF_{25-75} L/sec.	3.20	± 1.37	3.58	± 1.28
% pred.	94.0	± 34.3	96.2	± 28.3
FEF_{75-85} L/sec.	0.84	± .52	1.07	± .61 *
% pred.	91.8	± 46.7	95.6	± 45.3
TGV	7.60	± 1.05	7.19	± 1.10
% pred.	104.2	± 14.8	101.1	± 15.9

* $P < 0.05$

Table II

Correlations, R^2 and Coefficients from Regression Analysis of Effects of Years of Welding on Pulmonary Function as Percent Predicted of 226 Midwestern Construction Welders

Percent Predicted	ALL WELDERS Correlation	r_2	Regression Coefficient	NEVER SMOKED Correlations	r_2	Regression Coefficient
Number	226			43		
FVC	-.2096	.0439	-.0031 *	-.0841	.0071	-.0013
FEV_1	-.2114	.0447	-.0035 *	-.2351	.0553	-.0036
FEF_{25-75}	-.2326	.0541	-.0080 *	-.3479	.1211	-.0095 *
FEF_{75-85}	-.1182	.0140	-.0056	-.3929	.1544	-.0171 *

* P<0.05

Percent pred. FVC = 107.50 - .0031 x (40) years welding = 95.2

Percent pred. FEV_1 = 106.25 - .0035 x (40) years welding = 92.2

Percent pred. FEF_{25-75} = 111.20 - .0080 x (40) years welding = 79.2

Percent pred. FEF_{75-85} = 103.68 - .0056 x (40) years welding = 81.3

Figure 1a–1d. Regression equations for all welders and effect of 40 years of welding on percentage predicted.

Percent pred. FVC = 106.44 - .0013 x (40) years welding = 101.4

Percent pred. FEV_1 = 107.74 - .0036 x (40) years welding = 93.3

Percent pred. FEF_{25-75} = 115.57 - .0095 x (40) years welding = 77.6

Percent pred. FEF_{75-85} = 130.63 - .0171 x (40) years welding = 62.0

Figure 2. Regression equations for non-smoking welders and effect of 40 years of welding on percentage predicted.

and increase total lung capacity as does cigarette smoke, the welders have no significant increase in total lung capacity after adjusting for the effects of cigarette smoking.[22]

Earlier studies of the effects of welding were of cigarette smoking shipyard welders who are frequently exposed, in addition to asbestos in insulating materials, to silica from sand blasting, paint fumes, metal particles, and other toxins in closed spaces of hulls or compartments. In shipyard welders Hunnicutt in 1968[2] anticipated the smoking-welders synergism by showing that smokers but not nonsmokers had reduced peak flow, FEV_1 and FEF_{25-75}. In another study the shipyard effect was amply underscored by finding that 61 shipyard welders and 63 shipyard pipefitters had reduced FVC, FEV_1, TLC, RV and DLCO compared to "new pipefittters" and to current standard populations.[3] In engineering shop welders in Finland only diffusing capacity was reduced,[4] again emphasizing the shipyard effect. After lumping ex-smokers and nonsmokers, a Swedish shipyard study showed welders had significantly lower FEV_1, FVC and TLC than nonwelders.[5] A Newcastle, Great Britain shipyard study of 209 welders and 109 controls matched 2:1 for age, height, smoking habits, residence and social class showed that in both smokers and nonsmokers FVC and FEV_1 were significantly lower than in controls and DLCO was significantly lower in nonsmokers.[6] The most comparable study to this one found significant reductions of FVC, FEV_1, FEF_{25-85} and DLCO in 72 nonsmoking shipyard welders.[23]

This study has eliminated confounding exposures from work in shipyards,[8] ruled out asbestosis recognized by irregular opacities on chest radiographs and adjusted for the expected effects of years of cigarette smoking. Thus, for the first time arc welding gases and fumes can confidently be regarded as causing airway obstruction. However, the magnitude of decrements is smaller than that from cigarette smoke alone[9] or from asbestosis.[8] Welding fumes and gases, especially particles to which are adsorbed cytotoxic molecules, appear to elicit cellular and tissue reactions which narrow and distort small airways. Studies of lung pathology in welders or experimental studies of chronic exposure are needed to elicit the mechanisms.

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