LUNG FUNCTION DECLINE IN MAN-MADE FIBRE TEXTILE WORKERS: RELATIONSHIP TO PAST COTTON EXPOSURE

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Abstract

A five year prospective study, documenting respiratory symptoms, measurement of lung function and assessment of airborne dust concentrations, was performed in eight Lancashire textile mills. Six mills processed cotton and two mills processed man-made fibre. Provisional analysis of this cohort (Fletcher et al, 1999) demonstrated overall low levels of lung function decline, but with higher levels of decline witnessed in man-made fibre workers (-17mls/yr) and waste cotton workers (-21mls/yr) compared to blend (+4mls/yr) and medium cotton exposed workers (-5mls/yr). The reason for this could relate to a direct effect of man-made fibre dust, or be due to the fact that some workers appear to move from the cotton textile industry to man-made fibre manufacture. To study this in detail we have performed a subsidiary analysis of this data investigating lung function decline in man-made fibre workers only (n=652). Previous cotton dust exposure and current measures of man-made fibre exposure are included in the data set as separate predictor variables and its effect on the measured lung function decline compared to cotton naive workers. A random effects regression model was fitted to the data, firstly overall, then by ethnic group and age group (<35yrs and >35yrs). The statistical analysis demonstrated no association between lung function decline and measures of both past cotton dust exposure and current man-made fibre exposure. It appears that susceptible workers moving from cotton to man-made are not responsible for the excess in lung function decline demonstrated within this exposure group. Consistent statistically significant predictors of lung function decline were age and sex, which have previously been documented.

Introduction

Until recently, longitudinal studies have suggested a relationship between duration of exposure and accelerated lung function decline, with an excess in mean annual lung function decline detected in cotton workers compared with controls (Berry 1973, Zuskin and Valic 1975, Beck 1982, Christiani 1990). However, a large scale longitudinal study performed in the USA was unable to reproduce these findings and reported a greater lung function decline in synthetic workers compared with cotton exposed workers, which could not be explained by smoking or duration of employment (Glindmeyer, 1991). The authors postulated that within the manmade fibre exposed population the greater level of lung function decline could have been related to a unrecognised, unmeasured causative agent, or alternatively, due to selection bias of this population.

We reported a similar finding in a large longitudinal study recently performed in the Lancashire textile industry (Fletcher et al, 1999). Eight mills were included in the study, two processing waste cotton, two medium, two blend mills and two manmade fibre mills. The study documented rates of lung loss, in each of the fibre groups, which are consistent with the rates of lung function decline observed in prospective studies of normal populations. However, the pattern of lung function decline varied across each exposure group. Greater declines were found in those operatives processing man-made fibre and waste cotton. The reason for this is unclear and it may be multi factorial. Within the man-made fibre group, over half of the population have had past exposure to cotton. The basal measures of lung function were also statistically lower in this group compared to those in the waste exposed group. It is possible that susceptible workers are self-selecting from the more acutely toxic environment of cotton mills, and the excess decline is spurious because of a selected "unhealthy" worker concentration within the man-made fibre sector. These at risk workers are then postulated to continue to show larger lung function decline than the cotton survivors or controls never exposed to cotton dust. Alternatively as witnessed in similar epidemiological studies there may be a chronic effect of man-made fibre exposure on lung function.

To study this in detail we have performed a subsidiary analysis of this data investigating lung function decline in man-made fibre workers only (n=652). Previous cotton dust exposure and current measures of man-made fibre exposure are included in the data set as separate predictor variables and its effect on the measured lung function decline compared to cotton naive workers.

Study Population

Two man-made fibre spinning mills located in the North West of England agreed to take part over the five year study period. The mills processed polyester, viscose and nylon fibres. The two mills offered a target population between 442 and 501 workers on a yearly basis over the study period. For each year of the study all operatives of each individual mill were invited to take part, complete a computer administered respiratory questionnaire and perform assessment of lung

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function. A small number of workers were also asked to wear personal dust sampling equipment over a full working shift

Questionnaire

The questionnaire used was an adaptation of the Medical Research Council respiratory questionnaire published in 1966 and was administered via a Compaq computer to each operative by one of three investigators.

The questionnaire documented the presence or absence of the respiratory symptoms; cough, phlegm, chest tightness, wheeze and shortness of breath. In order to define whether any of symptoms experienced were work related they had to improve on rest days or holidays. Operatives reporting work related chest tightness which was worse on the first working day were classified as byssinotic and graded according to Schilling's classification (Schilling, 1956). In addition demographic details, present occupation and workroom, past work history related to the cotton industry and smoking history were recorded.

Lung Function Assessment

Lung function was measured each year using a Vitalograph dry wedge spirometer, according to the guidelines produced by the ATS Snowbird workshop (Gardner, 1979). Forced expired volume in one second (FEV_1) and forced vital capacity (FVC) were measured. Operatives were required to perform three blows, with two blows within 5% or 100 mls of each other and the largest measures of FEV_1 and FVC were recorded. All assessments were made with the operative standing and a nose clip was used in cases where spirometry technique was poor. Since the temperature in the workrooms throughout the mills ranged from 16˚C to 32˚C, spirometry results were read from the ambient temperature pressure saturated with water vapour (ATPS) scale and corrected to body temperature pressure saturated with water vapour (BTPS).

Work Area Dust Sampling

Work area dust sampling was performed according to the regulations laid down in the EH 25 guideline (British Health and Safety Executive, 1975). Rothero and Mitchell L60 samplers loaded with Whatman GFA 37 mm glass microfibre filter papers were used to measure work area dust concentrations. The samplers were enclosed by a pre-filter wire mesh 2 mm pitch gauze cage and positioned on the top of a aluminium ladder at a height of 1.5 metres. The L60 samplers were run for a period of approximately four hours. In addition to the sampler filter papers, control filters were also weighed before and after the sampling exercise. Differences in weight of the filter papers were corrected if any change in weight of the control filters was detected. The volume of air sampled was calculated from the time and flow rate recordings of each sampler. The work area dust concentration (SDMMF) was calculated by dividing the weight of dust by the volume sampled and was expressed as mgm^{-3}.

Personal Dust Sampling

Operatives from various occupational groups were asked to wear personal dust sampling equipment during their working shift. Casella AFC sampling pumps (adjusted to run at 2 litres per minute) and Institute of Occupational Medicine (IOM) open face sampling heads (Mark 1986) with 25 mm microglass fibre filter papers were worn at clavicle level. Changes in weight in the sample filters were corrected for any differences in weight of the control filter papers. Personal dust concentrations (PDMMF) were expressed as mgm^{-3}.

Current and Past Exposure Loadings

The documented current and past work history was used for the calculation of current man-made fibre exposure and past retrospective cotton dust exposure.

For current man-made fibre exposure the workers occupation, current workroom and mill was used to ascribe a level of both work area and personal dust concentrations.

A matrix for work area sampling was generated from ten cotton mills previously studied by the North West Lung Centre and also included data supplied by the mills themselves. The data reliably back dates to 1981 and measurements were performed following the HSE EH25 guideline. By using this matrix and the workers occupation, past workroom(s) and length of exposure(s) it was possible to calculate, by long hand, a cumulative work area cotton dust loading (SDPAST) for those individuals who reported to have previously worked in the cotton industry.

A conversion factor was applied to the matrix of retrospective work area dust levels to give estimates of personal dust exposure (Niven, 1991). Using this conversion factor and the documented work history a cumulative personal dust loading (PDPAST) was calculated for each operative who reported past exposure to cotton dust.

Statistical Analysis

Individual longitudinal regression models were fitted to the lung function data obtained from each study subject. As the maximum number of years available for the analysis was five, it was possible only to fit a linear relationship. By fitting linear regression lines in this way, no missing value estimates were required for any years that subjects were unavailable for assessment.

A random effects regression model was fitted, which assumes that the variances at each of the five assessment times were equal and that the covariances between all pairs of assessment times were equal.

The individual regression lines were then combined, using appropriate weightings based on the number of assessment points used for each line. Separate average regression lines were computed for each of the dust exposure parameters assessed in the study. From these analyses, the average regression slopes were extracted to provide estimates of the rate of change in FEV_1 and FVC over the study period.

The model was also fitted taking into account any influencing confounders (age, ethnic group, smoking pack yrs, use of face mask, use of inhaler). These confounding factors were included as covariates. The statistical data presented is fully adjusted for these potential influencing factors. All models were fitted using the STATA statistical computer software (StataCorp, 1997).

Results

Table one presents the target population, sample population and response rate for each year of the study. Approximately four hundred man-made fibre textile workers agreed to take part in the study on a yearly basis. In total 668 workers were seen on at least one occasion over the five year study period. 99.4% of the population were of Caucasian or Asian origin. For the purpose of this analysis the data has been stratified. Excluded from the analysis were those workers who may have moved mills during the study period and ethnic groups other than Caucasian and Asian.

In total 652 workers were included in the analysis, mean age 36.6yrs (CI 35.8-37.5), Caucasian=65.1% Asian=34.9%; male=62.8% and current smokers=44.5%, with a median pack years of smoking 0.3 (range 0-92). Past work history revealed that half of the study population (50.5%) had reported to have worked in the cotton industry. The median (range) of current man-made fibre exposure; PDMMF=0.55mg/m^3 (0-25), SDMMF=0.09mg/m^3 (0-0.46) and median (range) of past cotton exposure; PDPAST=2.6mg/m^3 (0-128.5), SDPAST=1.5mg/m3 (0-37.4).

Overall, the adjusted rate of change was low for both FEV^1 and FVC. No markers of past cotton dust or current man-made fibre exposure were predictive of lung function decline. Highly statistically significant confounders were ethnic group (being Asian), sex (being male), increasing age and increased smoking being predictive of lung function decline (Table 2).

In view of the above findings the data was sub grouped by ethnic group, then further sub grouped by age <35yrs and >35yrs. This was important as the physiology of the airways can be separated into three distinct phases, maturity of the airways up to 25yrs, a plateau of respiratory physiology between the ages of 25yrs to 35yrs, and the onset of lung function decline from 35yrs onwards.

Table 3 presents the findings of the Caucasian <35yrs group. Over the five year study period the adjusted rate of change for both FEV^1 and FVC are shown to increase; FEV_1 = +10mls/yr FVC= +9mls/yr. Of the known confounders, being male and increasing age was predictive of greater positive change in the annual rate of lung function. Within the Asian group <35yrs, a small annual decline was demonstrated in FEV_1 (-6mls/yr) whilst there was a small annual increase in FVC (+9mls/yr). Again, being male and increasing age are identified as statistically significant predictors (Table 4). These random effects regression model results for both the Caucasian and Asian <35yrs age group reflects the life cycle of the respiratory airways one would expect to find in this age band (maturity and plateau of the respiratory physiology). In both groups, no markers of past cotton dust exposure or current man-made fibre exposure were predictive of increased or decreased levels of lung function change.

The Caucasian and Asian greater than 35yrs sub groups demonstrated similar declines of lung function in FEV_1 and FVC; Caucasian FEV1= -25mls/yr, FVC= -17mls/yr; Asian FEV_1= -29mls/yr, FVC= -19mls/yr. Within the Caucasian group no markers of past cotton exposure were predictive of lung function decline. There was an association between the marker of current man-made fibre exposure as measured by personal dust sampling, but the finding demonstrated that greater decline in lung function occurred with lower levels of man-made fibre exposure. In the Asian group markers of past cotton dust exposure failed to reach significance at the 5% level but were significant at the 10% level for both FEV_1 and FVC which may be suggestive of a weak selection effect. The highly significant confounders in both groups were being male, and as one would expect, increased decline in lung function was associated with increasing age (Table 5 and Table 6).

Summary

This analysis was performed to investigate earlier findings of waste cotton workers, and man-made fibre workers, demonstrating similar rates of lung function decline. Within the cotton exposed population, the waste mills were found to have the highest levels of dust and contaminants, and a decline in lung function was associated with time exposed in the cotton industry and cumulative dust exposure. The reason for the lung function decline in man-made fibre workers was unclear, however, it was suggested that susceptible workers may have been selecting out of the toxic cotton environment. The data presented from this study does not support this theory. Past time in the cotton industry and markers of previous cotton dust exposure were not predictive of excess lung loss in these man-made fibre textile workers. Therefore, we believe it is not susceptible workers moving from cotton to man-made fibre that have caused the rate of decline to be

higher in the man-made fibre group compared to workers exposed to medium or cotton blend. It has also been suggested that lung function decline may be directly associated with the chronic effect of man-made fibre exposure. Again, this data found no evidence to support this in that markers of current manmade fibre exposure were not predictive of accelerated lung loss (in fact a negative association was found in one sub group analysis).

The rates of lung function decline in man-made fibre workers >35yrs, are in fact similar to the rates of lung function decline one would expect to find in the general population. At this time we are unable to explain the differential rate of lung function decline seen between

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Table 1. Study population over the five study years

2 MMF Mills	Year 1	Year 2	Year 3	Year 4	Year 5
Target population	486	475	501	474	442
Sample population	438	427	410	367	348
Response rate	90.1%	89.9%	80.1%	77.4%	78.7%

Table 2. Lung function decline - Overall analysis n=652. Rate of change FEV_1= -12mls/yr; FVC= -6mls/yr.

	FEV_1	FVC
Markers of past cotton dust exposure		
PDPAST	p=0.726	p=0.788
SDPAST	p=0.414	p=0294
Timecott	p=0.409	p=0.522
Markers of current MMF exposure		
PDMMF	p=0.945	p=0.952
SDMMF	p=0.715	p=0.484
Significant counfounders		
Ethnic group	p<0.001	p<0.001
Sex	p<0.001	p<0.001
Age	p<0.001	p<0.001
Pack yrs	p<0.002	p=0.342

Table 3. Lung function decline - Caucasian <35yrs n=236. Rate of change FEV_1= +10mls/yr; FVC= +9mls/yr.

	FEV_1	FVC
Markers of past cotton dust exposure		
PDPAST	p=0.311	p=0.173
SDPAST	p=0.524	p=0.173
Timecott	p=0.312	p=0.683
Markers of current MMF exposure		
PDMMF	p=0.222	p=0.748
SDMMF	p=0.888	p=0.463
Significant counfounders		
Sex	p<0.001	p<0.001
Age	p<0.050	p=0.615
Pack yrs	p=0.082	p=0.448

Table 4. Lung function decline - Asian <35yrs n=66. Rate of change FEV_1= -6mls/yr; FVC= +9mls/yr.

	FEV_1	FVC
Markers of past cotton dust exposure		
PDPAST	p=0.726	p=0.343
SDPAST	p=0.576	p=0.468
Timecott	p=0.545	p=0.955
Markers of current MMF exposure		
PDMMF	p<0.10*	p=0.147
SDMMF	p=0.624	p=0.983
Significant counfounders		
Sex	p<0.001	p<0.001
Age	p<0.001	p<0.050
Pack yrs	p=0.803	p=0.522

*Significant at the 10% level

Table 5. Lung function decline - Caucasian >35yrs n=188. Rate of change FEV_1= -25mls/yr; FVC= -17mls/yr.

	FEV_1	FVC
Markers of past cotton dust exposure		
PDPAST	p=0.497	p=0.833
SDPAST	p=0.726	p=0.747
Timecott	p=0.727	p=0.627
Markers of current MMF exposure		
PDMMF	p<0.050	p<0.050
SDMMF	p=0.718	p=0.807
Significant counfounders		
Sex	p<0.001	p<0.001
Age	p<0.001	p<0.001
Pack yrs	p=0.252	p=0.716

Table 6. Lung function decline - Asian >35yrs n=162. Rate of change FEV_1= -29mls/yr; FVC= -19mls/yr.

	FEV_1	FVC
Markers of past cotton dust exposure		
PDPAST	p=0.112	p<0.10*
SDPAST	p<0.10*	p<0.10*
Timecott	p=0.719	p=0.525
Markers of current MMF exposure		
PDMMF	p=0.120	p=0.546
SDMMF	p=0.408	p=0.846
Significant counfounders		
Sex	p<0.001	p<0.001
Age	p<0.001	p<0.001
Pack yrs	p=0.013	p=0.097

*Significant at the 10% level