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Editorials

Byssinosis—a form of occupational asthma?

Byssinosis is a form of occupational lung disease associated with exposure to cotton dust. Despite much epidemiological research the causative agent remains unknown and the nature of the disease and its ability to cause permanent respiratory disability is a matter of debate. At present few textile mills in Lancashire carry out pre-employment or periodic medical examinations on their employees and the industry itself has no idea whether current dust concentrations are causing new cases of byssinosis. It is a matter of particular concern that this state of ignorance exists at a time when the concentrations of dust to which workers are exposed are rising as a result of increasing spinning speeds and greater use of open ended spinning.

Byssinosis is characterised by a feeling of chest tightness on the first day of the working week, normally starting three to four hours from the beginning of a work shift. Characteristically, the chest tightness either disappears or becomes less intense on subsequent working days of the week; even when present on each working day the symptoms are always most severe at the beginning of the week. They then reappear with re-exposure to cotton dust on the first day at work after a weekend break. Symptoms generally appear after 20–25 years of exposure to cotton dust.[1] The terminology of byssinosis has been complicated by its division into "acute" and "chronic" forms. Acute byssinosis usually refers to the acute airway response seen in about a third of healthy volunteers exposed to cotton dust or cotton bract extracts for the first time. Falls in FEV_1 exceeding 30% have been described after such experimental exposure in the cardroom.[2] "Chronic" byssinosis is applied to byssinosis associated with respiratory disability—in other words "classical" byssinosis developing after many years' exposure to cotton dust. Whether the same pathophysiological process is responsible for both these observed effects is not clear. Acute airway constrictor responses occurring in one third of workers entering the cotton textile industry for the first time would be expected to lead to a rapid labour turnover during their first year of employment. There is anecdotal evidence for this in the Lancashire cotton spinning mills, where there is said to be a 30% labour turnover each year; and it has recently been documented in Finnish cotton spinning mills,[3] where one in 10 employees left within two weeks and one in four within three months of taking up employment.

The link between exposure to cotton and the development of chronic disabling lung disease and whether byssinosis should be classed as a form of occupational asthma are matters of controversy. The diagnosis of occupational asthma requires not only that bronchial asthma is diagnosed but that a work related effect on either respiratory symptoms or lung function is established. A review of early descriptions of byssinosis, at a time when workers were exposed to very high concentrations of cotton dust, shows that clinicians believed byssinosis to be a form of asthma. In his annual review to parliament in 1908 the medical officer of the Privy Council[4] reported the comments of Dr Greenwood, medical officer of health of Blackburn: "All strippers and grinders are bothered with chest trouble. He has no breath, and pants and gasps, and coughs and spits. The cough is six years developing, it is an asthmatic cough." He reviewed a group of 131 strippers and grinders; 33 were healthy and 98 either complained of or were found to suffer from some degree of asthma. The symptoms of byssinosis are now well documented by many studies of cotton spinners and weavers,[5 6] though recent work from this department has found that individuals with byssinosis also complain of various work related respiratory symptoms other than the classical chest tightness. These include paroxysmal wheeze, cough, and shortness of breath,[7] again usually most severe on the first day of the working week.

Changes in lung function associated with occupational asthma are variable and indeed lung function may be normal in some individuals at initial presentation.[8] Across shift falls in FEV_1 and serial peak expiratory flow (PEF) measurements are the two most common methods of assessing the effects of the workplace on airway physiology. Significant falls in FEV_1 may be misleading, however, and their absence does not exclude the diagnosis of occupational asthma, as noted by Burge and collegues in their study of colophony workers.[9] Serial PEF measurements may show various work related patterns in occupational asthma[10] but are in addition influenced by factors other than workplace exposure to allergens. The "acute" lung function changes associated with byssinotic symptoms are less well documented. Despite the fact that the symptoms of byssinosis have been well described for almost two centuries, data on the corresponding changes in lung function were not described until 1958. McKerrow *et al*[11] noted that, although mean FEV_1 declined progressively through the working week, the greatest fall in FEV_1 occurred on the first working day. This finding suggested that the chest tightness associated with this condition may well be related to the magnitude of the fall in FEV_1 rather than the absolute value. Merchant *et al*[12] subsequently confirmed these findings in a similar study of 25 cardroom workers.

There is a paucity of data on serial PEF measurements in cotton workers. Unpublished work from this department has shown that the serial PEF measurements of some cotton operatives with byssinosis are consistent with a diagnosis of asthma, with a large daily diurnal variation in peak expiratory flow during working days when they are exposed to cotton dust and with a reduction in this variation on rest days and mean PEF values lowest on the first day of the working week. This observation lends support to the theory that byssinosis is a form of occupational asthma, or at least that exposure to cotton dust may produce PEF changes consistent with asthma. Schachter and colleagues assessed the acute response to cotton dust in previously unexposed individuals. The first of two papers[13] reports on a group of healthy subjects exposed to cotton dust and classified according to their response (responders were defined as those with at least a 20% fall in mid expiratory flow (MEF_{40})). Both groups

(responders and non-responders) were then subjected to bronchial provocation with inhaled histamine. The responders and non-responders did not differ significantly in their response to histamine. Subsequent work by the same group,[14] using methacholine to assess bronchial responsiveness, noted that responders to cotton inhalation had significantly more reactive airways than their non-responding counterparts. It is difficult to explain the differences between the results of these essentially similar studies, though the authors postulated that part of the difference may be exaplained by the fact that cotton dust contains histamine and this may affect physiological responses of the airways. The results of the methacholine study suggests that the acute response to cotton dust may be related to airway reactivity, though it is difficult to extrapolate these findings in previously unexposed individuals to cotton workers, who have usually been exposed to cotton for many years. On the basis of the findings of Schachter and colleagues, if byssinosis is a form of occupational asthma an increase in bronchial reactivity would be expected in a substantial proportion of cotton operatives with byssinosis, as bronchial hyperreactivity is a particular hallmark of asthma.[15] Early work by Verbeke *et al*[16] found evidence in 10 of 13 workers with byssinosis of bronchial hyperreactivity, assessed by means of acetylcholine inhalation. Bouhuys,[17] however, was unable to show a relation between byssinosis and bronchial hyperreactivity in flax workers. Later in the same year Massoud *et al*[18] studied 26 cotton operatives, 11 of whom had been diagnosed as having pure byssinosis and 12 as having bronchitic byssinosis (the latter complaining of symptoms of chronic bronchitis in addition to those of byssinosis). Massoud *et al* found that seven of the 12 workers with bronchitic byssinosis had evidence of bronchial hyperreactivity whereas those with pure byssinosis had normal reactivity.

In one of our recent studies[19] we have found bronchial hyperreactivity in most cotton operatives with byssinosis, even though atopy was not significantly more prevalent in this group than in those operatives without byssinosis. This finding again provides support for classifying byssinosis as a form of occupational asthma. The presence of bronchial hyperreactivity may be a hallmark of a particular susceptibility to the effects of inhaled cotton dust, though clearly only truly longitudinal studies with pre-employment (and pre-exposure) assessment of reactivity will show whether the observed hyperreactivity predates exposure to cotton dust or is a consequence of it.

Although there is an established link between atopy and bronchial hyperreactivity, the role of atopy and skin reactivity to cotton antigens is poorly documented. Despite extensive investigations no consistent skin test or IgE response to a range of either cotton antigens or fungal antigens has been identified. There is limited evidence that atopy may influence the acute response to cotton dust. Jones *et al*[20] investigated 255 workers in a cotton seed crushing mill. Atopy was defined on the basis of skinprick tests with common inhalant allergens, and measurements of FEV_1 before and after shifts showed that atopic workers exposed to cotton linter had significantly greater falls in FEV_1 and forced expiratory flow at 25–75% of vital capacity (FEF_{25-75}) across shifts than non-atopic workers. The authors concluded that atopy may well interact with this particular type of cotton dust to accentuate the acute bronchoconstrictor response. Certain atopic individuals may therefore be unable to tolerate exposure to cotton dust in the workplace and leave the industry early, leaving behind a relatively non-atopic work force. This is borne out by work from Honeybourne *et al*,[21] who found a lower prevalence of atopy in a group of workers with byssinosis than in a control group. The atopic workers with byssinosis had also spent less time working in the cotton industry than their non-atopic fellow workers. Paradoxically therefore it may be the non-atopic individuals who develop work related respiratory symptoms, bronchial hyperreactivity, and byssinosis—because only they can tolerate exposure to cotton for long enough to develop disease. Clearly this point requires further research, in particular on the relation between the presence or development of bronchial hyperreactivity and work related respiratory symptoms and the changes in reactivity during the working shift and week. The question of disability in individuals with byssinosis remains controversial. Elwood and coworkers,[22] in a carefully conducted study, found a 2–8% decrement in lung function in ex-cotton textile workers by comparison with controls, with a small but increasing decrement with higher dust exposures. The loss of lung function in men with 15 years' heavy exposure to dust was equivalent to that seen in light smokers or ex-smokers. The mean duration of dust exposure in the study group, however, was only 15 years and this was therefore unlikely to have contained many individuals with byssinosis, who were not identified as a separate group in the study.

The failure also to find an excess mortality among ex-cotton textile workers[23] suggests that exposure to cotton dust does not cause chronic lung disease in most cotton workers. A study of female Finnish cotton workers[24] with at least five years' exposure to cotton dust showed a significant excess of disability pensions for respiratory disease (15 against an expected of 3·9). Interestingly, of the 15 cases, 11 were diagnosed as bronchial asthma, three as byssinosis, and one as allergic rhinitis. In a recent mortality study[25] of 3458 cotton workers both the total mortality and the mortality for respiratory disease were less than expected. In those reporting symptoms of byssinosis, however, the mortality from respiratory disease was raised.

In summary, there does now appear to be sufficient evidence to include byssinosis as a form of occupational asthma, particularly in the light of the acute physiological response to cotton dust, its interaction with atopy, and the presence of bronchial hyperreactivity in those workers with chronic, established symptoms.

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