# Long term respiratory health effects in textile workers

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## Abstract

**Purpose of review**—Over 60 million people worldwide work in the textile or clothing industry. Recent studies have recognized the contribution of workplace exposures to chronic lung diseases, in particular chronic obstructive pulmonary disease (COPD). Early studies in textile workers have focused on the relationship between hemp or cotton dust exposure and the development of a syndrome termed Byssinosis. The purpose of this review is to evaluate the effect of long term exposure to organic dust in textile workers on chronic respiratory disease in the broader context of disease classifications such as reversible or irreversible obstructive lung disease (i.e. asthma or COPD), and restrictive lung disease.

**Recent findings**—Cessation of exposure to cotton dusts leads to improvement in lung function. Recent animal models have suggested a shift in the lung macrophage:dendritic cell population as a potential mechanistic explanation for persistent inflammation in the lung due to repeated cotton-dust related endotoxin exposure. Other types of textile dust, such as silk, may contribute to COPD in textile workers.

**Summary**—Textile dust related obstructive lung disease has characteristics of both asthma and COPD. Significant progress has been made in the understanding of chronic lung disease due to organic dust exposure in textile workers.

## Keywords

byssinosis; textile; lung; chronic obstructive pulmonary disease (COPD); endotoxin; vegetable dust

## Introduction

Over 60 million people are employed in the textile or clothing industry worldwide [1]. There is growing interest in the contribution of workplace exposures to obstructive lung disease, given that 25-45% of patients with chronic obstructive pulmonary disease (COPD) worldwide have never smoked[2]. While a number of non-tobacco related environmental

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exposures may cause COPD, including biomass fuel, early childhood infections, and pulmonary tuberculosis, occupational exposure to dusts are a major contributor, with one U.S. based study citing a population attributable risk of 9%[3].

The adverse respiratory effects of exposure to cotton, flax, and hemp dust in the textile industry was first described several centuries ago as a syndrome later called byssinosis. Schilling first classified byssinosis severity based solely on symptoms: Grade I is characterized by chest tightness and shortness of breath on only the first day back to work after a weekend off, termed "Monday asthma". Grade II occurs when symptoms occurs on days other than the first day back to work, and Grade III is characterized by evidence of permanent loss of lung function often forcing the worker to retire [4]. In 1983 the World Health Organization proposed a modification of the Schilling classification that also incorporates spirometry[5], although this has not been broadly adopted in research publications. The purpose of this review is to evaluate the literature on long-term respiratory diseases due to non-synthetic textile work in the context of disease classifications such as reversible or irreversible obstructive lung disease (asthma and COPD, respectively), interstitial lung disease, and additionally, to highlight notable recent advances.

## Search strategy and selection criteria

We searched PubMed (English language publications, no date restriction) using the following search terms "textile[tiab] AND (respiratory[tiab] OR lung[tiab]) NOT asbestos[tiab]" and "byssinosis[tiab]". Additionally, reference lists from articles obtained via the PubMed search were hand-searched for additional relevant studies. We focused on reports published since 2010, but did not exclude highly cited or important studies prior to 2010.

### Limitations in studies on chronic respiratory diseases in textile workers

Several important limitations underlie studies in textile workers. Healthy people are both more likely to be employed ("healthy worker hire effect") and remain employed ("healthy worker survivor effect"), underestimating the impact of exposure on disease. Population based controls are problematic, and sick workers often transfer to less exposed jobs within the workforce if they continue to work. Thus paradoxically workers in areas of high exposure often have higher baseline lung function, and active workers have much higher lung function than retired workers [6]. The healthy worker effect may be particularly accentuated in cotton mills due to the noxious effect of contaminating endotoxin[7]. One experimental study involving exposure of healthy volunteers to cotton dust had to be prematurely terminated due to the onset of acute symptoms [8]. A study of 198 newly hired workers at a cotton spinning mill found that 20% left the mill environment in the first month and 53% in the first year, with the only significant predictor for early termination of employment being work-related lower respiratory symptoms[9]. Finally, standardized mortality ratios are significantly lower in cotton textile workers (0.79 and 0.87 for females and males) compared to the general population[10].

Many early studies did not adequately control for smoking when evaluating respiratory outcome in textile workers, which is problematic as exposure to tobacco and organic dust

may result in the same clinical symptoms or pathologic lesions. A number of cohort studies have noted additive interactions between dust and smoke exposure on respiratory symptoms and lung function decline in cotton[11] and hemp[12] workers.

There may also be differences in studies performed on textile cohorts who have been chronically exposed vs. experimental studies where healthy human subjects are exposed to textile dust. Differences in the effects of acute vs. chronic exposure to textile dusts may also occur. Finally, most textile studies involve cotton, although similar results in hemp and flax workers are seen.

## Clinical manifestations

Studies have reported several types of lung disease associated with textile dust exposure (Table 1).

### Obstructive lung disease: Asthma

Early stage byssinosis in many respects fulfills the criteria for the diagnosis of asthma: reversible airflow obstruction and airway hyper-responsiveness [13]. Large changes in $FEV_1$ before and after a work-shift (cross-shift drop in $FEV_1$) has been noted in a number of different studies[14-16], although pre-shift treatment with salbutamol appears to mitigate this response[17]. While the cross-shift drop in $FEV_1$ is seen in both atopic and non-atopic individuals, the magnitude of the drop appears to be greater in atopic individuals [18,19]. Nonspecific bronchial hyper-reactivity has been noted in a number of studies. A study of 225 newly hired textile workers in Shanghai found an increase in methacholine responsiveness at 1 year follow-up. A study of 22 cotton textile workers with and without a history of byssinosis based on symptoms found that while none of the workers had a change in $FEV_1$ due to 0.2mg methacholine prior to a regular work shift, all had a significant drop in $FEV_1$ to the same 0.2mg dose post-shift although unexpectedly not to higher doses[20]. A subsequent study of asymptomatic, symptomatic but not byssinotic, and byssinotic textile workers found that there was bronchial hyper-reactivity in only byssinotic workers[21]. Unlike occupational asthma where symptoms worsen later in the workweek, here symptoms occur on the first day back to work and improves with persistent exposure throughout the week, at least in early disease. There are no studies currently in textile workers that document whether bronchial hyper-reactivity persists after worker retirement; thus it is unclear if textile workers develop an exposure related asthma-like syndrome vs. the persistent airway hyper-reactivity that is present in asthma despite exposure cessation.

### Obstructive lung disease: COPD

Most studies in cotton and hemp workers report an increased incidence of chronic and progressive dyspnea, cough, and sputum production characteristic of symptoms seen in COPD[22-26]. Pathologic studies in cotton textile workers have been conflicting, reporting variable associations between cotton dust and emphysema but confirming the presence of airways disease[27-30]; all of these studies have been limited by lack of quantitative exposure assessment to cotton dust and tobacco. The definitive diagnosis of COPD calls for a post-bronchodilator $FEV_1/FVC$ ratio of 0.7, with disease severity further graded by

percent predicted $FEV_1$[31]. Unfortunately, most studies on textile workers lack either post-bronchodilator spirometry or percent predicted $FEV_1$, and of those that report the latter, different reference equations are used [16,32,33]. While early studies did make note of current vs. former smoking status, number of pack years smoked was not adjusted for, further complicating the effort to determine the exact contribution of textile work to COPD.

In an early U.S. based case series, of 13 retired subjects who reported symptoms of byssinosis, 12 had $FEV_1$ ranging from 17-58% predicted with minimal to no bronchodilator response[33]. This study suggests that severe and irreversible airflow obstruction due to cotton textile work does occur, although this study was affected by referral bias and lacked quantitative assessment for cumulative dust and tobacco exposure. A study in hemp workers found that of workers who died, 32% had predicted $FEV_1 < 50\%$[25]. Subsequent studies either do not report the $FEV_1/FVC$ ratio or report only the average % predicted $FEV_1$ [16]. Because of the healthy worker effect, many subjects enrolled have supernormal % predicted $FEV_1$ . With this in mind, an accelerated decline in $FEV_1$ rather than the proportion of workers with $FEV_1/FVC<0.7$, as originally defined by Fletcher and Peto in their seminal work on tobacco-related COPD[34], may be a better marker for COPD in this population.

Accelerated decline in $FEV_1$ has been observed in cotton and hemp workers. Berry et al found an accelerated annual decline in $FEV_1$ in cotton vs. synthetic mill workers (54 vs. 32 ml/yr)[32]. Zuskin et al documented an accelerated decline in $FEV_1$ in male cotton textile workers over a 10 year period [22]. Beck et al followed 383 cotton textile workers and 277 age matched controls from the general population and confirmed an accelerated decline in cotton workers vs. controls (42 vs. 25 ml/yr), even in nonsmokers and after retirement[23]. Similar findings have been noted in other cotton studies[11] and in hemp workers[25].

Schilling first suggested that presence of a cross-shift drop in $FEV_1$ or symptoms of acute byssinosis may predict long term respiratory morbidity from dust related textile work[15]. Two studies in 1995 reported the association between cross-shift drop and longitudinal loss in $FEV_1$. Glindemeyer et al. demonstrated in a 5-year study on 611 cotton textile workers that a cross-shift drop in $FEV_1$ of over 200ml was predictive of annual decline in $FEV_1$[35]. Christiani et al. reported that a 5% cross-shift drop was predictive of annual decline in $FEV_1$ at both 5 and 15 year follow-up[36,37]. The latter study was from The Shanghai Textile Workers Study, the longest ongoing longitudinal cohort of cotton and silk workers; survey are performed every 5 years starting from 1981 with a ~70% retention rate at 30 years, with measurement of cumulative dust, endotoxin, and tobacco use as well as respiratory symptoms and spirometry.

For symptoms, Zuskin et al[22] did not find a significant difference in annual decline in $FEV_1$ between byssinotics and non-byssinotics although the study may have been underpowered. Work from the Shanghai Textile Workers Study indicated that at the 15 year follow-up, both byssinosis symptoms and symptoms of chronic bronchitis, cough, and dyspnea were associated with accelerated decline in $FEV_1$[37]; at 20 year follow-up, byssinosis symptoms continued to predict accelerated $FEV_1$ decline[11].

The impact of exposure cessation on lung function has been controversial. An accelerated decline in $FEV_1$ was noted in retired Spanish hemp workers compared to controls from neighboring shoe and marble factories[25]. A second study of US cotton textile workers demonstrated a higher prevalence of byssinosis, chronic bronchitis, and dyspnea as well as accelerated $FEV_1$ decline in retired compared to both active workers and controls[23]. While these studies would suggest that progression of lung disease continues after retirement, the Shanghai Textile Workers Study demonstrated that at 25-year follow-up, there was an improvement in $FEV_1$ after retirement, with a greater improvement seen in cotton workers who were smokers compared to nonsmokers (25.4 vs. 5.2 ml/yr, respectively). An improvement in $FEV_1$ was also noted in retired silk workers[38]. Of interest, improvements in $FEV_1$ have also been reported after tobacco cessation in smokers [39].

A recent registry study based on the Shanghai Textile Industry Bureau involved 267,400 female textile workers, with mortality data obtained from 1989 to 2000. Mortality from COPD was assigned based on ICD-9 codes and review of death certificates. Compared to non-cotton and non-silk textile workers in the cohort (to adjust for the healthy worker effect), COPD-related mortality was elevated in cotton workers but unexpectedly highest in silk workers (HR=1.40 95% CI:1.03-1.89 for cotton, HR=2.54 95% CI 1.47-4.39)[40]. Interestingly, COPD deaths were observed only among silk workers who did not smoke; no COPD deaths were recorded in smoking silk workers. Given lack of exposure assessment and detailed medical history, it is difficult to determine the etiology of excessive deaths due to COPD in non-smoking silk workers. In contrast, increased mortality in silk compared to cotton textile workers was not observed in the longitudinal Shanghai Textile Workers Study; here silk workers with a prior history of active tuberculosis or asthma were excluded at the beginning of the longitudinal study in 1981.

### Interstitial lung disease

While an autopsy series of subjects with severe byssinosis qualifying for worker's compensation demonstrated the presence of "byssinosis bodies" in the lung parenchyma[27], this has not been replicated in subsequent autopsy studies[29,30]. In 2004 the case of a Japanese textile worker presenting with diffuse interstitial lung disease was reported, with infrared spectrophotometry confirming that string-like foreign bodies found on pathologic exam were consistent with cotton fibers. No other reports in the literature have been found, and to date no high resolution CT scans of textile cohorts have been performed to detect the presence of clinical or subclinical interstitial lung disease associated with textile dust exposure.

**Etiology—**While early studies correlated severity of illness in textile mills with dustier jobs within the mill [15], and an exposure-response relationship between dust levels and longer term changes in $FEV_1$ has been noted[41], there has been debate about the precise constituent(s) of cotton dust that is the etiologic agent. A number of microbes have been found in cotton, including gram negative and gram positive bacteria, actinomyces, and various fungi[42]. The evidence for endotoxin, a component of the gram-negative bacterial cell wall, is the most robust. As gram-negative bacterial contamination of cotton dust varies by the origin of the cotton used[43], an early study demonstrated that when healthy

volunteers were exposed to cotton dust with varying levels of endotoxin in a model card-room, there was a correlation between endotoxin and cross-shift drop in $FEV_1$ but not dust (correlation −0.71 vs. −0.08 for endotoxin vs. dust). In evaluating chronic changes in $FEV_1$, cumulative endotoxin, but not dust, exposure was predictive of 20-year changes in $FEV_1$ in the Shanghai Textile Workers Study[11].

In an animal model where guinea pigs were exposed to cotton dust, endotoxin, β-glucan (a fungal cell wall component), and tannin, endotoxin alone recapitulated the neutrophil recruitment, tachypnea, and decreased airway conductance seen with exposure to cotton dust, although endotoxin did not cause contraction of isolated tracheal smooth muscle seen with cotton dust[44]. Another component in cotton dust may cause the acute bronchoconstrictive response to cotton dust; a study in healthy human volunteers did not demonstrate a dose-response relationship between the endotoxin content of cotton bract extracts and acute drops in $FEV_1$ [45].

A recently developed animal model for cotton textile workers using endotoxin assessed differences in early vs. late responses (5 days vs. 8 weeks). There was significant bronchial hyper-reactivity with 5 day exposure, compared to significantly increased central airways resistance with 8 weeks exposure. This is consistent with the observation that organic dust may lead to reversible then fixed airflow obstruction over time. Interestingly, increasing duration of endotoxin exposure was associated with increasing pulmonary inflammation without endotoxin tolerance. An associated expansion of pro-inflammatory dendritic cells and reduction in macrophages important in resolving inflammation was observed, and may be a mechanism for the development of persistent obstructive lung disease seen in textile workers[46].

Noise pollution may also impact respiratory epithelium. Rats exposed to tape recordings of cotton mill room noise daily for 40 hours a week developed significant loss of tracheal ciliated cells at 1 month that persisted throughout the 7 month study period; the investigators postulate that this may lead to impaired mucociliary clearance and chronic cough[47].

**Conclusion**—Organic dust exposure in the textile industry leads to obstructive lung disease that has features of both asthma and COPD. Cessation of workplace exposure may lead to improved lung function. An inversion of the pulmonary macrophage:dendritic cell ratio may be a mechanistic explanation for persistent inflammation and obstructive lung disease seen in endotoxin-related textile exposures. Further research on the anatomic lesion (reversible or irreversible airways disease vs. emphysema) and the mechanism underlying textile dust related obstructive lung disease needs to be done.

## Acknowledgements

## Abbreviations

**COPD**          chronic obstructive pulmonary disease

**FEV$_1$**          forced expiratory volume in 1 second

**FVC**          forced vital capacity

**LPS**          lipopolysaccharide

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## Key points

- • Obstructive lung disease due to organic textile dust exposure has features of both asthma and chronic obstructive lung disease (COPD)

- • Cessation of exposure to organic textile dust due to worker retirement may lead to improvement in lung function

- • A reversal in the lung macrophage:dendritic cell ratio due to textile dust related endotoxin exposure may be a mechanistic explanation for the development of persistent airway inflammation and associated airflow obstruction

**Table 1**

Available evidence for lung disease in textile workers exposed to organic dusts

| Disease | Criteria | Evidence |
|---|---|---|
| Obstructive lung disease | | |
| Asthma[*] | Reversible airflow obstruction | Longitudinal cohort studies |
| | Airway hyper-responsiveness | Longitudinal cohort studies |
| COPD | Post-bronchodilator $FEV_1/FVC$ <0.7 | Case series<br>Longitudinal cohort studies |
| | Accelerated decline in $FEV_1$ | Longitudinal cohort studies |
| Restrictive lung disease | | |
| Interstitial lung disease | Lung biopsy with consistent features | Case report<br>Case series |

[*]
No studies available to distinguish between exposure-related asthma-like syndrome vs. asthma with persistent airway hyper-reactivity despite cessation of exposure