Long-term respiratory health effects in textile workers

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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 dust leads to improvement in lung function. Recent animal models have suggested a shift in the lung macrophage:dendritic cell population ratio 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, chronic obstructive pulmonary disease, endotoxin, lung, textile, 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]. Although a number of nontobacco-related environmental exposures may cause COPD, including biomass fuel, early childhood infections, and pulmonary tuberculosis, occupational exposure to dusts are a major contributor, with one US-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 were 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 (WHO) 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 nonsynthetic textile work in the

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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.

context of disease classifications, such as reversible or irreversible obstructive lung disease (asthma and COPD, respectively) and 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 articles 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 women and men) compared with 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 in which healthy human controls 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 forced expiratory volume in 1 second (FEV_1) before and after a work-shift (cross-shift drop in FEV_1) have 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 nonatopic 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 0.2 mg methacholine prior to a regular work shift, all had a significant drop in FEV_1 to the same 0.2 mg

Table 1. Available evidence for lung disease in textile workers exposed to organic dusts

Disease	Criteria	Evidence
Obstructive lung disease		
Asthma[a]	Reversible airflow obstruction	Longitudinal cohort studies
	Airway hyper-responsiveness	Longitudinal cohort studies
Chronic obstructive pulmonary disease	Postbronchodilator $FEV_1/FVC < 0.7$	Case series
	Accelerated decline in FEV_1	Longitudinal cohort studies
		Longitudinal cohort studies
Restrictive lung disease		
Interstitial lung disease	Lung biopsy with consistent features	Case report
		Case series

FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity.
[a]No studies available to distinguish between exposure-related asthma-like syndrome vs. asthma with persistent airway hyper-reactivity despite cessation of exposure.

dose postshift 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 in which symptoms worsen later in the workweek, here symptoms occur on the first day back to work and improve 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: chronic obstructive pulmonary disease

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 postbronchodilator FEV_1/forced vital capacity (FVC) ratio of ≤ 0.7, with disease severity further graded by percentage 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 US 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 less than 50% [25]. Subsequent studies either do not report the FEV_1/FVC ratio or report only the average percent predicted FEV_1 [16]. Because of the healthy worker effect, many subjects enrolled have supernormal percent predicted FEV_1. With this in mind, an accelerated decline in FEV_1 rather than the proportion of workers with FEV_1/FVC less than 0.7, as originally defined by Fletcher and Peto [34] in their seminal work on tobacco-related COPD, 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.* [32] found an accelerated annual decline in FEV_1 in cotton vs. synthetic mill workers (54 vs. 32 ml per year). Zuskin *et al.* [22] documented an accelerated decline in FEV_1 in male cotton textile workers over a 10-year period. Beck *et al.* [23] 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 per year), even in nonsmokers and after retirement. 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. Glindmeyer *et al.* [35] demonstrated in a 5-year study on 611 cotton textile workers that a cross-shift drop in FEV_1 of over 200 ml was predictive of annual decline in FEV_1. Christiani *et al.* [36,37] reported that a 5% cross-shift drop was predictive of annual decline in FEV_1 at both 5 and 15-year follow-up. The latter study was from The Shanghai Textile Workers Study, the longest ongoing longitudinal cohort of cotton and silk workers; surveys have been 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 nonbyssinotics 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 with 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 with 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 with nonsmokers (25.4 vs. 5.2 ml per year, 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 with 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 [hazard ratio = 1.40, 95% confidence interval (CI) 1.03–1.89 for cotton, hazard ratio = 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 cause of excessive deaths due to COPD in nonsmoking silk workers. In contrast, increased mortality in silk compared with 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

Although an autopsy series of patients 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 examination 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 causative 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, in which 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 with 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 h a week developed significant loss of tracheal ciliated cells at 1 month that persisted throughout the 7-month study period; the investigators postulated 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.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- ▪ of special interest
- ▪▪ of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 184).

1. Forstater M. Implications of the global financial and economic crisis on the textile and clothing sector. In: Sectoral coverage of the global economic crisis 2009, International Labour Organization: Geneva. pp. 1–26.
2. Salvi SS, Barnes PJ. Chronic obstructive pulmonary disease in nonsmokers. Lancet 2009; 374:733–743.
3. Trupin L, Earnest G, San Pedro M, *et al.* The occupational burden of chronic obstructive pulmonary disease. Eur Respir J 2003; 22:462–469.
4. Schilling RS. Byssinosis in the British cotton textile industry. Br Med Bull 1950; 7:52–56.
5. Kilburn K. Recommended health-based occupational exposure limits for selected vegetable dusts. World Health Organ Tech Rep Ser, 1983; 684: 1–78.
6. Pearce N, Checkoway H, Kriebel D. Bias in occupational epidemiology studies. Occup Environ Med 2007; 64:562–568.
7. Liebers V, Raulf-Heimsoth M, Brüning T. Health effects due to endotoxin inhalation (review). Arch Toxicol 2008; 82:203–210.
8. Castellan R, Olenchock S, Hankinson J, *et al.* Acute bronchoconstriction induced by cotton dust: dose-related responses to endotoxin and other dust factors. Ann Intern Med 1984; 101:157–163.
9. Bakirci N, Kalaca S, Fletcher AM, *et al.* Predictors of early leaving from the cotton spinning mill environment in newly hired workers. Occup Environ Med 2006; 63:126–130.
10. Su W, Chen Y, Liou S, Wu C. Meta-analysis of standard mortality ratio in cotton textile workers. Eur J Epidemiol 2004; 19:989–997.
11. Wang X-R, Zhang H-X, Sun B-X, *et al.* A 20-year follow-up study on chronic respiratory effects of exposure to cotton dust. Eur Respir J 2005; 26:881–886.
12. Bouhuys A, Schilling R, van de Woestijne K. Cigarette smoking, occupational dust exposure, and ventilatory capacity. Arch Environ Health 1969; 19:793–797.
13. NHLBI, Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma, 2007, National Heart Lung and Blood Institute. pp. 1–440.
14. Zuskin EE, Valić FF. Change in the respiratory response to coarse cotton dust over a ten-year period. Am Rev Respir Dis 1975; 112:417–421.
15. Schilling RS, Hughes JP, Dingwall-Fordyce I, Gilson JC. An epidemiological study of byssinosis among Lancashire cotton workers. Br J Ind Med 1955; 12:217–227.
16. Imbus H, Suh M. Byssinosis: a study of 10 133 textile workers. Arch Environ Health 1973; 26:183–191.
17. Fawcett IW, Merchant J, Simmonds S, Pepys J. The effect of sodium cromoglycate, beclomethasone diproprionate and salbutamol on the ventilatory response to cotton dust in mill workers. Br J Dis Chest 1978; 72:29–38.
18. Sepulveda M, Castellan R, Hankinson J, Cocke J. Acute lung function response to cotton dust in atopic and nonatopic individuals. Br J Ind Med 1984; 41:487–491.
19. Wang XR, Pan LD, Zhang HX, *et al.* Lung function, airway reactivity, and atopy in newly hired female cotton textile workers. Arch Environ Health 2003; 58:6–13.
20. Haglind P, Bake B, Belin L. Is mild byssinosis associated with small airways disease? Eur J Respir Dis 1983; 64:449–459.
21. Fishwick D, Fletcher A, Pickering C, *et al.* Lung function, bronchial reactivity, atopic status, and dust exposure in Lancashire cotton mill operatives. Am Rev Respir Dis 1992; 145:1103–1108.
22. Zuskin E, Ivankovic D, Schachter EN, Witek TJ. A ten-year follow-up study of cotton textile workers. Am Rev Respir Dis 1991; 143:301–305.

23. Beck GJ, Schachter EN, Maunder LR, Schilling RS. A prospective study of chronic lung disease in cotton textile workers. Ann Intern Med 1982; 97:645–651.

24. Glindmeyer HW, Lefante JJ, Jones RN, *et al.* Exposure-related declines in the lung function of cotton textile workers. Relationship to current workplace standards. Am Rev Respir Dis 1991; 144 (3 Pt 1):675–683.

25. Bouhuys A, Zuskin E. Chronic respiratory disease in hemp workers. A follow-up study, 1967–1974. Ann Intern Med 1976; 84:398–405.

26. Christiani D, Ye T, Zhang S, *et al.* Cotton dust and endotoxin exposure and long-term decline in lung function: results of a longitudinal study. Am J Ind Med 1999; 35:321–331.

27. Edwards C, Macartney J, Rooke G, Ward F. The pathology of the lung in byssinotics. Thorax 1975; 30:612–623.

28. Rooke GB. The pathology of byssinosis. Chest 1981; 79 (Suppl 4):67S–71S.

29. Pratt PC, Vollmer RT, Miller JA. Epidemiology of pulmonary lesions in nontextile and cotton textile workers: a retrospective autopsy analysis. Arch Environ Health 1980; 35:133–138.

30. Moran TJ. Emphysema and other chronic lung disease in textile workers: an 18-year autopsy study. Arch Environ Health 1983; 38:267–276.

31. Vestbo J, Hurd SS, Agusti AG, *et al.* Global strategy for the diagnosis, management and prevention of chronic obstructive pulmonary disease, GOLD Executive Summary. Am J Respir Crit Care Med 2012. [Epub ahead of print]

32. Berry G, McKerrow C, Molyneux M, *et al.* A study of the acute and chronic changes in ventilatory capacity of workers in Lancashire cotton mills. Br J Ind Med 1973; 30:25–36.

33. Bouhuys A, Heaphy L, Schilling R, Welborn J. Byssinosis in the United States. N Engl J Med 1967; 277:170–175.

34. Fletcher C, Peto R. The natural history of chronic airflow obstruction. Br Med J 1977; 1:1645–1648.

35. Glindmeyer HW, Lefante JJ, Jones RN, *et al.* Cotton dust and across-shift change in FEV1 as predictors of annual change in FEV1. Am J Respir Crit Care Med 1994; 149 (3 Pt 1):584–590.

36. Christiani D, Ye T, Wegman D, *et al.* Cotton dust exposure, across-shift drop in FEV1, and five-year change in lung function. Am J Respir Crit Care Med 1994; 150:1250–1255.

37. Christiani D, Wang X, Pan L, *et al.* Longitudinal changes in pulmonary function and respiratory symptoms in cotton textile workers. A 15-yr follow-up study. Am J Respir Crit Care Med 2001; 163:847–853.

38. Shi J, Hang J-Q, Mehta AJ, *et al.* Long-term effects of work cessation on respiratory health of textile workers: a 25-year follow-up study. Am J Respir Crit Care Med 2010; 182:200–206.

39. Willemse BW, Postma DS, Timens W, ten Hacken NH. The impact of smoking cessation on respiratory symptoms, lung function, airway hyperresponsiveness and inflammation. Eur Respir J 2004; 23:464–476.

40. ▪ Cui L, Gallagher L, Ray R, *et al.* Unexpected excessive chronic obstructive pulmonary disease mortality among female silk textile workers in Shanghai, China. Occup Environ Medi 2011; 68:883–887.

This is a registry-based study demonstrating that of all textile workers registered with the Shanghai Textile Industry Bureau, nonsmoking but not smoking silk workers have the highest COPD mortality.

41. Schachter E, Beck G, Maunder L. Chronic lung disease in cotton textile workers. Chest 1985; 87:406–408.

42. Salvaggio JE, O'Neil CE, Butcher BT. Immunologic responses to inhaled cotton dust. Environ Health Perspect 1986; 66:17–23.

43. Lane SR, Sewell RD. The bacterial profile of cotton lint from worldwide origins, and links with occupational lung disease. Am J Ind Med 2007; 50:42–47.

44. Castranova V, Robinson V, Frazer D. Pulmonary reactions to organic dust exposures: development of an animal model. Environ Health Perspect 1996; 104 (Suppl 1):41–53.

45. Buck M, Wall J, Schachter E. Airway constrictor response to cotton bract extracts in the absence of endotoxin. Br J Ind Med 1986; 43:220–226.

46. ▪▪ Lai PS, Fresco JM, Pinilla MA, *et al.* Chronic endotoxin exposure produces airflow obstruction and lung dendritic cell expansion. Am J Respir Cell Mol Biol 2012; 47:209–217.

Animal model of textile-related endotoxin exposure suggesting that an inverted macrophage:dendritic cell ratio is a mechanistic explanation for the persistent inflammation seen in textile-related obstructive lung disease.

47. Oliveira MJoR, Pereira AnS, es LG, *et al.* Chronic exposure of rats to cotton-mill-room noise changes the cell composition of the tracheal epithelium. J Occup Environ Med 2002; 44:1135–1142.