

## Cotton Dust and Gram-Negative Bacterial Endotoxin Correlations in Two Cotton Textile Mills

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Exposure to cotton dust is known to cause both acute and chronic respiratory illness. A specific pattern of symptoms called *byssinosis* is well described to occur among workers in the cotton processing (e.g., yarn preparation) industry. Recent studies have implicated Gram-negative bacterial endotoxin as one of the agents responsible for acute, and possibly chronic, respiratory illness. Laboratory experiments using a model cardroom have found poor correlations between airborne dust and associated endotoxin. This study reports the results of vertical elutriated dust and endotoxin levels in 11 work areas of 2 cotton textile mills in 1986 in Shanghai, China. The overall correlation between dust and endotoxin was strong,  $r_s = 0.66$  and 0.79 ( $p < 0.0001$ ) for mills 1 and 2, respectively. The dust-endotoxin correlation was relatively poor in early yarn preparation in the workshops and improved in the later preparation areas. Our findings suggest that in these mill settings, dust and endotoxin levels may be well correlated in most work areas. Therefore, dust may be a useful index for monitoring populations employed in the cotton textile industry throughout the world. Additional field studies need to be performed which consider the various determinants of dust and endotoxin levels.

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**Key words:** Gram-negative bacterial endotoxin, byssinosis, respiratory illness, cottondust exposures

### INTRODUCTION

Exposure to cotton dust is known to cause both acute and chronic respiratory illness. Symptoms associated with these illnesses include a number of acute pulmonary responses, in addition to chronic bronchitis and airflow limitation. One specific pattern of symptoms, called *byssinosis*, has been described among cotton textile workers. This syndrome is characterized by acute chest tightness and/or shortness of breath, initially occurring on the first day of exposure after 2 or more days away from work [Schilling, 1956]. Epidemiologic studies have revealed that the risk of byssi-

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nosis is highest in the early production areas of yarn preparation, e.g., carding, where cotton is mechanically brushed before being spun into yarn [Molyneux and Berry, 1968; Merchant et al., 1973].

In general, studies of byssinosis have been limited by the failure to identify in cotton dust a specific agent responsible for the symptom complex. Furthermore, assessment of exposure has been hampered by the absence of a personal sampler for cotton dust. The measurement of exposure has required the use of the vertical elutriator, an area monitor which only measures nonspecific airborne particulates. Nonetheless, the epidemiologic study used as the basis for the OSHA cotton dust standard provided evidence of a relationship between area dust levels and byssinosis prevalence in cotton textile mills [Merchant et al., 1973].

In the absence of a specific etiologic agent, the Occupational Safety and Health Administration (OSHA) was compelled to set limits for cotton dust exposure based on elutriated dust. Given the varying patterns of response observed in different settings, the standard also had to include separate exposure limits for different industries (i.e., ginning vs. yarn production) and different work areas (i.e., weaving vs. yarn preparation) [OSHA, 1978]. However, without the knowledge to select and control a specific etiologic component(s) of the particulate, a high residual disease prevalence (13%) had to be accepted. More stringent exposure control would have required reducing the nonspecific dust measurements to levels below outdoor background particulates.

During the past decade, cotton dust health effect studies have been improved by an effort to identify more precisely the agent responsible for the pulmonary effects. Recent experimental work in exposure chambers known as "model card rooms" in the United States and Sweden have played a central role in this effort to identify one or more etiologic agents responsible for the byssinosis syndrome. These studies have revealed a better exposure-response relationship for Gram-negative bacterial endotoxin than for dust when acute responses, such as the change in  $FEV_1$ , are examined [Castellan et al., 1987; Rylander et al., 1985]. Some epidemiologic investigations now provide evidence for Gram-negative bacteria or their associated endotoxins as the major causative agent of byssinosis [Cinkotai et al., 1977; Cinkotai and Whitaker, 1978; Rylander et al., 1979, 1983]. One study suggests a role for endotoxin in chronic, but not acute, disease [Kennedy et al., 1987].

Based on the poor correlation between dust and endotoxin levels in the model cardroom studies and the better association of byssinosis and endotoxin in these studies, it might follow that cotton workers would be better protected by an endotoxin standard in addition to, and possibly instead of, a dust standard. However, model cardroom investigators caution: ". . . of course, before an endotoxin based revision of the current OSHA regulations could be recommended, it would be important to determine and consider the ranges and correlation between dust and endotoxin concentrations in actual work areas in the cotton industry" [Castellan et al., 1987].

We were able to examine airborne cotton-dust and Gram-negative bacterial endotoxin levels from various yarn preparation areas in 2 cotton textile mills in Shanghai, The People's Republic of China. The mills used raw cotton from a variety of sources inside and outside of China. In order to examine dust-endotoxin correlations that might be stable and generalizable to similar mills around the world, we sampled the opening through spinning areas cross-sectionally in a large survey per-

formed over 3 months. We, therefore, did not pool data obtained from different surveys in an effort to minimize seasonal or time-dependent variation in air levels of dust and endotoxin.

## MATERIALS AND METHODS

Both textile mills were built in the 1920s. Exhaust ventilation was installed in the 1950s, with little change in process or exhaust controls since 1959. At the time of survey, raw material sources for mill number 1 included Southeast and Northwest China (combined = 91–92%), with the remainder imported from the United States and Africa. Sources for mill number 2 included Southeast, Northeast, and Northwest China (combined = 50–60%); Egypt, and other African countries (40–49%). Although the sources of the cotton varied considerably, grade did not, as most cotton used in these mills was of high to moderately high grade using commercial criteria. Bulk analysis of bale samples from these 2 mills revealed a wide range of endotoxin contamination, ranging from 2 ng/mg to over 500 ng/mg of cotton [Olenchock et al., 1990], indicating that geographic sources and level of contamination of these cottons are very heterogenous. There was no documented effort on the part of management to selectively buy from specific geographical regions. Rather, cotton was purchased on local and international commodities markets.

Environmental samples were taken in the yarn preparation areas of 2 cotton textile mills from October through December, 1986. In one mill, opening through spinning were sampled, while in the other, spinning was not sampled. Sampling was accomplished using vertical elutriators (General Metalworks Inc., Cleveland, Ohio, USA), which are designed to collect particles  $\leq 15 \mu\text{m}$  in aerodynamic diameter [DHEW-NIOSH, 1975]. All samples were taken on 5  $\mu\text{m}$  polyvinyl chloride hydrophobic filters of 37 mm diameter and were stored in petri dishes. They were packaged face-down in sterile Parafilm® and were shipped to Morgantown, West Virginia, where they were analyzed for endotoxin contamination. The filter was weighed on a balance sensitive to  $10^{-5} \text{ g}$  for gravimetric assessment before shipping and then were extracted with pyrogen-free water and assayed by the *Limulus* amebocyte assay [Nachum and Berdofsky, 1985] according to the method of Olenchock et al. previously described [Olenchock et al., 1989]. Briefly described, filters were extracted with 10 ml of sterile, nonpyrogenic water (Travenol Laboratories, Inc., Deerfield, IL) by rocking at room temperature for 60 minutes using sterile, nonpyrogenic plastic ware. The fluid was centrifuged at 1,000 g for 10 min, and the Gram-negative bacterial endotoxin content of the supernatant fluid was quantified in duplicate by a quantitative chromogenic modification of the *Limulus* amebocyte lysate gel test (QCL-1000; Whittaker Bioproducts, Walkersville, MD). Sample results are reported as Endotoxin Units (EU). The data were corrected mathematically to nanograms (10 EU = 1 ng), a conversion figure recommended by the manufacturer for the controls that were in use. Unused, blank filters and Parafilm "m" were used as negative controls in all assays.

Results for both types of exposure were calculated as time-weighted averages expressed in milligrams per cubic meter ( $\text{mg}/\text{m}^3$ ) for dust and in nanograms per cubic meter ( $\text{ng}/\text{m}^3$ ) for endotoxin. In addition, control samples collected from the nearby silk mill were collected and assayed in the same fashion.

TABLE I. Time-Weighted Averages of Cotton Dust and Endotoxin Concentrations in Area Samples Collected by Vertical Elutriator, Shanghai, 1986

	Dust (mg/m <sup>3</sup> )		Endotoxin (ng/m <sup>3</sup> )	
	Mill 1	Mill 2	Mill 1	Mill 2
Opening	1.69 <sup>a</sup> (.97–2.36) <sup>b</sup>	1.27 (1.09–2.64)	327 (289–795)	377 (267–446)
Cleaning	1.16 (.77–1.33)	1.73 (1.09–2.93)	354 (201–536)	578 (31.3–1338)
Carding	1.28 (.98–2.0)	1.52 (.74–2.58)	478 (168–783)	344 (251–1697)
Drawing	0.69 (.46–3.75)	1.52 (1.07–2.32)	428 (210–1462)	750 (520–1202)
Roving	0.33 (.14–.72)	0.45 (.31–.67)	99 (15.3–1060)	43 (20.2–112)
Spinning <sup>c</sup>	— —	0.24 (.04–.51)	— —	20.0 (.66–142)

<sup>a</sup>Median levels.

<sup>b</sup>Range: number of samples in parentheses below range.

<sup>c</sup>Plant 1 spinning not tested.

## STATISTICAL ANALYSIS

In order to determine whether data transformations or nonparametric statistical methods were appropriate, the distributions of both dust and endotoxin measurements were examined for normality. The Wilks-Shapiro test was used to test the distributions of the untransformed as well as the log transformed data for departures from normality. Nonparametric correlations between dust and endotoxin were estimated within specific departments as well as throughout each mill. Parametric linear regressions models were fit to the data to predict endotoxin on the basis of dust concentrations. Residuals were analyzed to examine violations of model assumptions.

## RESULTS

A total of 146 vertical elutriator samples were collected. Endotoxin analysis was performed on 99% of the samples. Air samples were collected in yarn preparation areas of both mills, with the exception of fine spinning in mill 1.

Dust and endotoxin concentrations from the 2 mills varied widely both within and between work areas (Table I). Time-Weighted Average (TWA) dust levels ranged from 0.14 to 3.75 mg/m<sup>3</sup> in mill 1 and from 0.04 to 2.93 mg/m<sup>3</sup> in mill 2. Endotoxin levels ranged from 15.3 to 1,462 ng/m<sup>3</sup> in mill 1 and from less than 1 to 1,697 ng/m<sup>3</sup> in mill 2. In general, dustier work areas included opening through draw frame areas. Median endotoxin levels were above 40 ng/m<sup>3</sup> in all work areas except fine spinning in mill 2. Six samples collected from the silk mill resulted in a median dust concentration of 0.12 mg/m<sup>3</sup>, with endotoxin concentrations less than 0.1 ng/m<sup>3</sup> (i.e., nondetectable).

Neither vertical elutriated dust nor endotoxin levels were normally distributed.

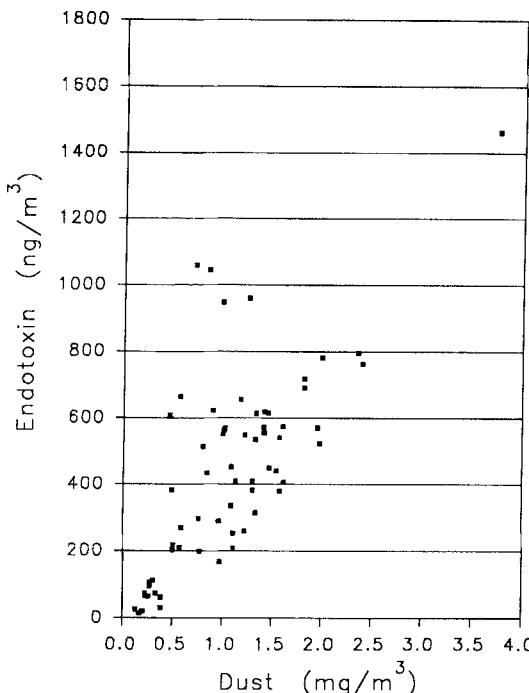


Fig. 1. Vertical elutriated cotton dust and endotoxin levels in mill no. 1, Shanghai, 1986.

When the natural logarithms were used to transform the data, the distributions still exhibited significant departures from normality. Therefore, rather than using logarithms to transform the data, nonparametric correlation coefficients were estimated to measure the association between the two untransformed measures of exposure.

The relationships between dust and endotoxin are presented, by mill, in Figs. 1 and 2. Cotton dust and endotoxin levels were significantly correlated in both mills,  $r_s = 0.66$  and  $0.79$  respectively ( $p < 0.0001$ ). The major processing departments in cotton yarn preparation are opening, cleaning, carding, and various spinning operations. These work areas were grouped into early (opening through carding) and late (drawing through spinning) stages. Grouped in this manner, the correlations remained high in both mills, with the exception of the early stage in mill 2 (Table II). When the work areas in the early stages were examined individually, the cardroom (where most of the employees in the early stage work) for mill 2 did not have the strong dust-endotoxin relationship observed in the cardroom of mill 1.

In order to estimate the linear model that best predicts endotoxin level on the basis of dust, a regression model was fit both separately to the data from each mill as well as to combined data. The distributions of residuals were unimodal and symmetric, indicating that although not strictly normal, the model parameters were validly estimated. The linear models predicted an endotoxin level of  $56 \text{ ng/m}^3$  (mill1) and  $68 \text{ ng/m}^3$  (mill2) associated with a dust level at the current PEL of  $0.2 \text{ mg/m}^3$  of respirable dust.

The correlations between dust and endotoxin were generally high in the later stages. However, since most of the byssinosis occurs in the early stage of cotton

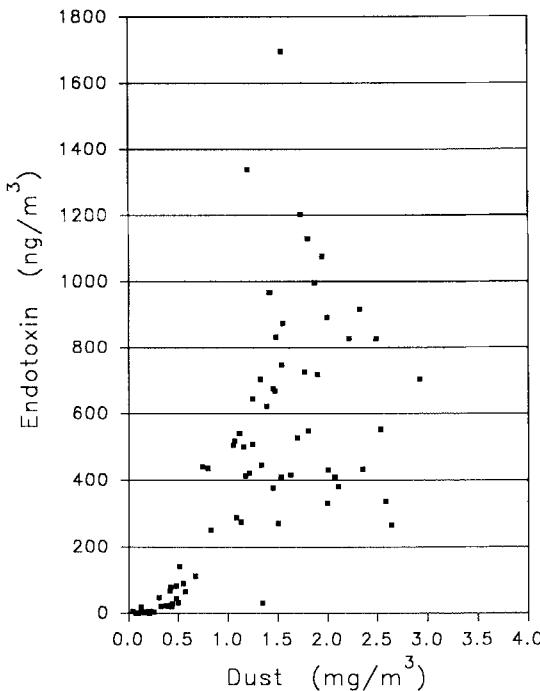


Fig. 2. Vertical elutriated cotton dust and endotoxin levels in mill no. 2, Shanghai, 1986.

**TABLE II. Cotton Dust-Endotoxin Correlations for Two Cotton Mills, Shanghai, 1986**

Work area	Mill 1			Mill 2		
	<i>n</i>	<i>r<sub>s</sub></i>	<i>p</i>	<i>n</i>	<i>r<sub>s</sub></i>	<i>p</i>
Early prep areas	32	0.67	0.0001	47	0.18	0.58
Opening	8 <sup>a</sup>	0.43	0.23	7	-0.07	0.88
Cleaning	8	0.36	0.39	8	-0.05	0.91
Carding	16	0.68	0.004	20	-0.06	0.82
Late prep areas	32	0.86	0.0001	47	0.94	0.0001
Drawing	16	0.51	0.04	16	0.65	0.006
Roving	16	0.75	0.0009	15	0.58	0.02
Spinning <sup>b</sup>	—	—	—	16	0.15	0.58
All areas	64	0.66	0.0001	81	0.79	0.0001

<sup>a</sup>*n* refers to paired samples tested for dust and endotoxin.

<sup>b</sup>Spinning in mill 1 not tested.

*r<sub>s</sub>* = Spearman's rank correlation.

manufacturing, the fit of the model was examined further. When standardized residuals from the regression model were plotted against predicted endotoxin values, the largest residuals were found to occur in the precarding stages of Mill 2. These were the areas with the lowest correlations using nonparametric methods (Table II).

## DISCUSSION

In an effort to isolate the causative agent(s) in byssinosis, experimental studies utilizing the model card room have demonstrated a relationship between exposure to endotoxin and acute respiratory symptoms [Haglind and Rylander, 1984] as well as, in some studies, spirometric function using both naive subjects and textile workers [Castellan et al., 1987; Rylander et al., 1985; Haglind and Rylander, 1984; Petsonk et al., 1986]. In contrast with results presented in this report, Castellan et al. demonstrated no correlation between dust and endotoxin concentrations ( $r = 0.07$ ,  $p = 0.46$ ) in a recent report of model cardroom studies [Castellan et al., 1987]. In this report, a better association was found between acute change in  $FEV_1$  and measures of airborne endotoxins than measures of dust exposure. Furthermore, 4 of 5 exposure sessions involving dust concentrations below the current OSHA permissible exposure limit ( $0.2 \text{ mg/m}^3$ ) resulted in substantial acute ventilatory decrements. Although the model cardroom findings are useful in furthering our understanding of byssinosis, these environments have been constructed to simulate only the carding area of a cotton yarn preparation mill [Castellan et al., 1987; Rylander et al., 1985; Haglind and Rylander, 1984]. Moreover, for purposes of the experimental design, cotton varieties were carefully selected from certain growing regions. Indeed, in this experimental setting it has been shown that the endotoxin level varies with the geographic source of cotton grown [Olenchock et al., 1985]. In individual challenge sessions, dust and endotoxin were well correlated, but when data from all exposure sessions were combined, there was no relationship [Castellan et al., 1987]. This would not be unexpected as (1) dust and endotoxin would tend to be correlated from the same cotton; (2) data from the area of growth and variety studies conducted within the same crop year tend to be better correlated than combined data from several crop years, since the level of endotoxin contamination is known to vary considerably from one growing season to the next for most regions of the cotton belt. In the typical mill setting, such as the 2 mills in this study, hundreds of bales of different cottons from various growing areas are blended, resulting in a mix within which there may a narrower range of endotoxin contamination of dust than between individual bales. Nevertheless, clear differences in the level of endotoxin contamination of dust between mills has been described [Castellan et al., 1988]. Indeed, the level of contamination differed between these 2 mills, although correlations between dust and endotoxin were genuinely good between both [Olenchock et al., 1990].

The authors of the model cardroom studies conclude that "consideration be given to limiting exposure to airborne endotoxin in work environments," while at the same time urging careful study of the correlations of dust and endotoxin in actual work areas in the cotton industry. Before modifying the standard to include endotoxin, it is important to note that measurement of elutriated dust is a relatively simple and inexpensive procedure. The measurement of airborne endotoxin, however, is complex. There is no accepted standard method for workplace sampling of airborne endotoxin, and the analysis requires a sophisticated laboratory not available in most industrial settings, particularly in developing countries. As a result, the overall cost of obtaining airborne endotoxin levels is far greater than the cost of an elutriated dust sample. Such considerations are especially important for China and other developing nations where the technology for sampling inhalable dust, let alone endotoxin, is often severely limited.

The results from this field study suggest that the correlation between dust and endotoxin in the typical cotton mill may be higher than seen in the model cardroom studies. In both mills, dust was highly correlated with endotoxin when all work areas were combined. When examining specific work areas in mill 1, the correlations were high in both early as well as the late stages. The correlation in the cardroom itself was 0.68 in mill 1. In mill 2, the correlations were high only for drawing and roving. In the one work area, spinning, where many dust samples levels were close to the current PEL of  $0.2 \text{ mg/m}^3$ , there was poor correlation between dust and endotoxin levels. This suggests that there may be a qualitative difference in the early stage environments between mills.

Dust and endotoxin levels in cotton mills vary as a result of several factors. The early stages of cotton processing (opening, cleaning, and carding) are intended to remove dirt and trash. It is in these 3 areas that dust and endotoxin levels tend to be high and variable. From the drawing areas on, the processes are designed to elongate and attenuate the cotton and usually generate lower, though finer particle size, dust levels. Other sources of variation in cotton mill dust levels include type and grade of cotton used, speed of production equipment, general ventilation, and local exhaust [DHEW-NIOSH, 1975]. Sources of variation in measured dust levels include the type and duration of dust sampling, and the location of samplers in production processes.

In addition to all of the above, endotoxin contamination of cotton has additional sources of variability. Climatic and botanical variables have been reported to affect endotoxin level [Delucca et al., 1979]. For example, the source of cotton growing [Petsonk et al., 1986, Castellan et al., 1986], as well as the time of harvest [Fischer et al., 1989], both influence bacterial growth and endotoxin concentrations on cotton leaf, bract, and fiber from opened bolls.

Given these additional factors which cause variation in endotoxin contamination, it would not be surprising to see poor correlation between the 2 measures of exposure unless there was a very similar level of bacterial contamination of the cottons used. In our study, the sources and levels of contamination varied considerably, a situation which probably mimics that of many cotton mills in the world today. It is therefore noteworthy that, in this field study, the 2 exposures exhibit a positive correlation both when results are combined overall as well as within a number of specific work areas. It is important to determine whether these correlations are more commonly high than low in the industry today.

To resolve the question of the correlation between dust and endotoxin levels, adequate numbers of samples need to be collected over several crop years in a number of different mills representing the full range of the climactic differences and botanical features of cotton. It may be that by accounting for the geographical source of the cotton, variety of cotton grade, time of harvest, or duration of storage, the degree of correlation expected in a given mill can be predicted. We agree with Castellan et al. [1987] that better characterization of actual plant conditions is needed in order to make an informed judgment about the adequacy of the cotton dust standard. Such study should include examination of dust-endotoxin relationships at and below the current OSHA dust standard of  $0.2 \text{ mg/m}^3$ . Interestingly, our data predict more than  $50 \text{ ng/m}^3$  would be present at the current OSHA dust standard of  $0.2 \text{ mg/m}^3$ . At this level significant mean responses in pulmonary function were found in subjects in the model cardroom [Castellan et al., 1987]. This finding of significant levels of endotoxin at the current PEL is consistent with other reports [Castellan et al., 1987].

We should also note that the relationships described in this study may not be indicative of exposures in other environments with "organic" aerosol exposure. Endotoxin levels have been estimated in similar fashion from various environments, such as poultry houses, swine confinement buildings, and grain elevators, where organic dust is the principal exposure [Rylander and Morey, 1983]. In the case of swine and poultry workers, airborne endotoxin exposure-response relationships for acute FEV<sub>1</sub> change have been reported [Donham et al., 1989]. Hence, other work environments where there is risk of exposure to endotoxin need to be studied in order to determine the relationships between airborne inorganic dust and Gram-negative bacterial endotoxin, and the relative importance of each in producing acute and chronic respiratory disease.

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