

# Byssinosis and Respiratory Diseases of Cotton Mill Workers

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Byssinosis is defined by the typical history of tightness in the chest, shortness of breath and cough occurring in cotton, flax and hemp workers on the first day back in the mill after an absence, hence, the name Monday tightness. Because byssinosis has been shown to be prevalent in cotton textile workers in the United States and because protracted exposure to cotton is associated with pulmonary impairment and chronic bronchitis producing higher rates of disability, the history and current concepts of the disease are reviewed. The study of byssinosis offers an important opportunity to relate the production of airway narrowing by an environmental agent to chronic pulmonary lung disease.

## The Importance of Byssinosis

Byssinosis has a world-wide distribution in cotton, flax and hemp workers, and has been observed in at least 16 countries. The reported prevalence of byssinosis varies from a few percent to practically all workers. Workers in British cotton mills concentrated in Lancashire have shown a consistently high prevalence of byssinosis.<sup>1,2</sup>

Byssinosis was made a compensable disease in England in 1941, but until recently there were only sporadic reports of byssinosis in the United States. In 1961, McKerrow and

Schilling found evidence of respiratory disease and impaired ventilation in cardroom workers in two U.S. textile mills.<sup>3</sup> In 1962 Schilling reported that only 14 cases of byssinosis had been recorded from the United States over a 15-year period.<sup>4</sup> Heaphy and Kilburn in 1965 described 18 hospitalized patients with pulmonary disease who had typical "Monday asthma" and cotton textile dust exposure indicating byssinosis.<sup>5</sup> In 1967, Bouhuys reported 22 nonhospitalized cotton workers with chronic respiratory disease, 14 of whom were diagnosed as byssinotic.<sup>6</sup> Increased interest in byssinosis was reflected in further reports beginning in 1969. In one survey, 29% of 214 workers in a prison cotton mill were found to have byssinosis.<sup>7</sup> Zuskin and coworkers found 25% of 59 carders and 12% of 99 spinners of two South Carolina cotton mills to have byssinosis.<sup>8</sup> Schrag and Gullett, in a North Carolina State Board of Health survey of 509 cotton mill workers, found byssinosis in 12%.<sup>9</sup> In a survey of 441 workers in a North Carolina textile plant, Merchant and associates found 6% had Grade 1 or 2 byssinosis, with a prevalence of 20% in preparation areas.<sup>10</sup> In another survey of 404 workers, byssinosis prevalence was 41% in carding, 22% in spinning and 21% in winding and twisting areas.<sup>11</sup> These prevalence rates are similar to those of foreign studies and indicate the magnitude of health impairment related to cotton dust inhalation in cotton mill workers in the United States.

Cotton remains the single most important textile fiber. Of nearly one million individuals currently working with textiles in the United States, it is

estimated that at least a third are exposed to cotton dust. The United States is the world's largest supplier of pure and mixed cotton yarns although the production of cotton fibers in many countries throughout the world is increasing. The problem of byssinosis, therefore, is not likely to be solved in the near future by the replacement of the cotton fiber by synthetic fibers.

## Historical Background

Occupational illness due to textile dusts was described by Ramazzini in 1705 "... those who hackle in the flax and hemp to prepare it for being spun and wove, afford frequent instances of the unwholesomeness of their trade; for there flies out of this matter a foul mischievous powder, that entering the lungs by the mouth and throat, causes continual coughs and gradually makes way for as asthma . . . But at the long run if they find their affliction grows upon them they must look out for another trade; for 'tis a sordid profit that's accompanied with the destruction of health."<sup>12</sup>

Health impairment among cotton mill workers of Lancashire, England was described in 1818.<sup>13</sup> Kay, describing "spinner's phthisis" in 1831, pointed out the relationship to dust, described the particularly high frequency of illness in cardroom workers, and reported that "methods of ventilation and of covering the machines have considerably diminished the evil."<sup>14</sup> In an official report presented to Parliament in 1860, Greenhow<sup>15</sup> described the illness we now know as byssinosis, but credit for coining the term "byssinosis" belong to Adrien Proust.<sup>16</sup> The word byssinosis is de-

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rived from the Greek word *βυσσος* or Latin *byssus*. For years the word *byssus* has meant a fine, soft, white fiber and probably originally referred to flax. *The Shorter Oxford Dictionary* gives the commonest use of *byssus* as "An exceedingly fine and valuable textile fiber and fabric known to the ancients, it denoted properly a kind of flax, but also of cotton, silk, etc."

In 1971 Ludwig Hirt, in discussing disease caused by inhalation of cotton dust, disliked Van Coetsen's term 'pneumonie cotonneuse' and used 'pneumoconiosis lyssinotica' and 'lyssinosis pulmonum'.<sup>17</sup> Proust, in 1877, realizing that Hirt had misused the *lyssa* (*λυσσα*) meaning rabies for *byssus* (*βυσσος*), called attention to the mistake and was the first to use the term *byssinosis*.

The word *byssinosis* became well known after its use by Sir Thomas Oliver in 1902 and 1908 in his books on occupational diseases. Although it was first used to describe a disease caused by the inhalation of cotton dust, *byssinosis* also occurs among flax and hemp workers and is used in this context.

The term "mill fever" was first used in 1892 by Arlidge<sup>18</sup> and outbreaks of

"weaver's cough" believed due to mildew of cotton threads were recognized as early as 1908.<sup>19</sup> Interest in these conditions seemed to wane until the report of the Departmental Committee on Dust in Cardrooms appeared in 1932.<sup>20</sup> This report described progression from early symptoms of incapacity and included ten post mortem studies. In 1936, Prausnitz conducted an extensive investigation of *byssinosis* and made a detailed report to the British Medical Council.<sup>21</sup> Carey's comprehensive 1965 report, "Byssinosis in Flax Workers in Northern Ireland", is an admirable, modern investigation of this illness.<sup>22</sup>

### Clinical Syndromes

In addition to *byssinosis* the respiratory symptoms of textile workers attributed to cotton mill dust comprise three syndromes which are not distinct. These are mill fever, weaver's cough and an acute illness among workers with low-grade, stained cotton that has been called "Mattress-maker's" fever. Because these syndromes have been associated with *byssinosis*, they will be described (Table I).

### Mattress-Maker's Fever

This condition, apparently not related to *byssinosis*, was reported by Neal<sup>23</sup> in 1942 as an acute, febrile illness associated with headache, gastrointestinal symptoms and retrosternal discomfort occurring among workers exposed to high dust concentration from low-grade cotton. Outbreaks occurred in several central depots where families gathered during the Depression to make mattresses for home use. The low-grade, stained cotton used was found to be contaminated with the gram negative bacillus, *Aerobacter cloacae*. The clinical symptoms were reproduced in humans by inhalation of contaminated cotton dust by clean cotton to which the contaminants had been added and by a filtrate of cotton dust extract. Although the clinical picture resembles "weaver's cough", no definite relationship has been established.

### Weaver's Cough

Since 1900, periodic outbreaks of an acute respiratory illness have occurred among weavers.<sup>19-24</sup> Various etiological agents associated with the cotton yarn have been implicated. Earlier reports were associated with

Table 1. — Principle Features of Respiratory Conditions of Cotton Workers

	Chronic Bronchitis	Byssinosis	Acute Febrile Syndromes		
			Mill-Fever	Weaver's Cough	Mattress-Maker's Fever
<b>Etiological Agents</b>	Multiple respiratory viruses and bacteria. Cigarette smoke. Chemical agents as SO <sub>2</sub> formalin, acrolein.	Unknown component in cotton, flax hemp dust.	Textile dust-ingredient	1. "Mildewed" yarn 2. Tamarind seed powder 3. Other sizings 4. Unknown contaminants	Endotoxin contaminated cotton dust
<b>Pathogenesis</b>	1. Viral infection 2. Bacterial infection 3. Chemical damage	1. Nonantigenic histamine release 2. Immunopathogenic 3. Endotoxin action	1. Atopy 2. Endotoxin action	1. Antigen-antibody 2. Endotoxin action	1. Endotoxin action
<b>Clinical Symptoms</b> 1. Fever 2. Cough 3. Dyspnea (or tightness) 4. Cough with sputum	Sometimes Yes  Frequent Yes	No Variable  Yes Frequent	Frequent Frequent  Rare No	Frequent Severe  Severe No	Yes Yes  Yes No
<b>Historical Features</b> 1. Acute onset 2. "Monday" onset 3. Acclimatization 4. New workers affected 5. Old workers affected	— — — — —	No Yes No  No Yes	Yes No Yes  Yes No	Yes No No  Yes Yes	Yes No No  Yes Yes

mildewed yarn.<sup>19 25 26 27</sup> Sizing added to thread prior to weaving has been incriminated in other outbreaks. Tamarind seed powder has been one culprit,<sup>28 29</sup> but the offender in yarn sized with other mixtures remains unidentified.

Characteristically, weaver's cough presents as a sudden epidemic of an acute asthma-like illness with a high attack rate. Both old and new workers are affected, acclimatization does not occur, and the illness does not recur when the offending agent is removed. In an outbreak studied in Italy in 1954, asthma-like symptoms lasted as long as six months.<sup>30</sup> Bacteriological and immunological studies were done but no definite conclusion was reached.

### Mill Fever

"Mill fever" is a term used to describe a symptom complex of unknown cause which occurs in susceptible persons unaccustomed to breathing cotton dust.<sup>18 31 32</sup> The terms cardroom fever, cotton fever, cotton cold, dust fever, dust chills and heckling fever in flax workers probably refer to the same condition. Individuals who are unaccustomed to these vegetable dusts may develop symptoms of malaise, cough, fever, chills and upper respiratory symptoms shortly after exposure. These symptoms may recur daily for days to months until acclimatization takes place and symptoms disappear. Tolerance may be temporarily lost after a period of absence from exposure or if exposure to a greater concentration of dust occurs. The exact prevalence of mill fever among new employees is unknown, but estimates range from 10 to 80%.

Some asthmatics react severely to cotton dust. Because of this dramatic response to cotton dust, many asthmatics undoubtedly avoid future exposure. Therefore the relationship of symptoms to the atopic state is difficult to interpret. However, there is no evidence that present cotton textile workers with byssinosis are more frequently atopic than those without byssinosis.<sup>33</sup>

The occurrence of an acute illness similar to "mill fever" in workers exposed to inhalation of bacterial contaminated dusts and in others exposed

to a high concentration of endotoxins has been said to suggest an etiologic relationship between endotoxin containing cotton dust and "mill fever."<sup>34</sup> The relationship between "mill fever" and the subsequent occurrence of the "Monday morning" illness of byssinosis is unknown. Some investigators believe they are stages of the same illness; others confuse the two or ignore "mill fever" completely. An understanding of the relationship between "mill fever" and byssinosis may provide clues to the pathogenesis of byssinosis.

## Byssinosis

### Definition and Natural History:

Byssinosis has been defined as a chronic respiratory disease associated with inhalation of cotton, flax and soft hemp dusts. Chest tightness, cough, wheezing and dyspnea in varying degrees of severity are the initial symptoms. The diagnosis depends upon the characteristic symptoms; the 1936 description of Prausnitz is classic:

The histories of their illness, given with a surprising uniformity by most of the patients, are as follows: After working for years without any appreciable trouble except a little cough, they notice either a sudden aggravation of their cough, which becomes dry and exceedingly irritating, or peculiar attacks of breathlessness. These attacks usually occur on Mondays, whilst the rest of the week finds them in fairly good condition. For a long time the trouble may be almost or entirely limited to this "Monday fever", but gradually the symptoms begin to spread over the ensuing days of the week; in time the difference disappears, and they suffer continuously. When, ultimately, they are forced to give up work, some improvement may occur. But if they resume work, they are forced sooner or later to give it up again. In some, this occurrence is most characteristic: A rest of some weeks or months gives them an opportunity of recovering so far that they feel perfectly fit; yet on the day that they return to the mill, they are struck down with the disease and are hardly able to return home unaided.<sup>21</sup>

Chest tightness, cough, wheezing or dyspnea are recognized on the first day of the work week. Symptoms usually disappear an hour or so after leaving work but may recur each Monday after a weekend away from dust exposure. With continued exposure,

the symptoms appear on Tuesday and may progress to other days of the week. Eventually, it is believed, irreversible obstructive airway disease appears. Initially, 20 or more years of dust exposure was required before respiratory impairment was attributed to byssinosis for purposes of workman's compensation in Great Britain. This requirement was later reduced to ten years.

Schilling<sup>35</sup> suggested a system for grading byssinosis which has become the standard classification for epidemiological surveys. The scheme shown below is based entirely on the history of symptoms which may be obtained by use of the British Medical Research Council questionnaire.

*Grade ½* — Occasional chest tightness or cough on the first day of the working week.

*Grade 1* — Chest tightness on every first day of the work week.

*Grade 2* — Chest tightness on the first and other days of the working week.

*Grade 3* — Grade 2 symptoms accompanied by evidence of permanent impairment.

Physical examination and chest x-rays of byssinotic individuals are initially normal. When pulmonary impairment develops these individuals are usually diagnosed as having chronic bronchitis and emphysema. A report in 1968 attributes a single case of pulmonary fibrosis of cotton dust inhalation but this must be considered unusual.<sup>36</sup>

Since McKerrow's 1958 report<sup>37</sup> that symptoms are frequently associated with reduced pulmonary function, objective measurements of such changes have become an integral part of the evaluation of the workers. A variety of methods have been used, but most epidemiological investigations have included the forced expiratory volumes in one second (FEV<sub>1.0</sub>). Recently flow-volume curves have been used.<sup>38</sup> Subjects with byssinosis usually show a decrease in expiratory flow rates and in vital capacity on Mondays after 3 or 4 hours of exposure to dust. There is less effect on the following days. The observed changes in airway resistance and decreased flow rates have been attributed to narrowing of small airways due to bronchoconstriction.<sup>39</sup>

Because in cross-sectional studies,

those workers who have decrements in flow rates with exposure show more evidence of pulmonary impairment than those with no significant flow rate response, it has been suggested that "reactors" are at greater risk from dust inhalation than "non-reactors".<sup>40</sup> Using decrement in flow rate as one major criterion and baseline FEV<sub>1.0</sub> value as the other, Bouhuys<sup>40</sup> proposed a functional grading system which he compared to the historical grading system of Schilling.<sup>41</sup> This scheme was later modified using FEV<sub>1.0</sub> before exposure as the major criteria and decrement in FEV<sub>1.0</sub> as a secondary criteria to classify exposed workers for the purpose of removing those showing evidence of pulmonary impairment and/or flow rate response from hazardous work areas, and preventing new workers with evidence of impairment or "reaction" from being assigned to hazardous areas.<sup>42</sup>

Although the association between acute symptomatic and spirometric responses to cotton dust and pulmonary insufficiency in textile workers is supported by strong clinical inference from cross-sectional studies, the only prospective study reported found no evidence that "reactors" or those with clinical byssinosis showed a greater decrease in pulmonary function over time than did other cotton workers.<sup>43</sup> Synthetic workers showed a decline of 29 ml per year while the decrement of all cotton workers was somewhat greater than 50 ml per year. However, when annual decrement in FEV<sub>1.0</sub> by diagnosis of byssinosis was considered, byssinotic and nonbyssinotic and "reactor" and nonreactor subjects had similar annual decrements. Unfortunately because it is not clear whether selection out of the population was taken into account interpretation of these findings is difficult.

### **Byssinosis, Chronic Bronchitis and Smoking:**

Both the limited pathological data and cross-sectional surveys of textile workers have indicated that chronic bronchitis is a frequent finding in these populations and specifically among those who have been diagnosed as byssinotic.<sup>10 44</sup> Those exposed to textile vegetable dusts show a strong association between gradient of respirable dust exposure and simple bron-

chitis.<sup>45</sup> This association has been similar to the association between dust exposure and byssinosis. When byssinosis and bronchitis have been related, the correlation has been strong and prompted Elwood<sup>44</sup> to suggest that byssinosis was essentially chronic bronchitis with superimposed acute symptoms. It appears that the two diseases are closely associated, but whether they share the same mechanism of lung damage and identical pathology is not known.

Many of the problems encountered in sorting out the multiple causal factors in coal worker's pneumoconiosis are met in studying byssinosis. Two problems in accurately classifying byssinosis to establish the etiology are the lack of definitive study of the pulmonary pathology of byssinotic patients, and variable definitions used for the clinical syndromes of byssinosis and of chronic bronchitis. If all grades of byssinosis are compared to all stages of chronic bronchitis, the association is expected to be less perfect than when Grade 3 byssinotics are compared with more severe bronchitics. The relationship with emphysema is more difficult to establish because there is no standard method to make a clinical diagnosis of emphysema. Clearly, the definitions used must be stated and qualified when such comparisons are made.

The role of cigarette smoking in causing chronic bronchitis is well known. The effect of smoking on byssinosis frequency and severity has not been as thoroughly studied, although most evidence indicates that smoking increases the workers risk of byssinosis. Carey<sup>22</sup> found that byssinosis occurred more frequently among smokers than nonsmokers as did Bouhuys<sup>7</sup> and Zuskin.<sup>8</sup> Zuskin found, however, that smoking habits did not affect the decrease in Monday FEV<sub>1.0</sub>. McKerrow and Schilling,<sup>3</sup> based on decrement in FEV<sub>1.0</sub>, suggested that smoking may potentiate the effect of cotton dust. Recent evidence suggests that the smoker is more likely to have byssinotic symptoms and that cotton dust and cigarette smoke may interact in their effect on byssinosis prevalence. For these reasons, it has been suggested that smokers be screened away from dusty work areas.<sup>10</sup> This apparent effect of cigarette smoking on byssino-

sis makes the determination of the relationship between byssinosis and chronic bronchitis more important.

### **Pathology:**

It is thought that patients who die with Grade 3 byssinosis have the pulmonary pathology of chronic bronchitis and emphysema, but no specific abnormality.<sup>20</sup> Specific dust bodies<sup>46</sup> said to be present in the lungs are probably non-specific ferruginous bodies unrelated to byssinosis or to bronchitis. Recent autopsies of card-room workers in whom neither smoking history nor clinical history was known, failed to reveal pathological changes other than the mucous gland hyperplasia and goblet cell metaplasia usually seen in chronic bronchitis.<sup>47</sup> Until a large, carefully controlled epidemiologic and pathologic study in which exposure to cigarette smoke, environmental and occupational exposures are known, as well as clinical histories, it will be difficult to differentiate between and understand the pathological interrelationships of end stage byssinosis, chronic bronchitis and emphysema.

### **Pathogenesis:**

One question in considering the pathogenesis of byssinosis is the nature of the pathology or other end-point to which experimental studies are to be directed. Should the investigator assume that elucidation of the mechanism of the acute airway response will disclose the pathogenesis of the chronic disease process as well? The conclusion that acute byssinosis becomes chronic is supported only by histories of patients who became disabled, but has not been studied prospectively over a sufficient number of years.<sup>38 43</sup> Because the chronic process is neither understood nor defined, the acute process manifested by chest tightness, bronchoconstriction and decrease in flow rate has been studied almost exclusively. Most investigators of the mechanism of byssinosis have assumed that the agent responsible for the acute effects of cotton dust is the same agent which is responsible for pulmonary disability and retirement. This is still an important question.

Other questions regarding the pathogenesis of byssinosis include the nature of the etiologic agent, its loca-

tion in the vegetable plant and the mode of transmission. Neither the active component of cotton dust nor its mechanism of action has been established although respirable dust particles, are considered to be the source and mode of transmission of the active agent.<sup>48</sup> Mechanical factors are not implicated because exposure to much higher dust concentrations of inert dusts and to the vegetable dusts of jute and sisal are not associated with clinical symptoms or physiological changes of byssinosis.<sup>37 49 50</sup> The quantities of inorganic components such as silica and other minerals are too small to be significant.<sup>21</sup>

At present there are three hypotheses for the mechanism of the acute process in byssinosis: (1) a nonantigenic histamine release; (2) endotoxin activity and (3) immunologic mechanism.

#### Histamine Release:

The role of pharmacologic histamine liberation in the pathogenesis of byssinosis is unsettled despite much discussion in recent articles. Two studies suggest that byssinotic subjects have no greater sensitivity to inhaled histamine than nonbyssinotic subjects.<sup>51 52</sup> The presence of a histamine-like substance in cotton dust was demonstrated by Maitland, Heap and MacDonald and subsequent investigators reported its presence in cotton and various other vegetable dusts.<sup>53 54 55</sup> Antweiler found the small amounts of histamine present in cotton and other vegetable dusts to be insufficient to cause symptoms.<sup>56</sup> Histamine release, from lung tissue by cotton dust was suggested as early as 1937 by Haworth and MacDonald.<sup>55</sup> They believed histamine accumulated in lungs over the weekend and was released by Monday's contact with an "allergen" in cotton dust. Daily exposure the remainder of the week prevented reaccumulation of histamine in the cotton worker's lungs. Biological activity of this factor has been demonstrated by Nicholls et al in extracts of raw cotton, cardroom dust and parts of the cotton plants particularly bracts, but no activity was found in extracts of pericarps.<sup>57</sup> There is no direct documentation of histamine release in vivo although a metabolic product of histamine does increase

after exposure.<sup>58 40</sup> Since it is well known that histamine produces acute changes in air flow because of its bronchoconstrictor and edemagenic activity, it has been assumed that this occurs in vivo as well. It is true that the same cotton extracts has in at least one study produced symptoms compatible with byssinosis and reduced flow on spirometric testing.<sup>59</sup>

Characteristics of the active agent which releases histamine from lung tissue include water solubility, resistance to boiling and sensitivity to boiling in acid or alkali.<sup>60</sup> Also, Hitchcock reported that a steam volatile component of cotton dust extract which had physicochemical behavior similar to methylpiperonylate.<sup>61</sup> In a study of textile workers, Hamilton et al demonstrated that the active principle was filterable at 0.22 $\mu$ , nonvolatile at 40C and nondialyzable using endobronchial challenge as the assay.<sup>52</sup> One objection to the theory of nonantigenic histamine liberation by cotton dust is the lack of a satisfactory explanation of permanent lung damage resulting from histamine release along. Antigenic or nonantigenic histamine release may explain the acute response but is hard to implicate in the chronic response. Several agents may be involved; one agent may produce the acute response mediated via histamine as a reversible protective phenomena without chronic effects, another agent or agents may be responsible for the chronic effects which resemble or are identical with chronic bronchitis. Additional studies of histamine or other chemical mediators released in human lungs and bronchi by plant extracts, dust and their chemical components may be helpful.

#### Endotoxins:

Because bacteria and fungi are present in large number in cotton dust, several investigators have attempted to show a significant relationship between viable agents and byssinosis.<sup>21 62 63</sup> The lack of an appropriate incubation period and predictable febrile illness, however, along with daily recurrence of chest tightness and rapid remission after leaving the mill was against this possibility. Endotoxin, a bacterial or fungal product, was considered as an alternative. Pernis et al<sup>34</sup> and Cavagna et al<sup>64</sup> proposed that the symptoms of byssinosis were due to release of a histamine-like substance following inhalation of gram negative bacterial endotoxin found in cotton dust. Their reasons included the consistent presence of endotoxin-like substances in cotton dust, correlation of endotoxin-like levels with byssinosis prevalence, the demonstration of inconstant pulmonary function changes upon challenge of human volunteers with purified endotoxin (not from cotton dust) in animals previously sensitized with endotoxin. Subsequent studies by the same authors demonstrated that rabbits develop hemagglutinating antibodies after inhalation of endotoxin or cotton dust extract, and that the challenge of endotoxin sensitized rabbits increases airway resistance. The usual endotoxin content of cotton dust, however, is considerably less than the experimental dose, furthermore, there is no convincing evidence that the response to the inhalation of appropriate endotoxin concentrations by byssinotics is different than that of nonbyssinotic subjects.<sup>65</sup> Cavagna et al also reported pathologic changes in lungs of animals exposed to endotoxin or to cotton extract by aerosol.<sup>64</sup> The changes included bronchial cell exfoliation, endobronchial secretion and peribronchial lymphocyte infiltrates.

Development of hypersensitivity to a component of cotton dust was suggested as a cause of byssinosis in the Departmental Committee Report of 1932.<sup>20</sup> Prausnitz thought there was a significant relationship between positive skin tests to cotton plant materials and symptoms of byssinosis.<sup>21</sup> Other studies have not supported a positive correlation of disease with skin tests.<sup>66</sup> In an extensive recent study of allergy to cotton, hemp, flax and jute assessed by skin tests immediate skin reactions were occasionally observed, delayed skin reactions were nearly always present in both byssinotic and nonbyssinotic workers and both types of skin tests were usually absent in normal, nontextile subjects.<sup>67</sup> Positive skin reactions to hemp, flax and jute were found in cotton workers. Although many textile workers and even some normals show skin reactions to textile dusts, toxic factors may be involved. Low correlations between skin reactivity

#### Immune Mechanism:

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and pulmonary responses except in asthmatic subjects do not support an allergic pathogenesis for byssinosis.

An immunopathologic mechanism unrelated to skin reactivity has been investigated. Taylor and associates<sup>68</sup> reported a condensed polyphenol as the component of cotton dust which reacted with agglutinating antibodies in byssinotic subjects as opposed to nonbyssinotic subjects. Byssinotic subjects had higher titers than nonbyssinotic subjects and normals. In a double blind endobronchial challenge study symptoms of byssinosis were reproduced only in those subjects with known byssinosis but no objective changes in pulmonary function could be demonstrated. The authors suggested that the symptoms of byssinosis were produced directly by the antigen-antibody complex or indirectly by the release of a pharmacologically active substance but did not rule out the possibility that antibody levels could be the result and not the cause of byssinosis. Popa could not demonstrate precipitating antibodies to extracts from cotton, flax, hemp and jute.<sup>67</sup> He found that subjects who were normal, asthmatic, bronchitic, and byssinotic had similar titers of circulating antibodies against textile dust antigens but, no definite causal relationship of these to disease has been established.

Early observers related the prevalence of disease to visible dust. This correlation has since been confirmed by measured dust levels. Attempts to control dust in cotton mills which began in England have continued in the United States. Vacuum exhaust systems on carding engines, hoods, exhaust fans and filtration of air are used extensively. Attempts to control dust by adding mineral oil or water and circulating and filtering air have not solved the problem. Because 20% of the weight of low grade raw cotton may be lost as dust and debris, the magnitude of the problem is obvious. Carding at increased rates also increases dust levels in work areas and often offsets exhaust ventilation. Symptoms were first thought to correlate with total airborne dust concentrations, then with medium-sized particles and later with fine (respirable) particles.<sup>48 69</sup> A dust threshold limit value (TLV) of 1 mg/M<sup>3</sup> for total dust has been recommended by the American

Functional Severity	FEV <sub>1.0</sub> * (% of predicted)	FEV <sub>1.0</sub> † (%)	Interpretation of FEV <sub>1.0</sub>	Recommendations for Employment
F <sub>0</sub>	>80 (no evidence of chronic ventilatory impairment)	a) -4 to 0; or +	minimal or no acute effect of dust on ventilatory capacity	No change; annual FEV <sub>1.0</sub>
		b) -9 to -5	moderate acute effect of dust on ventilatory capacity	No change; 6 mo. FEV <sub>1.0</sub>
		c) -10+	definite and marked acute effect of dust on ventilatory capacity	Move to lower risk area; 6 mo. FEV <sub>1.0</sub>
F <sub>1</sub>	60-79 (Evidence of slight to moderate irreversible impairment of ventilatory capacity)	a) -4 to 0; or +	As above	No change; 6 mo FEV <sub>1.0</sub>
		b) -5+	As above	Move to lower risk area; 6 mo. FEV <sub>1.0</sub>
F <sub>2</sub>	<60 (Evidence of moderate to severe irreversible impairment of ventilatory capacity)	-	-	Work requiring no cotton dust exposure, detailed pulmonary examination

\* FEV<sub>1.0</sub> in the absence of dust exposure (2 days of longer)

† Difference between FEV<sub>1.0</sub> before and after 6+ hours of cotton dust exposure on a first working day.

Conference of Governmental Industrial Hygienists but has not been achieved in many textile plants.<sup>70</sup> Evidence that the "respirable" or fine dust is the most important fraction has led to the development of sampling equipment for this size range.<sup>11</sup> It has been suggested that the concentration of respirable dust be compared with decrement in FEV<sub>1.0</sub> and bronchitis prevalence, as well as byssinosis prevalence to determine an improved TLV<sup>42</sup> Improved dust control may require redesign of ventilation systems and new methods of dust removal by exhaustion, condensation, or precipitation. A major technological advance in the spinning of cotton yarn could reduce this industrial hazard.

#### Dust Removal:

Dust removal from cotton before it enters the mill may well be the most important method to reduce dust-related diseases of textile workers. An economical method for removing or detoxifying cotton textile dust would eliminate byssinosis before the mechanism is understood. Studies on the application of steam to cotton show promise as one means to eliminate byssinosis without altering other

manufacturing processes.<sup>71</sup> If, as has been proposed, portions of the cotton plant, such as bracts, are responsible for the active component in cotton dust, the genetic development of a "safe" plant is possible. The admixture of debris resulting from the increased use of the mechanical cotton picker might be eliminated by improved design or by picking the cotton while green and removal from the boll without contamination by vegetable trash. New seed removal techniques applied before bracts and leaves dry and powder could also be effective. Until the active material can be removed from cotton before arrival at the mill or the dust removed from the worker's environment, byssinotic workers with significant pulmonary impairment must be removed before they have irreversible insufficiency.

#### Medical Approach:

Present methods of medical examination can identify those workers who react to dust and remove them before they have permanent impairment. Pre-employment examinations to identify and exclude from dusty areas those who already have evidence of respira-

tory disease, and periodic evaluations to detect workers with bronchitis and early byssinosis are feasible at this time. The initial medical evaluation should include detailed inquiry into occupational, environmental and cigarette smoke exposures, and measurements of flow rates during forced expiration including the well standardized FEV<sub>1.0</sub>. Periodic examinations should include ventilatory studies before and after dust exposures. Those workers with a marked response to dust should be removed before they have developed significant disability, Table 2. Application of present medical knowledge should prevent or delay development of pulmonary impairment among textile workers.

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**Moving Mountains** — Popular notions about Man's relationship to Nature seem to have reversed in the last century. Until then human power was so puny as to seem negligible when pitted against Nature, which was generally regarded as a hostile force to be subjugated whenever possible. Settlers who cleared the rocks from New England fields and sailed in clipper ships hardly had reason to worry about ruining Nature. They had to fight her every inch of the way. In those days the idea that human activities might conceivably bring an end to life on earth would have seemed ridiculous.

Now suddenly the tables have turned. People are coming to realize that they have the power collectively to move mountains, quite literally, and even contaminate the seas, or the atmosphere perhaps irretrievably. In this country the realization probably began with the disappearance of the buffalo and the carrier pigeon. The release of radioactive products into the environment and the widely publicized discussion of genetic effects after Hiroshima have doubtless contributed a great deal to the change in our attitudes about Nature as well. But in recent months the concept of our ecosystem as a fragile equilibrium in serious danger has been strikingly heightened by a whole series of events. The rupture of the oil tanker off the coast of England, the leaking oil wells off Santa Barbara, the evidence accumulated against residual pesticides, and the increasingly obvious smog are examples. — Hunter, TH: Apollo and the Environment (editorial), *The Pharos of Alpha Omega Alpha* 33:59 (Apr) 1970

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