# OCCUPATIONAL RESPIRATORY DISEASE IN NYLON FLOCK WORKERS

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## Abstract

The National Institute for Occupational Safety and Health (NIOSH) has recently been involved in investigations initiated after an astute physician identified a cluster of work-related interstitial lung disease (ILD) among workers at a small plant in Rhode Island that produces nylon flock and nylon-flocked upholstery fabric. A cluster of ILD had been independently reported from a similar plant in Canada, and additional sporadic cases have occurred at other nylon flock plants. This paper reviews results of the NIOSH epidemiological investigation at the Rhode Island plant, summarizes findings from a workshop convened to review lung biopsies and other clinical features of all cases known to have been diagnosed by January 1998, and reports on NIOSH plans for studies at other flock plants.

## Introduction

Flocking is a process in which fine particles, most commonly short textile fibers, are applied to surfaces for decorative or functional purposes. The term *flock* refers to the short fibers applied in flocking processes. Currently, most flock is made from nylon fiber (60%); lesser amounts are from rayon (30%), polyester (5%), or other fibers (5%) (Geppert 1997). Both three-dimensional objects (e.g., automobile window channels and glove boxes, toys, etc.) and rolled goods (e.g., upholstery fabric, material for blankets, contact paper, etc.) can be flocked. Until recently, there has been little concern about respiratory hazards associated with flocking. This lack of concern has apparently been rationalized, in part, on the fact that flock fibers themselves are generally too large to be respirable. This rationalization has either been accompanied by ignorance regarding the generation of fine occupational dust in flock processes or reinforced by an assertion that fine (non-flock) airborne particles present in a flocking plant are building dusts unrelated to the flocking process (Rettenmeier and Drysch 1997). Likewise, material safety data sheets for nylon fiber indicated not only a lack of hazard for the material as supplied, but also that respirable fibers are not generated from the material under normal conditions of use (Kern et al. 1998).

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## Background

### Canadian Plant

In the early 1990s, a cluster of occupational lung disease was recognized among employees at a plant in Canada that manufactures nylon flock and nylon-flocked upholstery fabric (Lougheed et al. 1995). Over a period of 18 months, five of 88 employees at the plant presented with interstitial lung disease (ILD). All affected workers had symptoms of cough and dyspnea, and two also had constitutional symptoms. Symptom onset occurred three months to five years prior to diagnosis, and employment tenure at the plant ranged from 18 months to 8 years. One patient required mechanical ventilation for several weeks.

Lougheed et al. (1995) reported that all five cases had inspiratory rales and mild leukocytosis. None were found to be febrile. Pulmonary function testing with spirometry and lung volumes documented restriction in all five. Diffusing capacity was reduced to between 30% and 70% of predicted. Chest radiographs revealed evidence of interstitial nodularity in all cases and alveolar filling in some.

The first three cases underwent lung biopsy; all three showed diffuse interstitial infiltrate with mononuclear cells (lymphocytes and plasma cells) but without granulomas (Lougheed et al. 1995). Clinical response to corticosteroids was noted as slow in all three cases. The following two cases, were not biopsied or treated; both improved after removal from work, but relapsed after returning to work (Lougheed et al 1995).

A limited environmental assessment of the plant revealed *Fusarium* contamination in the water-based adhesive used in the flocking process. On the speculation that inhalation of *Fusarium*-associated toxins may have caused the cluster of interstitial lung disease, the contaminated adhesive was discarded and steps were taken to prevent further contamination. The initial absence of additional cases reported by Lougheed et al. (1995) supported that speculation on etiology. However, in the continued absence of fungal contamination at the plant, four subsequent cases of work-related interstitial lung disease have occurred (see below under NIOSH Workshop).

### Rhode Island Plant

In 1996, an occupational medicine specialist became concerned about a possible occupational etiology after evaluating the second of two patients who had both developed interstitial lung disease while employed at a flock plant in Rhode Island. This plant employed about 160 workers; like the plant in Canada, it manufactures nylon flock and nylon-flocked upholstery. With the initial cooperation of the company, the physician initiated active case finding and advised the company to request that NIOSH conduct a Health Hazard Evaluation (HHE) to

investigate the cause of this disease (see below, under NIOSH Health Hazard Evaluation).

As of mid-1998, a total of 10 employees at this plant had been diagnosed with interstitial lung disease, including 9 cases diagnosed from 1992 through 1998, plus one other case diagnosed in 1985 and identified retrospectively (Kern et al. 1997; Kern et al 1998). Common major symptoms were cough and shortness of breath. Systemic symptoms (e.g., recurrent fevers and diffuse aches) were also reported by some, and were prominent in one affected worker whose work area had been enclosed in plastic sheeting several weeks before he became ill (NIOSH 1998). Time from symptom onset to diagnosis ranged from four months to 8 years, and employment tenure at the plant before symptom onset ranged from about 10 months to over 30 years (Kern et al. 1998).

Kern et al. (1998) reported that most cases had restrictive lung function and half had low lung diffusing capacity--as low as 29% of predicted. Chest radiographs were abnormal in some, and high resolution computerized tomography (HRCT) scans revealed evidence of interstitial abnormalities in nearly all.

Of the 10 cases, 9 underwent lung biopsy and were found to have interstitial abnormalities--peribronchovascular lymphocytic nodular infiltrates, most with germinal centers. No granulomas or birefringent particles were seen. In the case that did not undergo biopsy, bronchoalveolar lavage (BAL) fluid abnormalities (28% eosinophils and 35% neutrophils) were indicative of an active alveolitis.

All cases were reported to have improved substantially, though not completely, within weeks to months after leaving their jobs at the plant (Kern et al. 1998).

## NIOSH Health Hazard Evaluation

### Rhode Island Plant Description

Nylon flock is made at the Rhode Island plant by cutting wet nylon tow (i.e., bundles of continuous filaments) into very short segments using rotary precision cutters. Nylon tow processed at the plant contains titanium dioxide delusterant and ranges in diameter from approximately 10 to 20 micrometers depending on specifications for the final product. Prior to cutting, a flock finish (containing an ammonium ether of potato starch, tannic acid, and a fatty alcohol derivative) is applied in a continuous process bath to impart desired electrostatic properties. The cut flock is then dried, milled (to separate flock fibers that have been fused during cutting), screened (to exclude undesired residual fused flock and excessively long flock), and bagged. Flocked fabric is made at the plant in a roll-to-roll process using a woven cotton/polyester fabric substrate, water-based adhesive, and nylon flock. In the flocking process, flock is sifted and allowed to drop through an alternating current electrical field which aligns the flock more or less perpendicular to the fabric as it imbeds end-on into the adhesive. Beater bars under the adhesive-coated fabric help assure penetration of flock into the adhesive. After heat curing, the flocked fabric may be printed, embossed, or otherwise finished.

Many processes at the Rhode Island plant involved pneumatic conveyance of flock within air ducts terminating in cyclones intended to separate flock from the airstream. At bagging stations, for example, dried flock drops from the bottoms of cyclones into open bags, allowing escape of visible flock into the workplace air at the bag opening. Importantly, the tops of these cyclones were open to the workroom air, so that any small particles entrained within the airstream could be exhausted directly into the air within the plant. Bagged flock is manually dumped from bags into hoppers feeding the flocking process, and visible loose flock was noted to escape from the flocking modules, blanketing the floor and surfaces of equipment within the temperature- and humidity-controlled flocking rooms. Between process runs involving different flock of different color, compressed air hoses were used by workers to clear loose flock from equipment and other surfaces in a very dusty procedure referred to as a *blow-down*.

### Methods

NIOSH investigators carried out an environmental survey at the Rhode Island plant (NIOSH 1998; Burkhart et al. in press). To complement the clinical case series investigation carried out by Kern et al. (1998) and the environmental survey, NIOSH investigators also carried out a medical/epidemiological investigation of current employees at the Rhode Island plant (NIOSH 1998). The NIOSH medical survey included a standardized questionnaire, chest x-ray, spirometry, and lung diffusing capacity test offered to each current employee.

Workers were assigned to a departmental category on the basis of their job assignment. For many analyses presented below, departments were grouped as follows: production and maintenance (P/M) workers, as one group (i.e., those with generally higher exposures to air contaminants at the plant); and office, shipping, and warehouse (O/S/W) workers, as another group (i.e., those with generally lower exposures). For each individual symptom or symptom complex, a prevalence ratio (PR) (i.e., prevalence among the more highly exposed P/M workers divided by that among the less exposed O/S/W workers) was calculated.

### Environmental Survey Findings

Details are reported elsewhere (NIOSH 1998; Burkhart et al. in press). In summary, air concentrations of metals, various organic vapor compounds, viable bacteria, and viable fungi were not especially remarkable. Concentrations of airborne endotoxin were not especially high; by work area, the highest mean endotoxin concentration was measured in flocking rooms--approximately 85 EU/$m^3$, based on total dust sampling.

By process area, mean total dust concentrations were particularly high in the flocking rooms under normal operations (41.0 mg/m$^3$), and even higher during blow-down (76.2 mg/m$^3$). Mean total dust concentrations were lower in screening/milling areas (2.8 mg/m$^3$) and much lower in cutting (0.3 mg/m$^3$) and other areas (0.4 mg/m$^3$). Mean respirable dust concentrations were particularly high in the flocking rooms under normal operations (5.4 mg/m$^3$), and even higher during blow-downs (6.8 mg/m$^3$). Average respirable dust concentrations were lower in screening/milling areas (1.8 mg/m$^3$) and much lower in cutting (0.2 mg/m$^3$) and other areas (0.3 mg/m$^3$).

Microscopic examination of airborne dust samples revealed large flock fibers (approximately 15 micrometers in diameter and 1 mm in length), as well as much smaller particles. In respirable dust samples, numerous elongated particles were identified as nylon, primarily on the basis of melting temperature. Microscopic examination of flock samples collected after cutting but before milling revealed that tails of nylon were common at cut ends of flock; clumps of fused flock showed evidence of related effect, apparently caused by the flock cutting process.

**Medical Survey Findings**

Questionnaires were completed by 151 (89%) of current employees at the plant. Most workers reported working long hours and extended workweeks at the plant; 68% worked six days per week and 17% worked 7 days per week; 14% reported working 65 or more hours per week. The average tenure at the plant was 8.3 years (range <1 to 37 years); 52% of workers reported having worked for more than five years at the plant; 13% had worked less than one year. Fewer than 30% of workers reported having never smoked tobacco products. Compared to P/M workers, O/S/W workers were much less likely to report blow-down exposure (34.4% vs. 73.3%) or having worked on the flocking ranges (37.5% vs. 68.9%). On average, P/M workers reported working only two hours longer per week than O/S/W workers (54.4 vs 52.3). There were no major differences in either tenure or smoking status by departmental group.

Compared to O/S/W workers, P/M workers had a higher prevalence of each individual frequent respiratory, systemic, and irritant symptom (with onset after hire at the plant) (Table 1). The prevalence ratio (PR) (i.e., prevalence among the P/M workers divided by that among the less exposed O/S/W workers) exceeded 1.0 for each frequent symptom, and more than half these elevated PRs were statistically significant.

Many workers who reported frequent symptoms also reported improvement in symptoms when away from work. Work-related frequent eye irritation (PR = $\infty$; $p<0.05$), frequent throat irritation (PR = $\infty$; $p<0.02$), and frequent fevers (PR = $\infty$; $p<0.01$) were all reported only by P/M workers. Work-related frequent generalized aches were reported only by employees assigned to work on the flocking ranges (PR = $\infty$; $p=0.58$). None of the PRs for the other work-related frequent symptoms were significantly elevated, due in part to smaller underlying prevalences. However, all were larger in magnitude than the corresponding PRs shown in Table 1.

With respect to workers who reported having at least one frequent respiratory or systemic symptom (with onset since hire at the plant), significantly increased prevalence was found to be associated with assigned departmental category, with working on the flocking ranges, and with performing blow-downs, but not with either smoking or tenure (Table 2). Notably, these symptoms were also significantly associated with number of days worked per week and with number of hours worked per week, both demonstrating highly significant trends ($p<0.001$). Workers who reported working 7 days per week were about 3.5 times as likely to report at least one of these symptoms as workers who reported working five or fewer days per week. Likewise, those who reported working at least 65 hours per week were also more than three times as likely to report one or more of these symptoms as those who reported working no more than 45 hours per week.

Diagnosis of pneumonia in the last five years (excluding any diagnoses made before hire at the plant) were reported by 13 of 119 P/M workers and by only one of 32 O/S/W workers (PR = 3.5; 95% CI = 0.5 - 25.7). Multiple flu-like illnesses in the past year were reported by 35 P/M workers and by only four O/S/W workers PR = 2.4; 95% CI = 0.9 - 6.1). Multiple attacks of shortness of breath with wheeze in the past two years were reported by 33 P/M workers and by only three O/S/W workers (PR = 3.0; 95% CI = 1.0 - 9.0).

Analysis of lung function test results revealed a lower mean percent predicted forced vital capacity (FVC) ($p=0.07$) and a lower mean percent predicted diffusing capacity ($DL_{CO}$) ($p=0.02$), but an equivalent ratio of forced expiratory volume in the first second ($FEV_1$) to FVC (i.e., $FEV_1$/FVC) ($p=0.86$) among symptomatic workers compared to asymptomatic workers (Table 3). This lung function pattern (i.e., reduced $DL_{CO}$ and FVC, with maintained $FEV_1$/FVC ratio) among symptomatic individuals is consistent with a mild interstitial disease process.

Restricting analysis to never smokers (only 42 with spirometry and only 33 with diffusing capacity measurements), this symptom-associated lung function pattern was not evident, but mean $DL_{CO}$ did tend to be lower among symptomatic never-smokers than among asymptomatic never-smokers. Restricting analysis to the more numerous ever-smokers, this pattern of lower FVC ($p<0.04$) and lower $DL_{CO}$ ($p<0.02$) among those with symptoms was clearly evident (Table 3). [In an analysis restricted to ever-smokers (n=62), frequent respiratory/systemic symptom prevalence remained

significantly associated with both days and hours worked per week, with work on the flocking ranges, and with blow-down exposure; also, PRs for each of these factors were comparable in magnitude to those shown for the overall group in Table 2.]

None of 143 interpretable chest radiographs (out of 145 taken) were interpreted as abnormal.

## NIOSH Clinical Pathology Workshop

### Methods

In late January 1998, NIOSH convened a workshop of experts to review all known cases. Details are presented elsewhere (Eschenbacher et al. in press). In January 1998, a total of 20 workers from four different plants employing a total of less than 500 workers were known to have been diagnosed with ILD. These cases included 9 from the plant in Canada, 9 from the Rhode Island plant, and two other cases from other plants in Massachusetts (Eschenbacher et al. in press). At illness onset, each of these 20 cases worked in plants involved with nylon flock production and/or with application of nylon flock to rolled goods. Lung tissue specimens (8 wedge biopsies and 7 transbronchial biopsies) were available from 15 of these cases; five cases had not undergone biopsy. Cases with wedge biopsies were distributed by plant in proportion to the number of cases from each plant.

Workshop participants included three clinical pulmonary pathology consultants, the three hospital-based pathologists who had diagnosed most of the cases, clinicians who had diagnosed and/or managed most of the cases, a medical officer from the Ontario Ministry of Labour, and selected NIOSH staff. At NIOSH, slides of the tissue specimens from the 15 cases were masked, randomized, and coded to obscure the original hospital and worker identities. The consulting pathologists independently assessed each case and qualitatively scored specific histopathologic findings on a standardized form.

After all scoring was complete, slides were unmasked and the consulting pathologists were joined by other workshop participants. For each case, pathologists presented their biopsy findings after clinicians had presented history, clinical findings, management, treatment, and course of illness. Workshop participants discussed each case individually and all cases in aggregate.

### Histopathology Findings

The consulting pathologists, together with the pathologists who had been involved in the clinical diagnosis of most of the cases, reached a consensus that a distinctive pathological lesion was common to these cases. This underlying lesion was described as a lymphocytic bronchiolitis and peribronchiolitis with lymphoid hyperplasia represented by the presence of lymphoid aggregates. Other histological features were variably present. These included acute alveolar injury, showing patterns of diffuse alveolar damage (DAD) or bronchiolitis obliterans with organizing pneumonia (BOOP), and increased alveolar macrophages, with some areas suggestive of desquamative interstitial pneumonitis (DIP). Fibrosis was not a predominant feature, and giant cells and granulomas and were absent from all but one case, in which their presence was rare.

The pathologists agreed that 7 of the 8 cases with wedge biopsies met their consensus criteria for the distinctive lesion described above. The pathologists noted consistent but non-specific abnormalities, including lymphocytic cell infiltrates in all 7 cases with transbronchial biopsies. However, tissue quantities from transbronchial biopsies were inadequate to describe changes in bronchiolar and lobular architecture necessary to identify the distinctive underlying lesion seen in the wedge biopsies.

### Other Clinical Information

All 20 cases had experienced progressive shortness of breath and cough, present for months to years prior to diagnosis. In several cases, an exacerbation of symptoms over several days motivated a medical evaluation. In some cases, respiratory symptoms were accompanied by systemic symptoms including weight loss and generalized aches. Thirteen of the 20 cases were current or former smokers; several had quit smoking shortly after onset of their symptoms. Lung function testing provided clear evidence of restriction in half the cases; several others had lung capacities in the low normal range. Of those tested for lung diffusing capacity, more than two thirds (13 of 19) had results that were abnormally low (<75% of predicted). Radiographic examinations, including standard chest radiographs and/or HRCT examinations revealed abnormalities in the lung consistent with interstitial disease.

Removal from the workplace, with or without treatment with anti-inflammatory corticosteroid medication, resulted in gradual improvement in symptoms, lung function, and radiographic abnormalities. The period of time required for substantial clinical improvement varied, but generally required at least several weeks to several months. Apparent full recovery has occurred in some cases, but others have experienced years of persistent shortness of breath, exercise limitation, or requirements for supplemental oxygen, even after removal from work. Relapses in symptoms and objective abnormalities were experienced by all six cases who attempted to return to their jobs following initial recovery after removal from work. More than one affected worker has apparently been successfully accommodated with a job transfer away from production areas (i.e., to office or warehouse jobs).

## NIOSH Studies in Other Flock Plants

NIOSH is currently involved in investigations at facilities of two other flock industry companies: one company that produces flock from nylon and other tow, as well as from scraps of cotton fabric, in several different plants employing a total of approximately 166 workers; and another company that flocks rolled goods nylon and other types of flock in one plant employing about 100 workers.

Following walk-through examinations of the industrial processes at each of these plants, NIOSH industrial hygienists and medical epidemiologists completed data collection for the first phase in November 1998. This included air sampling for respirable dust and fiber counts to quantify exposures in each job title occupied by workers in these plants and administration of standardized health questionnaires to all current employees on a voluntary basis. Data will be analyzed to quantify worker exposures to respirable dust and fibers, to better understand how respirable shreds of flock are generated, and to determine if there is a relationship between health status reported by the workers and their exposure to respirable dust and/or fibers at work. Findings from this first phase will also be used to decide on a possible second phase of data collection involving more detailed examinations of worker health status. In complementary laboratory studies, dusts from these plants will be tested for inflammatory potential in the lungs of animals. Overall findings from these investigations will be used to help make appropriate recommendations to guide prevention of adverse respiratory health outcomes associated with occupational exposures in the flock industry.

## Discussion

Cases from the two plant-based clusters and from the other two plants all had similar symptoms, predominant restrictive lung function, and a distinctive histopathology, suggesting a common occupational etiology. Other evidence also indicates that this condition is work-related. The occurrence of 20 cases of this unusual disease represents an obviously impressive incidence among an estimated 500 workers in the aggregate cross-sectional workforces of the four plants. Also, the condition was temporally related to work at the flock plants. For a few cases, symptom onset or worsening occurred following changes in their jobs associated with increased occupational exposure to flock-associated dust. All affected workers who were removed from the workplace improved with respect to symptoms and objective abnormalities, even without other medical treatment; and, of the handful of affected workers who returned to their previous jobs after initial improvement following temporary removal from work, all experienced recurrence or worsening of symptoms and objective abnormalities. Some have tolerated transfer to low exposure jobs (e.g., to office or warehouse jobs) without recurrence or worsening.

The distinctive histopathological pattern identified at the NIOSH workshop involves bronchiolar and peribronchiolar lymphocytic inflammation and lymphoid hyperplasia, which suggests an inflammatory response to an inhaled toxic respirable particulate. The NIOSH environmental survey at the Rhode Island plant documented that respirable particulate is generated by processes in the plant and that air concentrations of respirable dust were particularly high in some production areas (e.g., flocking). Laboratory support for the hypothesis that this lung disease is caused by toxic respiratory particulate has been provided by recent animal studies, in which marked acute inflammation in the lungs of rats was induced by intratracheal instillation of airborne dust from the Rhode Island plant, as well as by intratracheal instillation of dust generated by milling nylon tow free of flock finish (Porter et al. in press; Castranova et al. 1999).

The NIOSH medical survey showed that exposed workers had frequent work-related symptoms that were consistent with an interstitial disease process, as evidenced by lung function pattern (i.e., reduced FVC and $DL_{CO}$ with maintained $FEV_1$/FVC ratio). The results of the medical survey also indicated that these symptoms were associated with exposure to dusty processes (e.g., flocking and compressed air blow-down of settled flock and dust),. with job assignments to areas of the plant shown to have generally higher dust exposures, as well as with increasing days and hours worked per week. All of this is consistent with a dust-related etiology.

The preliminary toxicologic studies (Porter et al. in press; Castranova et al. 1999) point to respirable fragments of nylon as a plausible etiologic agent of this disease. These fragments are probably generated when nylon flock is cut and then milled. Many of these fragments are fibrous (i.e., elongated) in morphology (Burkhart et al. in press), which may impede their clearance from alveoli. Components of the flock finish or other airborne agents cannot be completely ruled out as contributing in some way to the etiology of this condition.

Given the physical dimensions of nylon flock fibers processed at the Rhode Island plant (approximately 15 micrometers in diameter and 1000 micrometers in length), exposure to the flock itself (contrasted with flock-associated respirable particles) is not considered a risk for the distinctive interstitial disease experienced by nylon flock workers. However, it is entirely plausible that flock itself may cause adverse health effects through irritation of the eyes, nose, throat, and large pulmonary airways.

In the face of current uncertainty about the definitive etiology of this disease, prevention warrants limiting worker exposure to respirable particulate dust in nylon flock processing facilities (NIOSH 1998; Eshenbacher et al. in press; Burkhart et al. in press). Worker exposures to airborne dust can be reduced by work practice changes

(e.g., eliminating blow-downs), by engineering controls (e.g., filtering the exhaust air stream from process cyclones), by administrative controls (e.g., restricting workers' hours per day and days per week at work), and by personal respiratory protection (e.g., requiring effective air-purifying respirators in work areas with high dust levels and during and immediately following blow-downs). In addition, as details become known about the mechanisms by which fine dust is generated in nylon flock plants, it may be possible to modify the process to minimize fine dust generation through process changes. Finally, medical screening and workforce surveillance may prove helpful for identifying affected individuals before overt clinical illness develops, allowing administrative transfer to alternative jobs with low exposure.

Existing flock studies do not allow a quantitative risk assessment that can specify a recommended occupational exposure level for nylon flock-associated dust. In the absence of such data, existing guidance for non-specific dusts must be relied upon. Given the demonstrated toxicity of flock-associated dust, worker exposure to flock-associated dust, should logically be limited to well below guidance levels intended for "inert" dust. Thus, respirable dust exposures should be controlled to well below the permissible exposure limit of 5 $mg/m^3$ for *particulates not otherwise regulated* (NIOSH 1997), and it would be better to limit respirable dust exposure in flocking plants to well below the 3 $mg/m^3$ threshold limit value (TLV) for *particulates not otherwise classified* (ACGIH 1998). In fact, it may be best to limit exposure to well below the 1 $mg/m^3$ respirable dust limit that has been proposed for essentially non-toxic particles (Morrow et al. 1991). The limits presented are all intended as 8-hour time weighted averages and should be adjusted downward for extended work shifts and work weeks (Brief and Scala 1986). To prevent irritation to the eyes, nose, throat, and upper airways, worker exposure to total dust in nylon flock plants should also be limited.

It remains unknown if there is any risk of the same or similar disease among flock workers who produce or apply flock made from other (non-nylon) synthetic organic fibers. Additional research is being done to address this and other remaining questions.

## Summary

A recently recognized and histopathologically distinctive occupational lung disease--a bronchiolocentric lymphocytic interstitial pneumonitis--has affected nylon flock workers in several nylon flock plants. This disease is apparently caused by occupational exposure to airborne respirable particulate generated during flock production and processing. in nylon flock plants. With respect to specific etiology, nylon particulate is most suspect, although contribution of other agents in the airborne dust cannot be entirely ruled out at this time. Existing evidence warrants limiting worker exposures to airborne dust in plants that produce or apply nylon flock.

## References

ACGIH [American Conference of Governmental Industrial Hygienists] (1998): 1998 TLVs and BEIs; Threshold Limit Values for Chemical Substances and Physical Agents. American Conference of Governmental Industrial Hygienists, Cincinnati.

Brief RS, Scala RA (1986): Occupational health aspects of unusual work schedules: a review of Exxon's experiences. Am Ind Hyg Assoc J 47:199-202.

Burkhart J, Piacitelli J, Swegler-Berry D, Jones W (in press): Environmental study of nylon flocking process. J Tox Environ Health.

Castranova V, Porter D, Hubbs AF, Goldsmith T, et al. (1999): Acute response of rats to intratracheal instillation of airborne dust collected at a nylon flocking plant. (this proceedings)

Eschenbacher WL, Kreiss K, Lougheed MD, Pransky GS, Day B, Castellan RM (in press): Nylon flock-associated interstitial lung disease: clinical pathology workshop summary. Am J Respir Crit Care Med (manuscript accepted for publication).

Geppert F (1997): Market overview: nylon 6.6. (a handout provided at the *Fundamentals of Flocking* course presented by the American Flock Association, August 6-8, 1997).

Kern DG, Crausman RS, Durand KATH, Nayer A, Kuhn C (1998): Flock worker's lung: chronic interstitial lung disease in the nylon flocking industry. Ann Int Med 129:261-272.

Kern DG, Durand KTH, Crausman RS, Neyer A, et al. (1997): Chronic interstitial lung disease in nylon flocking industry workers--Rhode Island, 1992-1996. Morb Mort Weekly Rept 46:897-901.

Lougheed MD, Roos JO, Waddell WR, Munt PW (1995): Desquamative interstitial pneumonitis and diffuse alveolar damage in textile workers. Chest 108:1196-1200.

Morrow PE, Muhle H, Mermelstien R (1991): Chronic inhalation study findings as a basis for proposing a new occupational dust exposure limit. J Am Coll Tox 10:279-290.

NIOSH (1997): Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 97-140.

NIOSH (1998): Health Hazard Evaluation Report: Microfibres, Inc. Pawtucket, RI. Cincinnati, OH: U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, NIOSH Report No. HETA 96-0093.

Porter DW, Castranova V, Robinson VA, Hubbs AF, et al. (in press): Acute inflammatory reaction in rats after intratracheal instillation of material collected from a nylon flocking plant. J Tox Environ Health.

Rettenmeier AW, Drysch K (1997): First industrial medicine investigation of hazardous materials of hazardous materials pollution in electrostatic flocking. Flock 23:8-20.

Table 1. Number and percent of respondents with individual frequent symptoms (with onset since hire at plant) and prevalence ratios, by assigned departmental group.

| Symptom | Group | n | % | PR | 95% CI |
|---|---|---|---|---|---|
| Shortness of breath | P/M | 42 | 35.3 | 2.3 | 1.0 - 5.2 |
| | O/S/W | 5 | 15.6 | -- | -- |
| Dry cough | P/M | 38 | 31.9 | 3.4 | 1.1 - 10.3 |
| | O/S/W | 3 | 9.4 | -- | -- |
| Chest tightness | P/M | 41 | 34.4 | 2.8 | 1.1 - 7.1 |
| | O/S/W | 4 | 12.5 | -- | -- |
| Wheeze | P/M | 25 | 21.0 | 1.7 | 0.6 - 4.5 |
| | O/S/W | 4 | 12.5 | -- | -- |
| Phlegm | P/M | 38 | 31.9 | 2.0 | 0.9 - 4.8 |
| | O/S/W | 5 | 15.6 | -- | -- |
| Fevers | P/M | 5 | 4.2 | 1.3 | 0.2 - 11.1 |
| | O/S/W | 1 | 3.1 | -- | -- |
| Generalized aches | P/M | 30 | 25.2 | 8.1 | 1.1 - 56.9 |
| | O/S/W | 1 | 3.1 | -- | -- |
| Throat irritation | P/M | 28 | 23.5 | 7.5 | 1.1 - 53.3 |
| | O/S/W | 1 | 3.2 | -- | -- |
| Eye irritation | P/M | 37 | 31.1 | ∞ | -- * |
| | O/S/W | 0 | 0 | -- | -- |

Note: Questions were asked: "Do you frequently have....?"
P/M = production/maintenance workers (n=119)
O/S/W = office/shipping/warehouse workers (n=32)
PR = prevalence ratio using prevalence of O/S/W group as reference
95% CI = upper and lower limits of 95% confidence interval for PR ratio
*CI undefined, but $p<0.001$

Table 2. Number and percent of respondents with at least one frequent systemic/respiratory symptom (with onset since hire at plant) and prevalence ratios, by various factors.

| Factor | Groups | ≥ One Frequent Systemic or Respiratory Symptom* (with onset since hire at plant) | | | |
|---|---|---|---|---|---|
| | | n | % | PR | 95% CI |
| Smoking status | ES (n=106) | 62 | 58.5 | 1.1 | 0.8 - 1.5 |
| | NS (n=45) | 24 | 53.3 | -- | -- |
| Tenure at plant (years) | <3 (n=51) | 25 | 49.0 | -- | -- |
| | 3-10 (n=44) | 28 | 63.6 | 1.3 | 0.9 - 1.9 |
| | ≥10 (n=56) | 33 | 58.9 | 1.2 | 0.8 - 1.7 |
| Days worked per week | ≤5 (n=22) | 5 | 22.7 | -- | -- |
| | 6 (n=103) | 61 | 59.2 | 2.6 | 1.2 - 5.7 |
| | 7 (n=26) | 20 | 76.9 | 3.4 | 1.5 - 7.5 |
| Hours worked per week | ≤45 (n=21) | 5 | 23.8 | -- | -- |
| | 45-65 (n=109) | 64 | 58.7 | 2.5 | 1.1 - 5.4 |
| | ≥65 (n=21) | 17 | 80.9 | 3.4 | 1.5 - 7.5 |
| Blow-down (ever) | Yes (n=103) | 67 | 65.0 | 1.6 | 1.1 - 2.4 |
| | No (n=48) | 19 | 39.6 | -- | -- |
| Flocking range (ever) | Yes (n=94) | 62 | 66.0 | 1.6 | 1.1 - 2.2 |
| | No (n=57) | 24 | 42.1 | -- | -- |
| Depart-ment | P/M (n=119) | 77 | 64.7 | 2.3 | 1.3 - 4.1 |
| | O/S/W (n=32) | 9** | 28.1 | -- | -- |

ES = ever smoker; NS = never smoker
PR = prevalence ratio using prevalence of group with "--" as reference
95% CI = upper and lower limits of 95% confidence interval for PR
* shortness of breath, dry cough, chest tightness, wheeze, phlegm, fevers, generalized aches
**Of these 9, 3 were office workers who reported flocking range work.

Table 3. Comparison of mean lung function by symptom status and smoking status.

| | ≥ One Frequent Systemic or Respiratory Symptom (with onset since hire at plant) | | |
|---|---|---|---|
| | Yes | No | *p* |
| Overall | | | |
| $DL_{CO}$ (mean % predicted) | 94.0 | 100.9 | 0.02 |
| FVC (mean % predicted) | 99.1 | 103.2 | 0.07 |
| $FEV_1$/FVC (mean X 100) | 80.0 | 80.2 | 0.86 |
| Never Smokers | | | |
| $DL_{CO}$ (mean % predicted) | 98.3 | 103.1 | 0.31 |
| FVC (mean % predicted) | 102.1 | 102.0 | 0.97 |
| $FEV_1$/FVC (mean X 100) | 79.8 | 80.8 | 0.69 |
| Ever Smokers | | | |
| $DL_{CO}$ (mean % predicted) | 92.3 | 99.8 | 0.04 |
| FVC (mean % predicted) | 98.0 | 103.8 | 0.03 |
| $FEV_1$/FVC (mean X 100) | 80.1 | 79.9 | 0.89 |