HETA 96–0093–2685
Microfibres, Inc.
Pawtucket, RI

Rita Washko, MD
Joe Burkhart, CIH
Chris Piacitelli, IH

PREFACE

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ACKNOWLEDGMENTS AND AVAILABILITY OF REPORT

Primary field investigators were Dr. Rita Washko, Joe Burkhart, and Chris Piacitelli, of the Respiratory Disease Hazard Evaluation and Technical Assistance Program, Clinical Investigations Branch (CIB), Division of Respiratory Disease Studies (DRDS). Other DRDS staff were involved: Dan Yereb and Kurt Vandestouwe provided industrial hygiene field assistance; and Eileen Hayes, Marty Pflock, Jim Taylor, Ray Petsko, Mark Ryan, Diana Freeland, Brian Day, and Dr. Lu–Ann Beeckman provided medical field assistance. Mr. Day, Dr. Beekman, and Kathy Fedan assisted with data analysis; Dr. Bill Jones provided expertise and assistance in fiber microscopy; Dr. Jeff Kahn reviewed lung biopsies; and Drs. Bob Castellan and Kay Kreiss reviewed and contributed to this final report. In addition, Drs.Vince Castranova and Dale Porter of the Health Effects Laboratory Division (HELD) designed and directed toxicological studies.

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Health Hazard Evaluation Report 96–0093–2685
Microfibres, Inc.
Pawtucket, Rhode Island
April 1998

Rita Washko, MD
Joe Burkhart, CIH
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SUMMARY

On February 28, 1996, the National Institute for Occupational Safety and Health (NIOSH) received a request from the management of Microfibres, Inc. to investigate the occurrence of two cases of what was initially thought to be hypersensitivity pneumonitis (HP)–a type of interstitial lung disease (ILD)–among employees at its plant in Pawtucket, Rhode Island. This request was made at the urging of a local occupational medicine physician, who had clinically evaluated the patients and suggested a connection between this lung disease and exposure to air contaminants at the plant. This plant dyes, finishes, and cuts nylon (and some polyester) continuous fiber to produce flock and also applies the nylon flock to a backing fabric to produce flock–coated upholstery fabric, some of which is screen printed, embossed, or otherwise finished. The plant employs approximately 170 individuals, many of whom work substantial overtime.

Following a walk–through inspection, NIOSH industrial hygienists conducted initial qualitative air sampling in the plant, followed by more comprehensive work area air sampling throughout the plant to characterize potential exposures to dust, bioaerosols, and gases. Bulk samples were also collected for microbial analysis. A NIOSH medical officer reviewed individual medical records and interviewed individuals who worked at the same plant and had been previously or subsequently diagnosed with interstitial lung disease. NIOSH also conducted a medical survey, which included a standardized questionnaire, chest x–ray, spirometry testing, and lung diffusing capacity testing offered to each current employee. Finally, NIOSH has initiated experimental studies using animals to characterize the respiratory toxicity of dust from this plant.

The general and local ventilation exhaust systems were inadequate at a number of locations, and many process cyclones used to transfer flock exhausted directly into the workplace air without filtration. In addition, the flocking and screening rooms were noted to be the dustiest areas of the plant and particularly heavy deposits of loose flock settled on the floor and in and on equipment in the flocking rooms. To remove this settled flock material and associated dust between process runs, workers used compressed air to "blow–down" the settled flock, a process that was extremely dusty. The company's respirator program was found to be inadequate, and the only respirators in use were single–use dust masks.

Respirable dust concentrations were particularly high in the flocking rooms (as high as 39.9 milligrams per cubic meter [mg/m^3]) and screening room (as high as 5.02 mg/m^3); corresponding total dust concentrations in these same areas were 241 mg/m^3 and 7.6 mg/m^3. Due to a tendency for respirable and total dust sampler inlets to clog with loose flock in these high dust areas, these samples probably underestimate true dust levels. To avoid inlet clogging, vertical elutriators (VEs) were used for follow–up dust sampling in these two work areas. The VE sample from a flocking room in operation measured dust concentrations of 8.41 mg/m^3 during

a 3–hour period that included a blow–down and 0.81 mg/m^3 during a 2.5–hour period later on the same day. Some of the small airborne particulate was shown to be irregularly shaped nylon fibers in the respirable size range. Flocking room and screening room dust levels exceeded the OSHA PEL of 5 mg/m^3 for particulate not otherwise regulated (PNOR), which is intended for relatively inert dust and for typical 8–hour workdays and 40–hour workweeks. However, preliminary results of animal toxicology studies by NIOSH have shown that dust from this plant causes an intense inflammatory response in animal lungs, and most production workers at the Pawtucket plant worked extended workshifts and extended workweeks. For these reasons, the 5.0 mg/m^3 Occupational Safety and Health Administration (OSHA) Permissible Exposure Limit (PEL)for PNOR does not provide adequate guidance to protect the health of workers at this plant.

Besides dust exposures, the only other notable industrial hygiene sampling results were for formaldehyde. The highest formaldehyde concentrations, 0.44 parts per million (ppm) and 0.21 ppm, were measured on the Ranges. Much lower concentrations were found in areas where formaldehyde was expected to be found (i.e., the compounding area, the adhesive coating areas, and the flocking rooms). Eighty–three percent (10/12) of the formaldehyde concentrations measured exceeded the NIOSH Recommended Exposure Limit (REL) of 0.016 ppm. However, none of the formaldehyde concentrations exceeded the OSHA PEL of 0.75 ppm, and one exceeded the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV ®) of 0.3 ppm for a ceiling exposure.

After the request for NIOSH assistance was made, several additional cases of ILD were diagnosed among workers at this plant—most as a result of active case finding efforts by the local occupational medicine physician—bringing the total to eight cases diagnosed from 1992 through 1996. One additional case was identified in a former worker who had been diagnosed in 1985 while employed at the plant. Common major symptoms were cough and shortness of breath; major systemic symptoms were also reported by some and were prominent in one whose flock–cutting work area had been enclosed in plastic sheeting several weeks before diagnosis. Most cases had restriction on spirometric testing (Forced Vital Capacity [FVC] as low as 50% of predicted), and half had decreased lung diffusing capacity (as low as 29% of predicted). Standard radiography revealed abnormalities in half, and high resolution computerized tomography of the chest revealed abnormalities in nearly all. In seven of the nine cases, lung tissue biopsy confirmed the presence of ILD. Lung biopsies were not done in the other two cases, but bronchoalveolar lavage abnormalities in these two were consistent with alveolitis, which commonly accompanies ILD. Review of the totality of clinical evidence indicated that the ILD in these patients was not typical for hypersensitivity pneumonitis. All nine cases had worked either at or very near to RPC (rotary precision cutters, in the Raycote department) areas (where flock is cut) or the Ranges (where loose flock is applied to adhesive coated fabric).

A total of 151 (89%) of current employees completed the survey questionnaire. Frequent eye and throat irritant symptoms were reported more frequently by non–Office workers than by Office workers; over two–thirds of those affected reported improvement when away from work. Approximately one–third of non–Office workers reported frequent lower respiratory symptoms; in about half, improvement was noted away from work. Maintenance workers, in particular, had statistically significantly increased prevalences of respiratory symptoms compared with Office workers. Frequent systemic symptoms were less commonly reported; the majority were reported by Raycote, Coating, and Maintenance department workers. Respiratory/systemic symptom prevalence was significantly associated with days and hours worked per week, working on the Ranges, and performing blow–downs. Eleven (79%) of 14 workers who reported a diagnosis of pneumonia within the past 5 years worked either in the Coating or Raycote departments. Fifteen workers reported having been diagnosed with asthma; all worked in non–Office areas, and most (12, 80%) of these 15 worked in the Raycote, Coating, or Maintenance departments. In sum, the highest prevalences of symptoms (respiratory, systemic, and irritant) and respiratory diagnoses (pneumonia, asthma) were generally reported by workers from the Raycote, Coating, and Maintenance departments—the same areas in which all nine physician–diagnosed cases of ILD had worked.

None of the 143 technically acceptable chest x–rays were classified according to the International Labor Organization (ILO) system as having small opacities consistent with pneumoconiosis. Consistent with a restrictive interstitial lung disease process, spirometry test results from 145 workers showed generally lower mean percent predicted FVC and DL_{CO} (but not FEV_1/FVC ratio) among symptomatic workers. Even among ever smokers only, in which this finding was most apparent (and statistically significant), increased symptom prevalence was associated with days and hours worked per week, non–Office work (and specifically work on the Ranges), and blow–down exposure.

NIOSH investigators determined that a health hazard exists from occupational exposures to flock–associated dust. The presence of this risk is indicated by: (1) results of industrial hygiene sampling in this plant; (2) review of nine physician–diagnosed cases of ILD among workers at this plant; (3) results of a respiratory health survey of current employees at the plant; and (4) preliminary findings of an animal toxicology study of dust from this plant. Epidemiologic findings suggest a much wider presence of occupational lung disease than the physician–diagnosed cases. The hazard appears to be greatest in the flocking rooms and screening rooms, where dust levels are highest. (In addition, a potential health hazard exists from exposure to formaldehyde.) Reduction of worker exposures to airborne dust, together with implementation of a medical screening and surveillance program, is recommended to protect the health of the workers at this plant.

Keywords: SIC 2299 (Textile goods, Not Elsewhere Classified), nylon, fibers, flock, interstitial lung disease, flock workers lung, upholstery fabric, respiratory irritation, particulate not otherwise classified (PNOC), particulate not otherwise regulated (PNOR), formaldehyde.

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INTRODUCTION

On February 28, 1996, the National Institute for Occupational Safety and Health (NIOSH) received a request from the management of Microfibres, Inc. to investigate "two cases of hypersensitivity pneumonitis" (HP) among employees at its plant in Pawtucket, Rhode Island. This request was made at the urging of an occupational medicine physician who had evaluated the two affected workers, noted that their clinical presentations were consistent with HP, and suggested a possible connection between this lung disease and occupational exposures at the Microfibres plant.

Chronology of Events

On March 20, 1996, a NIOSH medical officer met with four workers from the Pawtucket plant, the two whose illness had prompted the request for NIOSH assistance and two additional workers who had been subsequently diagnosed with this lung disease in the interim period. (The local occupational medicine specialist had undertaken a screening program to identify additional cases.) Open–ended interviews, focusing on workplace exposures and symptoms, were conducted and individual medical records were reviewed. The local pulmonary pathologist who had been clinically involved in most of the cases reviewed the workers' lung biopsy slides with the NIOSH medical officer.

Because all four workers initially identified with lung disease had worked in or adjacent to the Ranges (see "Process Description" in the "Background" section of this report), the NIOSH medical officer also conducted open–ended interviews with Range workers.

NIOSH industrial hygienists returned to the plant on March 26, 1996, and conducted a walk–through inspection. On April 10, 1996, the industrial hygienists conducted qualitative air sampling of general work locations at the plant to identify potential exposures. In addition, bulk samples of the various process washes, finishes, and waste products were collected to assess potential microbial contamination.

A more comprehensive industrial hygiene survey was conducted during the week of May 6–10, 1996. General work location air samples were collected throughout the plant to assess concentrations of: respirable and total dusts, various volatile organic compounds (VOCs), nitrosamines, and endotoxins. Also, additional bulk samples were collected throughout the plant process for microbial analysis.

A medical survey was conducted May 5–14, 1996. This consisted of a questionnaire, chest x–ray, and spirometry testing, offered to each current employee at the plant. The medical survey team returned to the Pawtucket plant June 5–14, 1996, to perform additional (lung diffusion capacity, DL_{CO}) testing.

On June 18, 1996, the NIOSH investigators sent a letter reporting initial findings and interim recommendations to the company and union. The NIOSH letter noted that extremely high concentrations of airborne dust were measured in the flocking room and that respirators provided to workers were inadequate; corresponding recommendations were offered. A recommendation was also made to restrict smoking in the plant.

During August 20–22, 1996, the NIOSH industrial hygienists returned to the plant for further sampling in an effort to better characterize exposures to airborne dust and bioaerosols.

On October 28–29,1996, Microfibres hosted a two–day informational exchange meeting for their consultants (including the local occupational medicine specialist), the local pathologist, NIOSH investigators, and representatives from both the company and the local union. After presentation and discussion of clinical profiles and lung pathology of cases identified to date, the medical professionals in attendance agreed that the affected workers' disease was consistent with

interstitial lung disease (ILD), but that the aggregate clinical evidence did not clearly support a diagnosis of HP, a specific type of ILD. A similar outbreak of occupationally–related ILD, that had several years earlier affected workers at Microfibres' plant in Canada (see below in "Background"), was also discussed.

A second informational exchange meeting, held on February 27, 1997, involved the company and their consultants, local union representatives, NIOSH investigators, a representative from the State of Rhode Island Department of Health, and a representative from the international union. Investigators reported on new developments subsequent to the October meeting. NIOSH investigators commented on a review of cases' pathology slides by a NIOSH pathologist, the need to limit worker exposure to airborne dust, the need to enforce the respiratory protection program, and the need for continued vigilance to permit early identification of any new cases of ILD that may occur among workers at the plant so that the affected workers can be removed from further exposure. An outline of proposed animal toxicology studies to be conducted at NIOSH's Health Effects Laboratory Division (HELD) was presented, and comments on the protocol from those in attendance were solicited. At the meeting's conclusion, Microfibres requested NIOSH assistance in developing medical screening and surveillance protocols appropriate for their work force.

During the February 1997 visit to the plant, NIOSH industrial hygienists collected samples of inhalable dust from the flocking rooms and the flock screening rooms to support planned animal toxicology studies.

On June 23, 1997, recommended protocols for medical screening and surveillance, along with copies of pertinent references, were sent to Microfibres.

On September 26, 1997, a brief report prepared by the local occupational medicine specialist and submitted to NIOSH was published [Kern et al. 1997]. Limited findings from the NIOSH investigation were included in that published report.

On October 21, 1997, a third informational exchange meeting was hosted by Microfibres. In attendance were company and union representatives, company consultants, NIOSH investigators, other NIOSH staff, and a representative of the State of Rhode Island Department of Health. Among other things, preliminary results of the animal toxicology studies were presented. After the meeting, many of those present toured the plant.

BACKGROUND

The Pawtucket, Rhode Island Plant

The Microfibres, Inc. headquarters plant located in Pawtucket, Rhode Island, dyes, finishes, and cuts nylon (and a lesser amount of polyester) "tow" to produce flock for use in–house and at other flocking plants. This plant also applies nylon flock to a backing fabric to produce a proprietary line of flock–coated upholstery fabric. The company was originally founded in 1926 as the Rayon Processing Company of Rhode Island, and has produced cut fiber for flock (originally under the Raycote brand name) throughout its history. According to company literature, production at the Rhode Island plant had begun to resemble that of present day operations by the 1970s.

The Pawtucket, Rhode Island, plant is situated just outside of Providence, RI. At the time of the NIOSH medical survey, 170 workers were employed at this plant. Employees are represented by the Union of Needletrades, Industrial, and Textile Employees (UNITE). Operations are continuous, seven days a week with three daily 8–hour long production shifts. Many employees often work longer than 8–hour

work days and most work more than 5 days per week. Only a few work less than 5 days per week. During the standard work week, workers are typically assigned to a specific job/work station. On weekends, these same employees may be assigned to other jobs.

PROCESS DESCRIPTION

The flocked upholstery fabric manufactured by Microfibres consists of three basic components: a backing fabric, adhesive, and cut nylon fibers. A complex process brings these three basic components together into a final product. In recent years, only nylon flock has been used at the Microfibres plant, but both nylon and polyester flock is produced. All polyester flock produced at the plant is bagged for shipment to other plants. To produce flock, nylon (or polyester) "tow"– a loose rope of thin continuous nylon (or polyester) fibers–is dyed and coated with a finish before being cut, dried, screened, and bagged as flock. The flock is later deposited on cotton–polyester fabric backing coated with an acrylic latex adhesive by a process using an electrostatic field to align the falling flock fibers. After heat–curing of the adhesive, the flocked fabric may be subjected to finishing, mechanical embossing, and/or water–based ink–screen printing. A number of different departments perform specific tasks in producing the final product.

Dye House Department

At the Continuous Dyer, bulk "tow" (which arrives in boxes or on spools) is dipped in a dye bath, fixed with steam, and placed damp in large boxes for temporary holding. At the Recycle Area, located elsewhere in the plant, workers from the Dye House Department wash and dye waste flock from this and other Microfibres plants.

Raycote Department

"Tow" received from the Dye House Department is rinsed in a hot water bath, dipped in a finishing bath (containing tannic acid, an ammonium ether of potato starch, and a fatty alcohol derivative), and cut into flock by machines called rotary precision cutters (RPCs). The flock is then dried, screened, and stored in large bags for future use in this and other flocking plants.

Compounding Department

In the Compounding Department, acrylic latex adhesives are mixed. In addition, a fabric protector (Scotchgard®) and softening compounds are also prepared for use in finishing processes. Compounding Department workers deliver the prepared compounds to the areas of use and remove waste adhesive and empty containers.

Coating Department

The Coating Department consists of two roll–to–roll coating ranges (Ranges 1 and 2), two embossing ranges, and a printing range. *(Note: when the term "Range" is used in this report without specifying the particular type, it refers to the coating range.)* At each coating range, polyester/cotton fabric backing is coated with adhesive. The backing enters the flocking module located in a temperature– and humidity–controlled flocking room. Flock is manually dumped into hoppers and a desiccant powder may be added and mixed in to control moisture content and prevent clumping. In the module (an enclosed unit where flock is applied), the flock–desiccant mixture is dispersed above the adhesive–coated backing in an electrostatic field that aligns the falling flock fibers so they embed in the adhesive at an angle more or less perpendicular to the backing. Upon exiting the flocking room, the fabric is passed through a gas–fired curing oven, sprayed with either a fabric protector or a softening compound, returned through the oven, inspected, and taken up onto a roll. The fabric can then be sent to be processed at an embossing range or at a printing range that utilizes water–based inks, or can be sent directly to shipping.

Between process runs that differ with respect to flock texture (e.g., length and/or diameter) or color, the affected Range must be cleaned of residual flock prior to starting the new run. A "blow–down" involves the use of compressed air to clear loose flock from the flocking module, and from the flocking room walls, floor, and other equipment. Workers conducting blow–downs begin this process on the module and blow out any loose flock in or on the module, then blow the flock from one side of the room to the other, and finally bag the loose flock for disposal. The frequency of blow–downs varies with the production schedule. On occasion, it may be necessary to perform several blow–downs per shift.

Other Departments

Shipping Department personnel utilize forklifts to retrieve finished rolls of fabric from production areas and bring them to the loading area where they are placed in trucks for shipment. In addition to short–term storage space in the plant, an off–site warehouse is located in a nearby town. Maintenance Department workers maintain equipment throughout the facility. Office personnel are located within offices in the production building, as well as in a separate building nearby.

Prior Outbreak at Kingston, Ontario Plant

In 1990–91, five workers at the Microfibres' plant in Canada, which employs approximately 85 workers, were diagnosed with ILD [Lougheed et al. 1995]. Although a specific causative agent was not identified, that outbreak was attributed to occupational exposure at the plant. The investigators speculated that the disease may have been associated with inhalation of mold–associated toxins, as mold was found to be growing in stored adhesive at the facility. However, after mold–contaminated adhesive was discarded and interventions were undertaken to prevent mold growth in stored adhesive, the company's toxicology consultant reported at one of the informational exchange meetings that two additional cases of ILD occurred in 1995 among workers at the plant in Canada.

METHODS

Industrial Hygiene Evaluation

A preliminary industrial hygiene survey was conducted during the afternoon shift on April 10, 1996. During that survey, five air samples were collected for VOCs using thermal desorption tubes. The purpose of these samples was to qualitatively identify potential exposures to airborne chemical compounds from the manufacturing process in order to direct subsequent quantitative sampling efforts. Sampling locations for these five samples were: (1) Raycote area; (2) Range 1 oven by pre–coat; (3) Range 2 oven by pre–coat; (4) Range 1 flocking room; and (5) Range 2 Scotchgard® area.

During the week of May 6–10, 1996, a second industrial hygiene survey was conducted. This survey was designed to more thoroughly characterize the work environment. Based on information from the earlier survey, the following 12 area sampling sites were selected to represent typical workplace exposures:

Location

RPC 1–2 Area
RPC 3–7 Area
Basement Screening Area
Range 1 Pre–Coat Area
Range 2 Pre–Coat Area
Range 1 Flocking Room
Range 2 Flocking Room
Range 1 Scotchgard® Area
Range 2 Scotchgard® Area
Compounding Area
Embossing & Printing Range Area

At each sampling site, a basket of sampling equipment was placed at an area in close proximity to workers. Air samples were collected for respirable dust, total dust, endotoxin, elemental metals, formaldehyde, ethyl acrylate, ethyl propanoate, ethyl acetate, toluene, 1,1,1–trichloroethane, butyl cellosolve, oxides of nitrogen, propylene glycol, total hydrocarbons, nitrosamines, viable bacteria, and fungi. Additionally, several bulk liquid and flock samples were collected and submitted for bacterial and fungal culture. Also, several personal breathing zone (PBZ) dust samples were collected on employees performing blow–down operations. Finally, real–time dust monitors were placed in each of the flocking rooms to measure concentrations of both respirable and total dusts.

An additional survey was performed in August 1996. The purposes of the August survey were: (1) to better characterize the dust exposures in the flocking rooms and screening room; and (2) to collect viable bioaerosol samples from throughout the plant. In addition to standard dust sampling equipment, alternative sampling methods were used in an attempt to minimize clogging of sampler inlets by flock. Vertical elutriators (VEs) were used to collect a generally thoracic fraction dust with minimal potential for inlet clogging [Corn 1987]. (VEs are used extensively to sample cotton dust in the cotton textile industry because they tend not to clog with airborne cotton lint.) Samples from the VE, total dust, and a personal inhalable dust sampler (PIDS) cascade impactor were examined by both phase contrast light (PCL) microscopy and scanning electron microscopy (SEM). Respirable dust samples were also collected using BGI stainless steel cyclones operated at a flow rate of 2.2 liters per minute (lpm). (These cyclones were used in an attempt to reduce inlet clogging effects associated with static properties of the flock.) At each of the 12 sampling sites listed above, three air samples (30–second, 1–minute, and 5–minute) were collected for both bacteria and fungi using the N–6 Andersen viable sampler. This sequence was repeated three times at each location during the work shift.

From February 25–27, 1997, airborne dust was collected to support animal toxicology studies. To collect the large quantity of dust needed for these studies, air samples were collected in the flocking rooms of both Ranges and the basement screening room using VEs and high–volume, 1–inch respirable metal cyclones. For the 3 days of sampling, each sampler was operated continuously for 24 hours each day, at which time the sampling media in each sampler were changed.

Table 1 summarizes the air sampling methods used during this investigation.

Medical Evaluation

Physician–Diagnosed Cases: Interviews, Review of Medical Records, and Participation in the NIOSH Survey

Early in the NIOSH investigation, open–ended interviews were conducted with the four initial workers identified with ILD. All were asked to describe their activities at work, onset and characteristics of symptoms, and any temporal relationship of symptoms to work activities. Medical records and radiographic images (both standard chest x–rays and computerized tomography scans) were provided for review by the local occupational medicine specialist, and available lung biopsies were reviewed with the pulmonary pathologist who had seen the cases clinically.

Subsequent to the NIOSH medical survey (see below), an additional four workers diagnosed with ILD were brought to the attention of NIOSH by the local occupational medicine specialist, who had carried out a case–finding effort that included voluntary symptoms screening followed by progressive clinical testing through possible lung biopsy. The additional four cases were interviewed by telephone and medical records for three of these additional four were obtained and reviewed.

Among all eight physician–diagnosed cases, only two participated in both the questionnaire and objective medical testing. Additionally, one participated in the questionnaire only; one participated in all components except diffusion capacity testing; and one performed diffusion capacity testing only.

Interviews With Workers From Ranges

Information obtained from the initial cases suggested that the ILD among workers at this plant was associated with working on the Ranges. Therefore, first and second shift workers from the Ranges were interviewed in an open–ended format including questions on both respiratory and systemic symptoms (i.e., fever, body aches, flu–like illness symptoms). These interviews were conducted during the workers' shift in a private room at the plant.

Medical Survey

A survey conducted in May/June 1996 was intended to quantify respiratory morbidity among current employees and to identify work–related factors that may be implicated as possible causes of the observed respiratory morbidity.

Advance announcements of this survey were previously distributed to all workers by Microfibres management. With union–management consensus, Microfibres scheduled willing participants for this medical evaluation during their work shifts.

Questionnaire

Trained NIOSH interviewers administered a standardized questionnaire by computer utilizing "Epi Info" software [Dean et al. 1994]. The questionnaire asked about the following: demographic information, current and past job assignments, and work activities at Microfibres; previous work history; symptoms and health history; and tobacco use. Participants were asked about systemic symptoms (i.e., fever and generalized body aches) in addition to both acute and chronic respiratory symptoms and irritant symptoms. The following definitions were established for the purpose of questionnaire analysis:

FREQUENT SYMPTOMS	
SYSTEMIC SYMPTOMS:	***Answered "yes" to:***
Fever	Do you frequently have fever?
Body aches	Do you frequently have aches all over your body, similar to when you have the flu?
CHEST SYMPTOMS:	***Answered "yes" to:***
Dry cough	Do you frequently have a dry cough?
Cough with phlegm	Do you frequently have a cough with phlegm?
Wheeze	Do you frequently have a wheezing or whistling in the chest?
Chest tightness	Do you frequently have a chest tightness?
Shortness–of–breath	Do you frequently have shortness–of–breath?
IRRITANT SYMPTOMS:	***Answered "yes" to:***
Throat irritation	Do you frequently have throat irritation?
Eye irritation	Do you frequently have eye irritation?
OTHER SYMPTOMS	
Episodic flu–like illness	More than 2 episodes of flu–like illness during past year
Shortness–of–breath with wheeze	Attacks of shortness–of–breath with wheezing or whistling in the past 2 years
Chronic bronchitis	Morning cough with phlegm for 3 or more months for 2 or more years
MEDICAL CONDITIONS	
Pneumonia	Told by a physician had pneumonia within past five years.
Asthma	Told by a physician had asthma

Chest X–Ray

A single view, postero–anterior (PA) chest x–ray (CXR) was taken by NIOSH on a full–size (14 x 17 inch) film. CXRs were reviewed on–site by the NIOSH medical officer to determine if urgent notification of findings was necessary. At the conclusion of each shift, the company medical consultant (i.e., the local occupational medicine specialist) viewed films of participants who had signed medical release of information forms permitting his review of their chest x–ray.

Chest x–rays were sent for independent classification by two NIOSH–certified B readers (physicians trained and certified proficient in the classification of chest x–rays for the pneumoconioses) who, without knowledge of participants' ages, occupations, symptoms, or smoking histories, classified the films according to the current international classification system for pneumoconiosis [ILO 1980]. An additional independent classification was obtained from a third B reader for any films on which the first two readers disagreed on small opacity profusion.

A final small opacity profusion classification for each film was taken as the consensus (of two) or median (of three) classifications. Films with a final small opacity profusion classification of $\geq$1/0 were considered abnormal.

Spirometry

Spirometry, a type of lung function testing that measures air flow and volumes expelled from the lungs, was performed using a dry rolling–seal spirometer interfaced to a dedicated computer. Procedures conformed to standard guidelines [American Thoracic Society 1995a]. At least five maximal expiratory blows were recorded for each test. All measured volumes were corrected to BTPS (body temperature, ambient pressure, saturated with water vapor). The largest forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were the test parameters selected for analysis, regardless of the blows on which they occurred. Predicted values were calculated using published reference equations [Knudson et al. 1983]. Predicted values for blacks were determined by multiplying

the value predicted by the equations by 0.85 [Hankinson 1986]. To identify participants with abnormal results, test results were compared to the 95th percentile lower limit of normal (LLN) values obtained from the reference equations [Knudson et al. 1983; American Thoracic Society 1991]. Five percent of a large population of asymptomatic nonsmokers would be expected to have test results that fall below the LLN, while 95% would have test results above the lower limit. Abnormal test results were defined, and categorized as having a pattern suggesting obstruction or restriction, as follows:

***Restriction*:**
FVC < LLN and FEV_1/FVC % > LLN
***Obstruction*:**
FEV_1/FVC % < LNN

Diffusion Capacity (DL_{CO}) Testing

Diffusion capacity testing, a type of pulmonary function testing that measures the ability of a gas to pass across the lung tissue into the bloodstream, was determined using carbon monoxide as a test gas and an automated valve and timing device with a bag–in–a–box system (Spirometrics Inc., Auburn, ME). A breath holding time of 10 seconds was used [Ogilvie et al. 1957]. The alveolar sampling volume was approximately total lung capacity minus the washout volume of 750 ml. A correction factor for carbon monoxide back pressure (CO_{bp}) was used in the calculation of DL_{CO}. CO_{bp} was determined using a CO breath analyzer (Vitalograph, Inc., Lexena, KS). The average parts per million (ppm) value of three trials was used as the correction factor.

Procedures conformed to standard guidelines [ATS 1995b]. A maximum of five trials was attempted to obtain at least two DL_{CO} values that were within 5%. The mean value of at least two acceptable tests was the parameter reported and analyzed. Predicted values for DL_{CO} and the ratio of DL_{CO} to single–breath alveolar volume (DL_{CO}/V_A) were based on published prediction equations [Miller et al. 1983]. Because lung volume (V_A , as reflected by the inspired volume, or Vi) can result in a reduced DL_{CO} , American Thoracic Society (ATS) recommends that Vi be greater than 90% of FVC or VC (vital capacity) [ATS 1995b]. Typically, spirometry and DL_{CO} testing are conducted at the same time. However, for the medical survey at the Microfibres plant, FVC values measured in May were used in the application of this ATS criterion for DL_{CO} testing in June. DL_{CO} results for an individual were considered abnormal if they were < 80% of predicted.

Data Analysis

To analyze the medical survey data, Epi Info software [Dean et al. 1994] was used to generate prevalence ratios and confidence intervals. Mean values of continuous variables were compared using Student's t–test or, if variances differed, using the nonparametric Kruskal–Wallis test for means. Statistical significance levels were set at $p \leq 0.05$.

EVALUATION CRITERIA

Industrial Hygiene

To objectively assess workplace exposures with respect to known hazards, NIOSH investigators refer to a variety of environmental evaluation criteria. These criteria suggest exposure levels to which most workers may be exposed for a working lifetime without experiencing significant adverse health effects. However, they do not consider individual worker susceptibility (e.g., hypersensitivity, pre–existing medical conditions, concurrent medications, or personal habits such as smoking, etc.) or special environmental conditions (e.g., possible combined effects in the setting of mixed exposures to multiple agents, or via multiple routes of exposure). Therefore, workers may remain at risk of or actually experience occupational illness even when exposures are maintained below these limits. Evaluation criteria may change over time as new information on the

toxic effects of an agent become available. For all the above reasons, it is prudent for employers to maintain worker exposures well below established occupational health criteria.

The primary sources of evaluation criteria for the occupational exposures are: NIOSH recommended exposure limits (RELs) [NIOSH 1992], the Occupational Safety and Health Administration (OSHA) permissible exposure limits (PELs)[Code of Federal Regulations 1996], and the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs®) [ACGIH 1996].

Occupational health criteria are established based on available scientific information provided by animal or human experimental data or by observational epidemiologic studies of exposed workers. Differences between the NIOSH RELs, OSHA PELs, and ACGIH TLVs® relate to different philosophies and interpretations of technical information. RELs and TLVs® are guidelines, whereas PELs are legally enforceable standards. OSHA PELs are required to take into account technologic and economic feasibility of exposure control in various industries where the agents are found. NIOSH RELs are primarily based on disease prevention with less concern for feasibility issues, and therefore tend to be more conservative than OSHA PELs.

A Court of Appeals decision vacated the OSHA 1989 Air Contaminants Standard in *AFL–CIO v OSHA*, 965F.2d 962 (11th cir., 1992), and OSHA is now enforcing the previous 1971 standards (listed in 29 CFR 1910.1000, Table Z–1–A). However, some states which have OSHA–approved State Plans continue to enforce the more protective 1989 limits. NIOSH encourages employers to use the 1989 limits or the RELs, whichever is lower.

Evaluation criteria are usually based on an individual worker's average PBZ exposure to the airborne substance over an entire 8– to 10–hour workday, expressed as a time–weighted average (TWA). Exposure limits are usually expressed in ppm, milligrams per cubic meter (mg/m^3), or micrograms per cubic meter ($\mu g/m^3$). To supplement the full–shift TWA where there are recognized adverse effects from short–term exposures, some substances have a short–term exposure limit (STEL) for 15–minute peak exposure periods, and/or a ceiling limit not to be exceeded at any time. Additionally, some chemicals have a "skin" notation to indicate that the substance may be absorbed through direct contact with the skin and mucous membranes.

Table 2 summarizes many key occupational exposure criteria and health effects for the compounds sampled during this investigation. In the following several paragraphs, additional information is provided pertinent to evaluation criteria for exposures to particulates, viable microbes, and endotoxin.

Particulates Not Otherwise Classified/Regulated (PNOC/ PNOR)

In many work settings, the composition of airborne particulate does not have an established specific occupational health exposure criterion. Many such dusts were formerly referred to as "nuisance dust," based on their long use with little or no documented adverse health effects when exposures were kept under reasonable control. Excessive concentrations of such dust in workroom air may seriously reduce visibility, cause unpleasant deposits in the eyes, ears, and nasal passages, or cause injury to the skin or mucous membranes by direct chemical or mechanical action or, indirectly, by the rigorous skin cleansing procedures necessary for their removal. Few dusts, however, can be considered truly biologically inert; all dusts invoke at least some cellular response in the lung when inhaled in sufficient amount. Also, many dusts have not yet been carefully studied in terms of health effects associated with occupational exposure.

It has been the convention to apply generic exposure criteria in such cases. The preferred terminology for relevant ACGIH TLV ® criteria is

"particulates, not otherwise classified" (PNOC). The OSHA PEL terminology is "particulates, not otherwise regulated" (PNOR). The relevant OSHA PELs are 15.0 mg/m^3 for total PNOR and 5.0 mg/m^3 for the respirable fraction, each measured as an 8–hour TWA exposure. The ACGIH TLVs® for PNOC are 10.0 mg/m^3 (total) and 3 mg/m^3 (respirable), both measured as 8–hour TWAs. On the basis of animal studies, however, it has been suggested that limits for human exposure should be substantially lower [Morrow et al.1991].

Viable Microbes

Acceptable levels of airborne microorganisms have not been established. It is generally accepted that individuals differ with respect to immunogenic susceptibilities and that allergic reactions can occur even with exposure to relatively low concentrations of airborne allergens. The current strategy for on–site evaluation of environmental microbial contamination involves an inspection to identify sources (reservoirs) of microbial growth and potential routes of dissemination. In locations where contamination is visibly evident or suspected, bulk samples may be collected to identify the predominant species. In limited situations, air samples may be collected to document the presence of a suspected microbial contaminant.

Endotoxin

Endotoxin is a component of the outer cell wall of gram–negative bacteria. Endotoxins have a wide range of biological activities involving inflammatory responses. Of most importance to occupational exposures are the activities of inhaled endotoxin in the lung. Exposure to airborne endotoxin can result in acute fever, dyspnea, coughing, and reductions in lung function [Rylander 1997].

Occupational exposure criteria for airborne endotoxin have not yet been established by OSHA, NIOSH, or ACGIH. However, a committee of the International Commission on Occupational Health has recently proposed the following exposure guidelines for no–effect levels: 2000 EU/m^3 for toxic pneumonitis; 1000 EU/m^3 for systemic symptoms; and 100 EU/m^3 for airways inflammation [Rylander 1997]. Building in a safety factor of 2, a committee of the Health Council of the Netherlands is in the process of recommending an occupational exposure limit of 50 EU/m^3 [Dutch Expert Committee 1997].

Medical

The term “interstitial lung disease” refers to a heterogeneous group of acute and chronic lung diseases characterized by inflammation of the alveolar (i.e., breathing sac) walls and perialveolar (i.e., area surrounding the breathing sacs) tissue [Raghu 1995]. If the inflammation persists, interstitial fibrosis (scarring of the lung tissue) can result. Impaired pulmonary function (most typically compromised gas exchange and restrictive spirometry) can occur at any stage of the disease and may be irreversible once fibrosis is established [Redlich 1996; Schwarz 1994].

Some investigators have estimated the overall prevalence of all ILD to be 20–40 per 100,000 population in the United States [Crystal 1992; Redlich 1996]. However, some cases of ILD–mild undiagnosed cases and/or misdiagnosed cases–are not recognized as ILD. One recent study based on a registry of adult patients with ILD reported prevalences of 80.9 per 100,000 among males and 67.2 per 100,000 among females [Coultas et al. 1994]. That same study estimated incidences of ILD of 31.5 per 100,000/yr among males and 26.1 per 100,000/yr among females [Coultas et al. 1994].

There are over 100 known causes of ILD. Some cases of ILD are associated with collagen–vascular diseases (e.g., rheumatoid arthritis, scleroderma, systemic lupus erythematosus, or mixed connective tissue disease); others are infectious (e.g., viral pneumonia) or drug–induced (e.g., by various

antibiotics, antiarrhythmics, etc.); some result from inhalational exposures in occupational and environmental settings; and some are of unknown etiology (e.g., idiopathic pulmonary fibrosis).

Many occupational causes of ILD are known. These can be grouped into inorganic or organic agents. The most common inorganic agents that cause ILD are crystalline silica, asbestos, and coal dust. Other agents known to cause ILD when inhaled are metals (e.g., beryllium, cobalt) and irritant gases, fumes, and vapors. Organic agents, such as certain bacteria, fungi, animal and plant proteins, and some chemicals (e.g., anhydrides, isocyanates) have also been associated with ILD [Schwarz 1994; Lopez 1994; Redlich 1996; Rose 1996].

The clinical presentation of persons afflicted with this disease is that of dyspnea (i.e., shortness–of–breath), sometimes gradually progressive over months to years, with associated fatigue. Cough, either productive or more commonly nonproductive, is also a frequent symptom; wheezing is occasionally present [Schwarz 1994; Rose 1996]. While many of the ILDs are characterized by insidious onset and chronic symptoms, some forms of this disorder are marked by more abrupt onset with acute symptoms, including recurrent fevers. HP, for example, can present with acute respiratory and influenza–like systemic symptoms several hours after exposure to the offending agent [Parker et al. 1992].

On physical examination, end–inspiratory rales (i.e., fine crackles) may be heard with a stethoscope, especially at the lung bases. Resting tachypnea (i.e., rapid breathing) or tachycardia (i.e., rapid heart rate) may be present [Crystal 1992]. However, the examination may be unrevealing in mild cases.

CXRs are normal in 10–20% of individuals with ILD [Redlich 1996]. The extent and severity of the radiographic changes may not correlate with symptoms or physiologic abnormalities [Schwarz 1994]. Common radiographic patterns in those with idiopathic ILD (i.e., ILD of unknown etiology) include: a ground glass pattern; diffuse finely reticular, nodular, or reticulonodular patterns, most prominent at the lung bases. In those with longstanding disease, a honeycomb pattern with cystic areas may be seen [Crystal 1992]. With regard to physiology, reduced lung volumes on spirometry testing and impaired gas exchange on DL_{CO} testing are typically present in advanced cases.

For HP, which is generally understood to be an immunologically–mediated disease, a blood test may reveal the presence of serum antibodies specific for the offending antigen. However, such antibodies can also be detected in exposed but non–diseased individuals or might not be detected if the test does not include the relevant antigen, so antibody testing is often not diagnostically helpful in individual cases [Burrell and Rylander 1981; Roberts et al. 1976; McSharry et al. 1984].

Definitive diagnosis can be challenging; some individuals with ILD may present with mild dyspnea, a CXR that is normal, and routine pulmonary function studies (spirometry and DL_{CO}) that are also within normal limits [Schwarz et al. 1994]. High–resolution computerized tomography (HRCT) is a more sensitive technique and, in many cases, can identify parenchymal (i.e., lung tissue) abnormalities that are not evident on standard CXRs. More invasive testing is often necessary to make the diagnosis of ILD. Bronchoscopy with transbronchial biopsy (TBB) and bronchoalveolar lavage (BAL) and/or surgical lung biopsy (open lung biopsy, OLB) may be useful in this regard.

BAL allows for the examination of cells, cellular products, and proteins from the distal air spaces in the lung, and may be helpful in making a specific diagnosis [Rose 1996]. Guidelines for the clinical use of BAL in ILD have been published [BAL Cooperative Steering Committee 1990]. Expected findings with respect to BAL fluid differential cell counts include the following: (1) for healthy never smokers, 34.3% lymphocytes, 3.1% neutrophils, and 1.1% eosinophils represent 95th percentiles; (2) for healthy ex–smokers, corresponding values are 29.3%, 8.4%, and 3.7%;

and (3) for healthy smokers, corresponding values are 18.6%, 7.0%, and 3.0% [BAL Cooperative Steering Committee 1990].

A surgical lung biopsy is considered the "gold standard" for diagnosis of ILD [Raghu 1995]. Alternatively, TBB may be used to provide diagnostic lung tissue samples. However, because the tissue specimens obtained by TBB are small and the inflammatory process in ILD is often non–uniform (i.e., normal lung may be immediately adjacent to areas of diseased lung), the diagnosis can be missed with a TBB.

From an occupational and environmental standpoint, most agents identified as causing ILD result in one or more histopathologic patterns (patterns identified when viewing tissue specimens through a microscope). Redlich [1996] classified these common histopathologic patterns for occupational exposures as follows:

Common Histologic Patterns Found in Interstitial Lung Disorders

Histopathology	*Example(s)*
Bronchiolitis obliterans	Fumes, gases
Macules	Coal, iron, tin, graphite
Nodular fibrosis	Coal, silica, mixed dust
Diffuse fibrosis	Asbestos, talc, hard metal, mica
Fibrotic mass lesions	Coal, silica
Granulomatous disease	Beryllium, organic dusts
Emphysema	Cigarettes, coal

Source: Redlich [1996].

In general, there is an influx of inflammatory cells into the lung interstitium associated with edema (fluid accumulation and swelling). This process distorts the alveolar (air sac) walls. Prolonged, repeated exposures (or in some cases, shorter, more intense exposures) can lead to irreversible changes characterized by fibrotic (scarring) changes and honeycombing (disruption of the lung architecture with the formation of cystic spaces).

The granulomatous interstitial lung diseases may show mononuclear cell (white blood cells characterized by a single nucleus) infiltrates and small, scattered granulomas (microscopically distinctive collections of epithelioid cells that are surrounded by a peripheral collection of lymphocytes) on histologic examination. Granulomas are typical findings in organic dust diseases, such as HP [Coleman and Colby 1988], but are not observed in all cases [Reyes 1982].

In occupational ILD, there is often a long latency between initial occupational exposure and the development of clinically evident disease, and the disease may progress rather slowly, over months to years. However, some occupational ILD (e.g., some cases of HP and bronchiolitis obliterans with organizing pneumonia (BOOP)), can present acutely and progress rapidly [Schwarz 1994]. So called organic dust toxic syndrome (ODTS), a condition involving pulmonary interstitial inflammation not usually considered in general reviews of ILD, can present within several hours of a very intense organic dust exposure; acute ODTS associated with an obvious singular exposure event typically resolves within one to three days of removal from exposure [Parker et al. 1992].

Therapeutic options for occupational ILD are limited, and the course of illness can be quite variable. With some agents, particularly those that are not effectively cleared from the lung (e.g., silica dust), acute and chronic forms of disease can progress to disabling end–stage lung disease and eventual death despite medical treatment and removal from exposure. With other agents, particularly those that are cleared rapidly from the lungs (e.g., many organic dusts), complete recovery often occurs if the affected individual is removed from further exposure before irreversible fibrosis occurs in the lung [Parker et al. 1992]. Individuals with clear–cut ILD should cease further exposure if and when the offending agent(s) is identified or even strongly suspected.

RESULTS

Industrial Hygiene Evaluation

Observations

On March 26, 1996, a walk–through survey of the plant was conducted by two NIOSH industrial hygienists.

The general and local exhaust systems were observed to be inadequate at a number of locations within the plant. In particular, when Scotchgard® was applied, smoke from the ovens escaped into the general work area of the coating ranges resulting in apparent re–entrainment of smoke exhausted from the ovens into other areas of the plant. In addition, the ventilation system was imbalanced (evidenced by substantial air movement between areas within the plant, particularly in doorways and passageways).

Although controlled by a separate heating, ventilating, and air conditioning (HVAC) system separate from the production areas of the plant, it was observed that the office area was under a negative pressure (evidenced by difficulty in opening outside doors, popping of ceiling tiles in the office area when outside doors to that area were opened).

The flocking rooms and the screening room were observed to be the dustiest areas within the plant. In the flocking rooms, settled loose flock accumulated on the floors and equipment to a depth of several inches during process runs, and was only cleaned up during a process change. Settled loose flock was also observed outside the flocking room (e.g., on fabric rolls in and around the coating ranges). The blow–down process of using compressed air to clear loose flock from flock modules and flocking rooms was noted to be extremely dusty.

Numerous flock–handling process cyclones were noted to be open to the general plant air. Since these cyclone exhaust streams were not ducted outside the plant or filtered, fine dust particles in the flock process stream were being actively discharged into the air within work production areas of the plant.

Single–use respirators were used by a number of workers at various work locations throughout the plant. Microfibres' existing respiratory protection program was found to be inadequate in terms of medical clearance of workers for respirator use, fit–testing of respirators, and type of respirator selected for use.

Although not directly relevant to respiratory hazard, safety–toe shoes were not required in the plant, even though some workers are required to lift and manipulate heavy embossing rolls. Also, safety glasses were not required, even though many workers are in contact with a number of different liquid chemicals including, but not limited to adhesives, scours, and finishing compounds. Finally, many of the emergency eye–wash stations were found to be inoperable. (This latter finding was immediately relayed to management, and timely corrective action was taken.)

Particulate Sampling Results

In considering the following results, it is important to note that there was a tendency for inlets of respirable and total dust samplers and direct–reading monitors to become clogged with flock in the two work areas with higher levels of airborne flock, namely the flocking rooms and basement screening rooms. During sampling, sampler inlets in these areas were frequently unclogged by NIOSH industrial hygienists. The sampling results from the flocking rooms and basement screening room therefore probably underestimate true dust concentrations. Also, there appeared to be a tendency for the sampled particulate to cling to the inner surface of the filter cassette, thereby creating another potential reason that the measured dust levels are likely to be underestimates. Clogging was not a problem at the other ten work areas monitored (those results are not questioned).

Respirable Dust

Results for the 37 work area respirable dust samples collected in May 1996 and the three work

area respirable dust samples collected in August 1996 are shown in Table 3. Of the 37 samples collected in May, only one sample collected in the flocking room on Range 1 and one sample collected in the basement screening area exceeded the OSHA PEL for PNOR. An additional sample collected in the Range 1 flocking room exceeded the ACGIH TLV for PNOC. Measured concentrations ranged from 0.05 to 39.9 mg/m^3, with highest levels generally measured in the flocking rooms (0.53 to 39.9 mg/m^3) and basement screening room (0.08 to 5.02 mg/m^3).

Of the three samples collected in August (one in each of the flocking rooms and one in the basement screening room), the lowest measured respirable dust concentration was in the Range 1 flocking room, which was idle during most of the sampled work shift. Measured concentrations in the Range 2 flocking room and in the basement screening room were 0.72 and 0.90 mg/m^3, respectively.

The results of PBZ respirable dust sampling during blow–down operations in the Range 2 flocking room are shown in Table 3a. One set of results was collected during only the last 30–minutes or so of a blow–down that was already in progress when the samplers were placed on two workers involved in the blow–down. Measured respirable dust exposures were 1.96 and 0.18 mg/m^3. The other set of results is considered more representative of the task, having been collected throughout a blow–down that lasted approximately 90– minutes. Measured respirable dust exposures for the two involved workers were 9.8 and 15.3 mg/m^3. These samples represent partial–shift, task–specific exposure, and not a full–shift TWA. (No full–shift PBZ samples were collected in this investigation.) However, we observed that these workers spent most of their shift in the flocking room, so the measured task–specific exposures, together with the flocking room work area exposures (see above), suggest a clear potential for respirable dust exposures in excess of the ACGIH TLV, as well as the OSHA PEL, especially given the likelihood that these samples underestimated actual concentrations due to problems encountered with sampler clogging. During the blow–down operation, the involved workers wore disposable particulate respirators, but it is doubtful that this type of respirator afforded adequate respiratory protection.

Total Dust

Results for the 37 work area total dust air samples collected during the May 1996 survey and the three work area total dust samples collected in August 1996 are shown in Table 4. Measured concentrations ranged from 0.10 to 240.9 mg/m^3. As with the respirable dust samples, the highest total dust concentrations were measured in the flocking rooms and basement screening room. Concentrations measured in the flocking rooms ranged from 1.3 to 240.9 mg/m^3. Three of the four samples collected in the Range 1 flocking room exceeded the OSHA PEL of 15 mg/m^3.

One PBZ total dust sample was collected on a worker throughout a blow–down lasting about 90 minutes. The results showed a PBZ total dust exposure of 76.1 mg/m^3 (Table 3b). Especially given the likely underestimation of actual exposure associated with observed sampler clogging, these workers are considered to have been overexposed even if they wore properly fitted disposable respirators with an applied protection factor of 5.

Real–time Particulate Monitoring

Figure 1 shows the results of the real–time dust monitors collected on May 8, 1996, in the Range 1 flocking room. The monitors were activated at approximately 8:45 a.m., when the Range was not operating and blow–down had not yet begun. Initially, both respirable and total dust concentrations were relatively low within the room. However, at approximately 9:30 a.m., the blow–down started and dust levels began to increase substantially. Average total and respirable dust concentrations approached 20 and 30 mg/m^3, respectively. The dust concentrations within the room gradually dropped back to

baseline, mainly due to clogging of the instrument. At approximately 10:15 a.m. the blow–down was completed. The sampling statistics collected by the dust monitors during that particular time period and blow–down showed that total dust level within the room was as low as 0.03 mg/m^3 and peaked to over 200 mg/m^3. The overall average dust concentration for the sampling period was 1.24 mg/m^3. Respirable dust concentration for that period ranged from 0.07–30 mg/m^3 and averaged 2.51 mg/m^3. After blow–down, the dust monitors were unclogged, cleaned, re–calibrated and restarted at approximately 11:00 a.m. to continue measuring dust concentrations within the room.

Vertical Elutriator Sampling

Vertical elutriator dust sampling results measured during August 1996 in the two flocking rooms and in the basement screening room are shown in Table 5. No clogging was observed with the VE. The VE sampler filters were changed during the sampling period to avoid potential overloading. The Range 2 sampler filter changing was done after completion of a blow–down that occurred during the early part of the sampled shift. The dust concentration from the early portion of the shift that included the blow–down was 8.41 mg/m^3. The VE sample collected in the screening room was lost due to sampler pump malfunction

During the February sampling period, a total of 164 mg of dust was collected using a VE in the flocking room of Range 1 (32 mg), a VE in the Range 2 flocking room (74 mg), and a VE in the basement screening room (58 mg). Because the purpose of this sampling was to collect large quantities of dust for animal studies, dust filters were not changed frequently enough to avoid filter overloading. Therefore, no airborne dust concentration measurements have been calculated based on this sampling.

High Volume Cyclone Sampling

During the February 1997 sampling period, an additional total of 202 mg of dust was collected using high volume respirable dust cyclones placed in the flocking room on Range 1 (42 mg) and in the basement screening room (161 mg). The high volume cyclone did suffer some clogging problems, but the collected dust was still considered appropriate for use in the animal toxicology studies.

Microscopic Examination of Particulate

Microscopic examination of the respirable dust samples collected in the flock module rooms during August 1996 were observed to include oversized, non–respirable fibers that were the same size as the fibers seen in the bulk sample of flock. In contrast, the VE sample contained very few of these oversized flock fibers, resulting in a better measure of the respirable/thoracic concentration in the areas of high dust concentrations.

Total dust and VE dust samples collected in August 1996 both contained birefringent fibers which differed in size and overall geometry from the much larger nylon flock fibers. These were much smaller, more twisted, and less uniform in size and shape than the flock fibers. Cross–polar observations suggested an appearance like that of cellulose. (The backing material in the final product is a synthetic–cellulose blend.) However, despite striking differences in overall particle geometry, phase contrast observations revealed substantial similarity between the larger nylon flock fibers and the much smaller fibers in terms of color and surface appearance. This suggested that the smaller particles may have been formed by shredding of the larger nylon flock fibers during flock production. This, in fact, was suggested by scanning electron microscopy examination of the ends of nylon flock fibers (Figure 2). Although most of the ends showed a clean cut, some ends showed evidence of what appears to be shredding of nylon fibers in the respirable size range. As a follow–up analysis, these particles were concentrated using a cascade

impactor and their melting point was determined to be between 250–260° C. This is consistent with the reported melting point of nylon.

Endotoxin

Endotoxin assays were done on 35 work area air samples (16 respirable dust samples and 19 total dust samples) and on two PBZ samples (one respirable dust sample and one total dust sample). Results are shown in the rightmost columns of Tables 3, 3a, 3b, and 4. Endotoxin concentrations based on area respirable dust samples ranged from 0.7 to 138 endotoxin units per cubic meter of air (EU/m^3). Based on area total dust samples, concentrations ranged from 1 to 219 EU/m^3. The highest levels were measured in the flocking rooms (mean respirable = 70 EU/m^3; mean total = 85 EU/m^3) and the basement screening area (mean respirable = 38 EU/m^3; mean total = 92 EU/m^3). PBZ endotoxin concentrations measured during blow–downs were 32 EU/m^3 and 49 EU/m^3 based on assay of respirable dust and total dust samples, respectively (Tables 3a and 3b).

Endotoxin levels in the dust ranged from 2 to 30 EU/mg of respirable dust and from 1 to 24 EU/mg of total dust (Table 3, 3a, 3b, and 4). Variability in measurement is evident within the same sampling period and site. For example, for two nearly full–shift total dust samples collected at essentially the same time on May 8 in the Range 2 flocking room, measured EU/mg levels were 2.3 and 17.0 (Table 4).

Elemental Metals

The results for the 12 TWA samples collected for elemental metals are shown in Table 6. For the most part, only trace quantities of metals were found. None of the sampling results for any of the metals exceeded evaluation criteria for occupational exposure.

Gas/Vapor Sampling

Chromatograms showing individual volatile organic compound peaks detected by thermal desorption tube sampling in April 1996 are presented in Appendix I. Each peak detected on each sample is numbered 1–58. The table which accompanies these chromatograms in the appendix lists each peak number with its corresponding chemical identification. Each chromatogram has the same scale for comparison (same time and abundance axes). Major compounds identified on various samples were methanol, ethanol, isopropanol, methyl propenyl, and acetone. Other compounds detected included formaldehyde, 2–butoxyethanol, toluene, higher molecular weight aliphatic alcohols, branched alkanes, methyl propanoate, and ethyl butanoate.

Table 7 shows the May 1996 results of full–shift quantitative gas/vapor air samples collected in 12 work areas. The data presented in the table only show results of samples having detectable levels reported.

Total Hydrocarbons

Of the 12 full–shift air samples collected and analyzed for total hydrocarbons, only 4 samples showed detectable levels. The measured levels for those four samples were 0.34 ppm, 0.61 ppm, 2.6 ppm, and 0.17 ppm measured at Range 2 pre–coat area, Range 1 flock module room, Range 2 flock module room, and embossing; respectively. None of the measured concentrations exceeded the evaluation criteria for occupational exposure

Formaldehyde

Two of the highest formaldehyde concentrations, 0.44 ppm and 0.21 ppm, were measured at Range 1 and Range 2, respectively. Also, a concentration of 0.25 ppm was measured at the RPC 1–2 area, where no products containing formaldehyde are used. All other samples were below the minimum quantifiable concentration.

Measured formaldehyde concentrations were below the OSHA PEL of 0.75 ppm for a TWA exposure, as well as below the OSHA action level of 0.5 ppm. However, one measured value

(0.44 ppm) exceeded the ACGIH TLV® of 0.3 ppm (ceiling), and 10 of the 12 samples exceeded the NIOSH REL of 0.016 ppm TWA.

Ethyl Acrylate

Ethyl acrylate was not generally detected; only one of the 12 full–shift air samples had a quantifiable amount. This sample, collected in the Range 2 flocking room measured 0.16 ppm, but did not exceed occupational exposure evaluation criteria.

Ethyl Propanoate

Ethyl propanoate was detected in 8 of the 12 full–shift work area samples collected. Concentrations ranged up to 3.7 ppm, with the highest measured in the Range 2 flocking room. There are no occupational exposure criteria for ethyl propanoate.

Ethyl Acetate

Four of the 12 full–shift work area samples had detectable amounts of ethyl acetate. Three of the four samples with measurable amounts of ethyl acetate were collected in an area where adhesives are applied or used, and the other was collected in the area where adhesives are mixed. Even the highest concentration (1.26 ppm), measured in the Range 2 flocking room, did not exceed the evaluation criteria for occupational exposure.

Propylene Glycol

Five of the 12 full–shift work area samples had detectable amounts of propylene glycol. All five samples with measurable amounts of proplyene glycol were collected in an area where adhesives are applied or mixed. Even the highest concentration (0.09 ppm), measured at the Range 2 adhesive pre–coat area, did not exceed the evaluation criteria for occupational exposure.

Oxides of Nitrogen

No oxides of nitrogen were detected in any of the 12 full–shift workplace air samples.

Toluene and 1,1,1–trichloroethane

No toluene or 1,1,1 trichloroethane was detected on any of the 12 workplace air samples collected.

Butyl Cellosolve

No butyl cellosolve was detected in any of the 12 full–shift workplace air samples.

Nitrosamines

No nitrosamines were detected in any of the 12 full–shift workplace air samples.

Microbial Sampling

Fungi and Bacteria in Bulk Samples

The fungal and bacterial results from the 31 bulk samples are shown in Table 8. The majority of the fungi cultured and identified were various species of yeasts. Also, some samples contained assorted colonies of *Aspergillus*, *Fusarium*, and *Penicillium* species. The colony counts were relatively low, ranging from not detected to 10^9 colony forming units per gram (CFU/g) of solid material or CFU per cubic centimeter of fluid (CFU/cc). The bacteria cultures contained mostly *Bacillus*, *Pseudomonas*, and *Staphylococcus* species, and concentrations ranged from 10^1 to 10^9 CFU/g or CFU/cc. Gram–negative bacteria predominated.

Airborne Microbial Sampling

As mentioned in the "Evaluation Criteria" section of this report, there are no widely accepted guidelines regarding safe levels of exposure to airborne microorganisms. Allergic reactions can

occur in sensitized individuals, even with exposure to relatively low air concentrations.

Airborne Bacteria

Table 9 shows results of air sampling for viable bacteria. Average concentrations by work area ranged from 140 to 6080 CFU/m^3, with the highest found in the Range 1 flocking room. The highest time–specific concentration (7324 CFU/m^3) was measured during the morning in that same flocking room. The predominant species of bacteria identified was *Bacillus azotoformans*. The ambient (outside air) sample was entirely comprised of *Bacillus azotoformans* at a concentration of 4295 CFU/m^3.

Airborne Fungi

Table 9 also shows results of air sampling for viable fungi. Average concentrations by work area ranged from 129 to 1201 CFU/m^3, with the two highest in the compounding area (1201 CFU/m^3) and in the Range 1 Scotchgard® area (1133 CFU/m^3). The highest time–specific concentration (3039 CFU/m^3) was measured during the morning in the latter work area. The two predominant fungal species identified were *Cladosporium* and *Aspergillus fumigatus*, but a wide variety of fungi were identified, including many species of *Aspergillus* and some common indoor contaminant fungi (e.g., *Trichoderma, Gliomastix murorum, Paecilomyces varitoii*, and, to a lesser degree, *Penicillium)*. *Tritirachium* colonies were common in all the samples.

Medical Evaluation

Characteristics of Individuals with Physician–Diagnosed ILD

Medical information on the eight diagnosed cases of ILD among workers at the Microfibres plant is presented in Table 10. Seven were diagnosed by the local occupational medicine specialist. Initially, this physician thought the first few cases of ILD who came to his attention had HP. However, he later determined that, as a group, this cluster of cases did not satisfy diagnostic criteria for HP. The remaining worker was diagnosed with ILD by his personal pulmonary physician who felt that this patient had an early, "mild" case of ILD.

Two workers were diagnosed with ILD in 1992 and 1994, respectively; as a result of active case–finding undertaken by the local occupational medicine physician, the remaining six were diagnosed January–September 1996. The average age of these 8 cases was 39 years (range: 24 to 57). Only one had never smoked cigarettes, and one was a current smoker; six were ex–smokers, having quit smoking (from less than 1 year up to 7 years) prior to diagnosis.

Tenure at the plant at the time of symptom onset ranged from 10 months to 31 years (median 5 years). All but one (see below) had both shortness–of–breath and cough for several months to several years prior to diagnosis. Most described episodes of mild systemic symptoms, occurring within several hours after arriving at work.

One of the eight workers reported dramatic onset of illness with prominent systemic symptoms (including fever and chills; arthralgias in hands, elbows, knees; and severe fatigue). Onset occurred within two months of his immediate RPC work area being enclosed in plastic drapes to control moisture in the flock.

A restrictive pattern by spirometry was noted in five of the eight cases, and four of these five also

had a reduced diffusing capacity. Three of these five underwent OLB and histopathology in all three revealed evidence of ILD–BOOP in the worker with the dramatic onset of illness, and nonspecific interstitial pneumonitis (NSIP) in the other two. Two of the five with restrictive patterns did not undergo lung biopsy, but BAL revealed evidence of neutrophilic and eosinophilic alveolitis in both (35% neutrophils and 28% eosinophils in one, and 25% neutrophils and 15% eosinophils in the other).

Three of the eight cases did not have a restrictive pattern by spirometry. Two of these three had an obstructive pattern by spirometry, normal diffusing capacity, and normal standard CXR. However, HRCT revealed abnormalities, and biopsies (TBB in one and OLB in the other) were considered consistent with ILD–NSIP in both. One of the eight cases had normal spirometry, normal diffusing capacity, normal CXR, and questionable abnormality on HRCT. This worker underwent BAL, which revealed an acute eosinophilic alveolitis (10% neutrophils and 25% eosinophils), and TBB, which was consistent with ILD–NSIP with eosinophilia.

Biopsy slides for five of the six biopsied cases were reviewed by a NIOSH clinical pathologist, who concurred with the hospital clinical pathologists' findings. All showed lymphocytic interstitial inflammatory infiltrates.

Notably, work stations for these eight cases were localized to only two production areas: the Ranges in the Coating department (where flock is applied) and the RPCs (flock–cutting area) in the Raycote department. All eight workers improved in terms of both symptoms and objective clinical findings when removed from the workplace (n=7) or assigned to a non–production area (n=1). As of November 1997, two of the seven removed from work had returned to work in non–production areas. Two others attempted to return to work, but left again; the first one left permanently as a result of an apparent acute asthma attack after only a few days back at work, and the second one left permanently as a result of recurrence of cough and shortness–of–breath.

An Additional Earlier Case of ILD

In 1997, the NIOSH medical officer was informed by both the local occupational medicine physician and the pulmonologist of one of the eight cases, of a former worker from the Pawtucket plant who, while employed at the plant, had onset of an illness very similar to the more recent cases. This worker became symptomatic with a non–productive cough, fatigue, polymyalgias, and shortness–of–breath after more than 20 years of employment at the plant. A former smoker with 7.5 pack–years of smoking, had quit smoking 26 years prior to being diagnosed as having ILD. NIOSH interviewed this individual and reviewed his medical records. Objective studies showed a restrictive pattern on spirometry testing, diffuse reticulonodular infiltrates on CXR, and BAL eosinophilia. An open lung biopsy done in 1985 revealed a peribronchiolar lymphocytic and plasma cell infiltrate, consistent with a diagnosis of ILD. This worker was treated with steroids from 1985–1988 and with cytoxan from 1987–1990. Within 18 months of leaving the plant in 1988, he felt subjectively better, but his objective test results did not return to baseline status until a few years after leaving the plant. During his tenure at the Pawtucket plant, he reported working at the RPCs; he denied ever working on the Ranges.

Interviews With Range Workers

All 24 workers assigned to work the Ranges during the first and second shift, along with a Range supervisor and an employee who worked next to the Ranges, were interviewed. Almost two–thirds (16, or 62%) of these 26 workers reported having chronic dyspnea on exertion, over half (14, or 54%) reported having a chronic non–productive cough, and four (15%) reported having been diagnosed with pneumonia. One worker reported having been diagnosed with pneumonia twice within the preceding two years; in neither instance was a specific etiology determined. Chest pain and fatigue were reported by three workers. Two stated that their pulmonary function tests were abnormal; one of these two

was a physically active non–smoker. Only four (15%) of the 26 interviewed workers had no respiratory or systemic symptoms.

In addition to reports of extreme dustiness and the presence of loose flock in their working area, the interviewed Range workers suggested several other particular concerns that they felt warranted attention. One of their concerns was the desiccant powder that is added to the flock to control moisture content and prevent clumping of the flock; many workers complained of nosebleeds, sore throats, and skin irritation when working with this material. Another concern was the fabric protector that is sprayed onto the flocked fabric and then heat cured; this process was described as generating smoke, and sometimes creating a general "fog" in the work area. A final concern related to the hot–oil embossing process; this process also generates smoke, especially when the oil pump occasionally malfunctions.

NIOSH Medical Survey

Questionnaire

Among the 170 workers employed at the plant, 151 (89%) completed the questionnaire; 146 of the 151 did so during the May 1996 medical testing, and five others who participated in the June 1996 testing mailed self–completed questionnaires to NIOSH.

For purposes of data analysis, five workers who did not report working for one of the 7 major departments were assigned to departments as follows: two off–site warehouse workers were assigned to Shipping; one offsite office worker was assigned to Office; one print range worker was assigned to Coating; and, on the basis of job title, one other worker was assigned to Raycote. Only three participants were from Compounding; for analyses, these were aggregated with Dye House workers. With these assignments, 17% (n=25) of respondents currently worked in Dye House/Compounding; 16% (n=24) in Raycote; 39% (n=59) in Coating; 7% (n=11) in Maintenance; 7% (n=11) in Shipping/Warehouse; and 14% (n=21) in Office.

The median age of survey participants was 39 years (range: 18 to 71; average 40). Eighty–nine percent (134) were male; 108 (72%) were white, 24 (16%) Hispanic, 13 (9%) non–Hispanic black, and 4 (3%) Asian/Pacific Islander; 2 (1%) reported their race as "other."

One–third (n=49) of the participants were current smokers with a mean of 28 pack–years (range 1 to 126) of smoking; slightly more than one–third (n=57) were ex–smokers, with a mean of 17 pack–years (range <1 to 105) of smoking. Two–thirds (n=71) of participants reported having smoked while performing their job. Non–Office workers (production workers) reported smoking prevalences (70% ever smokers) that were very similar to those reported by Office workers (71% ever smokers). There were no major differences in smoking among departments (Table 11).

The median tenure working at the plant was 6 years (range <1 to 37 years). Thirteen percent (n=20) had worked less than one year, 35% (n=53) had worked from 1 to 5 years, and 52% had worked more than 5 years. Mean tenure was 8.3 years overall, ranging from 5.8 years in Coating workers to 11.7 years in Shipping/ Warehouse workers (Table 11).

Among non–Office participants, a typical work week was more than 5 days per week for 94% (n=119) of participants, and 19% (n=24) reported working 7 days per week; only two reported working fewer than five days per week. The mean number of days worked per week was 6.0 overall, ranging from 5.4 in Office workers to 6.3 in Raycote workers (Table 11).

Multiplying the number of days per week and the number of hours worked per day reported by each participant, the average number of hours per week worked by non–Office workers ranged from 16 to 112. The mean number of hours worked per week was nearly 54 overall, ranging from 48.4 in Office workers to nearly 52 in Raycote workers (Table 11).

One third (n=51) of all participants reported usually working on the Ranges, with department specific frequencies ranging from 10% among Office workers to 70% among Coating workers (Table 11). Over 60% (n=94) of participants stated that they had never worked on the Ranges, with department–specific frequencies ranging from 24% among Office workers to 86% and 90% among Coating workers and Maintenance workers, respectively.

Overall, 68% (n=103) of participants reported that they had never participated in blow–downs (Table 11). By department, blow–down participation ranged from 19% among Office workers to 97% among Coating department employees (Table 11). Two of the Office workers reporting having ever participated in blow–downs had been assigned to other departments (Coating and Raycote) in the past, one reported occasionally working on the Ranges even though assigned to the Office department, and the other reported having been present in the immediate vicinity while blow–down was performed by others.

Overall, 27% (n=41) reported having ever worked in the "pit," with department–specific frequencies ranging from 14% among Office workers to 82% among Maintenance workers (Table 11).

Five (3%) participants reported that, as a result of a respiratory problem, they had changed their assigned department or job at the Pawtucket plant. Four of these five changes in assignment occurred during 1991–1995; the other had occurred many years earlier. Two had transferred from Coating, two from Dye House, and one from Raycote. One additional worker, who had previously worked in Raycote, reported a departmental transfer due to eye, nose, and throat irritation. At the time of the medical survey, three other participants were on sick leave, having been recently diagnosed with ILD. (These three are included among the eight physician–diagnosed cases described above.)

Twelve (9%) participants reported that acute onset of respiratory symptoms (i.e., cough, wheeze, chest tightness, shortness–of–breath) had caused them to leave work suddenly on at least one occasion. Three reported that they went to the emergency room and another to a physician's office; diagnoses included bronchitis, pneumonia, and asthma. Dust was implicated by four affected workers as the cause of the sudden onset symptoms, and chemical fumes by three; the others did not specify a cause in describing the event. An additional four (3%) participants reported having left work because of: fever (n=2), irritation and swelling of eyes (n=1), and dizziness (n=1).

The prevalence of frequent respiratory symptoms with onset since employment at Microfibres is presented by department in Table 12. Overall, the following symptoms were reported by about 30% of participants: frequent shortness–of–breath; frequent dry cough; frequent chest tightness, and frequent cough with phlegm. Frequent wheeze was reported by about 20% of participants overall. Compared with Office workers, each of these symptoms were generally reported by higher percentages of workers in each of the production departments, as reflected by prevalence ratios greater than 1.0. The one exception was the Shipping/Warehouse department (which included off–site warehouse workers), for which prevalences of three of the five frequent respiratory symptoms were less than corresponding prevalences among Office workers. Prevalence ratios for Maintenance workers were elevated for all five frequent respiratory symptoms, and statistically significant for four of these five.

Many of those who reported frequent respiratory symptoms with onset since employment at Microfibres also reported that these symptoms improved when they were away from work. Overall, more than half of those with frequent dry cough, cough with phlegm, wheeze, or chest tightness, as well as more than 45% of those with frequent shortness–of–breath, reported symptom improvement when away form work. Compared to Office workers, improvement away from work was reported much more frequently by workers in production departments, with improvement most

frequently reported by Raycote, Coating, and Maintenance workers. Of note, the only Office worker to report improvement away from work also reported working on the Ranges and blow–down exposure.

Frequent systemic symptoms with onset since employment at Microfibres were reported less frequently than respiratory symptoms, but two–thirds or more of those reporting these symptoms also reported improvement when away from work (Table 13). All five Coating workers who reported frequent fevers also reported improvement when away from work. (The one Office worker who reported frequent fevers also reported working on the Ranges and blow–down exposure.) Generalized body aches with onset since employment at Microfibres were reported by one–fourth to nearly one–third of workers in the Coating, Raycote, and Maintenance departments, but by no Office workers. Again, many of those reporting this symptom, including three–fourths of affected Coating workers and all affected Raycote workers, reported improvement when away from work.

Frequent irritant symptoms with onset since employment at Microfibres followed a pattern similar to corresponding systemic and respiratory symptoms (Table 14). Overall, throat irritation was reported by one–fifth of participants and eye irritation was reported by one–fourth of participants. None of the Shipping/Warehouse workers reported eye or throat irritation; only one Office worker reported throat irritation. For the non–Office departments, two–thirds or more of affected workers reported improvement in frequent irritation symptoms when away from work.

Most of those with the frequent symptoms discussed above reported a duration of at least several months; the median reported duration for each of these symptoms ranged from 6 to 18 months.

A total of 86 participants reported at least one of the frequent systemic and/or respiratory symptoms (with onset since employment at Microfibres) discussed above (Table 15). Age, race, sex (data not shown), smoking status, tenure at the plant, and working in the pit were not significantly associated with these symptoms. Factors that were significantly associated with these symptoms were: days per week worked; hours per week worked; ever participating in blow–downs; and working on the Ranges (both usually and ever). Non–Office workers were significantly more likely to report frequent systemic and/or respiratory symptoms than were Office workers. By individual department, elevated prevalence ratios (using Office as the reference) were observed, all but one of which was statistically significant, for all non–Office departments except for Shipping/Warehouse (Table 16). There were statistically significant trends of increasing symptom frequency associated with days per week worked and with hours per week worked ($p<0.001$ for both). Those working seven days per week had a symptom prevalence nearly 3.5 times those working five or fewer days per week, as did those working 65 or more hours per week compared to those working 45 or fewer hours per week (Table 15).

Compared to Office workers, non–Office workers were nearly twice as likely to report having had two or more flu–like illnesses, and nearly three times as likely to report having had two or more attacks of shortness–of–breath with wheeze (Table 17).

Fifteen non–Office workers, including four each from Raycote, Coating, and Maintenance, reported having been told by a physician that they had asthma. In comparison, no Office workers reported having been told that they had asthma. Similarly, 14 non–Office workers, including five from Raycote, six from Coating, and two from Maintenance, reported having been diagnosed with pneumonia in the past 5 years. Again, none of the Office workers reported having been similarly diagnosed.

None of the Office workers reported symptoms of chronic bronchitis (i.e., productive morning cough for at least three months each year and for at least the last two years). Among non–Office workers,

symptoms of chronic bronchitis were reported by 20% (n=18) of the ever smokers and 13% (n=5) of the never smokers. Smoking–associated chronic bronchitis prevalence ratios (comparing prevalence among ever smokers to prevalence among never smokers) were 1.54 (0.6–3.9) among non–Office workers and 1.53 (0.6–3.9) among all participants.

Chest X–rays

Two of the 145 CXRs taken were considered uninterpretable due to technical problems. None of the other 143 interpretable films were considered abnormal with respect to consensus or median reading of small opacity profusion.

Spirometry

One hundred forty–five workers performed spirometry. Nine (6%) had a restrictive pattern and one had a minimally reduced FVC, but insufficient reproducible trials to document a definite restrictive pattern. Twelve (8%) had an obstructive pattern.

Forced vital capacity results are presented by department in Table 18. Mean FVC percent predicted values were less than 100% for both Coating workers and Maintenance workers, and the mean value for non–Office workers was slightly less than that for Office workers. However, compared to that for Office workers, none of these mean values were statistically significant. A restrictive spirometry pattern was observed more frequently among non–Office workers as a group than among Office workers (prevalence ratio=1.4), although neither this nor any other department–specific prevalence ratio was statistically significant. There were no statistically significant differences in mean FVC (percent predicted) in univariate analyses of the factors listed in Table 15.

Diffusion Capacity

Not including four workers (with normal test results) who did not complete the questionnaire, a total of 110 workers participated in diffusion capacity testing.

Thirteen non–Office workers, including six in Coating and three in Raycote, and no Office workers had DL_{CO} test results less than 80% of predicted (Table 19). Mean percent predicted DL_{CO} values exceeded 100 only for Office workers and for Shipping/Warehouse workers. Means for other departments ranged from 92% (for both Maintenance workers and Raycote workers) to 98% for Compounding/Dye House workers (Table 19). There were no statistically significant differences in mean DL_{CO} (percent predicted) in univariate analyses of the factors listed in Table 15.

Symptom Correlation with Test Results Among all participants, mean percent predicted values for DL_{CO} ($p<0.02$), FVC ($p<0.07$), and FEV_1 ($p<0.09$), but not the FEV_1/FVC ratio, were lower among those with at least one or more frequent systemic and/or respiratory symptoms compared to those without (Table 20). Stratifying by smoking status, this same pattern of association of symptoms with objective testing results remained quite evident and was statistically significant among ever smokers, but not among never smokers (Table 20). As shown in Table 21, symptom prevalence among ever smokers remained significantly associated with days worked per week, hours worked per week, blow–down (ever), range (ever), and department, with prevalence ratios for each of these factors similar in magnitude to those shown for all participants in Table 20.

Toxicology Studies

NIOSH has recently begun animal (rodent) toxicology studies to assess short–term pulmonary responses to airborne dust samples collected in the plant, as well as to fine particulates generated in the laboratory by milling unprocessed nylon tow fibers. Preliminary results have shown that both airborne dust from the plant and nylon fibers which were milled in the laboratory (these milled

fibers had properties similar to those at the plant), and therefore were without flock finish, caused an intense inflammatory response. Intratracheal (i.e., into the trachea, or windpipe) instillation (IT) of this dust, followed by sample times at 1 day and 29 days post IT, revealed a strong inflammatory response in the lungs of the rodents. Acute lung injury at 1 day post IT was evident through measurement of several markers of inflammation: (1) an influx of polymorhonuclear neutrophils (white blood cells); (2) evidence of red blood cells in the fluid from the lungs; and (3) identification of reactive products from the lung tissue cells.

The presence of these markers indicates lung inflammation and injury. NIOSH maintains a reference bank for various dusts which are not known to produce significant lung injury. Levels of these three biomarkers were much higher than that typically seen from our reference bank of dusts. Also, IT instillation of dust from this plant produced inflammation in the rodent lungs that was significantly greater than that seen from silica and coal (two reactive dusts of occupational significance).

DISCUSSION AND CONCLUSIONS

Physician–Diagnosed Cases of ILD

In carrying out this evaluation, NIOSH investigators responded to an unusual occurrence of two cases of interstitial lung disease occurring over a relatively short period of time in a relatively small work force. In fact, with subsequent case–finding efforts by the local occupational health specialist, a total of eight cases of ILD were diagnosed in the 1992–1996 period among workers at the Microfibres plant in Pawtucket (Table 10). (An additional earlier case, diagnosed in 1985, was also identified.) This cluster of physician–diagnosed cases of ILD among a current workforce of approximately 170 suggests a prevalence on the order of about 5% (about 5,000 cases per 100,000 population), far exceeding published estimates of ILD prevalence in the general population reported by Crystal [1992] of approximately 0.03% (30 per 100,000), as well as population estimates for ILD reported by Coultas et al. [1994] of approximately 0.08% (81 cases per 100,000) among males and about 0.07% (67 cases per 100,000) among females

For seven of the nine physician–diagnosed cases at the Pawtucket plant (including the case diagnosed in 1985) the presence of ILD was confirmed by lung biopsy (five had OLBs and two had TBBs only). The two cases that did not undergo biopsy both had clear BAL evidence of alveolitis, a common correlate of active ILD. (One of these latter two had a normal HRCT and was not included on the local occupational physician's final list of eight total cases, even though his worker was considered to have ILD by a local pulmonary physician.) BAL findings were clearly consistent with alveolitis in five of the six cases of ILD who underwent BAL. Three (50%) of these six had BAL eosinophilia, a finding consistent with a reported 42% (20 of 48) of patients with ILD who have BAL eosinophilia of at least 5% [Allen et al. 1990].

All but one of the workers with physician–diagnosed ILD at the Pawtucket plant worked in Raycote department jobs cutting flock (n=3), or in Coating department jobs on the Ranges where flock is applied to fabric (n=3), or in Maintenance department jobs involving repair of flock cutting and screening equipment or Range machines (n=2). This indicates a prevalence of ILD mong workers assigned to these jobs that exceeds the very high prevalence discussed among the overall workforce in this plant.

In the absence of any other explanation, the remarkably high prevalence of ILD among workers at the Pawtucket plant clearly suggests a causative association between the occurrence of this disease and exposure to some etiologic agent(s) at the plant. The distribution of ILD among workers in this plant suggests that exposure to the causative agent(s) is more

frequent and/or more intense in work areas primarily involving cutting and applying flock.

Additional evidence supporting the work–related nature of ILD diagnosed among workers at the Microfibres plant in Pawtucket derives from consideration of the general course of illness in affected workers. In this context, one of the several affected workers with systemic symptoms merits special attention. Consistent with a dose–related disease process, within weeks of having his work area enclosed in plastic sheeting, this worker developed remarkably acute systemic symptoms and clear disease progression. Also arguing strongly for an occupational etiology is the significant subjective and objective clinical improvement of most of the cases within weeks to months of removal from the workplace or reassignment to a non–production job. This particular evidence is strengthened substantially when considered along with the experience of those who returned to work at the plant. Cough and shortness of breath recurred in one individual who returned to work, while two others who returned to non–production jobs (presumably involving less exposure) have apparently fared well without recurrence.

With one exception, the first several cases of ILD to be diagnosed were more severely affected (in terms of objective functional and radiographic indicators of disease) than the subsequently diagnosed cases. This pattern of severity is not unexpected for a condition for which an occupational relationship was not initially suspected in the first cases, but for which active case–finding was used by a physician with a high index of suspicion to identify the later cases. The one exception to this pattern was the most recently diagnosed case, discussed in the preceding paragraph, in whom onset was rather dramatic following probable substantially increased exposure to flock–associated airborne dust following enclosure of his flock–cutting work area.

Respiratory bronchiolitis is seen rarely in heavy cigarette smokers and shares some clinical features with ILD [Myers et al. 1987]. However, only one of the biopsied cases was noted to have histopathologic characteristics warranting a diagnosis of respiratory bronchiolitis (noted in addition to a diagnosis of non–specific interstitial pneumonitis), and the cellular characteristics of the alveolitis reflected in the BAL findings clearly distinguishes the ILD in flock workers from respiratory bronchiolitis associated with cigarette smoking. Nevertheless, all but one of the nine workers from the Pawtucket plant who were diagnosed as having ILD were current or ex–smokers, raising the possibility that smoking may play a contributory role in this disease.

HP is one type of ILD caused by various inhaled agents. While a specific diagnosis of HP was entertained for more than one of the cases diagnosed at this plant, an assessment of clinical information from all the cases strongly suggests that this ILD is something other than HP. BAL eosinophilia is not a common finding in cases with HP. Also, the high prevalence of "ever" smoking among this group supports a diagnosis other than HP [Warren 1977]. Finally, no granulomas were identified in the lung biopsies of cases from this plant, providing additional evidence arguing strongly against HP as a diagnosis [Reyes et al. 1982].

Importantly, an excessive occurrence of ILD has also been reported among workers at a similar Microfibres plant in Canada [Lougheed et al 1995]. The occurrence of two independent outbreaks of ILD in two similar plants, one in Rhode Island and the other in Canada, very strongly implicates an occupational exposure common to both these plants in the etiology of this disease.

Exposure Evaluation

Alveolitis and ILD, when caused by an inhaled agent, require that the inhaled agent is of "respirable" size (i.e., small enough to be deposited in the alveolar regions of the lung). Aerodynamic principles and anatomy of the respiratory system dictate that larger particles are deposited in the nose, throat, or bronchi before

reaching the alveolar regions of the lung. Given the physical diameters of nylon "tow" processed in the Pawtucket plant (i.e., 15 to 20 microns), the flock fibers themselves cannot be considered respirable, and therefore cannot be plausibly implicated as causing the outbreak of ILD in this plant. While larger particles of flock itself cannot be plausibly implicated, airborne respirable dust including nylon fragments generated during flock production, flock application, and other flock–handling processes is certainly suspect.

Consistent with the predominant work locations of the physician–diagnosed cases, the highest levels of airborne respirable dust were measured in work areas associated with the Raycote department (specifically, the flock screening room) and with the Coating department (specifically, the flocking room). In both these areas, respirable dust levels clearly exceeded the ACGIH TLV of 3 mg/m^3 for PNOC, as well as the OSHA PEL of 5 mg/m^3 for PNOR. (Note that due to clogging of sampler inlets by flock fibers, these measured levels probably underestimated the actual levels of respirable dust in these areas of the plant.)

The OSHA PEL for PNOR is intended as a TWA exposure over a typical 8–hour workday and 40– hour work week. However, most workers at the Microfibres plant in Pawtucket worked substantially more than the typical 8–hours per day and five days per week work schedule, making the 5 mg/m^3 PEL inadequate as general guidance. In such situations, OSHA practice is for field inspectors to reduce applicable PELs to adjust for non–standard work shifts. For example, to protect exposed employees working 16 hours per day, five days per week, the 5 mg/m^3 PEL for PNOR would be effectively reduced to 2.5 mg/m^3 (an equivalent reduction of the ACGIH TLV® would yield an adjusted TLV of 1.5 mg/m^3). Likewise, if an individual would work a 16 hour shift, seven days a week, his reduced PEL would be 1.8 mg/m^3, and his TLV would be 1.1 mg/m^3 [Paustenbach 1993].

Based on preliminary NIOSH toxicology studies showing that airborne dust from this plant is remarkably inflammatory when instilled in the lungs of animals, the PEL for PNOR (and TLV for PNOC) probably does not represent adequate guidance to fully protect the health of exposed Microfibres employees, even when adjusted for extended work shifts/work weeks.

Airborne respirable dust concentrations during flocking room blow–down substantially exceeded PNOR/PNOC evaluation criteria. While the extreme levels measured during blow–down were somewhat transient, they contribute substantially to the overall exposure of those workers in the immediate vicinity of the blow–down.

Openness between various work areas of the plant, as well as the imbalance of ventilation between various work areas, both increase the likelihood that workers in other areas of the plant would also be exposed, albeit to lower levels, of dust generated during blow–down. In addition to more obvious sources of airborne flock–associated particulate (e.g., blow–downs, manual pouring of bagged flock and fugitive dust from automated processes in and around the flocking module), open–topped process cyclones within the Pawtucket plant represent a highly suspect source of respirable particulate.

Although conditions at the plant may have been somewhat conducive to microbial growth, environmental sampling conducted by NIOSH did not identify any obvious locus of microbial amplification and dissemination. Nevertheless, in both the basement screening room and flocking room, measured concentrations of airborne endotoxin were high enough to exceed occupational respiratory health evaluation criteria [Dutch Expert Committee 1997; Rylander 1997].

In contrast to airborne dust, essentially no evidence exists by which to implicate gases/vapors in this plant as a primary cause of the ILD observed in excess among workers at the Pawtucket plant. More on the nature of the respirable dust in this plant is discussed below (see "Specific Etiology").

NIOSH Medical Survey Findings

Despite limitations including the relatively small size of the population available for study at this plant, the lack of a sizeable unexposed subgroup of workers at this plant, and the cross–sectional nature of the survey, the NIOSH medical survey found evidence of excess of symptoms among Microfibres employees, as well as evidence that these excess symptoms are work–related.

Each non–Office department (except for Shipping/Warehouse) reported substantially higher prevalences of frequent eye and throat irritation–on the order of four to six–fold higher for throat irritation–than did Office workers. Over two–thirds of those reporting these symptoms also reported improvement when away from work.

Likewise, with respect to systemic symptoms, frequent "aches all over" were reported by nearly one–fourth of non–Office workers, contrasted with none of the Office workers, and two thirds of those with this symptom reported improvement when away from work. Although frequent fevers were reported less commonly, five of the six non–Office workers with this symptom also reported improvement when away from work.

Despite similarity in smoking between Office and non–Office workers, prevalences of these lower respiratory symptoms were about two to three times higher among non–Office workers than among Office workers. While only for Maintenance department workers were increased prevalences (relative to Office workers) of these symptoms statistically significant, it needs to be emphasized, that Office workers at the Microfibres plant in Pawtucket do not represent a truly unexposed comparison group. Questionnaire responses indicated that several workers assigned to the Office department were clearly exposed to production areas in the course of their work. To the extent that the Office workers' production area exposures may have been sufficient to have caused symptoms, this would have the effect of reducing prevalence ratios based on Office worker comparisons.

Depending on the symptom, nearly half or even more of non–Office workers reporting these frequent respiratory symptoms noted improvement when away from work, and the only Office worker reporting improvement away from work was one who also reported working on the Ranges, including blow–down exposure.

At least two of the physician–diagnosed cases had experienced systemic symptoms along with lower respiratory track symptoms, including wheezing. Additional analysis of these reported frequent symptoms was based on whether or not individual workers reported any one or more of these seven symptoms. The overall symptom prevalence among non–Office workers was more than twice that of Office workers; by individual department, only Shipping/Warehouse workers were not more likely than Office workers to have reported one or more of these symptoms (Table 16). All other departments were associated with prevalences on the order of two to three times as high as that among Office workers, and for all but the Raycote department was the elevated prevalence statistically significant.

Additional evidence for an association between symptoms and occupational exposure is summarized in Table 15. Days worked per week and hours worked per week were both strongly and statistically significantly associated with symptoms, those working 7 days per week or more than 65 hours per week having symptom prevalences nearly 3.5 times as high as those working five or fewer days per week or less than 45 hours per week. Moreover, this association was clearly "dose–related." Intermediate, yet statistically significantly elevated prevalence ratios of approximately 2.5 were observed among those who worked six days per week or between 45 and 65 hours per week compared with those working fewer days per week or fewer hours per week.

The association of symptom prevalence with tenure at the plant, though not statistically

significant, was negative (i.e., symptoms were less commonly reported by those with longer tenures). While not the only possible explanation for such a negative relationship, it is possible that this reflects a so–called "healthy–worker effect" [Arrighi and Hertz–Picciotto 1994; Choi 1992] very commonly observed in occupational disease studies that include only workers employed at a particular point in time (i.e., cross–sectional studies). Such studies exclude workers who have left employment due to clinical or even subclinical symptoms. With respect to the Microfibres plant in Pawtucket, it is well documented that several affected workers had already left employment due to symptomatic ILD before the NIOSH survey. It is not unreasonable to suspect that other affected workers with less severe symptoms may also have disproportionately left the plant before the NIOSH survey, especially given the finding that affected workers commonly perceived an improvement in their symptoms when away from work.

In terms of other work–related factors ascertained by the survey questionnaire, Range work and blow–down exposure were each associated with statistically significant symptom excesses. The questionnaire did not allow for distinguishing the frequency of blow–down exposures for individual workers, so it was not possible to evaluate a possible graded exposure–response relationship for exposure to blow–downs. Nevertheless, this finding of a significant association of blow–down exposure with symptom prevalence is particularly noteworthy. Because blow–down is a process characterized by extreme dustiness, this finding implicates dust exposure in this plant as a health hazard.

In addition to dust exposure, there were other *a priori* concerns regarding other possible hazards in the Pawtucket plant. The NIOSH survey questionnaire asked about working in "the pit" area, but a positive response to this question was not associated with a statistically significant symptom excess. The questionnaire did not include any questions ascertaining the frequency and or intensity of workers' exposure to "smoke" or "fog" generated during the heat curing of the fabric after it is sprayed with fabric protector or during hot oil embossing of fabric. As a result, there is no data with which to directly address *a priori* worker concerns regarding these exposures.

Smoking was not associated with symptom frequency (Table 15). Importantly, even in an analysis restricted to ever–smokers alone, the same work–related factors (i.e., days worked per week, hours worked per week, blow–down, Range work, and assigned department) all maintained statistically significant associations with symptoms, and those associations were in the same direction and of the same magnitude (Table 21).

One must question whether a substantial proportion of the physician–diagnosed pneumonia reported by survey participants represented the same non–infectious ILD process as that confirmed by lung biopsy among workers at this plant. Not only did reported physician–diagnosed pneumonia follow a distribution very similar to that observed for physician–diagnosed ILD, but interviews with the physician–diagnosed ILD cases revealed that one of them had been diagnosed as having "pneumonia" on two occasions before being definitively diagnosed as having occupationally–related ILD.

Likewise, multiple episodes of flu–like symptoms in the past year followed a similar pattern. Again, one must question whether these occurrences may also be related in some ways to the same ILD process. Although most of those diagnosed with ILD at the Pawtucket plant experienced insidious onset of their symptoms, more than one did have a relatively acute onset of systemic symptoms that perhaps could have been construed as flu–like episodes. (Acute exacerbation of symptoms was also reported by several of the physician–diagnosed cases of ILD from the Microfibres plant in Ontario.)

At least one of the physician–diagnosed cases of ILD at this plant had reported wheezing as a major symptom. The questionnaire survey data revealed higher (though not statistically significant) prevalences of recent multiple attacks of

shortness–of–breath with wheeze among non–Office workers overall, as well as among all individual non–Office departments except for Shipping/Warehouse. The questionnaire data also indicated that all 15 workers who reported having been told by a physician that they had asthma were non–Office workers, and that 12 of these 15 worked in Raycote, Coating, or Maintenance departments. Although the questionnaire did not ascertain when these workers were told they had asthma, it did ascertain both current frequent wheezing and recent attacks of shortness–of–breath with wheeze (see above). Although wheezing and, more specifically, attacks of shortness of breath with wheezing, are more commonly associated with primary airways disease (e.g., asthma) than with interstitial disease, it is well–documented that airways inflammation can accompany ILD caused by inhaled agents [Redlich 1996, Rose 1996].

Alternatively, even in the absence of exposure sufficient to cause deep lung (i.e., interstitial) inflammation, the same agent(s) responsible for causing the ILD (or an associated agent—perhaps non–respirable flock fibers themselves) could be responsible for airways irritation and/or inflammation manifested by wheezing in susceptible workers.

CXRs taken in the NIOSH survey were essentially completely negative, and pulmonary function test results were not so clearly associated with work–related factors as were symptoms. Nevertheless, though the relationships were not statistically significant, both restriction assessed by spirometry and low diffusion capacity were more frequent among non–Office workers, and mean values of FVC (percent predicted) and DL_{CO} (percent predicted) were lower among non–Office workers overall (as well as among workers in nearly all non–Office departments except Shipping/Warehouse), compared to Office workers.

The fact that objective pulmonary function testing done in the NIOSH survey was not so clearly supportive of a work–related adverse health effect as were the questionnaire findings is not reason to reject the more subjective questionnaire findings outright. The same should be said for the negative CXR findings from the survey. In fact, it would be entirely expected that the active case finding effort, which was ongoing at this plant before the NIOSH survey, would have tended to identify (and subsequently remove from exposure) the most affected workers. More importantly, some physician–diagnosed cases of ILD at this plant–those that were arguably milder cases–presented without a spirometric pattern of restriction, without a low diffusion capacity, and without an abnormal chest radiograph (Table 10).

The finding that mean FVC and DL_{CO} values, but not mean FVC/FEV_1 ratio values, were generally lower among those with symptoms compared to those without symptoms is consistent with a restrictive disease (such as ILD) rather than an airways disease. The strength of this finding among ever smokers, but not among never smokers, together with the preponderance of ever smokers among the physician–diagnosed cases of ILD at this plant, suggests that smokers may be especially susceptible to the risk of work–related ILD at this plant.

Specific Etiology

As discussed above, evidence from the physician–diagnosed cases, the NIOSH industrial hygiene survey, and the NIOSH medical survey most strongly implicates some respirable particulate in the plant as the etiologic agent.

Until the adverse health effects experienced by workers exposed at this plant can be specifically attributed to one or more clearly defined components of the respirable dust in this plant, the most effective approach to prevention would necessitate substantial reduction of workers' exposures to respirable dust, in general, through a combination of process changes, engineering and administrative controls, and (as needed) personal protective equipment (PPE). (See "Recommendations," below.)

Especially given the observed excessive airborne dust levels, substantial reduction of general respirable dust is warranted in some areas of the Microfibres plant in Pawtucket, regardless of the nature of that dust. In addition, it may be appropriate to consider eliminating any specific components of the dust (or modifying any characteristics of the dust), if feasible, that are shown to represent special respiratory health hazards.

Based on the initially recognized cases of ILD thought to be possibly work–related at this plant, the etiologic exposure was hypothesized to be predominantly concentrated along the Ranges. However, subsequent cases worked in the flock cutting areas (in the Raycote department), and case information and survey data implicate both the Ranges and RPCs (Raycote department). Components specific to the Range only–such as the desiccant powder, the spray–on fabric protector, or the adhesive–are therefore not likely to represent the specific etiologic agent. Furthermore, pathology specimens from the workers with this lung disease were not consistent with a silicatosis, arguing against the desiccant as the source. Also, according to Microfibres management, the spray–on fabric protector has not been used in the Canadian plant where an outbreak of ILD has also occurred [Lougheed et al. 1995], arguing against this material as the cause. Fragments of cellulose, observed in the respirable dust samples collected in the plant, are likewise not considered responsible for the disease seen in this workforce. In fact, purified cellulose has been used for "control" exposures in respiratory toxicological studies. Finally, the adhesive used in the process is quite viscous and is poured, not sprayed, so is not likely to become airborne as particulate.

A microbial air contaminant, high on an early list of suspected causes of the ILD among Microfibres workers at a time when the disease was thought to be HP, now seems unlikely to be the causative agent. Not only is the disease (including biopsy results) not clinically consistent with classic HP, but inspection of the plant and air sampling for viable microorganisms did not suggest a likely problem along these lines. Circumstantial evidence that had supported a published speculation that exposure to fungal toxin may have caused the ILD outbreak among workers at the Microfibres plant in Ontario [Lougheed et al. 1995] seems much less compelling now that additional cases have occurred among workers at that plant, even after remediation of the fungal contamination. (This information was shared with NIOSH investigators at a meeting on October 21, 1997.)

Though microbes may not be the primary respiratory hazard in this plant, airborne concentrations of bacterial endotoxin can reach levels that do present potential respiratory hazard (see "Exposure Evaluation," above). Endotoxin is a component of gram–negative bacteria, which may be a primary contaminant in the wet–processes involved in flock production or could be brought into the plant on raw materials (e.g., potato starch [Hollander et al. 1994]). Exposure to airborne endotoxin, at levels substantially higher than those measured in the Microfibres plant, has been implicated as playing a role in the etiology of so–called organic dust toxic syndrome [Rylander 1997], but levels of that magnitude have probably not occurred at the Microfibres plant .

Common to the Ranges and RPCs is the flock itself, comprised of the nylon fiber coated with various components including a proprietary finish containing tannic acid, ammonium ether of potato starch, and a fatty alcohol derivative. (The identity of the fatty alcohol derivative is proprietary information.) As mentioned above, the cut nylon flock fibers–typically 2000 microns in length and 15–20 microns in diameter–are too large to be considered respirable. However, samples of airborne particulate collected during the NIOSH investigation, documented the presence of small, irregularly–shaped, nylon fibers shown to be both morphologically and aerodynamically respirable in size.

Because nylon, in the diameters in which it is manufactured, is not respirable, no relevant research has been done to assess nylon respiratory

toxicity. As a result, nylon has been thought by many to be rather inert. Some evidence, however, raises some concern about the respiratory toxicity of nylon. As reported in a paper by Portuguese investigators [Pimentel et al. 1975], two workers who had been occupationally exposed for 10 or more years were found to have nylon fiber inclusions within lesions seen in lung biopsies. Although both had interstitial disease (fibrosis in one and cellular infiltrate in the other), suggesting a possible similarity with the ILD diagnosed among Microfibres workers, these two cases were apparently substantially different. One of these patients had bronchiectasis (severe inflammatory destruction of the airways) that was granulomatous, and the other patient presented with enlarged non–malignant hilar lymph nodes. No detailed information on work process or particulate exposures was provided by the authors.

Pimentel et al. [1975] followed up their clinical observations with experiments in which 28 guinea pigs were inhalationally exposed several times a day to pulverized nylon for 325 days. Granulomatous interstitial lung disease resulted in half the exposed animals. In all cases, nylon particles were found in these lesions.

The granulomatous nature of the nylon–associated lung pathology described by Pimentel et al. [1975] in patients occupationally exposed to nylon, as well as in experimentally exposed animals, would seem to distinguish it histopathologically from the ILD occurring among Microfibres workers. It may be that this difference relates to difference in exposure intensity. Alternatively, disease manifestations may differ as a result of differences in nylon finishes or other modifying effects in the respective workplace environments. The flock finish used at the Microfibres plant in Pawtucket contains several components.

Though tannic acid used in flock finish may have Food and Drug Administration (FDA) approval as a food additive, it is imprudent to consider it safe when inhaled. Published studies have shown that tannin influences alveolar macrophage function [Rohrbach 1994], induces epithelial cell changes [Cloutier and Rohrbach 1986], and causes an inflammatory response when inhaled [Kilburn et al. 1973]. Inhaled tannin–containing dusts may play a role in human disease [Lauque et al. 1988], but studies adequate to clearly document this have not been done [Lacey et al. 1994].

Potato starch, another component of flock finish, is not free of potential respiratory hazard. Occupational exposure to dust in the potato starch industry has been associated with asthmatic effects in workers; microbial contaminants and/or potato proteins are suspected specific agents [Hollander et al. 1994] in this dust.

With regard to the proprietary fatty alcohol derivative component of the finish, no information in the published literature was available on this chemical. The supplier (a supplier of dyes and chemicals for the textile industry) is unaware of any relevant toxicology studies. The Material Safety Data Sheets (MSDS) for this component of the finish indicates that no respiratory protection is required under normal use of the product.

Preliminary results of NIOSH toxicology studies indicate that respirable fragments of nylon, even without flock finish, causes a very intense inflammatory response when instilled in animal lungs. An equivalent mass of respirable dust collected from the air in the flocking and flock screening rooms at the plant was even more potent in the animal exposures. Moreover, when this dust collected from the plant was washed in water, the water extract also induced substantial inflammatory response when instilled in the animals in amounts that provided a dose of endotoxin equivalent to that delivered by the unwashed dust exposure. While much more data from these animal studies need to be considered before final conclusions can be developed from the toxicological studies, they do provide reason to be concerned about potential respiratory toxicity of both the nylon fragments themselves and the flock finish they carry, not to mention the endotoxin contamination that may also be present.

Summary Conclusions

Evidence communicated in this report leads to the following major conclusions:

! An excess of ILD has occurred among workers employed at the Microfibres plant in Pawtucket.

! Even among those workers not diagnosed with ILD, substantial excesses in various respiratory, systemic, and other symptoms have occurred.

! These excesses of ILD and respiratory symptoms are attributable to occupational exposure at the plant.

! Respirable dust exposures were found to be clearly in the hazardous range in some areas of the plant.

! Respirable dust generated during flock production, including respirable nylon fragments, causes very intense inflammation in animal lungs and represents the most likely cause of the excess ILD and symptoms observed among workers at this plant.

RECOMMENDATIONS

NIOSH provides the following recommendations to protect the health of workers employed at the Microfibres plant in Pawtucket. These recommendations cover primary prevention of illness through reduction of exposure to dust at the plant, and secondary prevention of illness through early detection of disease in individual workers to permit appropriate action to reduce the risk of clinically significant occupational disease in the affected individuals.

Microfibres management needs to take decisive, proactive action to install effective engineering controls, to enforce good work practices, to assure appropriate use of proper respiratory protection, to establish a medical screening/surveillance program, and to implement effective administrative controls. (The company has already made substantial progress in complying with these recommendations.)

1. Dust exposures should be reduced by means of engineering controls.

! A central vacuum system should be installed to permit removal of most settled loose flock and associated dust, thereby eliminating the use of compressed air for blow–downs. (Priority should be given to the flocking rooms, but all areas where compressed air is used to remove settled flock from equipment or other surfaces should eventually have vacuum systems.)

! Local exhaust ventilation should be installed around the flocking modules.

! The existing ventilation system should be inspected for leaks and broken seals and repaired as necessary.

! The existing ventilation system should be balanced and otherwise modified to minimize both reentry of plant exhaust and transfer of air contaminants between work areas.

! Open–topped process cyclones should be exhausted through particulate filters or discharged outside the plant

2. Dust exposures should be reduced by means of personal respiratory protection.

! A formal respiratory protection program should be instituted in accordance with OSHA regulations (CFR 1910.134).

! The flocking rooms and screening rooms should be designated as areas in which the use of personal respiratory protection is required.

! When in the respirator–designated areas, workers should be required to wear, at a minimum, NIOSH–certified approval class N95 dust respirator.

- Workers performing blow–downs, (whether it be in the flock module rooms, or in any of the screening/milling rooms), or otherwise using compressed air to move flock, should wear a full–facepiece, Powered Air–Purifying Respirator (PAPR) equipped with a high efficiency particulate air (HEPA) filter.

3. **Medical monitoring and health surveillance should be undertaken to help guide prevention efforts.** *(Information that may be helpful in these activities has previously been provided to Microfibres and union representatives).*

4. **Workers should be periodically informed about work–related disease observed among flock workers, as well as about how their risk of disease can be reduced or controlled.**

5. **Biocide should not be added to the adhesive used in the production process.** *(Microbial monitoring has not shown levels high enough to warrant such action, and exposure to biocides themselves carries health risks.)*

6. **A no–smoking policy should be implemented at the plant [NIOSH 1991]. If allowed at all, smoking at the plant should be restricted to designated, separately ventilated, smoking rooms, and workers should be encouraged to stop smoking altogether.**

ADDENDUM

To foster a better understanding of this disease, NIOSH convened a workshop that included a panel of experts in the pathology of interstitial lung disease, along with pathologists and other clinicians who diagnosed ILD cases in the two Microfibres plant outbreaks and in two additional cases of apparent occupational ILD in workers from other flock operations. A report from this workshop is in preparation. NIOSH also plans to continue animal toxicology studies to better define potential toxicities of respirable nylon fiber with and without flock finish components, and is continuing to work with Microfibres to determine the point of generation of respirable nylon fragments. Finally, NIOSH has initiated communications with representatives from other companies producing flock or flocked fabric, as well as with representatives of the flock industry, to explore further studies in the flocking industry.

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List of Abbreviations

ACGIH	American Conference of Industrial Hygienist
ATS	American Thoracic Society
BAL	bronchoalveolar lavage
BOOP	bronchiolitis obliterans with organizing pneumonia
CFR	Code of Federal Regulations
CFU	Colony forming units
CXR	chest x–ray, chest radiograph
DL_{CO}	diffusing capacity for carbon monoxide
EU/m^3	Endotoxin units per cubic meter
FEV_1	forced expiratory volume in one second
FVC	forced vital capacity
HETA	Hazard Evaluation and Technical Assistance
HP	hypersensitivity pneumonitis
HRCT	high resolution computed tomography (CT) scan
HVAC	Heating , ventilating and air conditioning
ILD	interstitial lung disease
ILO	International Labour Organization
LLN	Lower limit of normal
MDC	Minimun detectable concentration
mg/m^3	milligrams per cubic meter
MQC	Minimun quantifiable concentration
MSDS	Material Safety Data Sheet
NIOSH	National Institute of Occupational Safety and Health
ODTS	organic dust toxic syndrome
OLB	open lung biopsy
OSHA	Occupational Safety and Health Administration
PA	Postero–anterior
PBZ	Personal breathing zone
PCL	Phase–contrast light
PEL	Personal exposure limit
PNOC	Particulate not otherwise classified
PNOR	Particulate not otherwise regulated
ppm	parts per million
REL	Recommend Exposure Limit
RPC	Rotary precision cutter
SEM	Scanning electron microsocpy
STEL	Short–term exposure limit
TBB	transbronchial biopsy
TLV	Threshold Limit Value
TWA	Time–weighted average
$\mu g/m^3$	Micrograms per cubic meter
VE	Vertical elutriator

Table 1
Air Sampling and Analytical Methods
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Substance	Sample Type	Sampling Media	Flow Rate	Analytical Method[1]
PARTICULATES				
Particulates, Not Otherwise Classified (PNOC) Respirable Dust	Area Personal	1– Pre–weighed 37–mm diameter, 5–µm pore size PVC membrane filters housed in polystyrene cassettes in series with 10–mm Dorr–Oliver nylon cyclones	1.7 lpm	Gravimetric analysis according to NIOSH Method 0600 (with minor modifications). Microscopic analysis with phase contrast light (PCL) microscopy and scanning electron microscopy (SEM).
	Area	2– Similar filter with BGI stainless steel cyclone	2.2 lpm	Same
	Area	3– Similar filter mounted on vertical elutriators	7.4 lpm	Same Also collected for animal toxicity studies (probably approximates thoracic dust)
	Area	4– Active direct–reading dust monitors with respirable dust cyclone inlet with 1–minute averaging of 1–second samples	1.7 lpm	Direct measurement
	Area	5– Personal inhalable dust sampler (PIDS) impactor	2.0 lpm	Microscopic analysis
	Area	6– High volume 1" cyclone	90 lpm	Collected for animal toxicity studies
Particulates, Not Otherwise Classified (PNOC) Total Dust	Area Personal	1– Pre–weighed 37–mm diameter, 5–µm pore size PVC membrane filters housed in closed–face cassettes	2.0 lpm	Gravimetric analysis according to NIOSH Method 0500 (with minor modifications). Microscopic analysis with phase contrast light (PCL) microscopy and scanning electron microscopy (SEM).
	Area	2– Active direct–reading dust monitors with 1–minute averaging of 1–second samples	2.0 lpm	Direct measurement
Elemental Metals	Area	37–mm diameter, 0.8–µm pore size, cellulose ester membrane filters in open–face polystyrene cassettes.	2.0 lpm	Scanning inductively coupled plasma emission spectrometry in accordance with NIOSH Method 7300.
GASES AND VAPORS				
Volatile Organic Compounds	Area	Stainless steel thermal desorption tubes with 3 beds of sorbent material (90 mg Carbotrap Y, 115 mg Carbotrap B, 150 mg Carboxem 1003)	50 cc/min	Qualitative (screening) analysis using gas chromatography / mass spectrometry.
Formaldehyde	Area	2 impingers in series containing 1% sodium bisulfite ($NaHSO_4$) collection media for 2 consecutive 4–hour periods	400 cc/min	Visible absorption spectrometry NIOSH Method 3500.

Table 1 (continued)
Air Sampling and Analytical Methods
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Substance	Sample Type	Sampling Media	Flow Rate	Analytical Method[1]
Ethyl Acrylate Ethyl Propanoate Ethyl Acetate 1,1,1–Trichloroethane Toluene	Area	Coconut shell charcoal tubes (100mg/50mg)	25 cc/min	Gas chromatography with flame ionization detector (GC/FID) in accordance with NIOSH Methods 1450, 1457, 1003, and 1501
Butyl Cellosolve	Area	Coconut shell charcoal tubes (100mg/50mg)	25 cc/min	GC/FID with NIOSH Method 1403
Total Hydrocarbons	Area	Coconut shell charcoal tubes (100mg/50mg)	20 cc/min	GC/FID with NIOSH Method 1550
Nitrogen Oxides	Area	Palmes passive dosimeters	NA–diffusion	Visible absorption spectrometry via NIOSH Method 6700[2]
Propylene Glycol	Area	XAD–7 OVS™ sorbent tubes	1.0 lpm	GC/FID with NIOSH Method 5523
Nitrosamines	Area	Thermosorb–N™ solid sorbent tubes	1.0 lpm	TEA analyzer for nitro– compounds in accordance with OSHA Method 27
BIOAEROSOLS				
Bacteria and Fungi	Area	30–, 60–, and 180–second samples collected at each location directly onto tryptic soy agar (TSA) plates for bacterial counts and malt extract agar (MEA) plates for fungal counts in Anderson N–6 viable impactors	28.3 lpm	Mesophilic incubation: 23 ± 2ºC, 10 days Thermophilic and thermo–tolerant incubation: 52 ± 2ºC, 10 days
Endotoxins	Area Personal	Selected filters from respirable and total dust sampling submitted after gravimetric analysis	1.7 or 2.0 lpm	Chromogenic modification of Limulus amebocyte lysate gel test[3]

cc/min = cubic centimeters of air per minute
NA = not applicable
µm = micrometer
mm = millimeter
lpm = liters of air per minute
mg = milligram

Table 1 References

1 unless noted, all analytical methods were obtained from:
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Table 2
Summary of Selected Occupational Exposure Limits and Health Effects
Microfibres, Inc., Pawtucket, RI
HETA 96–0093

Substance	NIOSH [1] REL – TWA	OSHA [2] PEL – TWA	ACGIH [3] TLV – TWA	Primary Health Effects [1,4,5]
PARTICULATES				
Particulates, Not Otherwise Classified (PNOC)				Alveolar proteinosis, respiratory clearance inhibition[6]
Respirable Fraction	——	5 mg/m^3	3 mg/m^3	
Total Dust	——	15 mg/m^3	10 mg/m^3 §	
Elemental Metals	Metal–specific limits			Metal–specific
GASES AND VAPORS				
Volatile Organic Compounds	Compound–specific limits			Compound–specific
Formaldehyde	Ca 0.016 ppm 0.1 ppm 15–min C	Ca 0.75 ppm 2 ppm STEL	Ca 0.3 ppm C	Nasal cancer [7]
Ethyl Acrylate	Ca LOQ	25 ppm	Ca 5 ppm 15 ppm STEL	Potential for cancer; tumors of the forestomach in animals
Ethyl Propanoate	——	——	——	——
Ethyl Acetate	400 ppm	400 ppm	400 ppm	Eye and respiratory irritation
1,1,1–Trichloroethane (Methyl Chloroform)	350 ppm 15–min C	350 ppm	350 ppm 450 ppm STEL	Central nervous system, liver, and cardiovascular effects
Toluene	100 ppm 150 ppm STEL	200 ppm 300 ppm C	50 ppm skin	Central nervous system depression [8]
Butyl Cellosolve	5 ppm skin	50 ppm skin	25 ppm skin	Adverse effects on blood and hematopoietic system, tissue irritation, central nervous system depression [9]
Total Hydrocarbons	——	——	——	Compound–specific
Nitrogen Oxides: NO	25 ppm	25 ppm	25 ppm	Effects on blood and respiratory systems
NO_2	1 ppm STEL	5 ppm C	3 ppm 5 ppm STEL	
Propylene Glycol	——	——	——	——
Nitrosamines	——	——	——	For the nitrosamine compound N–Nitrosodimethylamine: Potential for cancer; tumors of the liver, kidney, lung, and nasal cavity in animals – NIOSH and OSHA recommend/require that OSHA handling regulations be followed [10]

Table 2 (continued)
Summary of Selected Occupational Exposure Limits and Health Effects
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Substance	NIOSH [1] REL – TWA	OSHA [2] PEL – TWA	ACGIH [3] TLV – TWA	Primary Health Effects [1,4,5]
BIOAEROSOLS				
Bacteria and Fungi	——	——	——	Allergic asthma, allergic rhinitis, hypersensitivity pneumonitis [11–21]
Endotoxins	——	——	——	Fever, airway constriction, acute respiratory symptoms (flu–like illness) [22–29]

§	=	Inhalable fraction	mg/m^3	=	milligrams per cubic meter of air
REL	=	recommended exposure limit	C	=	Ceiling limit.
PEL	=	permissible exposure limit	STEL	=	Short–term exposure limit.
TLV	=	threshold limit value	Ca	=	Carcinogen
TWA	=	time–weighted average	LOQ	=	Limit of quantitation
ppm	=	parts per million parts air	skin	=	Potential exposure through skin

Table 2 References

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Table 3
Work Area Airborne Respirable Dust and Endotoxin
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Date	*Sample Number*	*Time On*	*Time Off*	*Volume (liters)*	*Dust (mg)*	*Dust (mg/m^3)*	*Endotoxin (EU/mg)*	*Endotoxin (EU/m^3)*
RPC 1–2 Area								
5/7/96	24813	851	1507	639.2	0.12	**0.19**	22.5	**4.28**
5/8/96	24824	817	1509	700.4	0.08	**0.11**	—	**—**
5/9/96	24838	801	1506	722.5	0.11	**0.15**	—	**—**
RPC 3–7 Area								
5/7/96	24814	814	1459	688.5	0.20	**0.29**	14.5	**4.21**
5/8/96	24818	811	1525	737.8	0.13	**0.18**	—	**—**
5/9/96	24792	809	1511	717.4	0.19	**0.27**	—	**—**
Basement Screening Room								
5/7/96	24816	845	1502	640.9	0.05	**0.08***	—	**—**
5/8/96	24804	834	1504	663.0	0.79	**1.19***	29.5	**35.09***
5/9/96	24819	755	1506	732.7	3.68	**5.02***	8.1	**40.66***
8/21/96	25306	816	1543	980.0	0.88	**0.90***	—	**—**
Compounding Area								
5/7/96	24803	838	1511	668.1	0.29	**0.43**	—	**—**
5/8/96	24836	824	1514	697.0	0.28	**0.40**	20.7	**8.28**
5/9/96	24789	803	1509	724.2	0.57	**0.79**	8.6	**6.79**
Range 1 Pre–Coat Area								
5/7/96	24801	748	1515	759.9	0.13	**0.17**	20.0	**3.40**
5/8/96	24829	813	1520	725.9	0.10	**0.14**	—	**—**
5/9/96	24832	755	1508	736.1	0.04	**0.05**	—	**—**
Range 2 Pre–Coat Area								
5/7/96	24794	746	1504	744.6	0.09	**0.12**	—	**—**
5/8/96	24811	820	1531	732.7	0.34	**0.46**	2.1	**0.95**
5/9/96	24797	803	1525	751.4	0.04	**0.05**	—	**—**

*clogging of sampler inlets by loose flock occurred in flocking room and basement screening room samples
— endotoxin assay not done

Table 3 (continued)
Work Area Airborne Respirable Dust and Endotoxin
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Date	*Sample Number*	*Time On*	*Time Off*	*Volume (liters)*	*Dust (mg)*	*Dust (mg/m^3)*	*Endotoxin (EU/mg)*	*Endotoxin (EU/m^3)*
Range 1 Flocking Room								
5/7/96	24826	755	1515	748.0	2.63	**3.51***	30.4	**106.55***
5/8/96	24828	1105	1511	418.2	0.22	**0.53***	—	**—**
5/8/96	24798	826	1104	268.6	10.72	**39.91***	3.5	**138.09***
5/9//96	24806	800	1504	720.8	0.96	**1.33***	9.4	**12.48***
8/21/96	25298	805	1442	870.0	0.46	**0.53****	—	**—**
Range 2 Flocking Room								
5/7/96	24799	750	1459	729.3	0.54	**0.74***	—	**—**
5/8/96	24793	806	1520	737.8	0.80	**1.08***	18.0	**19.44***
5/8/96	24833	834	1525	698.7	0.43	**0.62***	—	**—**
8/21/96	25273	811	1500	1050.0	0.76	**0.72***	—	**—**
Range 1 ScotchGard® Area								
5/7/96	24796	803	1509	724.2	0.13	**0.18**	—	**—**
5/8/96	24830	836	1509	668.1	0.09	**0.13**	—	**—**
5/9/96	24822	755	1505	731.0	0.15	**0.21**	3.3	**0.70**
Range 2 ScotchGard® Area								
5/7/96	24800	755	1518	753.1	0.16	**0.21**	—	**—**
5/8/96	24809	833	1514	681.7	0.21	**0.31**	2.6	**1.03**
5/9/96	24807	757	1505	727.6	0.16	**0.22**	—	**—**
Embossing Area								
5/7/96	24808	822	1503	681.7	0.72	**1.06**	2.6	**2.80**
5/8/96	24795	829	1517	693.6	0.08	**0.12**	—	**—**
5/9/96	24827	803	1509	724.2	0.26	**0.36**	—	**—**
Printing Range Area								
5/7/96	24834	831	1504	668.1	0.20	**0.30**	—	**—**
5/8/96	24837	811	1520	729.3	0.08	**0.11**	—	**—**
5/9/96	24791	809	1504	705.5	0.27	**0.38**	8.9	**3.38**

* clogging of sampler inlets by loose flock occurred in flocking room and basement screening room samples
**Range 1 flocking room idle most of this sampling period
— endotoxin assay not done

Table 3a
Personal Breathing Zone Respirable Dust and Endotoxin
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Date	*Sample Number*	*Time On*	*Time Off*	*Volume (liters)*	*Dust (mg)*	*Dust (mg/m^3)*	*Endotoxin (EU/mg)*	*Endotoxin (EU/m^3)*
Blow–down in Range 2 Flocking Room								
5/8/96	24817	1205	1330	144.5	2.22	**15.36***	—	**—**
5/8/96	24821	1205	1330	144.5	1.42	**9.82***	—	**—**
5/9/96	24802	907	940	56.1	0.11	**1.96***	16.4	**32.07***
5/9/96	24805	908	940	54.4	0.01	**0.18***	—	**—**

*clogging of sampler inlets by loose flock occurred in flocking room samples
— endotoxin assay not done

Table 3b
Personal Breathing Zone Total Dust and Endotoxin
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Date	*Sample Number*	*Time On*	*Time Off*	*Volume (liters)*	*Dust (mg)*	*Dust (mg/m^3)*	*Endotoxin (EU/mg)*	*Endotoxin (EU/m^3)*
Personal Breathing Zone – Blow–down in Range 2 Flocking Room								
5/9/96	24840	901	1037	192	14.62	**76.15***	0.6	**48.74***

* clogging of sampler inlets by loose flock occurred in flocking room samples

Table 4
Work Area Airborne Total Dust and Endotoxin
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Date	*Sample Number*	*Time On*	*Time Off*	*Volume (liters)*	*Dust (mg)*	*Dust (mg/m^3)*	*Endotoxin (EU/mg)*	*Endotoxin (EU/m^3)*
RPC 1–2 Area								
5/7/96	24881	850	1507	754	0.24	**0.32**	—	**—**
5/8/96	24863	817	1509	824	0.18	**0.22**	—	**—**
5/9/96	24861	800	1506	852	0.14	**0.16**	—	**—**
RPC 3–7 Area								
5/7/96	24873	816	1459	806	0.32	**0.40**	—	**—**
5/8/96	24885	811	1525	868	0.32	**0.39**	13.3	**5.19**
5/9/96	24879	809	1511	844	0.44	**0.52**	14.8	**7.68**
Basement Screening Room								
5/7/96	24846	845	1502	754	0.12	**0.16***	—	**—**
5/8/96	24869	834	1504	780	1.34	**1.72***	21.4	**36.84***
5/9/96	24853	755	1506	862	6.58	**7.63***	19.4	**147.87***
8/21/96	25280	816	1543	760	1.34	**1.76***	—	**—**
Compounding Area								
5/7/96	24844	837	1511	788	0.65	**0.82**	21.8	**17.84**
5/8/96	24849	824	1514	820	0.56	**0.68**	—	**—**
5/9/96	24858	803	1509	852	0.90	**1.06**	23.6	**24.97**
Range 1 Pre–Coat Area								
5/7/96	24875	748	1515	894	0.29	**0.32**	—	**—**
5/8/96	24857	813	1520	854	0.14	**0.16**	—	**—**
5/9/96	24856	755	1508	866	0.36	**0.41**	10.8	**4.44**
Range 2 Pre–Coat Area								
5/7/96	24845	746	1504	876	0.26	**0.30**	20.0	**5.99**
5/8/96	24876	820	1531	862	0.09	**0.10**	—	**—**
5/9/96	24870	803	1525	884	0.14	**0.16**	—	**—**

* clogging of sampler inlets by loose flock occurred in flocking room and basement screening room samples
— endotoxin assay not done

Table 4 (continued)
Work Area Airborne Total Dust and Endotoxin
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Date	*Sample Number*	*Time On*	*Time Off*	*Volume (liters)*	*Dust (mg)*	*Dust (mg/m^3)*	*Endotoxin (EU/mg)*	*Endotoxin (EU/m^3)*
Range 1 Flocking Room								
5/7/96	24847	755	1515	880	22.57	**25.65***	2.0	**51.81***
5/8/96	24851	1104	1511	494	38.43	**77.79***	2.1	**163.35***
5/8/96	24866	826	1104	316	76.12	**240.89***	0.9	**216.80***
5/9/96	24884	800	1504	848	4.19	**4.91***	16.3	**79.93***
8/21/96	25291	805	1442	680	0.68	**1.34***	—	**—**
Range 2 Flocking Room								
5/7/96	24841	750	1459	429	1.69	**3.93***	4.5	**17.83***
5/8/96	24874	834	1525	822	6.29	**7.65***	2.3	**17.90***
5/8/96	24880	806	1520	868	2.37	**2.73***	17.0	**46.30***
8/21/96	25284	811	1500	820	3.57	**4.35***	—	**—**
Range 1 ScotchGard® Area								
5/7/96	24843	802	1509	854	0.35	**0.41**	—	**—**
5/8/96	24860	836	1509	786	0.16	**0.20**	—	**—**
5/9/96	24850	755	1505	860	0.26	**0.33**	10.4	**3.42**
Range 2 Scotchgard® Area								
5/7/96	24864	755	1518	886	0.12	**0.13**	—	**—**
5/8/96	24848	833	1514	802	0.26	**0.32**	—	**—**
5/9/96	24888	757	1505	856	0.28	**0.32**	3.3	**1.06**
Embossing Area								
5/7/96	24859	823	1503	800	0.31	**0.39**	—	**—**
5/8/96	24887	829	1517	816	0.24	**0.29**	—	**—**
5/9/96	24855	803	1509	852	0.41	**0.48**	6.6	**3.16**
Printing Range Area								
5/7/96	24842	831	1504	786	0.46	**0.59**	9.3	**5.49**
5/8/96	24886	811	1520	858	0.15	**0.17**	—	**—**
5/9/96	24883	809	1504	830	0.20	**0.24**	—	**—**

* clogging of sampler inlets by loose flock occurred in flocking room and basement screening room samples
** Range 1 flocking room idle most of this sampling period
— endotoxin assay not done

Table 5
Vertical Elutriator Airborne Dust
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Date	*Sample Number*	*Time On*	*Time Off*	*Volume (m^3)*	*Dust (mg)*	*Dust (mg/m^3)*
Range 1 – Flocking Room						
8/21/96	25272	805	1115	1.41	1.43	**1.01***
8/21/96	25305	1115	1325	1.04	1.08	**1.03***
Range 2 – Flocking Room						
8/21/96	25271	811	1120	1.41	11.86	**8.41****
8/21/96	25277	1120	1343	1.06	0.86	**0.81**
Basement Screening Room						
8/21/96	25309	816	1543	void	void	**void**

* Range 1 flocking room was idle most of this sampling period
** Blow–down occurred during this sampling period

Table 6
Work Area Airborne Elemental Metals
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Sampling Site	Sample Number	Volume (m^3)	Aluminum (ug/m^3)	Antimony (ug/m^3)	Arsenic (ug/m^3)	Barium (ug/m^3)	Beryllium (ug/m^3)	Calcium (ug/m^3)	Cadmium (ug/m^3)	Cobalt (ug/m^3)	Chromium (ug/m^3)	Copper (ug/m^3)
Compounding Area	24894	0.79	ND	{1.77}	ND	ND	ND	ND	ND	ND	ND	ND
RPC 1–2 Area	24906	0.75	ND	{1.32}	ND	ND	ND	ND	ND	ND	ND	ND
RPC 3–7 Area	24921	0.81	ND	{2.35}	ND	{0.07}	ND	ND	ND	ND	ND	ND
Basement Screening Room	24893	0.75	ND	{2.67}	ND	ND	ND	ND	ND	ND	ND	{0.12}
Range 1 Pre–Coat Area	24908	0.89	{2.36}	{2.81}	ND	ND	ND	ND	ND	ND	ND	ND
Range 2 Pre–Coat Area	24902	0.88	ND	{2.05}	ND	{0.06}	ND	ND	ND	ND	ND	ND
Range 1 Flocking Room	24918	0.88	204.55	ND	ND	0.94	ND	14.77	ND	1.59	{2.16}	{0.31}
Range 2 Flocking Room	24917	0.7	6.43	{1.36}	ND	{0.16}	ND	{9.29}	ND	ND	3.00	{0.17}
Range 1 ScotchGard® Area	24920	0.85	{3.41}	ND	ND	ND	ND	ND	ND	ND	ND	ND
Range 2 ScotchGard®Area	24925	0.88	{1.82}	{1.25}	ND	ND	ND	ND	ND	ND	ND	ND
Embossing Area	24922	0.81	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Printing Range Area	24891	0.79	ND	ND	ND	{0.11}	ND	ND	ND	ND	ND	ND
Minimum Detectable Concentration (MDC)			1.43	1.14	4.29	0.07	0.01	4.29	0.11	0.29	0.71	0.11
Minimum Quantifiable Concentration (MQC)			5.00	3.57	10.71	0.24	0.05	10.71	0.36	0.61	2.43	0.36

ND = not detected
All were full–shift samples
{ } = Trace

Table 6 (continued)
Work Area Airborne Elemental Metals
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Sampling Site	Sample Number	Volume (m^3)	Iron (ug/m^3)	Lithium (ug/m^3)	Magnesium (ug/m^3)	Manganese (ug/m^3)	Molybdenum (ug/m^3)	Nickel (ug/m^3)	Lead (ug/m^3)	Phosphorus (ug/m^3)
Compounding Area	24894	0.79	{3.42}	ND	ND	{0.03}	ND	ND	ND	{3.04}
RPC 1–2 Area	24906	0.75	4.40	ND	{0.72}	0.17	ND	ND	ND	ND
RPC 3–7 Area	24921	0.81	{1.36}	ND	ND	ND	ND	ND	{1.10}	ND
Basement Screening Room	24893	0.75	{3.47}	ND	ND	{0.01}	ND	ND	{0.71}	{3.20}
Range 1 Pre–Coat Area	24908	0.89	{1.24}	ND	ND	{0.01}	ND	ND	ND	ND
Range 2 Pre–Coat Area	24902	0.88	{2.95}	ND	ND	{0.01}	ND	ND	ND	ND
Range 1 Flocking Room	24918	0.88	{3.18}	ND	{2.09}	0.85	ND	ND	{1.02}	{5.00}
Range 2 Flocking Room	24917	0.7	11.00	ND	{1.63}	0.08	ND	ND	{1.06}	{2.86}
Range 1 ScotchGard® Area	24920	0.85	{3.41}	ND	ND	{0.01}	ND	ND	{1.17}	{3.06}
Range 2 ScotchGard®Area	24925	0.88	{1.59}	ND	ND	{0.01}	ND	ND	ND	{4.77}
Embossing Area	24922	0.81	{1.60}	ND	ND	{0.02}	ND	ND	{0.98}	{3.95}
Printing Range Area	24891	0.79	{1.52}	ND	ND	{0.01}	ND	ND	{1.00}	ND
Minimum Detectable Concentration (MDC)			1.14	0.04	0.71	0.01	0.43	0.71	0.71	2.86
Minimum Quantifiable Concentration (MQC)			3.57	0.11	2.43	0.05	1.21	1.43	2.43	6.14

ND = not detected
All were full–shift samples
{ } = Trace

Table 6 (continued)
Work Area Airborne Elemental Metals
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Sampling Site	Sample Number	Volume (m^3)	Platinum (ug/m^3)	Selenium (ug/m^3)	Silver (ug/m^3)	Sodium (ug/m^3)	Tellurium (ug/m^3)	Thallium (ug/m^3)	Titanium (ug/m^3)	Vanadium (ug/m^3)	Yttrium (ug/m^3)
Compounding Area	24894	0.79	ND	ND	ND	{5.91}	ND	ND	ND	ND	ND
RPC 1–2 Area	24906	0.75	ND	ND	ND	{4.89}	ND	ND	ND	ND	ND
RPC 3–7 Area	24921	0.81	ND	ND	ND	ND	ND	ND	ND	ND	ND
Basement Screening Room	24893	0.75	ND	ND	ND	ND	ND	ND	ND	ND	ND
Range 1 Pre–Coat Area	24908	0.89	ND	ND	ND	{3.00}	ND	ND	ND	ND	ND
Range 2 Pre–Coat Area	24902	0.88	ND	ND	ND	ND	ND	ND	ND	ND	ND
Range 1 Flocking Room	24918	0.88	ND	ND	ND	215.53	ND	ND	4.43	{0.14}	ND
Range 2 Flocking Room	24917	0.7	ND	ND	ND	22.38	ND	ND	ND	ND	ND
Range 1 ScotchGard® Area	24920	0.85	ND	ND	ND	{4.31}	ND	ND	ND	ND	ND
Range 2 ScotchGard®Area	24925	0.88	ND	ND	ND	ND	ND	ND	ND	ND	ND
Embossing Area	24922	0.81	ND	ND	ND	ND	ND	ND	ND	ND	ND
Printing Range Area	24891	0.79	ND	ND	ND	ND	{1.90}	ND	ND	ND	ND
Minimum Detectable Concentration (MDC)			4.29	2.86	0.11	2.86	1.14	4.29	0.29	0.11	0.03
Minimum Quantifiable Concentration (MQC)			10.71	6.14	0.36	9.71	3.57	10.71	0.61	0.36	0.06

ND = not detected
All were full–shift samples
{ } Trace

Table 7
Work Area Gases/Vapors
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Sampling Site	Formaldehyde (ppm)	Ethyl Acrylate (ppm)	Ethyl Propanoate (ppm)	Ethyl Acetate (ppm)	Propylene Glycol (ppm)	1–1–1 Trichoroe thane (ppm)	Toluene (ppm)	Butyl Cellosolve (ppm)	Total Nitrosamines (ug/m^3)	Oxides of Nitrogen (ppm)	Total Hydrocarbons (ppm)
Compounding Area	{0.07}	ND	0.31	{0.11}	{0.04}	ND	ND	ND	ND	ND	ND
RPC 1–2 Area	0.25	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
RPC 3–7 Area	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Basement Screening	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Range 1 Pre–Coat	{0.05}	ND	0.65	0.25	ND	ND	ND	ND	ND	ND	ND
Range 2 Pre–Coat	{0.04}	ND	0.46	{0.15}	0.09	ND	ND	ND	ND	ND	0.34
Range 1 Flocking	{0.08}	ND	ND	ND	{0.02}	ND	ND	ND	ND	ND	0.61
Range 2 Flocking	{0.06}	0.16	3.7	1.28	ND	ND	ND	ND	ND	ND	2.6
Range 1 ScotchGard®	0.44	ND	{0.04}	ND	{0.06}	ND	ND	ND	ND	ND	ND
Range 2 ScotchGard	0.21	ND	{0.04}	ND	0.07	ND	ND	ND	ND	ND	ND
Embossing Area	{0.07}	ND	{0.05}	ND	ND	ND	ND	ND	ND	ND	0.17
Printing Range Area	{0.05}	ND	{0.05}	ND	ND	ND	ND	ND	ND	ND	ND
Minimum Detectable Concentration (MDC)	0.04	0.02	0.02	0.05	0.01	0.03	0.03	0.08	0.01	0.03	0.001
Minimum Quantifiable Concentration (MQC)	0.13	0.10	0.08	0.17	0.07	0.11	0.08	0.24	0.08	0.05	0.004

ND = not detected , { } = Trace

Table 8
Summary of Bulk Microbial Results

HETA 96-0093 - Microfibres, Pawtucket, RI

Sample Number	Sample Location	Fungal Content	Bacteria Content
8442-822B	Dying - Waste H_2O under dryer	7.0×10^5 *Exophiala* 4.0×10^5 pink yeast 3.0×10^5 brownish green yeast like mold 1.0×10^5 white, round yeast 1.0×10^4 *Geotrichum* 3.0×10^3 *Trichoderma* 1.0×10^2 *Mucor*	2.2×10^7 *Acinetobacter* genospecies 15 1.8×10^7 Unidentified Gm-rod[2] 7.0×10^6 CDC Group IVC-2 7.0×10^6 *Pseudomonas nitroreducens* 6.0×10^6 *Pseudomonas fragi* 1.0×10^6 *Pseudomonas stutzeri*
8442-823B	Dying - Sample of green dye being applied	None detected	5.0×10^3 *Bacillus thuringiensis/cereus* 5.0×10^3 *Staphylococcus capitas* ss capitis
8442-828B	Dying - Final rinse solution	None detected	6.7×10^3 Unidentified Gm-rod 6.0×10^2 *Flavobacterium indologenes* 2.0×10^2 *Curtobacterium luteum* 2.0×10^2 *Pseudomonas vesicularis* 1.0×10^2 *Acinetobacter calcoaceti* 1.0×10^2 *Staphylococcus capitas ss capiti*
8442-824B	Dying - Floor H_2O sample of final rinse runoff	1.1×10^3 *Trichophyton* 2.0×10^3 *Aureobasidium* 1.0×10^3 *Fusarium* 1.0×10^3 *Scedosporium*	2.5×10^7 *Staphylococcus capitas ss capitis* 1.3×10^7 Unidentified Gm-rod 7.0×10^6 *Pseudomonas fragi* 3.0×10^6 *Aeromonas caviae* DNA group 4 2.0×10^6 *Pseudomonas vesicularis*
8442-825B	Compounding - Dumping H_2O waste from Ranges	2.5×10^5 white, rhizoid yeast 5.0×10^2 *Acremonium*	1.2×10^7 *Klebsiella pneumoniae ss rhinoscleroma* 6.0×10^6 *Enterobacter agglomerans Biogroup 7* 5.0×10^6 *Shewanella putrefaciens B* 1.0×10^6 *Enterobacter agglomerans Biogroup 2B*
8442-829B	Compounding - Adhesive (waste)	None detected	4.0×10^7 *Pseudomonas mucalicola*
8442-848B	Compounding - Adhesive (waste)	None detected	

Table 8 (continued)
Summary of Bulk Microbial Results

IIETA 96-0093 - Microfibres, Pawtucket, RI

Sample Number	Sample Location	Fungal Content	Bacteria Content
8442-831B	Cutting - Scour 1	1.3×10^4 white, round yeast 3.4×10^2 orange yeast 2.0×10^2 white, spreading yeast 2.0×10^2 white, rhizoid yeast 4.0×10^2 Aureobasidium	2.2×10^7 *Acinetobacter genospecies 15*
8442-854B	Cutting - Run off from floor	1.6×10^6 white, round yeast 3.2×10^5 cream yeast 3.2×10^5 *Fusarium*	4.8×10^6 *Sphingobacterium thalophilum*
8442-840B	Cutting - Scour from tank, last	None detected	3.4×10^7 *Pseudomonas pseudoalcaligenes*
8442-833B	Coating - waste material under adhesive app.	1.5×10^4 *Aspergillus versicolor* 1.5×10^4 *Aspergillus flavus* 7.4×10^3 *Geotrichum* 7.4×10^3 *Penicillium*	2.2×10^7 *Chryseomonas luteola* 1.1×10^7 *Pseudomonas maculicola*
8442-839B	Coating - Range 1 wall scraping	5.9×10^3 *Penicillium* 1.2×10^3 *Fusarium* 1.2×10^3 *Scedosporium* 1.2×10^3 pink yeast	1.6×10^9 *Curtobacterium flaccumfaciens PV poinA* 4.7×10^8 *Pseudomonas vesicularis* 2.4×10^8 *Psedomonas corrugata*
8442-836B	Coating - Range 1 wall scraping by H_2O spray	8.2×10^3 *Penicillium* 5.9×10^4 *Scedosporium* 5.9×10^4 *Fusarium*	1.8×10^8 *Curtobacterium flaccumfaciens PV poinA* 1.2×10^8 *Psedomonas corrugata* 1.2×10^7 *Leuconostoc oenos*
8442-857B	Coating - Range 1 scraping from A/C grill	1.0×10^6 pink yeast 5.0×10^4 *Scedosporium* 1.3×10^4 *Penicillium* 1.1×10^4 *Cladosporium* 2.5×10^3 *Aureobasidium*	8.8×10^8 *Curtobacterium flaccumfaciens PV poinA* 3.3×10^8 *Pseudomonas syringae PV aptata*
8442-838B	Coating - Range 1 scraping from A/C grill	3.4×10^6 orange yeast 2.2×10^6 peach yeast 2.2×10^6 *Acremonium* 2.2×10^6 *Cladosporium* 2.2×10^6 *Penicillium*	8.7×10^{10} *Crynebacterium aquaticum A*

Table 8 (continued)
Summary of Bulk Microbial Results

BETA 96-0093 - Microfibres, Pawtucket, RI

Sample Number	Sample Location	Fungal Content	Bacteria Content
8442-827B	Dying - Floor runoff of final rinse.	1.8×10^5 cfu/cc of *Candida parapsilosis* 1.4×10^4 cfu/cc of *Blastoschizomyced capitus* 3.6×10^3 cfu/cc of *Aspergillus fumigatus* group	2.4×10^7 cfu/cc of *Klebsiella pneumoniae* 5.8×10^6 cfu/cc of *Aeromonas hydrophila* 3.6×10^5 cfu/cc of *Flavobacterium meningosepticum* 1.2×10^5 cfu/cc of most closely resembles *sphingobacterium spiritivorum*
8442-826B	Compounding - dumping waste H2O from Range	2.5×10^6 cfu/cc *Candida Lipolytica* 5.0×10^4 cfu/cc of *Aspergillus niger* group 1.5×10^4 cfu/cc of *Candida parapsilosis*	5.0×10^5 cfu/cc of *Bacillus* spp. 5.0×10^2 cfu/cc of *Bacillus* spp. (thermotoletant) (Note: No actinomycetes isolated.)
8442-830B	Compounding- Adhesive (waste)	1.5×10^4 cfu/cc of *Aspergillus fumigatus* group	4.7×10^5 cfu/cc of *Alcaligenes faecalis* (Note: No thermophilic bacteria isolated No actinomycetes isolated.)
8442-851B	Coating - Waste slime under Sctochgard application on Range 2.	6.9×10^5 cfu/g of *Candida lipolytica* 1.1×10^5 cfu/g of *Candida parapsilosis* 1.5×10^5 cfu/g of *Rhodotorula rubra*	No mesophilic, thermophilic or actinomycetes isolated.
8442-835B	Coating - Waste slime under Sctochgard application on Range 1.	5.1×10^5 cfu/g of *Candida parapsilosis* 1.7×10^5 cfu/g of *Candida lipolytica*	1.7×10^5 cfu/g of *Ochrebactrum anthropi* (Note: No thermophilic bacteria isolated No actinomycetes isolated.)
8442-855B	Drip pan (H_2O) sample from Range 1 HVAC unit.	1.0×10^5 cfu/cc of *Penicillium* spp. (morphotype 1) 1.0×10^5 cfu/cc of *Crytococcus* spp. (Resembles albidus) 2.0×10^2 cfu/cc of *Rhodotorula* spp. 1.0×10^2 cfu/cc of *Penicillium* spp. (morphotype 2) 5.0×10^1 cfu/cc of *Penicillium* spp. (morphotype 3) 5.0×10^1 cfu/cc of *Fusarium* spp. 5.0×10^1 cfu/cc of a sterile hyaline mould 5.0×10^1 cfu/cc of *Aspergillus fumigatus* group (thermotolerant) 52°C	1.6×10^6 cfu/cc *Corynebacterium* spp. 2.5×10^3 cfu/cc of a gram-negative bacilli; non-viable after subculture. 5.0×10^2 cfu/cc of most closely resembles *Corynebacterium* spp.

Table 8 (continued)
Summary of Bulk Microbial Results

BETA 96-0093 - Microfibres, Pawtucket, RI

Sample Number	Sample Location	Fungal Content	Bacteria Content
8442-827B	Dying - Floor runoff of final rinse.	1.8×10^5 cfu/cc of *Candida parapsilosis* 1.4×10^4 cfu/cc of *Blastoschizomyced capitus* 3.6×10^3 cfu/cc of *Aspergillus fumigatus* group	2.4×10^7 cfu/cc of *Klebsiella pneumoniae* 5.8×10^6 cfu/cc of *Aeromonas hydrophila* 3.6×10^5 cfu/cc of *Flavobacterium meningosepticum* 1.2×10^5 cfu/cc of most closely resembles *sphingobacterium spiritivorum*
8442-826B	Compounding - dumping waste H2O from Range	2.5×10^6 cfu/cc *Candida Lipolytica* 5.0×10^4 cfu/cc of *Aspergillus niger* group 1.5×10^4 cfu/cc of *Candida parapsilosis*	5.0×10^5 cfu/cc of *Bacillus* spp. 5.0×10^2 cfu/cc of *Bacillus* spp. (thermotoletant) (Note: No actinomycetes isolated.)
8442-830B	Compounding- Adhesive (waste)	1.5×10^4 cfu/cc of *Aspergillus fumigatus* group	4.7×10^5 cfu/cc of *Alcaligenes faecalis* (Note: No thermophilic bacteria isolated No actinomycetes isolated.)
8442-851B	Coating - Waste slime under Sctochgard application on Range 2.	6.9×10^5 cfu/g of *Candida lipolytica* 1.1×10^5 cfu/g of *Candida parapsilosis* 1.5×10^5 cfu/g of *Rhodotorula rubra*	No mesophilic, thermophilic or actinomycetes isolated.
8442-835B	Coating - Waste slime under Sctochgard application on Range 1.	5.1×10^5 cfu/g of *Candida parapsilosis* 1.7×10^5 cfu/g of *Candida lipolytica*	1.7×10^5 cfu/g of *Ochrebactrum anthropi* (Note: No thermophilic bacteria isolated No actinomycetes isolated.)
8442-855B	Drip pan (H_2O) sample from Range 1 HVAC unit.	1.0×10^5 cfu/cc of *Penicillium* spp. (morphotype 1) 1.0×10^5 cfu/cc of *Crytococcus* spp. (Resembles albidus) 2.0×10^2 cfu/cc of *Rhodotorula* spp. 1.0×10^2 cfu/cc of *Penicillium* spp. (morphotype 2) 5.0×10^1 cfu/cc of *Penicillium* spp. (morphotype 3) 5.0×10^1 cfu/cc of *Fusarium* spp. 5.0×10^1 cfu/cc of a sterile hyaline mould 5.0×10^1 cfu/cc of *Aspergillus fumigatus* group (thermotolerant) 52°C	1.6×10^6 cfu/cc *Corynebacterium* spp. 2.5×10^3 cfu/cc of a gram-negative bacilli; non-viable after subculture. 5.0×10^2 cfu/cc of most closely resembles *Corynebacterium* spp.

Table 9
Airborne Viable Microbes
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Sampling Site	Bacteria (CFU/m^3)				Fungi (CFU/m^3)			
	Morning	Mid–Day	Afternoon	Avg.	Morning	Mid–Day	Afternoon	Avg.
Compounding Area	704	282	318	435	2403	353	848	1201
RPC 1–2 Area	1408	985	1408	1267	735	777	989	834
RPC 3–7 Area	1971	636	4014	2207	989	438	346	591
Basement Screening	2253	1901	1126	1760	1208	742	261	737
Range 1 Pre–Coat Area	210	704	563	492	219	191	226	212
Range 2 Pre–Coat Area	352	281	352	328	495	219	318	344
Range 1 Flocking Room	7324	7183	3732	6080	353	120	141	205
Range 2 Flocking Room	4	986	141	377	106	141	141	129
Range 1 ScotchGard®	211	70	140	140	3039	184	177	1133
Range 2	210	281	422	304	2085	226	205	839
Embossing Area	140	352	282	258	219	290	155	221
Printing Range Area	6337	704	777	2606	813	219	304	445
Blow–down		4295				191		
Outside			1901				813	

Table 10
Characteristics of Eight Workers Diagnosed with Interstitial Lung Disease from 1992 to 1996
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

	Case #1	Case #2	Case #3	Case #4	Case #5	Case #6	Case #7	Case #8
Major Symptoms	dry cough, SOB	productive cough, SOB	dry cough, SOB, chest pain	productive cough, SOB	flu–like, dry cough	dry cough, SOB, wheezing	productive cough, SOB	SOB, fatigue, polyarthralgias dry cough
Spirometry (% predicted or ratio)	Restriction (FVC =72%)	Restriction (FVC = 59%)	Restriction (FVC = 50%)	Restriction (FVC = 74%)	Obstruction (FEV_1/FVC = .67)	Normal	Obstruction (FEV_1/FVC = .64)	Restriction (FVC = 64%)
DL_{CO} (% predicted)	Reduced (46%)	Reduced (29%)	Reduced (50%)	Normal (91%)	Normal (99%)	Normal (98%)	Normal (94%)	Reduced (40%)
Chest x–ray	Honeycombing	Diffuse reticulonodular infiltrates	Diffuse reticulonodular infiltrates	Normal	Normal	Normal	Normal	Patchy consolidation
HRCT	Patchy ground glass; honeycombing	Patchy ground glass; consolidation	Patchy ground glass; micronodularity	Normal	Patchy ground glass	Possible patchy ground glass	Diffuse tiny nodules at bases	Patchy ground glass; consolidation
Broncho–alveolar lavage	Not done	35% neutrophils 28% eosinophils	58% lymphocytes 8% neutrophils	25% lymphocytes 15% eosinophils	35% lymphocytes	10% neutrophils 25% eosinophils	Normal	Not done
Biopsy findings (biopsy type)	NSIP (OLB)	Not done	NSIP with lymphoid hyperplasia (OLB)	Not done	NSIP (TBB)	NSIP with eosinophilia (TBB)	NSIP with respiratory bronchiolitis (OLB)	Lymphoid hyperplasia; BOOP (OLB)

SOB = shortness–of–breath; DL_{CO} = diffusing capacity; HRCT = high resolution computerized tomography; NSIP = nonspecific interstitial pneumonitis; OLB = open lung biopsy; TBB = transbronchial biopsy; BOOP = bronchiolitis obliterans with organizing pneumonia

Table 11
Smoking Status and Other Factors by Department
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Department (n)	Smoke (ever)	Tenure (years)	Days/wk	Hours/wk	Range (usually)	Range (ever)	Blow–down (ever)	Pit (ever)
	n %	mean SD	mean SD	mean SD	n %	n %	n %	n %
Dye House/ Compounding (n=25)	19 76.0	10.2 6.7	6.0 0.45	55.7 9.6	2 8.0	11 44.0	11 44.0	5 20.0
Raycote (n=24)	17 70.8	10.1 8.8	6.3 0.53	51.8 5.7	2 8.3	10 41.6	17 70.8	9 38.0
Coating (n=59)	38 64.4	5.8 5.6	6.0 0.87	54.9 13.8	41 69.5	51 86.4	57 96.6	12 20.3
Maintenance (n=11)	8 72.7	11.2 8.4	6.2 0.40	54.0 7.2	4 36.4	10 90.0	7 63.6	9 81.8
Shipping/ Warehouse	9 81.8	11.7 8.2	6.0 0.45	59.7 11.1	0 —	7 63.6	7 63.6	3 27.3
Non–Office Subtotal (n=130)	91 70.0	8.4 7.3	6.1 0.68	54.8 11.2	49 37.7	89 68.5	99 76.2	38 29.0
Office (n=21)	15 71.4	7.4 8.7	5.4 0.67	48.4 9.8	2 9.5	5 23.8	4 19.0	3 14.3
Total (n=151)	106 70.2	8.3 7.5	6.0 0.72	53.9 11.3	51 33.8	94 62.3		103 68.241

Table 12
Frequent Respiratory Symptoms (With Onset Since Working at Microfibres) by Department
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Department (n)	Shortness of Breath				Dry Cough				Chest Tightness			
	n %	PR	95% CI	improves* n %	n %	PR	95% CI	improves* n %	n %	PR	95% CI	improves* n %
Dye House/ Compounding (n=25)	12 48.0	2.5	0.95–6.66	4 33.0	6 24.0	2.5	0.57–11.2	1 17.0	8 32.0	2.2	0.68–7.392	25.0
Raycote (n=24)	8 33.3	1.8	0.61–4.99	6 75.0	4 17.0	1.8	0.36–8.61	3 75.0	7 29.2	2.0	0.60–6.91	4 57.1
Coating (n=59)	16 27.1	1.4	0.54–3.78	8 50.0	21 35.6	3.7	0.96–14.59	15 71.4	20 33.9	2.4	0.78–7.18	14 70.0
Maintenance (n=11)	6 54.5	2.9	1.02–8.05	2 33.3	7 63.6	6.7	1.66–26.88	4 57.1	6 54.5	3.8	1.18–12.41	4 66.7
Shipping/ Warehouse (n=11)	1 9.1	0.5	0.06–3.77	0 —	1 9.1	1.0	0.10–9.40	1 100.0	1 9.1	0.6	0.07–5.42	0 —
Non–Office Subtotal (n=130)	43 33.1	1.7	0.70–4.34	20 46.5	39 30.0	3.2	0.82–12.08	24 62.0	42 32.3	2.3	0.77–6.64	24 57.1
Office (n=21)	4 19.0	—	—	1** 25.0	2 9.5	—	—	1** 50.0	3 14.3	—	—	1** 33.3
Total (n=151)	47 30.5			21 44.7	41 27.2			25 61.0	45 29.8			25 55.6

PR=prevalence ratio using Office prevalence as reference
95% CI=upper and lower limits of 95% confidence interval for prevalence ratio
***symptom improves away from work**
****this worker reported Range and blow–down exposure**

Table 12 (continued)
Frequent Respiratory Symptoms (With Onset Since Working at Microfibres) by Department
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Department (n)	Wheeze				Cough with Phlegm			
	n %	PR	95% CI	improves* n %	n %	PR	95% CI	improves* n %
Dye House/ Compounding (n=25)	5 20.0	2.1	0.45–9.74	1 20.0	7 28.0	2.9	0.68–12.67	2 28.6
Raycote (n=24)	2 8.3	0.9	0.13–5.68	1 50.0	7 29.2	3.1	0.71–13.16	5 71.4
Coating (n=59)	16 27.1	2.9	0.71–11.35	12 75.0	19 32.2	3.4	0.86–13.30	12 63.2
Maintenance (n=11)	2 18.2	1.9	0.31–11.77	0 —	5 45.5	4.8	1.10–20.73	3 60.0
Shipping/ Warehouse (n=11)	2 18.2	1.9	0.31–11.77	1 50.0	3 27.3	2.9	0.56–14.67	1 33.3
Non–Office Subtotal (n=130)	27 20.1	2.2	0.56–8.50	15 55.6	41 31.5	3.3	0.87–12.68	23 56.1
Office (n=21)	2 9.5	—	—	0 —	2 9.5	—	—	0 —
Total (n=151)	29 19.2			15 51.7	43 28.5			23 53.5

PR=prevalence ratio using Office prevalence as reference
95% CI=upper and lower limits of 95% confidence interval for prevalence ratio
***symptom improves away from work**
****this worker reported Range and blow–down exposure**

Table 13
Frequent Systemic Symptoms (With Onset Since Working at Microfibres) by Department
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Department (n)	Fevers				Aches All Over			
	n %	PR	95% CI	improves* n %	n %	PR	95% CI	improves* n %
Dye House/ Compounding (n=25)	0 —	—	—	0 —	2 8.0	undef	—	0 —
Raycote (n=24)	1 4.2	0.9	0.06–13.79	0 —	6 25.0	undef	—	6 100.0
Coating (n=59)	5 8.5	1.8	0.22–14.37	5 100.0	19 32.2	undef	—	14 73.7
Maintenance (n=11)	0 —	—	—	0 —	3 27.3	undef	—	1 33.3
Shipping/ Warehouse (n=11)	0 —	—	—	0 —	1 9.1	undef	—	0 —
Non–Office Subtotal (n=130)	6 4.6	1.0	0.12–7.65	5 83.3	31 23.8	undef	—	21 67.7
Office (n=21)	1** 4.8	—	—	0 —	0 —	—	—	0 —
Total (n=151)	7 4.6			5 71.4	31 20.5			21 67.7

see Table 12 footnotes
undef = undefined

Table 14
Prevalence of Frequent Irritant Symptoms (With Onset Since Working at Microfibres) by Department
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Department (n)	Throat Irritation				Eye Irritation			
	n %	PR	95% CI	improves* n %	n %	PR	95% CI	improves* n %
Dye House/ Compounding (n=25)	5 20.0	4.2	0.53–33.19	3 60.0	8 32.0	undef	—	4 50.0
Raycote (n=24)	5 20.8	5.3	0.69–40.15	2 40.0	8 33.3	undef	—	7 87.5
Coating (n=59)	16 27.1	5.7	0.80–40.35	12 75.0	18 30.5	undef	—	17 94.4
Maintenance (n=11)	2 18.2	3.8	0.39–37.59	2 100.0	3 27.3	undef	—	3 100.0
Shipping/ Warehouse (n=11)	0 —	—	—	0 —	0 —	undef	—	0 —
Non–Office Subtotal (n=130)	28 21.5	4.5	0.65–31.50	19 67.9	37 28.5	undef	—	31 83.8
Office (n=21)	1 4.8	—	—	0 —	0 —	—	—	—
Total (n=151)	29 19.2			19 65.5	37 24.5			31 83.8

see Table 12 footnotes
undef = undefined

Table 15
Prevalence of At Least One Frequent Systemic or Respiratory Symptom
(With Onset Since Working at Microfibres) by Various Factors
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Factor		At Least One Frequent Systemic and/or Respiratory Symptom*			
		n	%	Prevalence Ratio	95% CI
Smoking Status	Ever Smoker (n=106)	62	58.5	1.1	0.80 – 1.51
	Never Smoker (n=45)	24	53.3	—	—
Tenure at plant	≤3 yrs (n=51)	25	49.0	—	—
	3–10 yrs (n=44)	28	63.6	1.3	0.91 – 1.86
	≥10 (n=56)	33	58.9	1.2	0.84 – 1.71
Days/wk	≤5 (n=22)	5	22.7	—	—
	6 (n=103)	61	59.2	2.6	1.19 – 5.72
	7 (n=26)	20	76.9	3.4	1.52 – 7.52
Hrs/wk	≤45 (n=21)	5	23.8	—	—
	45–65 (n=109)	64	58.7	2.5	1.13 – 5.39
	≥65 (n=21)	17	80.9	3.4	1.54 – 7.51
*Pit** (ever)*	Yes (n=41)	28	68.3	1.3	0.98 – 1.69
	No (n=109)	58	53.2	—	—
Blow–down (ever)	Yes (n=103)	67	65.0	1.6	1.13 – 2.40
	No (n=48)	19	39.6	—	—
Range (usually)	Yes (n=51)	35	68.6	1.4	1.03 – 1.76
	No (n=100)	51	51.0	—	—
Range (ever)	Yes (n=94)	62	66.0	1.6	1.12 – 2.19
	No (n=57)	24	42.1	—	—
Department	Non–Office (n=130)	80	61.5	2.2	1.08 – 4.29
	Office (n=21)	6***	28.6	—	—

PR=prevalence ratio using group with "—" as reference
95% CI=upper and lower limits of 95% confidence interval for prevalence ratio
**** information missing on one participant**
*****three of these six reported working on the Range**

Table 16
Prevalence of at Least One Frequent Systemic or Respiratory Symptom
(With Onset Since Working at Microfibres) by Department
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Department (n)	At Least One Frequent Systemic or Respiratory Symptom*		
	n %	Prevalence Ratio	95% CI
Dye House/ Compounding (n=25)	17 68.0	2.4	1.15 – 4.93
Raycote (n=24)	13 54.2	1.9	0.88 – 4.09
Coating (n=59)	38 64.4	2.3	1.12 – 4.55
Maintenance (n=11)	9 81.8	2.9	1.38 – 5.95
Shipping/ Warehouse (n=11)	3 27.3	1.0	0.29 – 3.10
Non–Office Subtotal (n=130)	80 61.5	2.2	1.08 – 4.29
Office (n=21)	6*** 28.6	—	—
Total (n=151)	86 60.0		

PR=prevalence ratio using Office prevalence as reference
95% CI=upper and lower limits of 95% confidence interval for prevalence ratio
*** see Tables 12 and 13 for symptoms**
***** three of these six Office workers reported working on the Range**

Table 17
Prevalence of Other Symptoms by Department
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Department (n)	Flu–Like Illness (more than one episode in past year)			Attacks of SOB with Wheeze (more than one episode in past two years)		
	n %	Prevalence Ratio	*95% CI*	n %	Prevalence Ratio	*95% CI*
Dye House/ Compounding (25)	8 32.0	2.2	0.68 – 7.39	6 24.0	2.5	0.57 – 11.20
Raycote (24)	4 16.7	1.2	0.29 – 4.63	8 33.3	3.5	0.83 – 14.69
Coating (59)	19 32.2	2.3	0.74 – 6.85	15 25.4	2.7	0.67 – 10.71
Maintenance (11)	4 36.4	2.6	0.69 – 9.41	4 36.4	3.8	0.82 – 17.68
Shipping/ Warehouse (11)	1 9.1	0.6	0.07 – 5.42	1 9.1	1.0	0.10 – 9.40
Non–Office (130)	36 27.7	1.9	0.70 – 6.05	34 26.2	2.8	0.71 – 10.59
Office (21)	3 14.3	—	—	2 9.5	—	—
Total (151)	39 25.8			36 23.8		

Prevalence ratios use Office prevalence as reference

Table 18
Forced Vital Capacity Results by Department
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Department (n)	FVC (% predicted)		Restriction		
	Mean SD	*p*	n %	Prevalence Ratio	95% CI
Compounding/ Dye House (n=25)	100.0 14.2	0.51	2 8.0	1.7	0.16 – 17.26
Raycote (n=24)	104.4 12.8	0.67	2 8.3	1.8	0.17 – 17.95
Coating (n=53)	98.5 13.0	0.24	4 7.4	1.6	0.19 – 13.37
Maintenance (n= 11)	98.0 13.2	0.36	0 —	—	—
Shipping/ Warehouse (n=11)	105.5 17.5	0.57	0 —	—	—
Non–Office Subtotal (n=124)	100.5 13.7	0.51	8 6.5	1.4	0.18 – 10.28
Office (n=21)	102.7 13.0	–	1 4.8	—	—

Note: Six of the 151 workers who participated in the questionnaire did not perform spirometry.
Prevalence ratios uses Office prevalence as reference

Table 19
Diffusion Capacity Results by Department
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Department	DL_{CO} (% predicted)		DL_{CO} <80% predicted		
	Mean SD	*p*	n %	Prevalence Ratio	95% CI
Compounding/ Dye House (n=19)	97.9 13.3	0.54	2 10.5	undef.	–
Raycote (n=16)	92.1 15.3	0.12	3 18.8	undef.	–
Coating (n=45)	96.7 16.0	0.39	6 13.3	undef.	–
Maintenance (n=11)	91.7 12.3	0.14	1 9.1	undef.	–
Shipping/ Warehousing (n=7)	103.2 15.3	0.74	1 14.3	undef.	–
Non–Office Subtotal (n=98)	96.1 15.0	0.29	13 13.3	undef.	–
Office (n=12)	100.9 13.0	—	0	—	—

Prevalence ratios use Office prevalence as reference

Table 20
Comparison of Mean Pulmonary Function Results by Presence of Symptoms and Smoking Status
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

		At Least One Frequent Systemic or Respiratory Symptom (with onset since working at Microfibres)		
		Yes	No	p
All				
DL_{CO} (% predicted)	mean SD n	94.0 15.1 68	100.9 13.4 42	0.02
FVC (% predicted)	mean SD n	99.1 13.3 84	103.2 13.6 61	0.07
FEV_1/FVC (X 100)	mean SD n	80.0 6.5 84	80.2 6.5 61	0.86
Never Smokers				
DL_{CO} (% predicted)	mean SD n	98.3 14.3 19	103.1 11.9 14	0.31
FVC (% predicted)	mean SD n	102.1 12.5 23	102.0 14.7 19	0.97
FEV_1/FVC (X 100)	mean SD n	79.8 8.4 23	80.8 7.5 19	0.69
Ever Smokers				
DL_{CO} (% predicted)	mean SD n	92.3 15.2 49	99.8 14.2 28	0.04
FVC (% predicted)	mean SD n	98.0 13.6 61	103.8 13.2 42	0.03
FEV_1/FVC (X 100)	mean SD n	80.1 5.6 61	79.9 6.1 42	0.89

Table 21
Prevalence of at Least One Frequent Systemic and/or Respiratory Symptom (With Onset Since Working at Microfibres) by Various Factors Among Ever Smokers
Microfibres, Inc., Pawtucket, RI
HETA 96–0093–2685

Factor		At Least One Frequent Systemic and/or Respiratory Symptom			
		n	%	Prevalence Ratio	95% CI
Tenure at plant	≤3 yrs (n=31)	18	58.1	—	—
	3–10 yrs (n=30)	19	63.3	1.1	0.73 – 1.63
	≥10 (n=45)	25	55.6	1.0	0.64 – 1.42
Days/wk	≤5 (n=12)	3	25.0	—	—
	6 (n=74)	43	58.1	2.3	0.86 – 6.31
	7 (n=20)	16	80.0	3.2	1.17 – 8.74
Hrs/wk	≤45 (n=11)	3	27.3	—	—
	45–65 (n=76)	44	57.9	2.1	0.79 – 5.68
	≥65 (n=19)	15	78.9	2.9	1.07 – 7.81
*Pit** (ever)*	Yes (n=29)	19	65.5	0.9	0.66 – 1.26
	No (n=72)	43	59.7	—	—
Blow–down (ever)	Yes (n=68)	45	66.2	1.5	1.00 – 2.19
	No (n=38)	17	44.7	—	—
Range (usually)	Yes (n=34)	23	67.6	1.3	0.91 – 1.71
	No (n=72)	39	54.2	—	—
Range (ever)	Yes (n=65)	45	69.2	1.7	1.12 – 2.49
	No (n=41)	17	41.5	—	
Department	Non–Office (n=91)	58	63.7	2.4	1.02 – 5.61
	Office (n=15)	4	26.7	—	—

PR=prevalence ratio using group with "—" as reference
95% CI=upper and lower limits of 95% confidence interval for prevalence ratio
**** information missing on one participant**

Figure 1.
Real-time Dust Concentrations
HETA 96-0093
Microfibres, Inc.

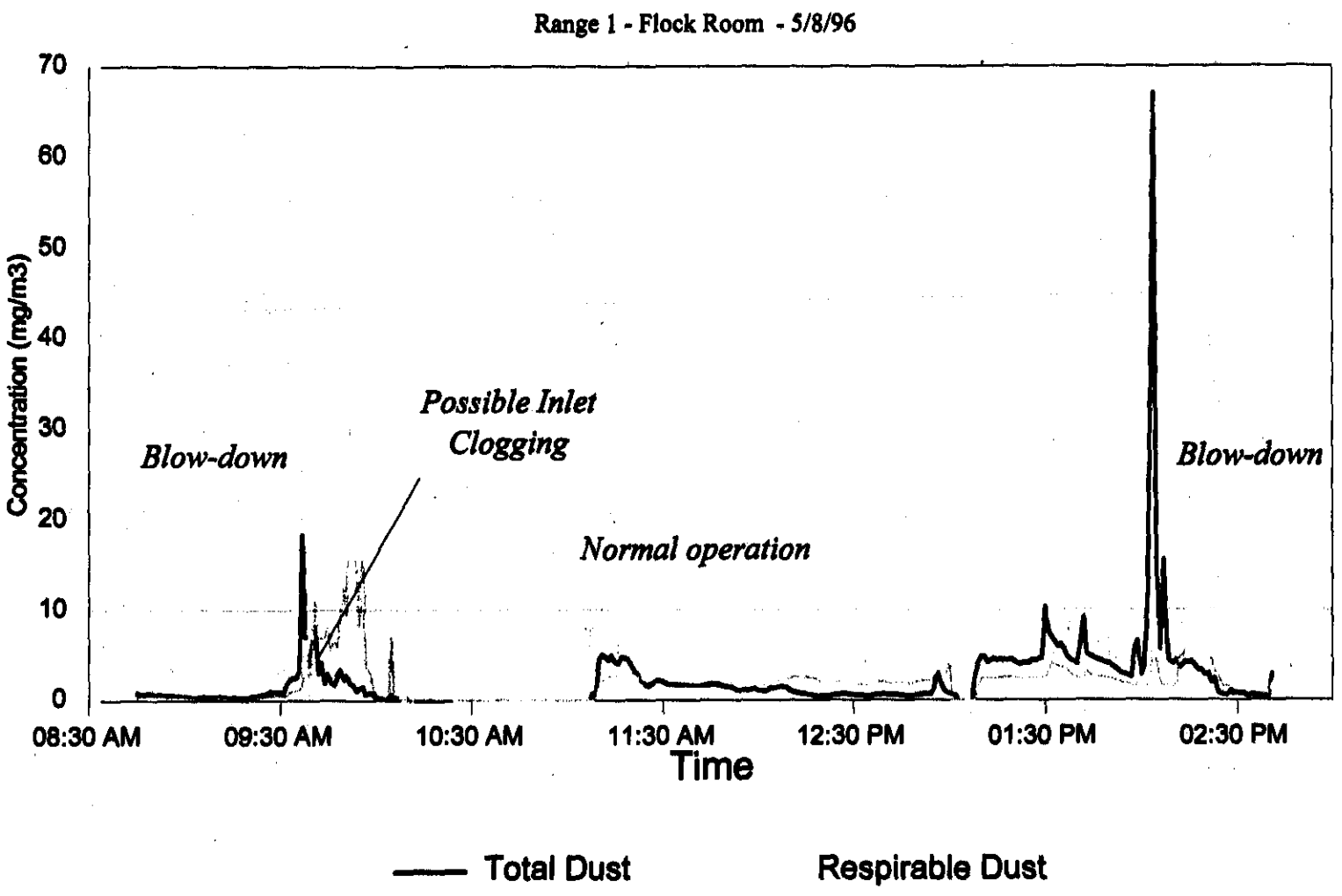
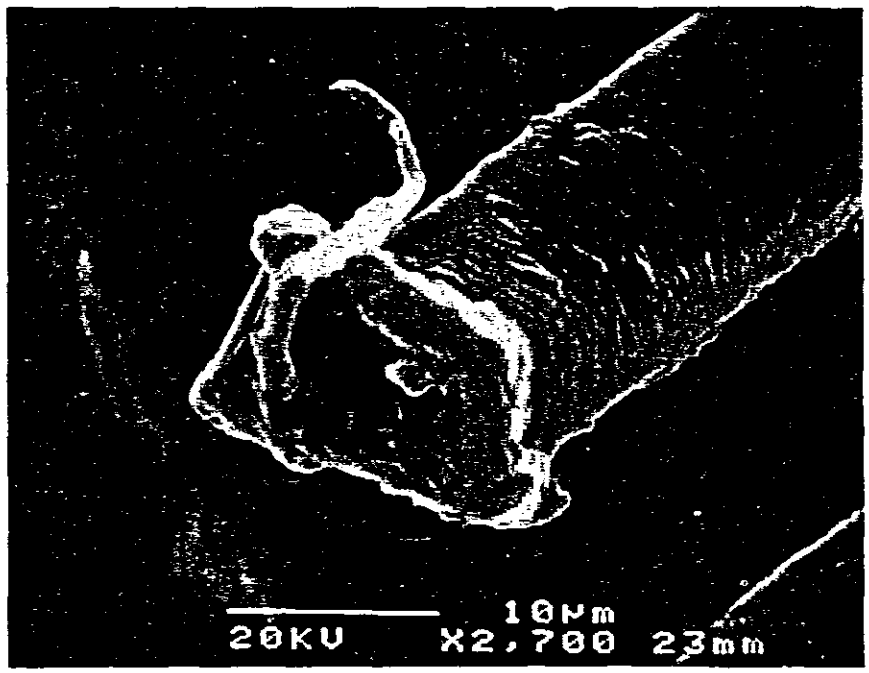
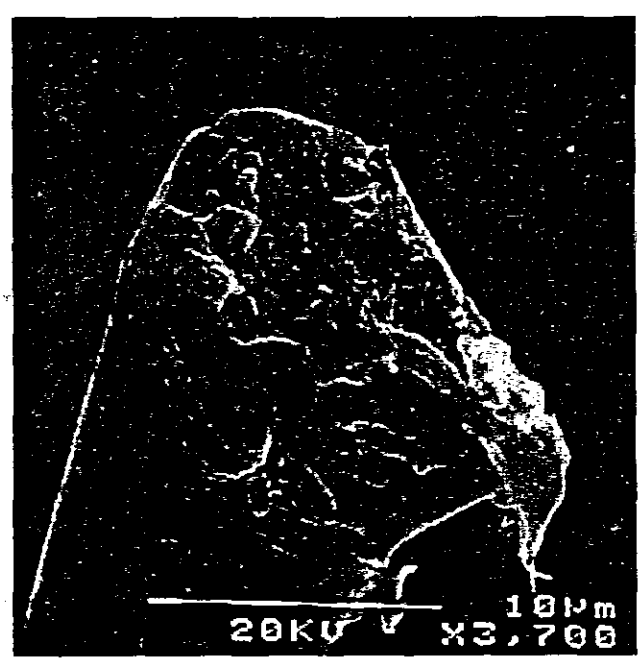
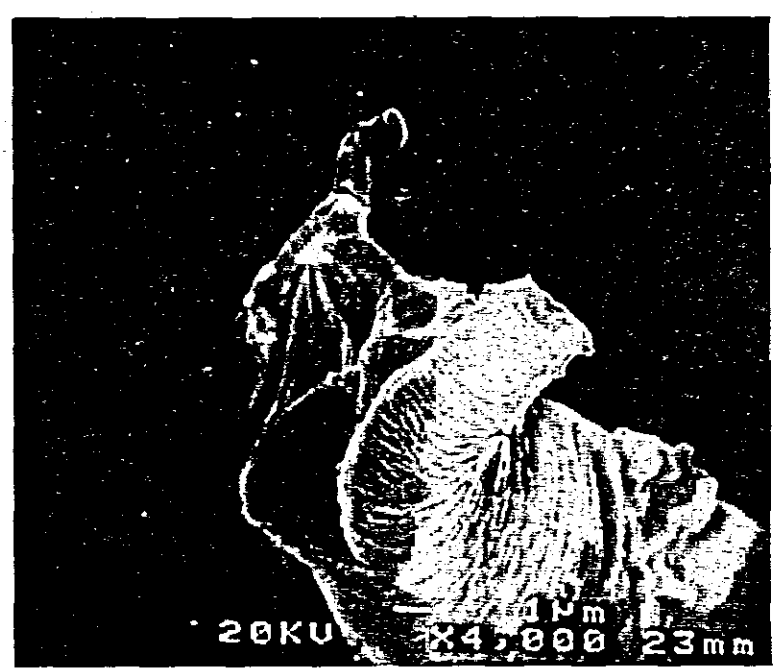


Figure 2.
Scanning Electron Microscope Images
of Bulk Nylon Fiber Ends

HETA 96-0093
Microfibres, Inc.
Pawtucket, RI







HETA 96-0093
Microfibres, Inc.
Pawtucket, RI

Appendix I

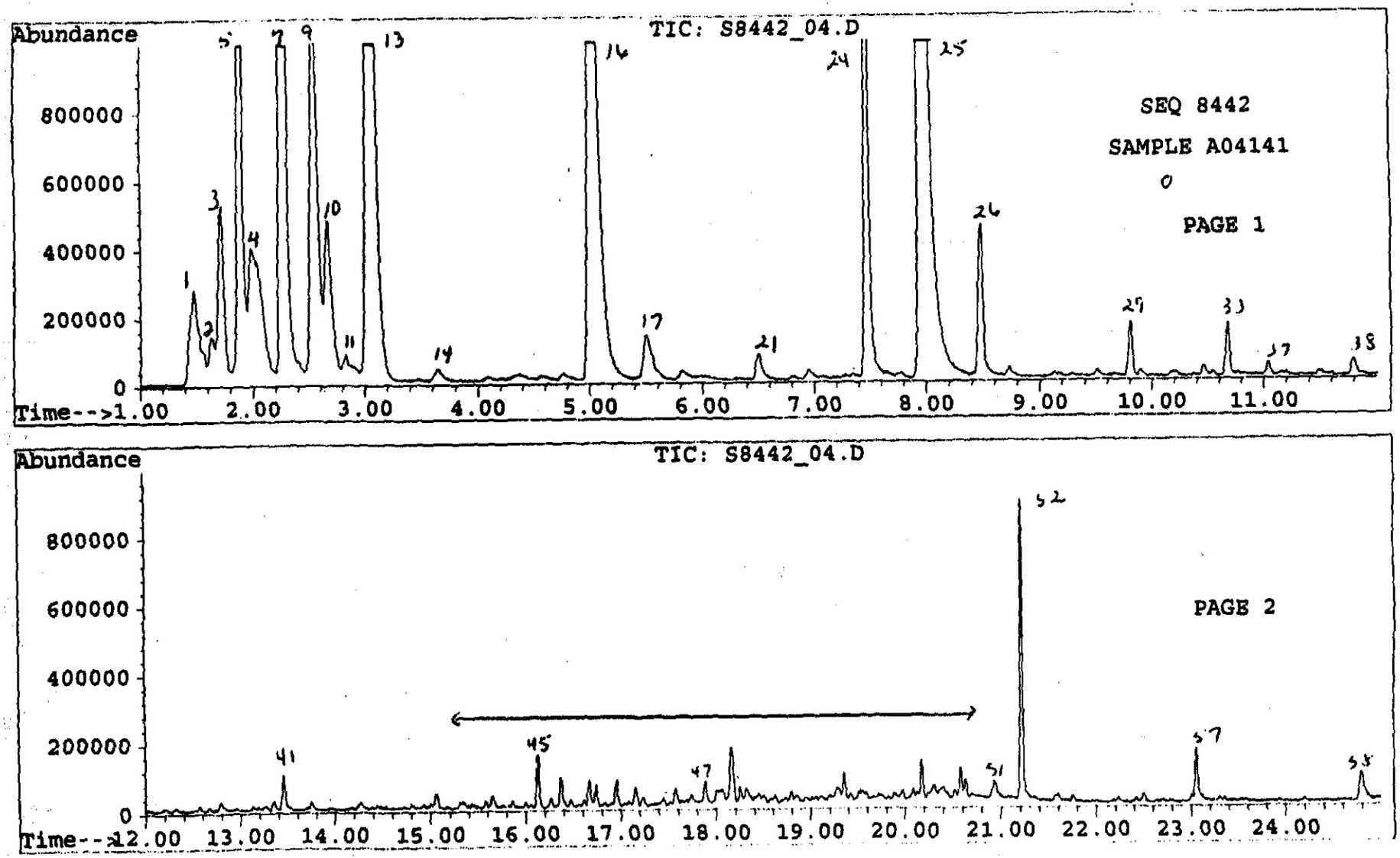
SEQ 8442
THERMAL DESORPTION TUBES
PEAK IDENTIFICATION

1) Air*
2) Formaldehyde**
3) Propane
4) Butene
5) Methanol/trace isobutane
6) Butane
7) Ethanol
8) Acetonitrile
9) Acetone
10) Isopropanol
11) Acrylonitrile
12) Unknown, amine?
13) 2-Methyl-2-propanol plus dimethoxy methane
14) 1-Propanol
15) Acetic acid
16) Ethyl acetate
17) Methyl propanoate
18) Methylcyclopentane
19) 1,1,1-Trichloroethane
20) Methyl dioxolane
21) Isopropyl acetate/butanol/ benzene
22) Trioxane (formaldehyde trimer)
23) Ethylene glycol
24) Ethyl acrylate
25) Ethyl propanoate (ethyl propionate)
26) Propylene glycol
27) Methyl isobutyl ketone (MIBK)
28) Fatty acid ester
29) Toluene
30) Cyclopentanone
31) Aliphatic acetate?
32) Hexanal*
33) Ethyl butanoate (ethyl butyrate)
34) Ethyl acrylate
35) Ethyl crotonate
36) Dimethylacetamide?
37) Ethyl methacrylate
38) Hexamethylcyclotrisiloxane*
39) Xylene
40) Nonane
41) Butyl cellosolve
42) Diethylene glycol
43) C_9-C_{16} aliphatics, mostly branched alkanes
44) Decane
45) Limonene plus branched alkane
46) Undecane
47) Ethyl benzoate
48) Dodecene
49) Tetradecene
50) Tetradecane
51) Fatty acid
52) Dodecanol
53) Pentadecane
54) Diphenyl ethane
55) Hexadecene
56) Diethyl biphenyls
57) Tetradecanol
58) Hexadecanol

*Also present on some media blanks; no field blanks were submitted with this set.
**Formaldehyde may be present as a decomposition product and/or impurity in methanol.

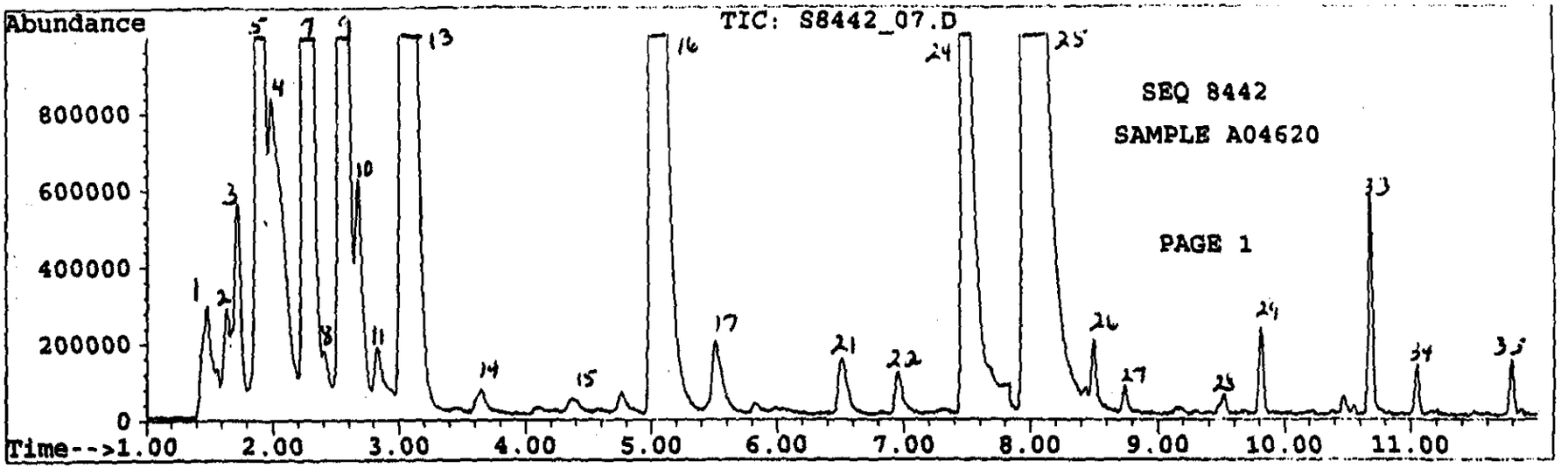
File : C:\HPCHEM\1\DATA\S8442\S8442_04.D
Opera. : AAG
Acquired : 18 Apr 96 10:12 am using AcqMethod ATL
Instrument : 5970 - In
Sample Name: SAMPLE A04141
Misc Info : 30 M DB-1 SC20-300 TP35-300
Vial Number: 4

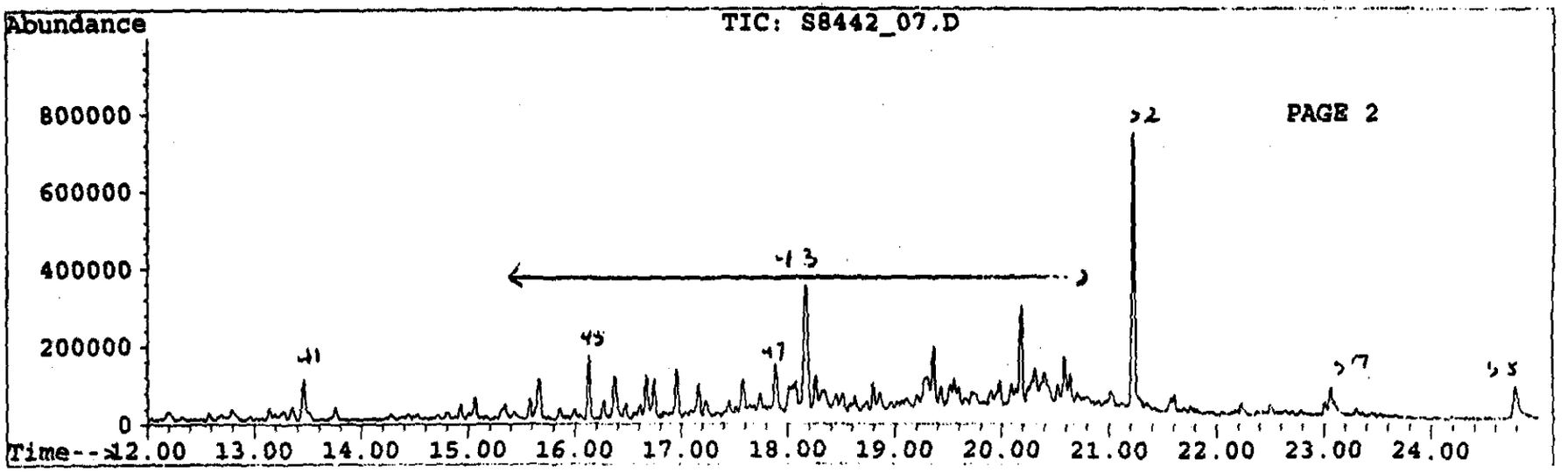
10 Apr 96
Range 1 - Precoat Oven
(4.2 liter sample)



File : C:\HPCHEM\1\DATA\S8442\S8442_07.D
Opera. : AAG
Acquired : 18 Apr 96 12:40 pm using AcqMethod ATD
Instrument : 5970 - In
Sample Name: SAMPLE A04620
Misc Info : 30 M DB-1 SC20-300 TP35-300
Vial Number: 7

10 Apr 96
Range 1 - Flock Room
(2.8 liter sample)





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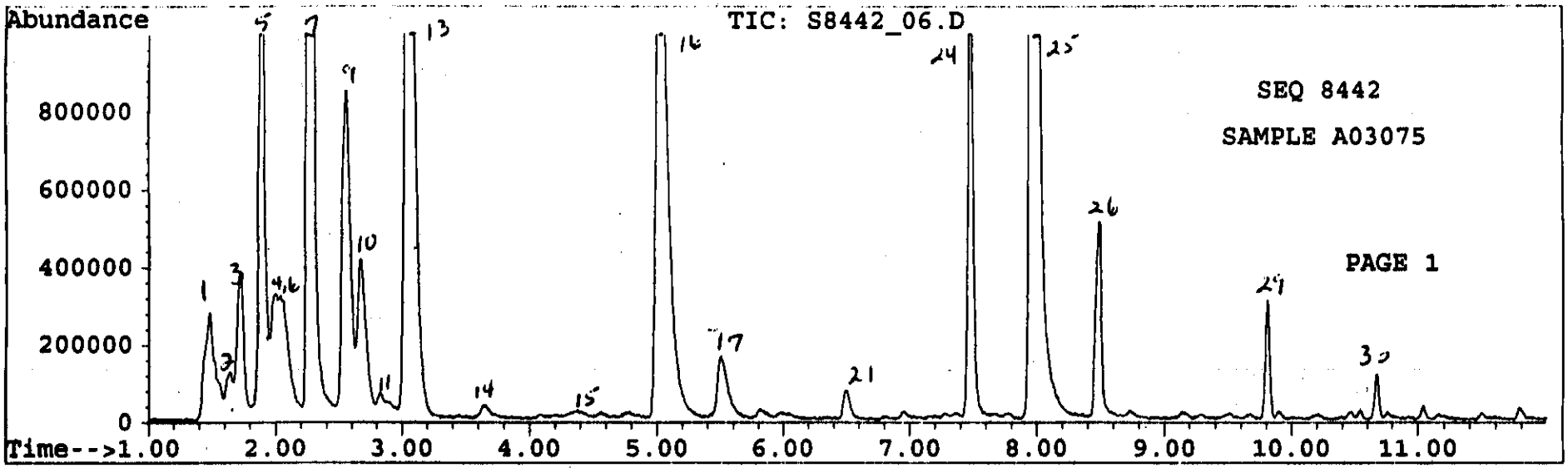
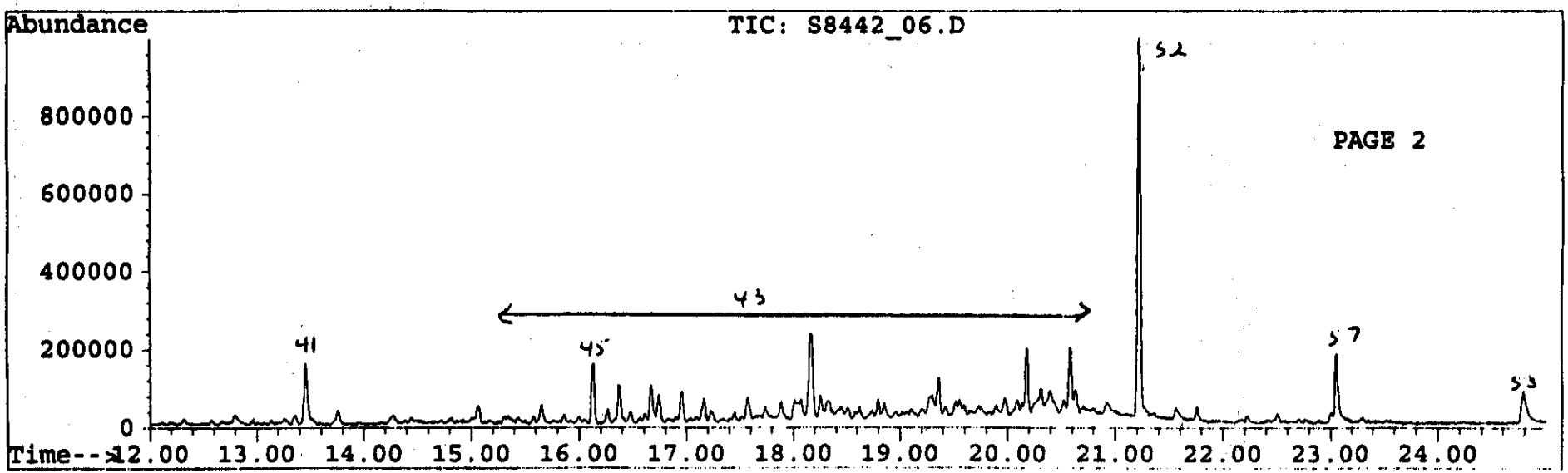
Opera. : AAG
Acquired : 18 Apr 96 11:51 am using AcqMethod ATD
Instrument : 5970 - In
Sample Name: SAMPLE A03075
Misc Info : 30 M DB-1 SC20-300 TP35-300
Vial Number: 6

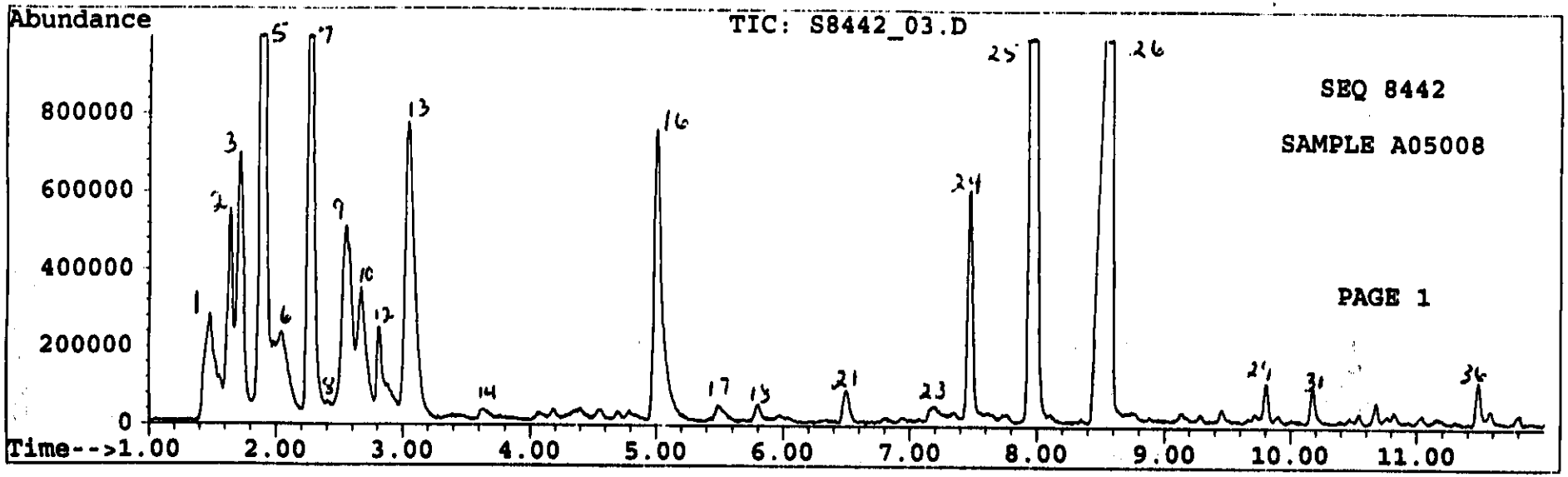
10 Apr 96
Range 2 - Precoat Oven
(3.7 liter sample)

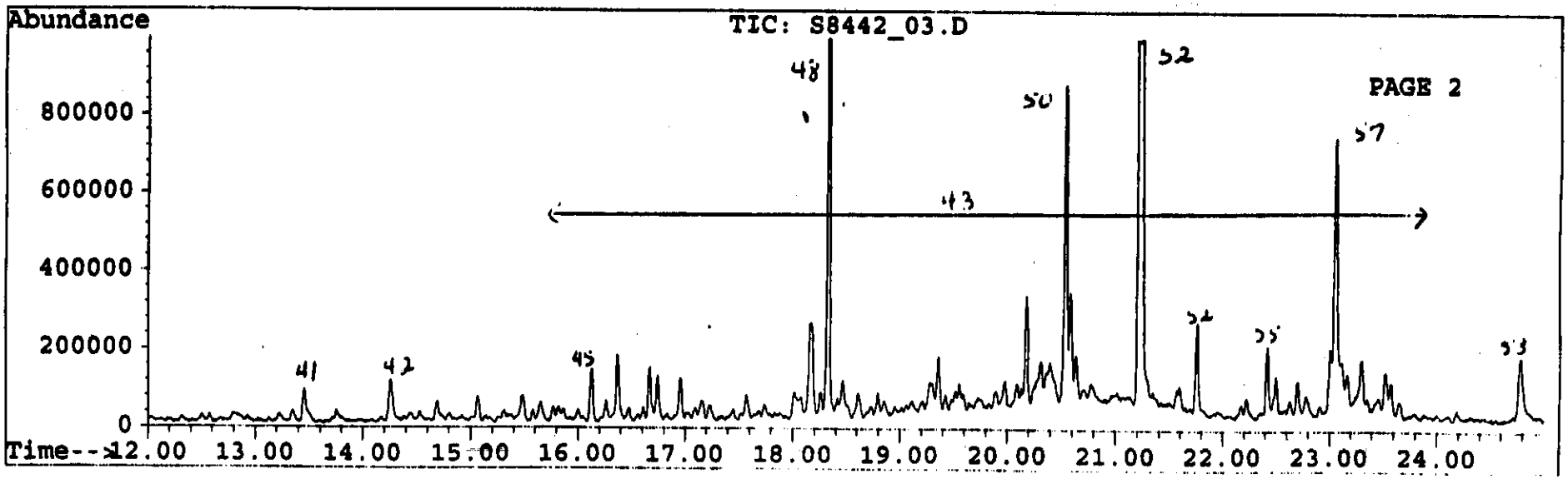




File : C:\HPCHEM\1\DATA\S8442\S8442_03.D
Opera. : AAG
Acquired : 18 Apr 96 9:23 am using AcqMethod ATD
Instrument : 5970 - In
Sample Name: SAMPLE A05008
Misc Info : 30 M DB-1 SC20-300 TP35-300
Vial Number: 3

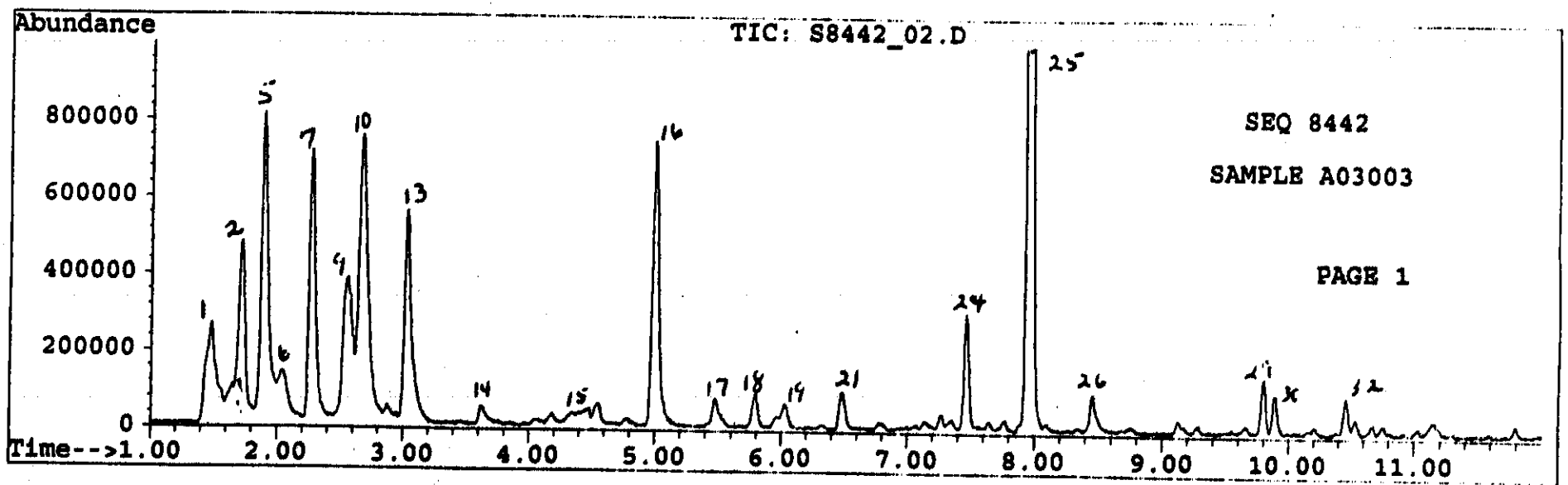
10 Apr 96
Range 2 - Scotchgard
(4.2 liter sample)

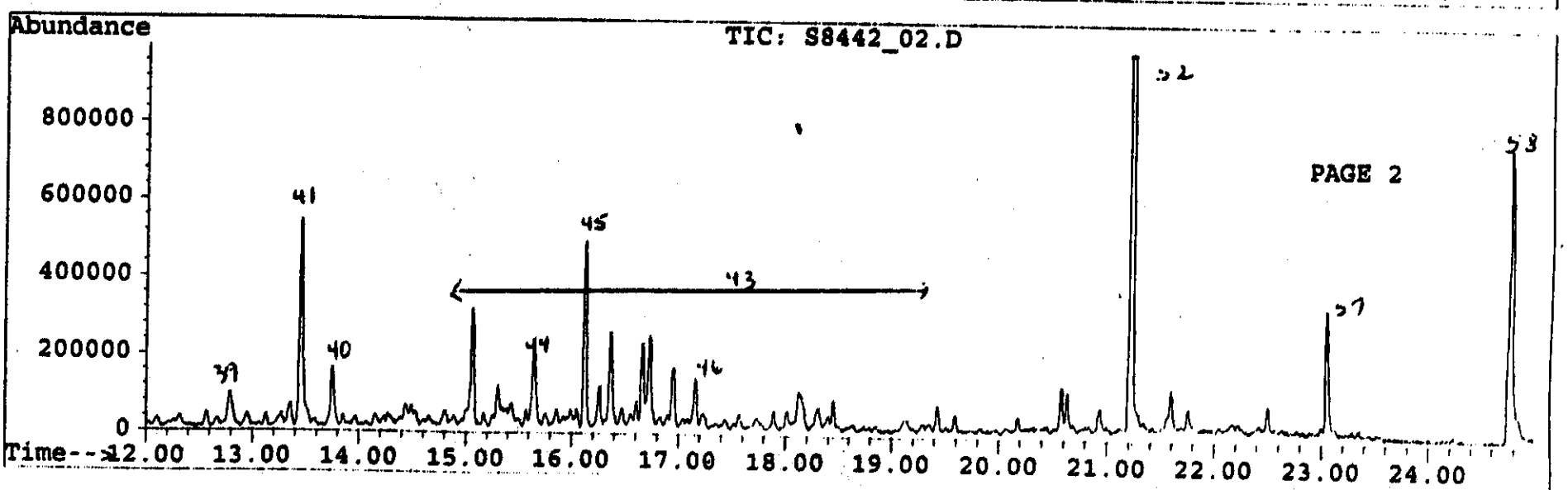




File : C:\HPCHEM\1\DATA\S8442\S8442_02.D
Opera. : AAG
Acquired : 18 Apr 96 8:33 am using AcqMethod ATD
Instrument : 5970 - In
Sample Name: SAMPLE A03003
Misc Info : 30 M DB-1 SC20-300 TP35-300
Vial Number: 2

10 Apr 96
Raycout - RPC
(4.8 liter sample)





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Safety and health at work for all people through research and prevention