HETA 98-0011-2801
Woodbridge Corporation
Brodhead, Wisconsin

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PREFACE

The Hazard Evaluations and Technical Assistance Branch (HETAB) of the National Institute for Occupational Safety and Health (NIOSH) conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health (OSHA) Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from any employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

HETAB also provides, upon request, technical and consultative assistance to Federal, State, and local agencies; labor; industry; and other groups or individuals to control occupational health hazards and to prevent related trauma and disease. Mention of company names or products does not constitute endorsement by NIOSH.

ACKNOWLEDGMENTS AND AVAILABILITY OF REPORT

This report was prepared by Helga Daftarian, Kevin Roegner, and Christopher Reh of HETAB, Division of Surveillance, Hazard Evaluations and Field Studies (DSHEFS). Statistical assistance was provided by Charles Mueller, HETAB, DSHEFS. Laboratory support was provided by Daniel Lewis, Chief, Analytical Services Branch, Health Effects Laboratory Division (HELD), NIOSH Morgantown, and Toni Bledsoe, chemist, HELD, NIOSH Morgantown. Field assistance was provided by Boris Lushniak, Joel McCullough, Loren Tapp, Ann Krake, BJ Haussler, Tim Bushnell, Barbara MacKenzie, Deborah Sammons, Joshua Harney, Gregory Kinnes, and Rob McCleary. Desktop publishing was performed by Patricia C. McGraw, HETAB, DSHEFS. Review and preparation for printing were performed by Penny Arthur.

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For the purpose of informing affected employees, copies of this report shall be posted by the employer in a prominent place accessible to the employees for a period of 30 calendar days.

Highlights of the NIOSH Health Hazard Evaluation

Medical/Industrial Hygiene Evaluation - Woodbridge Corporation

NIOSH conducted a medical/industrial hygiene evaluation of health concerns among Woodbridge employees.

What NIOSH Did

Questionnaire survey for health effects.

Breathing tests for asthma.

Blood and skin tests to study toluenedissocyanate (TDI) sensitivity.

Air sampling for TDI & other chemicals.

Urine testing for evidence of TDI exposure.

What NIOSH Found

Low levels of TDI & other chemicals.

Urine tests showed TDI exposure.

Questionnaire results and breathing tests found a high rate of asthma noted in workers exposed to TDI; however, definite conclusions cannot be drawn due to low participation rate.

Blood and skin tests did not find evidence of sensitivity to TDI.

What Managers Can Do

Improve ventilation on both production lines by making modifications to the passive exhaust system and relocating supplemental fans.

Maintain current health & safety guidelines, and provide regular health & safety training to employees.

Instruct employees of the hazards associated with all chemicals used at the facility (including TDI).

Encourage regular use of appropriate personal protective equipment (PPE) by employees working in the production area.

Provide a comprehensive medical surveillance program in order to reduce the health problems associated with TDI.

What the Employees Can Do

Don't put fans near hoods.

Use safe work practices & appropriate PPE.

Don't eat, drink or smoke in work areas.

Report work-related symptoms to the plant medical clinic.

CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

What To Do For More Information:
We encourage you to read the full report. If you would like a copy, either ask your health and safety representative to make you a copy or call 1-513/841-4252 and ask for HETA Report #98-0011-2801

NIOSH National Institute for Occupational Safety and Health

Health Hazard Evaluation Report 98-0011-2801
Woodbridge Corporation
Brodhead, Wisconsin
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SUMMARY

On October 14, 1997, the National Institute for Occupational Safety and Health (NIOSH) received a Health Hazard Evaluation (HHE) request from the Union of Needle trades, Industrial and Textile Employees (UNITE!) Local 1871 on behalf of employees at the Woodbridge Corporation in Brodhead, Wisconsin. The request indicated that five Woodbridge employees had been diagnosed with cancer over the last four years, and employees were concerned that these cancers might be caused by workplace exposures at Woodbridge Corporation, namely exposure to toluene diisocyanate (TDI), the primary chemical constituent used to make the flexible foam used for automotive seat cushions. The HHE requestor later noted that 83 employees had recently completed a health and safety survey, and various health symptoms were reported which were consistent with exposure to diisocyanates.

On November 19 - 20, 1998, NIOSH investigators conducted an initial site visit, which included an opening conference, employee interviews, interviews with Woodbridge health and safety personnel, medical records review, and a walk-through inspection of the Woodbridge foam-production facility. During March 3 - 5, 1999, NIOSH investigators returned to the Woodbridge facility to conduct environmental air sampling for hydrocarbon solvents (naphthas).

Based on the findings from the first two site visits, NIOSH investigators conducted a combined medical and industrial hygiene study at the plant during the week of May 24, 1999, which was designed to evaluate employees' exposures to TDI, and to determine the relationship between TDI exposure and the prevalence of occupational asthma, airway hyper responsiveness, allergic sensitization to TDI, and diisocyanate-related allergic contact dermatitis. NIOSH investigators also conducted an evaluation of the five reported cancers.

One hundred fourteen (39%) of the 290 Woodbridge employees completed medical questionnaires, 100 provided blood samples for measuring TDI-specific antibodies, 65 provided serial peak flow records for assessing airway hyper responsiveness, and 26 participated in skin patch testing to assess allergic contact dermatitis.

Asthma and work-related asthma were defined from questionnaire responses using standard epidemiologic definitions; cases defined in this way may not meet standard clinical definitions of asthma. Twenty-two percent (25/114) of the participants met the case definition for asthma, and 18% (20/114) met the case definition for work-related asthma. Production work [prevalence rate ratio (PRR)=3.40; 95% confidence interval (CI)= 0.92-39.52] and ever working with TDI (PRR=2.31; 95% CI 0.34-123.20) were both associated with asthma. Production work (PRR=2.66; 95% CI=0.65-29.16) and ever working with TDI (PRR=1.83; 95% CI 0.25-92.75) were also associated with work-related asthma. However, these associations were not statistically significant ($p<0.05$).

Of the 59 peak flow participants whose peak flow records were suitable for analysis, 25 (42%) met the definition for airway hyper responsiveness. Of the 25, 8 had a work-related pattern, 5 had a non-work related pattern, and no pattern could be discerned for the remaining 12.

Eighty-two (72%) of participants met the case definition for work-related mucous membrane (nose and eye) irritation symptoms. Production line work (PRR=1.57; 95% CI 1.05-10.05) and ever working with TDI (PRR=1.88; 95% CI 0.97-23.08) were associated with mucous membrane symptoms ($p<0.05$).

Antibody test and skin patch testing results did not show an immune response to TDI, or the presence of TDI-related allergic contact dermatitis. Of the 100 individuals providing blood for antibody testing, two had an elevated TDI-specific immunoglobulin class G (IgG) antibody level, and none had an elevated TDI-specific immunoglobulin class E (IgE) antibody level. Of the 26 individuals participating in skin patch testing, none developed skin reactions to any of the test allergens either 48 or 96 hours after patch test application.

Personal breathing zone (PBZ) air samples were calculated for each worker participating in the medical evaluation. Additionally, PBZ samples were obtained for a random sample of workers who did not participate in the medical evaluation. TDI area air sampling was also conducted. Workers who participated were also asked to provide an end-of-shift urine sample, which was analyzed for a metabolite of TDI exposure, toluene diamine (TDA).

The highest TDI (2,4-, 2,6- and total TDI) exposures were found among production line workers. Demold workers had the highest mean total TDI exposures (2.75 micrograms per cubic meter [$\mu g/m^3$]), followed by insert workers (2.37 $\mu g/m^3$), mechanics (1.49 $\mu g/m^3$), and utility (1.40 $\mu g/m^3$) workers. However, TDI concentrations for all PBZ and area samples were below the current American Conference of Governmental Industrial Hygienists (ACGIH®) Threshold Limit Value (TLV®) of 36 $\mu g/m^3$. A sample stability problem arose when TDI air samples collected in May 1999 underwent storage for a 3-month period; a 12-14% decline in TDI concentration between analysis in September and October 1999 was identified in these samples. However, reanalysis of the samples did not support a continuous sample stability problem, and only minor concentration declines were identified between the initial and subsequent TDI analyses. Analysis of urine TDA concentrations in workers demonstrated that production line workers (primarily demold and insert workers) had the highest TDA levels; creatinine-corrected mean urine total TDA levels among demold workers and insert workers were 1.77 micrograms per liter (µg/l) and 1.74 µg/l, respectively. Statistically significant correlations were found between total TDI exposure and both uncorrected ($r=0.30$, $p=0.007$) and creatinine-corrected ($r=0.35$, $p=0.002$) urine 2,4-TDA levels.

PBZ and area air samples were collected for formaldehyde on May 22, 1999, (during the cold blast mold cleaning operation) and on May 24, 1999, (during typical operation). PBZ and area samples were also collected for hydrocarbon solvents (naphthas), as well as for bis (2-dimethylaminoethyl) ether (DMAEE) on March 4, 1999, and May 24, 1999, respectively. All concentrations of formaldehyde and hydrocarbon solvents (naphthas) were below applicable exposure limits. One of 8 PBZ DMAEE concentrations exceeded the ACGIH TLV of 0.33 mg/m^3.

Although airborne exposures to TDI were below recommended exposure limits, respiratory, mucous membrane, and skin problems were noted in this worker population and these symptoms were associated with indicators of TDI exposure. The strength of this association, however, was limited by the low participation rate of the study. Insert and demold workers had higher environmental TDI exposure levels compared with offline workers and non-production personnel, and subsequently demonstrated higher urine TDA levels. Exposures to formaldehyde and hydrocarbon solvents were also below the applicable exposure criteria. DMAEE was measured in excess of the TLV in one PBZ sample. The reported cancers among Woodbridge employees are not consistent with a work-related etiology, due to the variety of cancers noted, the limited carcinogenic potential of the compounds identified, and the low exposure levels measured for each compound. Recommendation include following proper medical surveillance procedures for employees exposed to TDI, improving ventilation, improving the availability and usage of personal protective equipment, and following existing health and safety guidelines.

KEYWORDS: SIC 3714 (Motor vehicle parts and accessories), diisocyanates, occupational asthma, diisocyanate-induced sensitization, allergic contact dermatitis, toluene diisocyanate (TDI), toluene diamine (TDA), formaldehyde, hydrocarbon solvents, Bis (2-dimethylaminoethyl) ether, NIAX, respiratory irritants, foam-manufacturing.

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INTRODUCTION

On October 14, 1997, the National Institute for Occupational Safety and Health (NIOSH) received a Health Hazard Evaluation (HHE) request from the Union of Needle trades, Industrial and Textile Employees (UNITE!) Local 1871 on behalf of employees at the Woodbridge Corporation in Brodhead, Wisconsin. The request indicated four union members and one management person had been diagnosed with cancer over the last four years, and employees were concerned that these cancers might be caused by workplace exposure to toluene diisocyanate (TDI), the primary chemical constituent used to make the flexible foam manufactured at the plant.

On November 19 - 20, 1998, a NIOSH medical officer and two NIOSH industrial hygiene officers conducted an initial site visit, which included an opening conference, confidential medical interviews with eight employees, interviews with the health and safety representative and occupational health nurses, a medical records review, and a walk-through inspection of the Woodbridge Brodhead plant. Results of a 1998 health and safety survey (which was conducted by the union) were also reviewed. The survey results indicated that production employees were experiencing a variety of health effects, including difficulty breathing; eye, nose and throat irritation; skin rashes; nausea; dizziness; and headaches. Based upon this information, NIOSH investigators determined that a more comprehensive evaluation was needed in order to evaluate the variety of health concerns noted by the employees.

Between March and September 1999, NIOSH investigators made three more visits to the plant. On March 3 - March 5, 1999, NIOSH investigators made a second site visit. During this visit, NIOSH industrial hygienists conducted area air sampling for total hydrocarbons in various areas of both production lines.

During the weeks of May 22 - 28 and September 27 - October 1, 1999, NIOSH investigators evaluated employees' exposures to TDI, formaldehyde, and Bis (2-dimethylaminoethyl) ether (DMAEE), and conducted a medical evaluation to determine whether TDI exposure at this plant was associated with occupational asthma, airway hyper responsiveness, allergic sensitization to TDI, or diisocyanate-related allergic contact dermatitis. Study participants were notified of their individual test results by letter, and an interim report outlining the initial study findings was submitted by NIOSH to the management and union in April 1999.

BACKGROUND

The Woodbridge Brodhead plant produces flexible polyurethane foam cushions for automobile seats. The process involves common polyurethane foam chemistry, the core reaction of which is a diisocyanate reacted with a polyol/resin mixture to form a carbamate, commonly referred to as a polyurethane. The diisocyanate used at the plant is the monomeric form of a mixture of 2,4- and 2,6-isomers of TDI. TDI is delivered to the plant by either rail car or tank truck and is offloaded under positive pressure into one of five, 5000-gallon storage tanks.

Woodbridge Brodhead operates two production lines: A-line and B-line, both of which are in nearly continuous operation. Each line consists of a chain of heated molds which continuously cycle through a series of stations. The stations are as follows: 1) wax application, 2) insert, 3) pourhead, 4) heating/curing oven, and 5) demold. A robot applies the wax to molds on the B-line. On the A-line wax is applied manually. Workers in the A-line demold station (described below) rotate through the wax application booth in 60-minute intervals. The term "wax dispersion" is used to describe the liquid form in which wax is applied to the mold bowls. The liquid formulation is a wax dispersed in naphtha solvent. The wax dispersion is applied to the bowl of the mold using

an air atomization spray gun. The organic solvent evaporates upon contact with the heated mold, leaving a wax residue. This residue prevents the foam cushion from adhering to the mold.

Workers in the insert area are responsible for inserting components such as metal frames/wires and velcro patches, into the bowl of the mold before it reaches the pourhead. Typically, five employees work at each of the insert stations. One of the five insert workers is rotated off the line, in turn, into what is termed a supply job, which entails bringing needed materials to the other workers at the insert station. This rotation occurs at 60-minute intervals.

The pourhead is the point where the TDI and polyol components come together and the mixture is dispensed by a robot into the bowl of an open mold. Each of the pourheads is fully automated, and a worker is not required to be in the immediate vicinity. The molds close as they move away from the pourhead and into the heating/curing oven, which is maintained at a nominal temperature of 150 degrees Fahrenheit. A mold moves through the oven in four minutes as the foam should fully set. The lid of the mold opens as the mold moves from the oven to the demold area.

Persons in the demold area are responsible for removing the foam cushion from the mold and cleaning the mold in preparation for the next cycle. The demold work station is subdivided into pullers, tossers, and mold cleaners. Pullers loosen the foam cushions from the bowls of molds. The tosser removes the foam cushion from the mold and places it on a conveyor belt. Mold cleaners pull tape and residual foam flash out of the mold. After the foam cushions are removed from the molds, employees in the offline area trim, sort, inspect, and repair the finished foam product.

At the time of the first site visit in November 1998, the Woodbridge Brodhead plant employed 240 hourly workers, distributed equally across three shifts. Sixty-eight hourly workers per shift work directly on the production line, 34 on each line. Of these 68 employees, 16 work directly on the foam production line (i.e., insert or demold), and the remainder work in the offline area. Other hourly employees work in the warehouse area (where baskets of cushions are stacked), or as tooling or maintenance specialists.

Eight employees were interviewed by NIOSH investigators during the first site visit. Employees were selected for an interview by representatives of the union, based on their symptoms, availability, and desire to talk to NIOSH investigators. Among interviewed employees, the most frequently occurring symptoms were breathing problems (7), sinus symptoms (1), headache (1), dizziness, (1) and skin rash (1). Two of the employees interviewed stated that they had been evaluated by their physicians for their symptoms; one was diagnosed with TDI-induced chemical bronchitis, and the other with TDI-induced asthma. Four of the interviewed employees stated that their symptoms worsened when they were working in the plant, and would improve once they left the plant premises. One employee reported that her symptoms were worse while working in the plant during the winter months, when natural outdoor ventilation was not readily available.

METHODS

Medical

Overview, Questionnaire, and Peak Expiratory Flow (PEFR) Measurement

The medical study was designed to assess the relationship between TDI exposure at this plant and the prevalence of occupational asthma, airway hyper responsiveness, allergic sensitization to TDI, and diisocyanate-related allergic contact dermatitis. All employees were invited to

participate. Participants were asked to complete a self-administered questionnaire which addressed work and health history, as well as a history of work-related symptoms. In addition, participants were asked to provide a blood sample for measurement of antibodies to TDI, as well as to common environmental allergens and total immunoglobulin E (IgE) antibody. Also, employees were asked to periodically measure their peak expiratory flow rate (PEFR) by using a mini-Wright peak flow meter (manufactured by Clement Clarke, Inc., Mason, Ohio).

Antibody Testing

Approximately 5 cc of whole blood was obtained from each participant who consented to antibody testing. After collection, each blood specimen was centrifuged in order to separate serum from the cellular component. The resulting serum samples obtained were submitted in a frozen state to the NIOSH Health Effects Laboratory Division for immunoassay testing. Serum samples were tested using both the Pharmacia CAP System™ fluoroenzymeimmunoassay (FEIA) (manufactured by Pharmacia & Upjohn Diagnostics AB, Uppsala, Sweden) (for determining TDI-specific IgE levels), and enzyme-linked immunosorbent assay (ELISA) (for determining TDI-specific IgG levels). Measurement of TDI-specific antibody levels is helpful in identifying those individuals who may have sensitization (allergy) to TDI.

Serum samples were also tested for total IgE levels using the Pharmacia CAP System™ FEIA (Pharmacia AB, Uppsala, Sweden). Total IgE levels were reported in kU/l. Each serum sample was also assayed against six CAP® environmental allergen mixes: grass mix (gx1), house dust mix (hx2), mold mix (mx1), tree mix (tx2), weed mix (wx1), and epidermal mix (ex1). The environmental allergen mixes were chosen because they contained allergens typically found in the region of southern Wisconsin. Reaction to each mix was scored as either positive or negative, and a positive reaction to one or more of the mixes was considered evidence of atopy (allergy). Measurement of total IgE and IgE antibodies specific to environmental allergens are useful in identifying those individuals with a positive atopic status.

Serial Peak Flow Testing and Analysis

Peak flow instruction was conducted by two NIOSH medical officers, who demonstrated to participants correct usage of the mini-Wright peak flow meter, and instructed participants in the proper completion of the serial peak flow log forms (i.e., the recording of PEFR measurements and medical symptoms). NIOSH instructors then directly observed each participant's use of the mini-Wright peak flow meter, to ensure that each participant understood and could demonstrate proper peak flow measurement technique. Participants were instructed to measure and record their PEFRs five times daily (i.e., upon awakening or before leaving for work, upon arriving at work, lunchtime or mid-shift break, before leaving work, and four hours after leaving work). Additionally, participants were asked to measure and record their PEFRs three consecutive times during each of their five peak flow measurement sessions (totaling 15 PEFR measurements daily) for a period of seven consecutive days.

Participants' peak flow records were analyzed using a peak expiratory flow software program, developed by the Division of Respiratory Disease Studies, NIOSH, Morgantown. A period percent amplitude mean (defined as $[PEFR_{max} - PEFR_{min}/PEFR_{mean}$) was calculated for each set of peak flow records. A period percent amplitude mean greater than or equal to 20% is indicative of airway hyper responsiveness, which can be an indicator of asthma. If the peak flow patterns showed evidence of 20% or greater variability during the work week and this variability was not evident during days away from work or during the weekend, then the airway hyper responsiveness was considered work-related.

Questionnaire Analysis

Questionnaire responses were analyzed using SAS® Version 6.12 statistical software (SAS Institute, Cary, North Carolina). Employees were classified in a variety of ways, which served as indicators of potential TDI exposure. These included:

1) Production (demold/insert/offline/other) area vs. non-production area;
2) Job class (hourly line A or B vs. salaried; this classification did not include those individuals who classified themselves as working in either the maintenance, warehouse, tooling, or "other" job categories);
3) Percent of time spent in the production area (<75% vs. ≥75%); and
4) Self-reported exposure to TDI (ever exposed to TDI vs. never exposed to TDI).

Health outcome variables based upon questionnaire responses included the following:

1) Asthma (defined as wheezing, plus one of the following: shortness of breath, cough, or chest tightness);
2) Work-related asthma (defined as questionnaire-based asthma which improves on non-work days);
3) Work-related mucous membrane irritation (defined as an itchy, stuffy, or runny nose or frequent sneezing or eye irritation at work); and
4) Work-related skin symptoms (defined as dermatitis, eczema, or other red rash reported in the last 12 months which improves away from work).

The case definitions used for asthma and work-related asthma are based on standard epidemiologic definitions, and reflect self-reported symptom information obtained from the medical symptoms questionnaire. These case definitions are distinct from the clinical case definition for asthma, which is based on specific clinical and diagnostic criteria. Questions regarding exposures to isocyanates in previous jobs or from home use of isocyanate-containing materials were also included in the questionnaire.

Skin Patch Testing

Participants who reported skin problems (consistent with allergic contact dermatitis) in the last 12 months were asked to participate in skin patch testing. Skin patch testing was performed by a board-certified dermatologist during the week of September 27, 1999, using a six-component isocyanate skin patch testing series marketed by the Chemotechnique Diagnostics Company (Malmo, Sweden; distributed by Dormer Laboratories, Inc., Rexdale, Ontario, Canada). Allergens used as part of the isocyanate series included TDI, diphenylmethane-4,4-diisocyanate (MDI), 1,6-hexamethylene diisocyanate (HDI), diaminodiphenylmethane, isophorone diisocyanate (IPDI), and isophorone diamine (IPD). Skin patches were applied to the participant's upper back for a period of 48 hours, after which time the patches were removed and an initial reading was made. Participants returned 96 hours after initial application for a second reading. Interpretation of patch testing results involved the use of a standard scale of 1+ to 3+, with 1+ representing erythema and edema at the site of the patch test, 2+ representing vesicles, and 3+ representing a severe reaction with bullae. A reaction of 2+ or 3+ was considered indicative of an allergic reaction.

Statistical Analyses

Prevalence rate ratios (PRRs) were used to measure the association between health outcomes and exposures. When the PRR is 1 or less, we say that people with the exposure are no more likely to have the health outcome than people without the exposure. That is, there is no evidence that being exposed is related to an increase in the occurrence of the health outcome. When the PRR is greater than 1, we say that people with the exposure are more likely to have the condition than people without the exposure. That is, there is evidence that being exposed is related to an

increase in the occurrence of the health outcome. We estimate the PRR from the data collected, but because all estimates have some uncertainty, we also calculate the confidence interval (CI) for the PRR. A CI that does not include the number 1.0 means that the evidence of an association between a disease and the exposure is especially convincing. A CI that includes the number 1.0 indicates that the evidence of an association between disease and exposure is less convincing.

Cancer Evaluation

NIOSH investigators obtained further information for each of the six cancer cases that were reported in the HHE request. This information included: name, date of birth, gender, year of hire, employment information (including current job title, year present position began, department/area worked), type of cancer, year cancer was diagnosed, and vital status (alive vs. deceased). Based on the information provided, we calculated the latency periods (defined as the time between starting work at Woodbridge and the diagnosis of cancer) for each of the six reported cases.

Industrial Hygiene

TDI Inhalation Exposure Assessment

The TDI inhalation exposure assessment consisted of personal breathing zone (PBZ) air sampling of workers at the facility. The focus of this air sampling was to collect one full-shift TDI air sample from each worker participating in the medical evaluation. Additionally, workers (not in the medical evaluation) were randomly selected to participate in the exposure assessment. Also, some TDI area air sampling was conducted.

The air sampling and analytical method used in this study was a new NIOSH method for isocyanate-containing compounds.[1] In this method, area and PBZ air samples were collected using battery-operated air sampling pumps calibrated to a flow rate of 1 liter per minute (Lpm). These pumps were used to draw air through a 37 millimeter (mm) quartz fiber filter (QFF) impregnated with 1-(9-anthracenylmethyl) piperazine (MAP). The QFFs were connected to the inlet port of the air sampling pump with Tygon® tubing. For PBZ air sampling, the QFFs were placed as near each worker's breathing zone as possible (the collar or lapel), and the sampling device was worn for the entire shift. Immediately after the cessation of air sampling (usually at the end of the shift), the QFFs were removed from the filter cassette and placed in a clean jar containing 5 milliliters (mL) of a MAP in acetonitrile solution. All samples were shipped and stored in a cold environment prior to analysis.

The QFFs were analyzed by pH-gradient high pressure liquid chromatography (HPLC) with ultraviolet and fluorescence detection for both 2,4-TDI and 2,6-TDI. Upon receipt at the analytical laboratory, 10 microliters of acetic anhydride were added to each filter sample, and allowed to react overnight with the excess MAP. Next, the sample solutions were filtered and concentrated to 1 mL. The HPLC analysis used a 150 x 4.6 mL C_8 Inertsil column containing 5 micron particles. The mobile phase flow rate was 1.5 mL per minute. The mobile phase for this analysis actually consists of two separate solutions (A and B). Solution A is 65% acetonitrile and 35% pH 6.0 buffer, and solution B is 65% acetonitrile and 35% pH 1.6 buffer. The gradient involved beginning the analysis at 100% A, and holding for 7 minutes. Then the mobile phase was gradually changed to a mixture of 70% A and 30% B over a 4 minute period, and then held for 3 minutes. Finally, the mobile phase was changed to 100% B, and held for 6 minutes. Thirty microliters of each sample were injected into the instrument. Analysis of MAP-derivatized monomer standards in the appropriate concentration range were interspersed with the sample analyses. Monomers (2,4-TDI and 2,6-TDI) were quantified based on comparison of their fluorescence peak heights to those of

monomer standards. Also, each worker's 2,4-TDI and 2,6-TDI exposure concentrations were summed to get a total TDI exposure measurement.

The limit of detection (LOD) and limit of quantification (LOQ) for 2,4-TDI were 42 and 110 nanograms (ng) per sample, respectively. In addition, the LOD and LOQ for 2,6-TDI were 15 and 28 ng per sample, respectively. LODs and LOQs are values determined by the analytical procedure used to analyze the samples, and are not dependent on air sample volume. Minimum detectable concentrations (MDCs) and minimum quantifiable concentrations (MQCs) are determined by dividing the LODs and LOQs by respective air sample volumes appropriate for the given set of samples. In determining the MDC and MQC for these exposure data, the NIOSH industrial hygienist used the highest sample volume collected during this survey (556.6 liters). This results in a 2,4-TDI MDC and MQC of 0.08 and 0.20 micrograms per cubic meter of air ($\mu g/m^3$), and a 2,6-TDI MDC and MQC of 0.03 and 0.05 $\mu g/m^3$, respectively. The MDC and MQC reflect the sensitivity of the air sampling and analysis protocol; *i.e.*, the lowest 2,4-TDI and 2,6-TDI exposure concentrations that could be reliably detected and quantified by the procedures used in this study.

TDI Dermal Exposure Assessment

Dermal TDI exposures were assessed using the Permea-Tec™ detectors produced by Omega Specialty Instrument Company (Chelmsford, Massachusetts).[2] These detectors have an adhesive backing that adheres to the skin, and an impregnated pad on the exposed surface that changes color when exposed to isocyanates. The limit of detection for these detectors is 3 micrograms of isocyanate per pad, and they provide a qualitative (yes/no) dermal exposure measurement. The detectors were placed on the palmar side of the index finger, and/or on the palm of the worker, and read in the field. If the worker was wearing a light-weight cotton glove, the detector(s) was placed inside the glove.

Biological Monitoring for Urine TDA

Every worker who participated in the TDI inhalation exposure assessment was asked to provide the NIOSH investigators with an end-of-shift urine sample. These samples were analyzed for 2,4-toluene diamine (2,4-TDA), 2,6-toluene diamine (2,6-TDA), and creatinine. These samples were collected to determine the level of correlation between TDI exposure measurements and the corresponding urine TDA levels.

The workers' urine samples were analyzed for 2,4-TDA and 2,6-TDA using a method developed simultaneously by a contract laboratory.[3] Each urine sample was collected in a sterile bottle containing citric acid as a preservative, and shipped and stored in a cold environment prior to analysis. An aliquot of the urine was hydrolyzed with sodium hydroxide to convert conjugated amines back to the original amine. Then the amines were extracted from the urine matrix with butyl chloride, and back-extracted from the organic solvent layer with an aqueous acid. Finally, 2,4-TDA and 2,6-TDA were separated using HPLC with a 4.6- x 150-mLr C_{18} reversed-phase column with 5-micron particles, and determined by electrochemical detection. The MDC and MQC for 2,4-TDA were 0.4 and 1.3 micrograms per liter ($\mu g/L$), respectively. In addition, the MDC and MQC for 2,6-TDA were 0.5 and 1.6 $\mu g/L$, respectively. Finally, each worker's 2,4-TDA and 2,6-TDA urinary levels were summed to get a total urine TDA measurement.

One issue associated with spot urine samples is the effect of urinary water output on the volume portion of the urine TDA concentration. Since urinary water output varies according to water intake, activity, environmental conditions, and other factors, a dilution correction can be used to normalize the volume portion of the urine TDA

concentration; that is, to control for variations in urinary water output. One method of dilution correction is to express the urine concentrations in micrograms of analyte per gram of creatinine (µg/g–Cr). Creatinine is a substance normally found in urine and is excreted at a fairly constant rate, independent of the urinary water output. Hence, the urine 2,4-TDA, 2,6-TDA and total TDA were also determined using the creatinine correction method.

Whenever measuring workplace exposures and urinary markers of exposure, there is a possibility that a given sampling and analytical method will not detect any of the analyte. Hence, the analyte concentration will be reported as "none detected" (ND) or below the MDC. As such, these values cannot be used in descriptive statistics (such as means or standard deviations), or in correlation analyses (*e.g.*, correlation between TDI exposures and urine TDA levels). Hornung *et al*.[4] provided guidance on how to estimate average analyte concentrations when dealing with censored data (*i.e.*, the results are below the MDC, but not zero). This guidance was used to estimate 2,4-TDI, 2,6-TDI, 2,4-TDA, and 2,6-TDA concentrations for those workers with levels below the MDC. Considering this, the number of air or urine samples below the MDC will be provided in this report, but an actual number/concentration (expressed in µg/m^3 for TDI and in µg/l for TDA) is used for these samples when presenting and discussing the TDI and TDA data.

Goodness-of-fit testing was performed on the TDI and TDA data sets to determine the underlying distribution of the data. Past studies of chemical exposures have determined that these data tend to be lognormally distributed.[5] If the data were found to be lognormally distributed, then all statistical analyses were performed using the log-transformed data.

Correlation analyses were conducted to examine the relationship between TDI exposures and uncorrected and creatinine-corrected urine TDA levels. All statistical analyses were performed using either SAS Version 6.12 (SAS Institute, Inc., Cary, North Carolina) or Winstat Version 3.1 (Kalmia Company, Inc., Cambridge, Massachusetts). In all cases, a result was considered statistically significant when the probability of obtaining a more extreme finding was less than or equal to 5% ($p \leq 0.05$).

Formaldehyde

Area and PBZ air samples were collected for formaldehyde on May 22, 1999, during the cold blast mold cleaning operation, and on May 24, 1999, during typical operation. PBZ samples were collected on the worker's lapel during the full shift. Samples collected for less than 8 hours were computed into 8-hour time-weighted average (TWA) values applying the assumption that exposures during the unsampled portion of the shift equaled exposures for the sampled period. Four employees rotated through the ice-blasting job. Each employee wore a supplied-air hood while using the ice gun. PBZ samples were collected under the hood while employees performed the task. Air samples were collected by drawing air through a silica gel-containing cartridge coated with 2,4-dinitrophenylhydrazine. Cartridges were connected via flexible Tygon® tubing to sampling pumps calibrated at a nominal flow rate of 0.1 Lpm. Samples and field blanks were analyzed for formaldehyde using NIOSH analytical Method 2016.[6]

Hydrocarbon Solvents (Naphthas)

Seven PBZ and two area air samples were collected for solvent vapors emitted from the wax application process on March 4, 1999. The samples were collected over a full shift during typical operations. Samples collected for less than 8 hours were computed into 8-hour TWA values applying the assumption that exposures

during the unsampled portion of the shift equaled exposures during the sampled period. Sampling media for a sample collected in the A-line wax application booth was changed out after four hours of sampling to limit potential breakthrough losses. The results of these two, four-hour samples have been time-weighted, and are reported as an 8-hour sample. Air samples were collected by drawing the air through a sorbent tube containing 100 milligrams (mg) front bed/50 mg rear bed of coconut shell charcoal. Sorbent tubes were connected via flexible Tygon® tubing to sampling pumps calibrated at a nominal flow rate of 0.1 Lpm. Samples and field blanks were analyzed for total hydrocarbons, as stoddard solvent, per NIOSH analytical Method 1550.[6]

Niax® Catalyst A-99 (Bis [2-dimethylaminoethyl] ether, DMAEE)

Tertiary amine catalysts are commonly used in polyurethane foam production. Employees expressed concern to NIOSH investigators about the amine catalyst used at the plant, which has DMAEE as its major component. To assess potential exposure, NIOSH collected nine PBZ and seven area air samples on May 24, 1999, for DMAEE. These samples were collected during the full duration of the first shift on a typical production day. Samples collected for less than 8 hours were computed into 8-hour TWA values applying the assumption that exposures during the unsampled portion of the shift equaled exposures for the sampled period. Air samples were collected by drawing the air through a glass fiber filter (GFF) in series with an XAD-2 sorbent tube. The GFF was housed in a 37 mL diameter polystyrene cassette. Sampling media were connected via flexible Tygon® tubing to sampling pumps calibrated at a nominal flow rate of 1.0 Lpm. The GFF and XAD2 tubes were shipped and stored under refrigeration until the media were prepared for analysis.

Analyses of the QFFs and XAD2 tubes were conducted separately, but by the same method. Five field blanks were submitted with the sample set. The analyte was extracted from the respective media with an extraction solvent composed of 5% acetone in 95% ethanol. Both the front and back media beds were removed from the sampling tubes for analysis and treated as separate samples (to account for breakthrough). After the addition of the extraction solvent, the samples were sonicated for 30 minutes to enhance the extraction efficiency. All samples, calibration standards, blanks, and QC samples were then analyzed by gas chromatography (GC) with a nitrogen chemiluminescence detector (NCLD). Quantitation was performed with a calibration curve generated from the analysis of liquid standards of the Bis-DMAEE in 95% ethanol.

The method used in the analysis of these samples has not been fully developed. A recovery study was not carried out on the glass fiber filters. Because of the high variability in the recovery of this analyte from the sampling tubes, the concentrations presented in the data table can be considered estimates with an approximate deviation of plus or minus 25%. The nature of this method development project did not allow an in-depth determination of the variability associated with the recovery of DMAEE for the XAD-2 sampling tubes.

Ventilation Assessment

The A and B lines are serviced by two independent exhaust ventilation systems. Both systems fully cover the respective production lines, and incorporate an overhead canopy hood configuration. These systems were evaluated qualitatively and quantitatively to determine how effectively the hoods were containing and exhausting gasses emitted from the process. General airflow direction and hood turbulence were evaluated using smoke tubes. Measurements of face velocity were obtained at several locations around each hood using a VelociCalc® Plus, Model 8360 (TSI Inc., St. Paul, Minnesota).

Pedestal fans were turned off while face velocity measurements were obtained.

EVALUATION CRITERIA

As a guide to the evaluation of the hazards posed by workplace exposures, NIOSH field staff employ environmental evaluation criteria for the assessment of a number of chemical and physical agents. These criteria are intended to suggest levels of exposure to which most workers may be exposed up to 10 hours per day, 40 hours per week for a working lifetime without experiencing adverse health effects. It is, however, important to note that not all workers will be protected from adverse health effects even though their exposures are maintained below these levels. A small percentage may experience adverse health effects because of individual susceptibility, a pre-existing medical condition, and/or a hypersensitivity (allergy). In addition, some hazardous substances may act in combination with other workplace exposures, the general environment, or with medications or personal habits of the worker to produce health effects even if the occupational exposures are controlled at the level set by the criterion. These combined effects are often not considered in the evaluation criteria. Also, some substances are absorbed by direct contact with the skin and mucous membranes, and thus potentially increases the overall exposure. Finally, evaluation criteria may change over the years as new information on the toxic effects of an agent become available.

The primary sources of environmental evaluation criteria for the workplace are: (1) NIOSH Recommended Exposure Limits (RELs),[7] (2) the American Conference of Governmental Industrial Hygienists' (ACGIH®) Threshold Limit Values (TLVs®),[8] and (3) the U.S. Department of Labor, Occupational Safety and Health Administration (OSHA) Permissible Exposure Limits (PELs).[9] Employers are encouraged to follow the OSHA limits, the NIOSH RELs, the ACGIH TLVs, or whichever are the more protective criterion.

OSHA requires an employer to furnish employees a place of employment that is free from recognized hazards that are causing or are likely to cause death or serious physical harm [Occupational Safety and Health Act of 1970, Public Law 95–596, sec. 5.(a)(1)]. Thus, employers should understand that not all hazardous chemicals have specific OSHA exposure limits such as PELs and short-term exposure limits (STELs). An employer is still required by OSHA, however, to protect their employees from hazards, even in the absence of a specific OSHA PEL.

A TWA exposure refers to the average airborne concentration of a substance during a normal 8- to 10-hour workday. Some substances have recommended STEL or ceiling values which are intended to supplement the TWA where there are recognized toxic effects from higher exposures over the short-term.

Diisocyanates (TDI)

The unique feature common to all isocyanate-containing compounds (hereinafter referred to as isocyanates) is that they contain one or more -N=C=O (isocyanate) functional groups attached to an aromatic or aliphatic parent compound. When a parent compound contains two isocyanante functional groups, it is referred to as a diisocyanate. Because of the highly unsaturated nature of the isocyanate functional group, the diisocyanates readily react with compounds containing active hydrogen atoms (nucleophiles). Thus, the diisocyanates readily react with water (humidity), alcohols, amines, *etc.*; the diisocyanates also react with themselves to form either dimers or trimers. When a diisocyanate species reacts with a primary, secondary, or tertiary alcohol, a carbamate (-NHCOO-) group is formed which is commonly referred to as a urethane. Reactions involving a diisocyanate species and a polyol result in the formation of cross-linked polymers; *i.e.,* polyurethanes. Hence, they are used in surface coatings, polyurethane foams, adhesives, resins, elastomers, binders, and

sealants. Some common examples of diisocyanates include 1,6-HDI, 2,4- and 2,6-TDI, 4,4'-MDI, methylene bis(4-cyclohexylisocyanate (HMDI), IPDI, and 1,5-naphthalene diisocyanate (NDI). Commercial-grade TDI is an 80:20 or 65:35 mixture of the 2,4- and 2,6- isomers of TDI, respectively. It should be noted that the low molecular weight diisocyanates (including TDI) tend to volatilize at room temperature, creating a vapor inhalation hazard.

Exposure to isocyanates is irritating to the skin, mucous membranes, eyes, and respiratory tract.[10,11] The most common adverse health outcome associated with isocyanate exposure is asthma; less prevalent are contact dermatitis (both irritant and allergic forms) and hypersensitivity pneumonitis (HP).[11,12,13] Skin contact with isocyanates can result in symptoms such as rash, itching, hives, and swelling of the extremities.[10,11,13] A worker suspected of having isocyanate-induced asthma will exhibit the traditional symptoms of acute airway obstruction, *e.g.*, coughing, wheezing, shortness of breath, tightness in the chest, and nocturnal awakening.[10,12,13] An isocyanate-exposed worker may first develop asthma-like symptoms or an asthmatic condition after a single (acute) exposure, but sensitization usually takes a few months to several years of exposure.[10,12,14,15,16] The asthmatic reaction may occur minutes after exposure (immediate), several hours after exposure (late), or as a combination of both immediate and late components after exposure (dual).[12,15,17] An improvement in symptoms may be observed during periods away from the work environment (weekends, vacations).[10,12,15] After sensitization, any exposure, even at levels below an occupational exposure limit or standard, can produce an asthmatic response which may be life threatening. Experience with isocyanates has shown that monomeric, prepolymeric and polyisocyanate species are capable of producing respiratory sensitization in exposed workers.[18,19,20,21,22,23,24,25,26,27,28,29,30,31,32,33,34] Prevalence estimates for isocyanate-induced asthma in exposed worker populations vary considerably: from 5% to 10% in diisocyanate production facilities[14,35] to 25% in polyurethane production plants[35,36] and 30% in polyurethane seatcover operations.[37] The scientific literature contains a limited amount of animal data suggesting that dermal exposure to diisocyanates may produce respiratory sensitization.[38,39,40,41] This finding has not been tested in dermally-exposed workers.

The percentage of sensitized workers with persistent symptoms of asthma after years of no exposure may be 50% or higher. Studies have shown that workers with persistent asthma had a significantly longer duration of symptoms prior to diagnosis, larger decrements in pulmonary function, and a severe degree of nonspecific bronchial hyperreactivity at diagnosis.[15] These data suggest that prognosis is improved with early diagnosis of diisocyanate-induced respiratory sensitization and early removal from diisocyanate exposure. This emphasizes the need to minimize workplace exposure concentrations, and for active medical surveillance of all workers potentially exposed to diisocyanates.

There is no OSHA PEL for TDI based on an 8-hour, TWA exposure.[9] The ACGIH recently added TDI to the "Notice of Intended Changes for 2000" list, signifying a change in the TLV. The new TLV is an 8-hour TWA exposure of 36 $\mu g/m^3$ (5 parts per billion) for 2,4-TDI, 2,6-TDI, or a mixture of the two isomers.[8] In the TLV documentation, a mixture is defined as being an 80:20 or 65:35 mixture of the 2,4- and 2,6-TDI isomers.[42]

A limited number of animal studies have shown that commercial-grade TDI is carcinogenic in both rats and mice.[43] Significant excesses of liver and pancreatic tumors were observed in male and female rats and female mice that received TDI by gavage (administered directly into the stomach). TDI was also found to have a dose-dependent mutagenic effect on two strains of *Salmonella typhimurium* in the presence of a metabolic activator (S-9 liver fractions from rats or hamsters

treated with Aroclor 254).[44] Based on these animal and *in vitro* studies, NIOSH concluded that sufficient evidence exists to classify TDI as a potential occupational carcinogen, and recommends that exposures be reduced to the lowest feasible concentration (LFC).[45] It is important to note that no epidemiologic data exist linking TDI exposure to elevated cancer rates in exposed workers.

Urine TDA Levels

Recent studies have shown that urine TDA levels are considered biological markers of recent TDI exposure.[46,47,48,49,50,5152] These studies found that the urine TDA excretion is biphasic, with the excretion occurring in both a fast and slow phase. The predominant fast phase is indicative of recent exposure, whereas the lesser slow phase reflects urinary elimination of degradation products of TDI-modified proteins and erythrocytes. The observed half lives for the fast phase were 5.3 - 6.2 hours for 2,4-TDA and 7.4 - 8.4 hours for 2,6-TDA. Conversely, the observed half-lives for the slow phase were 18 days for 2,4-TDA and 19-days for 2,6-TDA. The cumulative amount of 2,4-TDA excreted during the 24-hour period, including and following the exposure period, was 15-19% of the estimated inhaled 2,4-TDI dose. Similarly, the 2,6-TDA excretion over the same period was 17-23% of the estimated inhaled 2,6-TDI dose. Hence an end-of-shift spot urine TDA sample may be used as a biological marker for recent TDI exposure. It is unclear whether the use of the creatinine volume correction method to express urine TDA levels (compared to no correction) improves the correlation between TDI exposure measurements and the observed urine TDA levels.

Formaldehyde

Formaldehyde is a colorless gas with a strong odor. Exposure can occur through inhalation and skin absorption. The acute effects associated with formaldehyde exposure are irritation of the eyes and respiratory tract and irritant and allergic dermatitis. The first symptoms associated with formaldehyde exposure, at concentrations ranging from 0.1 to 5 parts per million (ppm), are burning eyes, tearing, and general irritation of the upper respiratory tract. Individual tolerances and susceptibility to acute exposures of the compound can vary.[53]

In two separate studies, formaldehyde induced a rare form of nasal cancer in rodents.[54,55] Formaldehyde exposure has been identified as a possible causative factor in cancer of the upper respiratory tract in a proportionate mortality study of workers in the garment industry.[56] Based on this information, NIOSH has identified formaldehyde as a suspected human carcinogen and recommends that exposures be reduced to the LFC. The OSHA PEL is 0.75 ppm as an 8-hour TWA, and 2 ppm as a STEL.[57] ACGIH has designated formaldehyde as a suspected human carcinogen, and therefore recommends that worker exposure by all routes should be carefully controlled to levels "as low as reasonably achievable" below the TLV.[58] ACGIH has set a ceiling limit of 0.3 ppm.

Hydrocarbon Solvents (Naphtha)

Petroleum distillates (naphtha), also referred to as refined petroleum solvents, is a general term used to describe a class of complex hydrocarbon solvent mixtures.[59] Petroleum naphtha is composed mainly of aliphatic hydrocarbons (as distinguished from coal tar naphtha, which is a mixture composed primarily of aromatic hydrocarbons).[60,61] Petroleum distillates are further characterized by the boiling range of the mixture; typically, the larger hydrocarbon chain length equates to a higher distillation fraction.[59] Specific names for some typical petroleum distillate mixtures in order of increasing temperature of boiling ranges are: petroleum ether, rubber solvent, varnish makers' and painters' (VM & P) naphtha, mineral spirits, stoddard solvent, and kerosene.[59] Boiling ranges of these

mixtures overlap; therefore, some of these mixtures contain the same hydrocarbons, but in different proportions.

Effects from exposure to refined petroleum solvents are primarily acute, unless significant amounts of substances that have chronic toxicity are present, such as benzene or glycol ethers. Epidemiologic studies have shown that exposure to similarly refined petroleum solvents (i.e., mineral spirits, stoddard solvent) can cause dry throat, burning or tearing of the eyes, mild headaches, dizziness, central nervous system (CNS) depression, respiratory irritation, and dermatitis.[59]

Petroleum naphtha appears to have weak skin cancer causing potential in laboratory mice.[62] The International Agency for Research on Cancer (IARC) has determined that there is only limited evidence implicating petroleum naphtha as a carcinogen in animals and insufficient evidence associating exposure to petroleum naphtha and the development of cancer in humans.[63] However, depending upon the manufacturing process, petroleum naphtha may sometimes contain varying amounts of aromatic hydrocarbons such as benzene.

Many petroleum naphtha mixtures used throughout industry contain *n*-hexane or other simple alkanes. Prolonged and repeated exposure to *n*-hexane may damage peripheral nerve tissue and result in muscular weakness and loss of sensation in the extremities.[59] Studies indicate that methyl ethyl ketone may potentiate peripheral neuropathy caused by *n*-hexane.[64]

Since naphthas are mixtures of aliphatic hydrocarbons, the evaluation criteria are based upon the mixture composition in relation to the most commonly available products - petroleum ether, rubber solvent, VM&P naphtha, mineral spirits, and stoddard solvents. The NIOSH REL for all of the petroleum distillate mixtures is 350 milligrams per cubic meter of air (mg/m^3) as a full shift TWA exposure, for up to 10 hours per day, providing a 40-hour work week is not exceeded. In addition, a ceiling concentration limit (for a 15 minute duration) of 1800 mg/m^3 is recommended by NIOSH. The OSHA PEL for petroleum distillates (naphtha) is 2000 mg/m^3 TWA, while the PEL for stoddard solvents is 2900 mg/m^3. The ACGIH has also established a TLV-TWA (for eight hours) of 1600 mg/m^3 for rubber solvent, 1370 mg/m^3 for VM & P naphtha, 525 mg/m^3 for stoddard solvents (and mineral spirits), and a 15-minute STEL of 1800 mg/m^3 for VM & P naphtha. The NIOSH, OSHA, and ACGIH exposure limits for *n*-hexane are all 180 mg/m^3, for an 8-hour TWA.

DMAEE

DMAEE is a pale yellow liquid that is used primarily as an amine catalyst in the manufacturing of polyurethane foam. As a chemical group, amines tend to be irritating to the skin upon dermal contact and irritating to the respiratory tract upon inhalation. Repeated inhalation exposure studies in rats concluded that DMAEE may cause respiratory irritation at concentrations as low as 0.22 ppm ($1.5 \mu g/m^3$).[65] Based on these data, ACGIH has a TLV of 0.05 ppm (0.33 mg/m^3) for full-shift exposure to DMAEE. Additionally, ACGIH has a STEL of 0.15 ppm (1.0 mg/m^3).

NIOSH and OSHA jointly recommend that NIAX® Catalyst ESN and its components, dimethylaminopropionitrile and DMAEE, as well as formulations containing either component, be handled in the workplace as exceedingly hazardous materials. Investigations of outbreaks of urinary dysfunction among workers at a number of facilities that manufacture flexible polyurethane foam strongly suggest an association between NIAX® Catalyst ESN and the urological disorders. There is no current Federal standard for occupational exposure to NIAX® Catalyst ESN or either of its components. However, on April 7, 1978, OSHA issued a Health Hazard Alert and indicated "it is imperative that worker

exposure to ESN and its components be completely avoided."[66]

RESULTS

Medical

Characteristics of the Study Population

One hundred fourteen (39%) of 290 employees participated in at least the questionnaire portion of the medical evaluation. Seventy-four (65%) of participants were female, and the mean age of all participants was 40 years (range 19 to 71 years). The mean duration of employment among participants was 13 years. The job classification breakdown of the medical study participants included: salaried employees (comprising 15% of the total participant pool); hourly employees (i.e., production line workers and other hourly workers, including vacation replacements, forklift drivers, and line technician assistants, comprising 76% of the total); maintenance workers (comprising 3% of the total); warehouse workers (comprising 4% of the total); and workers in "other" categories (including tooling and resource management, comprising 2% of the total) (Table 1). Of the 114 employees completing questionnaires, 100 provided blood samples, 65 provided serial peak flow records, and 26 of 40 eligible workers participated in skin patch testing.

Asthma

Twenty-two percent (25/114) of the participants met the case definition for asthma, and 18% (20/114) met the case definition for work-related asthma (Table 2). Production workers were more than three times as likely as non-production workers to meet the case definition for asthma (PRR=3.40; 95% CI 0.92-39.52) (Table 3). Workers who had ever worked with TDI were more than twice as likely as workers with no previous history of TDI exposure to have asthma (PRR=2.31; 95% CI 0.34-123.20) (Table 4). None of these associations, however, were statistically significant. The prevalence of asthma was not associated with length of employment at the plant or time spent on the production line.

When environmental sampling results for 2,4-TDI, 2-6-TDI, and total TDI were analyzed for those participants providing PBZ sampling data, it was noted that the means and geometric means for 2,4-TDI, 2,6-TDI, and total TDI were slightly higher among those meeting the case definition for asthma; however, the differences in the means were not found to be statistically significant.

The relationship between TDI exposure and asthma was also evaluated by grouping TDI exposure measurements by quartiles of exposure (Table 5). Using the lowest levels of exposure (quartile 1) as the referent group, individuals in the second, third and fourth quartiles (which were combined, because the prevalence of asthma was the same in these three groups [i.e., 21%]) were four times more likely than the referent group to report symptoms consistent with asthma (PRR=4.00; 95% CI 0.56-29.00). This association, however, was not statistically significant.

Work-Related Asthma

Production workers reported more than twice the prevalence of work-related asthma as non-production workers (PRR=2.66; 95% CI 0.65-29.16) (Table 6). A similar elevation was also found among workers who had ever worked with TDI, compared to workers who had never worked with TDI (PRR=1.83; 95% CI 0.25-92.75) (Table 7). Evaluation of work-related asthma by quartiles of exposure to TDI (Table 8) demonstrated that those individuals in the second, third and fourth quartiles combined were almost three times more likely than the referent group (quartile one) to report work-related asthma (PRR=2.70; 95% CI 0.36-20.00). However, none of these reported associations between indicators

of TDI exposure and work-related asthma were statistically significant.

Airway Hyper responsiveness

Fifty-nine of the 65 serial peak flow records were suitable for analysis. Of the 59 records analyzed, 25 (42%) met the definition for airway hyper responsiveness (Table 2). Of these, 8 demonstrated a work-related pattern, 5 had a non-work related pattern, and no specific pattern was discernable for the remaining 12. We were unable to do a meaningful epidemiological analysis of the peak flow data because of the relatively small number of workers who participated in this phase of the study.

Mucous Membrane Symptoms

Eighty-two (72%) of the participants met the case definition for work-related mucous membrane irritation (Table 2). The prevalence of work-related mucous membrane irritation was 1.6 times higher among production line workers than among non-production line workers (PRR=1.57; 95% CI 1.30-10.05; p=0.01) (Table 9). Workers who had a positive TDI exposure history had 1.8 times the rate of mucous membrane irritation as compared to workers with no TDI exposure history (PRR=1.88; 95% CI 0.97-23.08; p=0.03) (Table 10).

Work Related Skin Symptoms

Sixteen participants (15%) met the case definition for work-related skin symptoms (Table 2). All sixteen workers were production line employees (PRR= ∞; the infinity symbol "∞" indicating an undefined PRR) and worked 75% or more of the time on the production line (Table 11). Of the 26 individuals who participated in skin patch testing, none developed skin reactions to any of the test allergens upon skin examination at 48 and 96 hours after skin patch tests had been applied.

Antibody Testing Results

TDI - Specific IgG/IgE Results

Of the 100 individuals who had blood tests, two had an elevated TDI-specfic IgG antibody level, and none had an elevated TDI-specific IgE antibody level (Table 2).

Total IgE Antibody and Environmental Allergen Test Results

Of the 100 individuals tested, 11 had elevated levels of total IgE, and 35 had positive antibodies to one or more of the six environmental allergen mixes.

History of Previous Work with Isocyanates

Of the 114 participants, 53 (46%) reported having worked in an occupation or industry (other than Woodbridge) with potential exposure to isocyanates, having worked in a job with known exposure to isocyanates, having used plastic foam kits at home, or having used polyurethane varnishes at home.

Frequency of exposure to isocyanates outside of Woodbridge was examined for the four main outcomes (asthma, work-related asthma, mucous membrane irritation, and work-related skin symptoms), in order to determine whether any differences existed between those individuals who had each outcome and those who did not. For three of these outcomes (i.e., asthma, work-related asthma, and mucous membrane irritation), individuals who had the outcome of interest were less likely to report exposure to isocyanates outside of Woodbridge, compared with those who did not have the outcome. For the work-related skin symptom outcome, individuals with work-related skin symptoms were more likely to report

exposure to isocyanates outside of Woodbridge compared with those who did not have work-related skin symptoms. Therefore, exposures to isocyanates outside of Woodbridge could not account for the associations which were observed between asthma, work-related asthma and mucous membrane irritation outcomes and exposure to isocyanates at Woodbridge. For the work-related skin outcome, exposure to isocyanates outside of Woodbridge cannot be excluded as a contributing factor.

Cancer Evaluation

Since 1986, six individuals were reported or were known to have developed cancer among the approximately 290 employees working at the Woodbridge Brodhead facility. The six employees who developed cancer worked in a variety of positions at the plant: production line, tooling, maintenance, and janitorial services. Three of the six cases were female, and three were male. These individuals had five different types of cancer: two cases of colon cancer, one case of breast cancer, one case of bladder cancer, one case of testicular cancer, and one case of leukemia. For the six listed employees for whom information was provided regarding the year they began their current job and the year of cancer diagnosis, the mean time between starting work at the Woodbridge Brodhead facility and the diagnosis of cancer was 18.7 years (range: 11-27 years). Three of these cases had latency periods less than 15 years.

Industrial Hygiene

TDI

The data from the TDI exposure determinations are presented in Table 12**,** and summary results based on these data are in Table 13. Full-shift TDI exposure measurements were collected from 104 workers over 3 shifts: 37 were collected from first shift workers, 36 from second shift workers, and 31 from third shift workers. The number of workers participating in the exposure assessment by job title was as follows: demold-27, insert-23, sort-7, trim-10, repair-8, bagging-7, mechanic-6, forklift operator-6, administrative (non-production, salaried participants)-4, and miscellaneous-2.

The mean (average) 2,4-TDI, 2,6-TDI, and total TDI exposures for all 104 workers were 0.86, 0.75, and 1.61 $\mu g/m^3$, respectively. The 2,4-TDI exposures ranged from 0.06 to 4.3 $\mu g/m^3$, the 2,6-TDI exposures ranged from 0.02 to 3.77 $\mu g/m^3$, and the total TDI exposures ranged from 0.08 to 8.07 $\mu g/m^3$. Eight of the 2,4-TDI concentrations and four of the 2,6-TDI concentrations were below the MDC. On average, the highest 2,4-, 2,6-, and total TDI exposures were found in the demold workers, followed by the insert workers, mechanics, and utility workers.

In addition to these data, five partial shift TDI exposure measurements were collected from workers at the facility. Two of these air samples were from the warehouse/maintenance area. An air sample was started on this worker at 7:00 a.m. Soon after starting the sample, a TDI tank truck arrived to offload bulk TDI, and a small leak was discovered in the transfer lines. Because a monitored worker responded to the leak, his first sample was removed at 8:25 a.m. and replaced with a new sample to capture the TDI exposure associated with the leak. The second sample ran until 11:13 a.m., at which point the leak repair task was finished and the worker left work for the day. The 2,4-TDI, 2,6-TDI, and total TDI concentrations measured on the first exposure sample were 1.06, 0.46, and 1.52 $\mu g/m^3$, respectively. The respective TDI concentrations for the second sample during the leak response and repair were 3.07, 1.06, and 4.14 $\mu g/m^3$, respectively.

The other partial shift samples were collected on two forklift operators and a lab technician, all of whom only worked a partial shift on the day of the NIOSH exposure assessment. The elapsed sample

time and exposure concentrations for these workers were as follows:

•Forklift operator - elapsed sample time of 154 minutes
2,4-TDI exposure of 0.18 μg/m^3
2,6-TDI exposure of 0.07 μg/m^3
Total TDI exposure of 0.25 μg/m^3

•Forklift operator - elapsed sample time of 343 minutes
2,4-TDI exposure of 0.13 μg/m^3
2,6-TDI exposure of 0.11 μg/m^3
Total TDI exposure of 0.24 μg/m^3

•Lab technician - elapsed sample time of 240 minutes
2,4-TDI exposure of 0.11 μg/m^3
2,6-TDI exposure of 0.05 μg/m^3
Total TDI exposure of 0.16 μg/m^3

In addition to the exposure assessment, nine full-shift area air samples were collected to characterize TDI concentrations in various areas or associated with specific processes or equipment. The data from these samples are shown in Table 14. Two area air samples were collected in the lobby area to determine background TDI concentrations in the administrative areas. In addition, 3 area air samples were collected in the QA lab after some employees requested TDI monitoring in that room.

Four process air samples were collected to identify potentially high exposure areas and to evaluate the effectiveness of existing engineering controls. Total TDI concentrations measured above the two B-line oven passive vents (19.4 and 66.6 μg/m^3) were generally one to two orders of magnitude greater than the personal TDI exposures. The total TDI concentration in the air sample collected above the door to B-line vacuum vessel #1 (1.74 μg/m^3) was not higher than personal exposures for workers in that area. Finally, an air sample collected where molds exit the A-line oven detected a total TDI concentration of 6.20 μg/m^3, which was within the range of exposures reported for demold workers.

Due to a backlog in isocyanate sample analysis at the NIOSH laboratory, the TDI air samples for this survey were stored in a freezer for approximately three months. After this, the samples were processed and analyzed during a four week period in September and October 1999. For quality assurance purposes, several samples were re-analyzed at the end of this period. The 2,6-TDI and 2,4-TDI values from the re-analyses were found to be on average 12% and 14% lower, respectively, than those from the original analyses. There was therefore a concern that these differences may indicate a sample stability problem. To investigate this, all of the samples that had been re-analyzed in October 1999, plus several others from this sample set, were analyzed again in early December 1999. These TDI air samples are referred to as sequence 9191 samples. In addition, TDI air samples from a March 4, 1999 survey at Woodbridge Brodhead that were first analyzed in late March 1999 were re-analyzed in early December 1999 (sequence 9157 samples).

The re-analysis of the sequence 9191 samples in early December did not support a continuous sample stability problem. Plots of concentration of TDI as a function of sample storage time showed very little, if any, decline in concentration between the initial re-analyses in October and the final re-analyses in December. After nine months of storage, the 2,6-TDI and 2,4-TDI values in the sequence 9157 samples were found to be on average 24% and 16% lower, respectively, than the original analyses.

Only a limited amount of data is available from the dermal exposure assessment. Most of the Permea-Tec™ detectors failed within 1 hour of use, with the impregnated pads separating from the adhesive backing and falling to the floor. We tried positioning the pads at different locations of the palm or fingers, but the pads continued to separate from the backing. Three of the pads turned color, which indicated dermal exposure to

TDI. All three pads were located on the palms of demold workers, and were under the light-weight cotton gloves worn by these workers.
An end-of-shift urine sample was collected from some of the workers who participated in the TDI exposure assessment. The TDI exposure and urine TDA data for each worker are presented in Table 15, and summary results based on these data are in Table 16. A total of 80 workers from the TDI exposure assessment agreed to provide an end-of-shift urine sample. The job titles from these 80 workers were as follows: demold, insert, forklift operator, offline (includes all workers who list their job as sort, trim, repair, and bagging), mechanic, and administrative. Among the 80 participants, the mean 2,4-TDI exposure was 0.79 $\mu g/m^3$ (range from 0.06 to 4.30 $\mu g/m^3$), the mean 2,6-TDI exposure was 0.68 $\mu g/m^3$ (0.02 to 3.77 $\mu g/m^3$), and the mean total TDI exposure was 1.47 $\mu g/m^3$ (0.08 to 8.07 $\mu g/m^3$). Seven of the 80 2,4-TDI exposure measurements, and three of the 2,6-TDI measurements were below the MDC. The highest TDI exposures were among the demold workers, followed by workers in the insert and mechanic job titles.

Urine TDA levels were expressed as uncorrected urine TDA levels, and as creatinine-corrected urine TDA levels. The corresponding urine TDA levels for the 80 participants were as follows: mean 2,4-TDA level - 0.83 µg/L (range 0.20 to 9.70 µg/L), mean 2,6-TDA level - 1.46 µg/L (0.25 to 7.30 µg/L), and mean total TDA level - 2.29 µg/L (0.45 to 17.0 µg/L). Of the 80 urine samples, 29 had 2,4-TDA levels below the MDC, and 24 had 2,6-TDA levels below the MDC. In 21 of these urine samples, both the 2,4- and 2,6-TDA measurements were below the respective MDCs. The highest uncorrected total TDA levels were among insert workers, followed by workers in the demold, offline, and mechanic job titles.

Mean urine TDA levels determined using the creatinine volume correction can also be found in Table 16. The mean creatinine-corrected urine TDA levels are as follows: mean 2,4-TDA level - 0.54 µg/g-Cr (range 0.07 to 2.88 µg/g-Cr), mean 2,6-TDA level - 0.99 µg/g-Cr (0.10 to 4.69 µg/g-Cr), and a mean total TDA level - 1.53 µg/g-Cr (0.18 to 6.02 µg/g-Cr). The highest total TDA levels determined using the creatinine correction were in the demold workers, followed by insert workers, offline workers, and the mechanics.

No correlation was found between 2,4-TDI exposure and corrected urinary 2,4-TDA (r=0.18, p=0.1), but a statistically significant correlation was found between 2,4-TDI exposure and the creatinine-corrected urine 2,4-TDA levels (r=0.23, p=0.04). Significant correlations were found between 2,6-TDI exposure and both uncorrected urine TDA levels (r=0.37, p=0.0007) and creatinine-corrected urine TDA levels (r=0.41, p=0.0002). In addition, statistically significant correlations were also found between total TDI exposure and both uncorrected (r=0.30, p=0.007) and creatinine corrected (r=0.35, p=0.002) urine TDA levels.

Formaldehyde

Formaldehyde sampling results are summarized in Tables 17 and Table 18. Samples were collected on May 22, to evaluate formaldehyde concentrations during mold cleaning; and on May 24, during a typical production shift. Four PBZ samples obtained on May 22, ranged from 0.01 to 0.06 ppm. Five area samples collected the same day measured from 0.01 to 0.02 ppm. Slightly higher formaldehyde concentrations were measured during a typical production shift. Five PBZ samples collected on May 24, ranged from 0.055 to 0.070 ppm; and the four area samples ranged from 0.034 to 0.065 ppm. Generally, the formaldehyde concentrations were low and did not exceed health-based exposure limits. Formaldehyde concentrations in most areas did, however, exceed the NIOSH REL of 0.016 ppm, which is based on analytical capabilities. Formaldehyde was consistently measured throughout the plant at levels greater than background (outside), indicating that a source of formaldehyde exists in the plant. No spatial differences were noted in formaldehyde

concentrations on either day. The source of the formaldehyde could not be identified.

Hydrocarbon Solvents

Sampling results for solvents in the wax dispersion application areas are summarized in Table 19. PBZ samples were collected on those persons having the greatest potential for exposure to the solvent. A-line demold workers, who rotate through the manual wax application booth, and B-line inserts, who work less than 20 feet down-line from the automated wax application booth, were sampled. All PBZ concentrations were below the exposure limit for naphtha solvents, with the highest concentration of 13 mg/m^3 measured on a person working in the A-line demold area. Two area samples were collected in what were thought *a priori* to be higher concentration areas. A full-shift area sample collected in the A-line wax application booth measured 190 mg/m^3 of naphtha solvent vapors. A sample collected on the B-line at the curtain separating the wax application booth from the insert area measured naphtha solvent vapors at a concentration of 11 mg/m^3.

These sampling results indicate that exposure to low levels of organic solvent are occurring in the A-line demold and B-line insert areas. Exposure concentrations were well below current exposure limits, and adverse health effects would not be expected at these levels. Though solvent vapor concentrations in the A-line wax application booth were below the applicable exposure limit, the measured concentration was considerably higher than other area and PBZ concentrations. Because persons applying the wax used a supplied air hood, and samples were collected under the hood, personal exposures should approximate the values reported for A-line demold/wax applicators. The measured concentrations demonstrate the effectiveness of wearing the supplied air hood to reduce the inhalation exposures of persons working in the wax application booth.

DMAEE

Sampling results for DMAEE are summarized in Table 20. Analytical results from a number of the sorbent tubes indicated that the rear media bed had an amount greater than 10% of that found on the front media bed. These samples are marked in the data table. This condition indicates that there was sample breakthrough and potential loss of DMAEE. DMAEE was detected on all eight PBZ samples collected during typical operations. Exposure concentrations on one of the eight (13%) samples met or exceeded the ACGIH TLV of 0.33 mg/m^3 for DMAEE. The exposure in excess of the TLV was from a worker in the B-line demold area. As noted below, turbulence in the exhaust ventilation hood is reducing the control's effectiveness, and may have been a contributing factor resulting in these exposure values.

Ventilation Assessment

Persons working in the demold and insert areas use pedestal and overhead-mounted fans to provide additional airflow through their work areas. These workers indicated that the fans help to keep them cool when the plant is warm. Air flow patterns through the ventilation hood were evaluated with smoke tubes at several work locations with the supplemental fans on, and again with the fans turned off. In general, air moved from the work area into the hood whether the supplemental fans were on or off. With the fans on, however, air flow into the hood was turbulent, allowing some smoke to escape from the hood into the immediate work area. Air flow was laminar (streamline), and no smoke escaped the hood when the fans were turned off. This indicates that the turbulence generated by supplemental fans operating in close proximity to the ventilation hood is sufficient to overcome the capture of the hood. Accordingly, some portion

of the chemicals emitted from the process escape the ventilation hood.

Capture velocity measurements were obtained across the hood face in the A- and B-line demold areas and in the B-line insert area, and also at the pour heads. Extreme air flows generated by downdraft ventilation above the A-line insert area precluded us from obtaining meaningful capture velocity data at this location. The average air velocity across the hood face in the respective work areas was: A-line demold, 119 ft/min; B-line demold, 70 ft/min; B-line insert, 75 ft/min; A-line pourhead, 75 ft/min; B-line pourhead, 100 ft/min. Capture velocities for processes such as conveyor belts, where contaminants are emitted into moderately still air, should be maintained at a minimum of 100 ft/min.[67]

DISCUSSION AND CONCLUSIONS

Medical

The prevalence of asthma among the study participants was 22%, which is higher than in the general population, which is estimated to range from 5-10%.[68] While the prevalence seems high, our findings should be interpreted cautiously. If those who participated in the study were an unbiased subset of the worker population, then these findings would reflect the true prevalence. However, if those who participated in this study were not representative of the workforce, then the true prevalence is unknown. If workers with respiratory symptoms decided to participate while those without such symptoms abstained from the study, then the prevalence would be inflated and suggest an increased prevalence when no such condition exists. Conversely, our estimate of the occurrence of asthma may be low if workers who develop asthma are more likely to stop working at Woodbridge. In addition, the case definitions used in this study, while consistent with those used elsewhere in the epidemiologic literature, may not be specific enough to differentiate asthma from other respiratory conditions. Thus, some of the people who have been identified with asthma for epidemiologic purposes, may not actually have asthma. Because the study participation rate was low, it is difficult to say with any certainty that the medical findings among those who participated in the study accurately reflect the disease prevalence of those who did not participate.

After we identified an apparently higher rate of asthma in this work population, we examined whether asthma was related to TDI exposure. In all situations, those who had a history of exposure to TDI had a higher prevalence of asthma. Although these associations were not statistically significant, they are internally consistent, and the point estimates for the prevalence ratios are all elevated. This is consistent with published studies that show that persons with exposure to TDI are at increased risk for asthma.[69,70,71] Evaluation of other chemicals currently in use at the plant did not identify exposures to any other asthmagenic compounds which might explain the increased prevalence of asthma at this facility.

The isocyanate-based skin patch testing showed no evidence of skin hypersensitivity to TDI, or to the other isocyanate compounds tested. The use of various waxes and adhesives in the mold-cleaning and foam repair areas are other potential causes of skin problems reported by employees, but these materials were not evaluated as part of the study.

The immunologic results obtained are consistent with results from other studies of TDI-exposed workers. Where TDI-specific IgE antibodies were measured, the prevalence ranged from 0% in some studies to less than 20% in others, although in one study it was 80%.[72] Similarly, the prevalence of demonstrable IgG antibody in studies of TDI-exposed workers has been shown to be as low as

0% in some studies, in contrast with an increased prevalence associated with exposure to other classes of diisocyanates, namely HDI and MDI.[73] Thus, there may be other, non-immunologically mediated mechanisms for TDI-related asthma that have not yet been characterized and that may play a role in the pathogenesis of this type of asthma.

Overall, the results of this study cannot determine conclusively whether there is an increased prevalence of asthma in this workforce and whether TDI exposure is responsible for the apparently increased prevalence we identified. The equivocal results, while multifaceted, may be chiefly the result of the low participation rate. Additionally, the lack of conclusive findings may be related to the fact that area and PBZ sampling levels of TDI measured at the Woodbridge Brodhead facility were low, and did not exceed the ACGIH TLV for TDI. This factor may explain, in part, the inconclusive nature of the study results.

Evaluation of Cancers

Cancer is a group of different diseases that share one common feature: the uncontrolled growth and spread of abnormal cells. Each different type of cancer may have its own set of causes. Cancer is common in the United States. One in two men and one in three women will develop some type of cancer in their lifetime. One out of every four deaths in the United States is from cancer. Among adults, cancer is more frequent among men than women, and it is more frequent with increasing age. Many factors play a role in the development of cancer. The importance of these factors is different for different types of cancer. Most cancers are caused by a combination of several factors. Some of these factors include: (a) personal characteristics such as age, sex, and race; (b) a family history of cancer; (c) diet; (d) personal habits such as cigarette smoking and alcohol consumption; (e) the presence of certain medical conditions; (f) exposure to cancer-causing agents in the environment; and (g) exposure to cancer-causing agents in the workplace. In many cases, these factors may act together or in sequence to cause cancer. Although specific causes of some types of cancer are known, many cancers have causes which are as yet unknown.

Cancers often appear to occur in clusters, which scientists define as an unusual concentration of cancer cases in a defined area or time period. A cluster also occurs when cancers are found among workers of a different age or sex group than is usual. These cases of cancer may have a common cause or may be the coincidental occurrence of unrelated causes. The number of cases may seem high, particularly among the small group of people who have something in common with the cases, such as working in the same building. In many workplaces the number of cases is small. This makes it difficult to determine whether the cases have a common cause, especially when there are no apparent cancer-causing exposures.

When cancer in a workplace is described, it is important to learn whether the type of cancer is a primary cancer or a metastasis (spread of the primary cancer into other organs). Usually, only primary cancers are used to investigate a cancer cluster. To assess whether the cancers among employees could be related to occupational exposures, we consider the number of cancer cases, the types of cancer, the likelihood of exposure to potential cancer-causing agents, and the timing of the diagnosis of cancer in relationship to the exposure (otherwise referred to as the latency period). A latency period is technically defined as the time between first exposure to a cancer-causing agent and clinical recognition of the disease. Latency periods vary by cancer type, but are usually 15 to 20 years. In some instances, the latency period may be shorter, but it is rarely less than 10 years. Given the insufficient latency period for three of the six cancers, the variety of cancer types reported, and dissimilar exposure potentials among the

individuals identified, a common-source exposure at Woodbridge Brodhead is unlikely to be the cause for these cancers.

The information which was provided regarding the chemicals to which Woodbridge production employees may be exposed on a regular basis include TDI, formaldehyde, and various hydrocarbon solvents (naphthas). Findings from a previous NIOSH HHE conducted at the Brodhead facility in May 1981 (HETA 81-128-1107, Janesville Products, Brodhead, Wisconsin) also indicated that MDI and methylene chloride (dichloromethane) were being used at that time for the foam manufacturing process.[74] Methylene chloride is listed by the United States National Toxicology Program as a suspected carcinogen, and is classified by the IARC as a Group B chemical (i.e., an agent that is possibly carcinogenic in humans).[75] Although a limited number of animal studies have demonstrated that commercial-grade TDI is carcinogenic in both rats and mice, no current epidemiologic data exist which link TDI exposure to elevated cancer rates in exposed workers.[43] Consequently, TDI has been classified as a potential occupational carcinogen by NIOSH. With this in mind, NIOSH recommends that occupational exposures to TDI be reduced to the LFC.[45] Based on the results of both animal studies and epidemiologic studies in humans, NIOSH has identified formaldehyde as a suspected human carcinogen, and also recommends that exposures to this chemical be reduced to the LFC.[53] Hydrocarbon solvents (such as petroleum naphtha) appear to have weak skin cancer causing potential in laboratory mice. IARC has determined that there is only limited evidence implicating petroleum naphtha as a carcinogen in animals, and insufficient evidence associating exposure to petroleum naphtha and the development of cancer in humans.[63]

The occurrence of a small number of cases of different types of cancer over a period of years, as is the case at the Woodbridge Brodhead plant, does not necessarily suggest that the cancers are due to exposure to a specific cancer-causing agent in the workplace, since most cancer-causing substances are known to cause only one or two different types of cancer. A common occupational exposure is more likely to be involved when several cases of the same type of cancer occur, and it is more readily documented when the cancer type is not common in the general population (a good example would be the occurrence of mesothelioma in shipyard workers who have been exposed to asbestos over a period of decades).

Also, given the size and age distribution of the Woodbridge workforce, neither the number of cases nor types of cancers appears to be unusual. In addition, the 15-year latency period required for most occupational cancers was not satisfied in all six cases. Given this information, it is unlikely that the cancers which have been reported among this population of workers, as a group, are the result of employment at Woodbridge Brodhead.

Industrial Hygiene

The TDI air sampling data indicates that the workers at Woodbridge are exposed to low to trace levels of 2,4-TDI, 2,6-TDI, and total TDI. The highest total TDI exposure concentration measured in this study was 8.07 $\mu g/m^3$ (in a demold worker), which is less than one-fifth of the ACGIH TLV of 36 $\mu g/m^3$. The average total TDI exposure concentration for this data set is 1.61 $\mu g/m^3$, which is 22 times below the TLV. Also, TDI was not detected in a few of the air samples, mostly from workers who did not work on the A- or B-lines. The NIOSH investigators believe the above TDI exposures do not constitute a significant carcinogenic risk to workers at Woodbridge.

In addition, the TDI concentrations found in the lobby and QA lab were also low to trace levels, and indicate that the TDI concentrations in these

areas do not pose a health hazard to workers in those areas. The TDI concentration data taken near the B-line passive vents indicate that the vents are a source of contaminant emanation into the plant.

The NIOSH laboratory does not have a good explanation for the differences in TDI values for air samples initially run in September and October 1999 versus the values found when those samples were re-analyzed between late October and early December 1999. The differences are fairly small, but almost all samples that were re-analyzed gave lower values than in the original analysis. The results of the two re-analysis investigations do not support the hypothesis that the apparent analyte loss represented a continuous problem over the four month storage period preceding analysis. Thus, the NIOSH investigators believe that the TDI exposure data in this report are reasonable estimates of the TDI exposures occurring at Woodbridge.

The failure of the Permea-Tec™ detectors in this study was unfortunate, and was probably related to the detector's inability to withstand a high degree of hand and finger activity over an extended period of time. The fact that three detectors from demold workers were found to be positive may indicate that some residual isocyanate groups are present when the foam exits the molds. We were unable to determine how long these groups are available on the surface of the foam, or whether this exposure poses a health risk to the workers. Further dermal exposure assessment studies should be conducted to determine the extent of this exposure. In the meantime, it may be prudent to provide the workers in the demold and offline areas with appropriate dermal protection for TDI.

Finally, urine TDA levels were measured in many of the TDI-exposed workers. In most cases, statistically significant correlations were found between TDI exposure and urine TDA levels. This suggests that workers with urinary TDA levels were probably exposed to TDI during the work shift, and the level of TDA excretion may be indicative of the worker's airborne (inhalation) TDI exposure over this work shift. It should be emphasized that urine TDA levels only reflect recent TDI exposure, and do not indicate whether a worker has been over- or under-exposed, nor suggest that a worker is at risk for developing TDI-related symptoms or disease. Also, the fact that only a few workers had TDI exposures below the respective MDCs, and several workers had urine TDA levels below the TDA method's MDCs, probably reflects the fact that the TDI air sampling and analysis method was more sensitive than the urine TDA method.

The only case where a correlation was not found involved the test of a relationship between 2,4-TDI exposure and uncorrected 2,4-urine TDA levels. Conversely, a significant correlation was found between 2,4-TDI exposure and the creatinine-corrected 2,4-TDA data. It is interesting to note that the strength of correlation (as indicated by the r-values) was greater for the relationships between TDI exposure metrics and creatinine-corrected urine TDA levels when compared to the relationships between the TDI exposure metrics and uncorrected urine TDA data. This likely indicates that when collecting spot urine samples, creatinine-corrected TDA measurements are better indicators (when compared to uncorrected TDA data) of recent TDI exposure.

To summarize, the exposure assessment portion of this study documented that the workers at Woodbridge are exposed to low levels of TDI. Urine TDA levels were found among the TDI-exposed workers, and these levels reflect recent TDI exposure. Using the creatinine correction to express urine TDA data appears to be more appropriate when using these data as biological markers for recent TDI exposure. Finally, a small amount of qualitative data indicate that demold

workers may have dermal isocyanate exposures. Further studies should be conducted to better document this possible exposure.

Formaldehyde was measured consistently throughout the plant at levels greater than background, indicating that a source of formaldehyde exists in the plant. No spatial differences were noted in formaldehyde concentrations on either day. This would indicate that the formaldehyde is not emanating from a point source (e.g., a pour head). Rather, it is more likely that the source is more widespread (e.g., slow off-gassing from the foam). Although formaldehyde is a known air contaminant associated with the production of formaldehyde-urea foams, no previous industrial hygiene data or rationale could be cited to indicate that formaldehyde should be evolving from polyurethane foam production processes.[76] The source of the formaldehyde at the Brodhead plant is unknown at this time.

Hydrocarbon solvent exposures from the wax dispersion process are controlled to below current exposure limits. Solvent vapor concentrations in the A-line manual wax application booth exceeded half of the REL. Current methods to control solvent exposure include exhaust ventilation, worker rotation, and respiratory protection for workers applying the wax dispersion.

Employee exposures to DMAEE were below the current TLV for all sampled employees except those working in the A-line demold area. The two samples collected on workers in this area were 100 % and 124 percent of the TLV, respectively. A possible contributing factor for this exposure includes turbulence in the local exhaust ventilation system due to the use of pedestal fans in close proximity to the hood.

NIOSH investigators evaluated the exhaust ventilation systems servicing the A- and B-lines. Measurements of the airflow across the face of the ventilation systems in several different work areas revealed laminar air flow of 70 to 119 ft/min across the face in most A- and B-line work areas when supplemental fans are not in use. When the pedestal and overhead fans are in use, a turbulence is created in the exhaust hood which limits the hood's efficiency in containing gasses emitted by the process. Overcoming these air-disturbing effects would require higher air flow rates through the hood.

RECOMMENDATIONS

1. It is important to eliminate sources of external air motion at the A- and B-lines to achieve effective emissions control without the need for excessive air flow.[67] To obtain greater capture and containment of air contaminants, Woodbridge and the employees should move supplemental fans further away from the production line. Fans should be moved to a distance from the line that limits the in-hood turbulence, and permits laminar flow of air from the work area into the hood. Improvements in airflow can be observed qualitatively using smoke tubes. Capture velocities should be re-evaluated and maintained at a minimum of 100 ft/min in all work areas.

2. The current configuration of the B-line ventilation includes one passive exhaust vent above the cure oven. This vent exhausts in the space between the top of the oven and the ceiling, allowing for heat and air contaminants to re-circulate into the work environment. Air concentrations of contaminants measured during this survey were slightly higher in the space above the passive exhaust vent. However, concentrations were below current exposure limits. Extending the B-line passive exhaust vent to the roof so that air contaminants off-gassed in the cure oven will be exhausted outside of the plant would reduce concentrations of air contaminants in the space above the cure ovens.

3. NIOSH recommends that employers conduct industrial hygiene surveys on all workers potentially exposed to isocyanate-containing compounds. These surveys should be conducted on an annual basis, or whenever there are changes in the process or engineering controls. Samples should be collected to characterize each employee's exposure, and to characterize isocyanate emissions from a given process, operation, machine, etc. These surveys should encompass both routine (e.g., normal operations and scheduled maintenance) and non-routine (e.g., repair activities associated with breakdowns or malfunction) work activities. Task-oriented exposure assessments should be used to determine the isocyanate exposure levels associated with specific tasks within an operation or shift.

4. Formaldehyde sampling results suggest that a source of formaldehyde generation exists inside the plant. Although only low concentrations of formaldehyde were measured, Woodbridge should continue efforts to identify and control the source.

5. The limited amount of qualitative dermal exposure data suggests that workers in the demold area handling freshly-cured foam may be receiving dermal isocyanate exposures. Woodbridge should provide these workers with gloves that are impervious to isocyanate-containing compounds. The gloves should be made of a permeation-resistant material, such as nitrile rubber, butyl rubber, neoprene, PVC, or flexible laminates (e.g., 4H™ [PE/EVAL] and Silver Shield™).

6. Woodbridge management should continue developing detailed written health and safety programs and to instruct all employees in the hazards associated with the chemicals used in the facility and the proper usage of personal protective equipment. Use of respirators must be in adherence with the Respiratory Protection Standard (29 CFR 1910.134).

7. Eating, drinking and smoking should continue to be prohibited in all work areas. These activities should be restricted to designated break areas which are separate from the work areas. If smoking is permitted, it should be restricted to dedicated rooms that have no other common purpose, and have air exhausted directly outdoors. Workers who smoke should be counseled on how smoking may exacerbate the adverse effects of respiratory hazards, such as TDI and the other diisocyanates.

8. Appropriate personal protective equipment should be made available to all Woodbridge employees who work directly with TDI-containing materials.

9. Each employee who has a potential for exposure to TDI or the other diisocyanates should receive a thorough preplacement medical examination, which includes a history of exposure to diisocyanates, a smoking history, and a history of respiratory illnesses. The purpose of the history and physical examination is to detect any pre-existing conditions (such as asthma, or exposure to respiratory irritants, such as tobacco smoke) that might place the exposed employee at increased risk for developing diisocyanate-related asthma, and to establish a baseline for future health monitoring. The preplacement physical should include pulmonary function testing (including FEV1 and FVC), as well as a chest X-ray before beginning work in a plant which uses diisocyanates. During preplacement examinations, applicants or employees found to have medical conditions that could be directly or indirectly aggravated by exposure to diisocyanates should be counseled on their increased risk of sensitization, should they be exposed to these compounds. These individuals should be offered employment in diisocyanate exposure-free areas of the plant.

10. Each employee with occupational exposure to TDI or other diisocyanates should also receive a

physical examination and pulmonary function test on a yearly basis. Because of seasonal and diurnal variations in pulmonary function, that part of the periodic examination for each employee should be performed at about the same time each year and at the same time of the day. Records of medical examinations should be kept for at least 30 years after the employee's last exposure to diisocyanates

11. Employees presenting to the plant clinic with complaints of work-related respiratory symptoms that are consistent with asthma (i.e., shortness of breath, wheezing, or chest tightness) should be promptly referred for further evaluation by a physician who has experience in the diagnosis and treatment of occupational asthma. Those employees receiving a diagnosis of occupational asthma secondary to diisocyanate exposure should be given appropriate work accommodations in order to avoid further exposure to TDI. Employees transferring to another job for work-related medical reasons should retain wages, seniority, and other benefits to which they would have been entitled had they not been transferred.

12. Employees with other work-related symptoms suggestive of TDI exposure (e.g, mucous membrane or skin irritation) should be evaluated promptly by the occupational medicine provider.

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Table 1
Job Classification of Medical Study Participants
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Job Classification	Number of Participants	Number Eligible	Percent of Eligibles Participating	Percent of Participant Total
Salaried employees	17	52	33	15
Hourly line employees	87	209	42	76
Maintenance	3	10	30	3
Warehouse	5	16	31	4
Other	2	10	20	2

Table 2
Summary of Prevalence for Medical Outcomes
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Outcome	Number of Cases	n	Prevalence
Asthma (Questionnaire)	25	114	22%
Work Related Asthma (Questionnaire)	20	114	18%
Airway Hyper responsiveness (PEFR)	25	59	42%
Work Related Airway Hyper responsiveness (PEFR)	8	59	14%
Mucous Membrane Irritation (Questionnaire)	82	114	72%
Work Related Skin Symptoms (Questionnaire)	16	110	15%
TDI Specific IgG	2	100	2%
TDI Specific IgE	0	100	0%

Table 3
Relationship between Production Area (Production vs. Non Production) and Asthma
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Exposure Group	Prevalence of Asthma	Prevalence Rate Ratio (PRR)	95% Confidence Interval
Production Workers	23/88 (26%)	3.40	(0.92, 39.52)
Non Production Workers	2/26 (8%)	Referent	- - - -

Table 4
Relationship between Self Reported Exposure to TDI (Ever vs. Never) and Asthma
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Exposure Group	Prevalence of Asthma	Prevalence Rate Ratio (PRR)	95% Confidence Interval
Ever Worked with TDI	24/104 (23%)	2.31	(0.34, 123.20)
Never Worked with TDI	1/10 (10%)	Referent	- - - -

Table 5
Relationship between TDI Exposure Quartile and Asthma
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Quartile	TDI Concentration (range in $\mu g/m^3$)	# of Cases	n	Prevalence	PRR	95% CI
1	0.081-0.425	1	19	5%	Referent	- - - -
2	0.426-1.05	4	19	21%	4.00	(0.49, 32.57)
3	1.06-2.24	4	19	21%	4.00	(0.49, 32.57)
4	2.25-8.07	4	19	21%	4.00	(0.49, 32.57)

Table 6
Relationship between Production Area
(Production vs. Non Production) and Work Related Asthma
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Exposure Group	Prevalence of Work Related Asthma	Prevalence Rate Ratio (PRR)	95% Confidence Interval
Production Workers	18/88 (20%)	2.66	(0.65, 29.16)
Non Production Workers	2/26 (8%)	Referent	- - - -

Table 7
Relationship between Self Reported Exposure to TDI (Ever vs. Never) and Work Related Asthma
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Exposure Group	Prevalence of Work Related Asthma	Prevalence Rate Ratio (PRR)	95% Confidence Interval
Ever Worked with TDI	19/104 (18%)	1.83	(0.25, 92.75)
Never Worked with TDI	1/10 (10%)	Referent	- - - -

Table 8
Relationship between TDI Exposure Quartile and Work-related Asthma
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Quartile	TDI Concentration (range in $\mu g/m^3$)	# of Cases	n	Prevalence	PRR	95% CI
1	0.081-0.425	1	19	5%	Referent	- - - -
2	0.426-1.05	3	19	16%	3.00	(0.34, 26.33)
3	1.06-2.24	3	19	16%	3.00	(0.34, 26.33)
4	2.25-8.07	2	19	11%	2.00	(0.20, 20.24)

Table 9
Relationship between Production Area (Production vs. Non Production)
and Mucous Membrane Irritation Symptoms
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Exposure Group	Prevalence of Mucous Membrane Symptoms	Prevalence Rate Ratio (PRR)	95% Confidence Interval
Production Workers	69/88 (78%)	1.57	(1.30, 10.05)
Non Production Workers	13/26 (50%)	Referent	- - - -

Table 10
Relationship between Self Reported Exposure to TDI
(Ever vs. Never) and Mucous Membrane Irritation Symptoms
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Exposure Group	Prevalence of Mucous Membrane Symptoms	Prevalence Rate Ratio (PRR)	95% Confidence Interval
Ever Exposed to TDI	78/104 (75%)	1.88	(0.97, 23.08)
Never Exposed to TDI	4//10 (40%)	Referent	- - - -

Table 11
Relationship between Production Area (Production vs. Non Production Area)
and Work Related Skin Symptoms
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Exposure Group	Prevalence of Work Related Skin Symptoms	Prevalence Rate Ratio (PRR)	95% Confidence Interval
Production Workers	16/86 (19%)	∞ [1]	N/A (p=0.02)[2]
Non Production Workers	0/24 (0%)	Referent	- - - -

[1] The symbol "∞" indicates a PRR approaching infinity (i.e., undefined PRR with zero as the denominator)
[2] P-value calculated using chi-square analysis.

Table 12: Workers' Personal Breathing Zone Exposure Data for 2,4-TDI, 2,6-TDI and Total TDI
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

JOB/LOCATION	SAMPLE TIME	SAMPLE VOLUME[1]	2,4-TDI[2]	2,6-TDI[2]	TOTAL TDI[2]
Administrative	0753-1440	411.1	0.07*	0.04†	0.12
Administrative	0715-1442	447.0	0.27	0.15	0.42
Administrative	0647-1445	492.3	0.11†	0.11	0.22
Administrative	0651-1456	523.8	0.19†	0.12	0.31
Bagging/A	1459-2249	512.3	0.14†	0.14	0.28
Bagging/B	0743-1451	423.7	0.26†	0.18	0.44
Bagging/B	2250-0657	479.7	0.33	0.25	0.58
Bagging/Pallet Prep/A	0100-0657	385.6	0.49	0.39	0.88
Bagging/Repair/B	0654-1501	482.1	0.39	0.29	0.68
Bagging/Repair/B	0652-1454	527.8	0.28	0.23	0.51
Bagging/Trim/Sort/B	2305-0652	467.0	0.54	0.45	0.99
Clerk/Warehouse	1410-2221	505.7	0.06*	0.05†	0.10
Demold/A	0004-0638	401.9	1.14	1.02	2.16
Demold/A	0002-0640	409.9	1.46	1.29	2.76
Demold/A	0723-1450	415.7	1.37	1.27	2.65
Demold/A	0000-0640	416.0	1.56	1.37	2.93
Demold/A	2358-0639	421.1	1.95	1.64	3.59
Demold/A	0712-1451	449.8	1.56	1.49	3.05
Demold/A	0725-1450	469.5	1.51	1.47	2.98
Demold/A	1450-2246	478.3	1.94	2.09	4.04
Demold/A	1450-2246	483.1	0.79	0.75	1.53
Demold/A	1500-2246	487.0	1.27	1.15	2.42
Demold/A	0649-1450	488.2	0.59	0.53	1.13
Demold/A	0648-1450	489.2	0.67	0.72	1.39

Table 12 Continued: Workers' Personal Breathing Zone Exposure Data for 2,4-TDI, 2,6-TDI and Total TDI Woodbridge Corporation Brodhead, Wisconsin HETA 98-0011-2801

JOB/LOCATION	SAMPLE TIME	SAMPLE VOLUME[1]	2,4-TDI[2]	2,6-TDI[2]	TOTAL TDI[2]
Demold/A	0648-1450	508.5	1.91	2.16	4.07
Demold/A	1446-2246	508.8	1.12	1.10	2.22
Demold/B	0833-1441	371.7	4.30	3.77	8.07
Demold/B	2338-0633	427.5	1.66	1.57	3.23
Demold/B	2335-0633	430.5	1.81	1.60	3.41
Demold/B	0716-1441	431.7	0.88	0.90	1.78
Demold/B	2341-0635	436.8	2.13	1.76	3.89
Demold/B	1530-2239	439.7	1.18	1.07	2.25
Demold/B	1532-2241	448.3	0.62	0.62	1.25
Demold/B	0716-1441	451.7	0.89	0.89	1.77
Demold/B	1535-2241	453.7	1.30	1.23	2.53
Demold/B	0719-1440	458.6	0.92	0.87	1.79
Demold/B	0717-1440	478.4	0.94	0.84	1.78
Demold/B	2258-0633	482.3	1.82	1.49	3.32
Demold/B	1443-2239	492.7	0.95	1.18	2.13
Forklift Operator/A	1459-2245	466.0	0.06*	0.06†	0.12
Forklift Operator/B	2255-0659	479.2	0.42	0.33	0.75
Forklift Operator/B	1500-2250	491.2	0.09†	0.08	0.16
Forklift Operator/Warehouse	1504-2210	426.0	0.16†	0.12	0.27
Forklift Operator/Warehouse	1502-2246	459.6	0.06*	0.02*	0.09
Forklift Operator/Warehouse	1444-2246	501.3	0.06*	0.04†	0.10
Insert/A	0710-1450	439.3	0.66	0.50	1.16
Insert/A	0708-1450	457.8	0.61	0.55	1.16

Table 12 Continued: Workers' Personal Breathing Zone Exposure Data for 2,4-TDI, 2,6-TDI and Total TDI Woodbridge Corporation Brodhead, Wisconsin HETA 98-0011-2801

JOB/LOCATION	SAMPLE TIME	SAMPLE VOLUME[1]	2,4-TDI[2]	2,6-TDI[2]	TOTAL TDI[2]
Insert/A	2309-0642	459.8	1.52	1.22	2.74
Insert/A	0710-1450	462.3	0.69	0.56	1.25
Insert/A	2244-0642	480.4	1.60	1.19	2.79
Insert/A	1456-2250	507.2	0.63	0.51	1.14
Insert/A	1510-2251	516.3	0.77	0.58	1.36
Insert/A	1505-2258	527.4	0.17†	0.19	0.36
Insert/A	1445-2257	528.9	1.15	1.04	2.19
Insert/B	0036-0630	380.6	2.63	2.39	5.02
Insert/B	1450-2148	420.1	0.60	0.52	1.12
Insert/B	0713-1437	444.0	1.24	0.90	2.14
Insert/B	2325-0633	447.3	2.46	2.46	4.92
Insert/B	0806-1436	448.5	1.90	1.47	3.37
Insert/B	2300-0632	456.5	1.03	0.66	1.69
Insert/B	2329-0633	460.0	2.39	1.87	4.26
Insert/B	1442-2239	469.8	1.96	1.77	3.72
Insert/B	1529-2239	473.0	0.68	0.63	1.31
Insert/B	0704-1436	485.9	2.26	1.73	3.99
Insert/B	0711-1441	495.0	0.77	0.75	1.52
Insert/B	0706-1436	497.3	2.21	1.65	3.86
Insert/B	2250-0701	500.8	1.64	1.26	2.90
Insert/B	1445-2302	556.6	0.22	0.22	0.43
Mechanic/Maintenance	0045-0700	433.1	0.21†	0.14	0.35
Mechanic/Maintenance	0725-1435	445.1	3.37	2.47	5.84

Table 12 Continued: Workers' Personal Breathing Zone Exposure Data for 2,4-TDI, 2,6-TDI and Total TDI Woodbridge Corporation Brodhead, Wisconsin HETA 98-0011-2801

JOB/LOCATION	SAMPLE TIME	SAMPLE VOLUME[1]	2,4-TDI[2]	2,6-TDI[2]	TOTAL TDI[2]
Mechanic/Maintenance	2305-0645	471.5	0.85	0.51	1.36
Mechanic/Maintenance	0727-1435	475.1	0.72	0.53	1.24
Mechanic/Maintenance	0737-1440	478.0	0.06*	0.02*	0.08
Mechanic/Maintenance	1540-2252	481.7	0.06*	0.02*	0.08
Repair/A	2350-0648	418.0	0.72	0.60	1.32
Repair/A	2345-0639	455.4	0.31	0.21	0.51
Repair/A	1514-2249	487.2	0.20†	0.17	0.37
Repair/A	1515-2249	524.4	0.23	0.21	0.44
Repair/B	0714-1501	469.3	0.19†	0.13	0.32
Repair/B	1527-2244	472.0	0.16†	0.14	0.31
Repair/Bagging/A	2347-0642	439.9	0.41	0.36	0.77
Repair/Bagging/B	0021-0657	409.9	0.37	0.27	0.63
Sort/A	0827-1444	418.5	0.33	0.31	0.65
Sort/A	1549-2244	446.1	0.13†	0.19	0.31
Sort/A	1500-2249	485.4	0.16†	0.19	0.35
Sort/B	1510-2243	448.5	0.25†	0.27	0.51
Sort/B	0715-1454	475.1	0.19†	0.21	0.40
Sort/Trim/B	1458-2245	464.7	0.32	0.34	0.67
Sort/Trim/B	1500-2240	473.8	0.32	0.36	0.68
Technician/B	0755-1438	429.2	0.42	0.35	0.77
Trim/A	0025-0646	379.1	0.66	0.55	1.21
Trim/A	0020-0652	390.0	0.38	0.31	0.69
Trim/A	1526-2243	432.6	0.16†	0.16	0.31

Table 12 Continued: Workers' Personal Breathing Zone Exposure Data for 2,4-TDI, 2,6-TDI and Total TDI Woodbridge Corporation Brodhead, Wisconsin HETA 98-0011-2801

JOB/LOCATION	SAMPLE TIME	SAMPLE VOLUME[1]	2,4-TDI[2]	2,6-TDI[2]	TOTAL TDI[2]
Trim/A	2319-0652	453.0	0.66	0.51	1.17
Trim/A	1520-2243	454.1	0.20†	0.21	0.41
Trim/B	0728-1500	463.3	0.56	0.41	0.97
Trim/Repair/A	0824-1445	384.8	0.55	0.52	1.07
Trim/Repair/A	0825-1444	399.8	0.40	0.33	0.73
Trim/Repair/A	1510-2249	488.8	0.29	0.27	0.55
Trim/Sort/B	2305-0652	520.7	0.58	0.46	1.04
Utility/A	0000-0648	414.1	0.65	0.48	1.13
Utility/B	0715-1451	453.7	0.37	0.29	0.66
Utility/B	2303-0632	458.0	2.10	1.62	3.71
Utility/Warehouse	1433-2221	500.8	0.06*	0.02*	0.08
PARTIAL-SHIFT TDI EXPOSURE DATA					
Chemical Handler	0700-0825	90.5	1.06†	0.46	1.52
Chemical Handler	0825-1113	178.9	3.07	1.06	4.14
Forklift Operator/Warehouse	1502-1736	160.9	0.18*	0.07*	0.25
Forklift Operator/Warehouse	0910-1453	353.3	0.13†	0.11	0.24
Technician/QA Lab	1440-1841	260.4	0.11*	0.05*	0.16

[1] Sample volumes are in liters of air.
[2] These columns contain the worker's full-shift, breathing zone exposure concentrations in micrograms of analyte per cubic meter of air.
* Exposure value is an estimation, actual exposure was below the MDC.
† Exposure concentration is between the MDC and MQC.

Table 13: Summary Statistics for the TDI Full-Shift Exposure Data

Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

JOB CATEGORY	n[1]	MEAN TDI EXPOSURE[2] (RANGE OF EXPOSURES)			# EXPOSURES < MDC[3]	
		2,4-TDI	2,6-TDI	TOTAL TDI	2,4-TDI	2,6-TDI
ALL DATA	**104**	**0.86** (0.06-4.30)	**0.75** (0.02-3.77)	**1.61** (0.08-8.07)	**8**	**4**
DEMOLD	27	1.42 (0.59-4.30)	1.33 (0.53-3.77)	2.75 (1.13-8.07)	None	None
INSERT	23	1.30 (0.17-2.63)	1.07 (0.19-2.46)	2.37 (0.36-5.02)	None	None
SORT	7	0.24 (0.13-0.33)	0.27 (0.19-0.36)	0.51 (0.31-0.68)	None	None
TRIM	10	0.44 (0.16-0.66)	0.37 (0.16-0.55)	0.82 (0.31-1.21)	None	None
REPAIR	8	0.32 (0.16-0.72)	0.26 (0.13-0.60)	0.58 (0.31-1.32)	None	None
BAGGING	7	0.35 (0.14-0.54)	0.28 (0.14-0.45)	0.62 (0.28-0.99)	None	None
MECHANIC	6	0.89 (0.06-3.37)	0.62 (0.02-2.47)	1.49 (0.08-5.84)	2	2
UTILITY	4	0.80 (0.06-2.1)	0.60 (0.02-1.62)	1.40 (0.08-3.71)	1	1
FORKLIFT OPERATOR	6	0.14 (0.06-0.42)	0.11 (0.02-0.33)	0.25 (0.09-0.75)	3	1
ADMINISTRATIVE	4	0.16 (0.07-0.27)	0.11 (0.04-0.15)	0.27 (0.12-0.42)	1	None
MISCELLANEOUS	2	0.24 (0.06-0.42)	0.20 (0.05-0.35)	0.44 (0.10-0.77)	1	None
NIOSH REL		LFC[4]	LFC[4]	LFC[4]		
ACGIH TLV		36	36	36		

[1] n - sample size, number of exposure measurements in the given "job category."
[2] 2,4-, 2,6- and total TDI exposure data are in micrograms per cubic meter of air. The "range of exposures" (data in parentheses) are the minimum and maximum exposure measurements for each job category.
[3] These columns indicate the number of 2,4- and 2,6-TDI exposure measurements (in the given "job category") that were below the minimum detectable concentration (MDC).
[4] LFC - lowest feasible concentration.

Table 14: Area Air Sampling Data for TDI
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

SAMPLE LOCATION	SAMPLE	SAMPLE	2,4-TDI[2]	2,6-TDI[2]	TOTAL
Near oven in QA Lab	1505-2237	454.3	0.17	0.02	0.19
Front Lobby	0700-1440	469.2	0.06	0.02	0.09
Office near lobby	0704-1440	456.0	0.07	0.04	0.11
Bookshelf in QA Lab	1505-2237	456.5	0.14	0.06	0.20
At computer in QA Lab	1533-2237	426.1	0.16	0.04	0.20
A-line demold area	1510-2152	409.2	2.93	3.27	6.20
Above small B-line passive vent near pour head	1455-2150	435.8	27.5	39.0	66.6
B-line, vacuum vessel #1	0734-1452	449.0	0.76	0.98	1.74
Above grate for B-line oven passive exhaust	0738-1452	438.3	7.99	11.4	19.4

[1] Sample volumes are in liters of air.
[2] These columns contain the 2,4-, 2,6-, and total TDI concentrations in micrograms of analyte per cubic meter of air.

Table 15:Worker-specific TDI Exposure Data and Urine TDA Levels
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

JOB/LOCATION	TDI EXPOSURE DATA[1]			URINE TDA DATA[2] NO VOLUME CORRECTION			URINE TDA DATA[3] CREATININE VOLUME CORRECTION		
	2,4-TDI	2,6-TDI	TOTAL TDI	2,4-TDA	2,6-TDA	TOTAL TDA	2,4-TDA	2,6-TDA	TOTAL TDA
Administrative	0.27	0.15	0.42	0.20*	0.25*	0.45	0.11	0.14	0.25
Administrative	0.07*	0.04	0.12	0.20*	0.25*	0.45	0.21	0.27	0.48
Administrative	0.19	0.12	0.31	0.20*	0.25*	0.45	0.12	0.16	0.28
Administrative	0.11	0.11	0.22	1.00	1.20	2.20	0.56	0.68	1.24
Bagging/A	0.14	0.14	0.28	0.20*	1.00	1.20	0.10	0.51	0.61
Bagging/A	0.49	0.39	0.88	0.50	0.25*	0.75	0.31	0.16	0.47
Bagging/B	0.26	0.18	0.44	0.20*	0.25*	0.45	0.63	0.78	1.41
Bagging/B	0.67	0.72	1.39	0.20*	0.25*	0.45	0.18	0.23	0.41
Bagging/Repair/B	0.39	0.29	0.68	0.20*	0.25*	0.45	1.11	1.39	2.50
Bagging/Repair/B	0.28	0.23	0.51	0.20*	0.25*	0.45	0.32	0.40	0.72
Bagging/Trim/Sort/B	0.54	0.45	0.99	0.60	0.80	1.40	0.55	0.73	1.28
Clerk/Warehouse	0.06*	0.05	0.1	0.20*	0.25*	0.45	0.65	0.81	1.46
Demold/A	1.91	2.16	4.07	0.20*	0.25*	0.45	0.14	0.17	0.31
Demold/A	0.59	0.53	1.13	0.20*	0.25*	0.45	0.17	0.21	0.38
Demold/A	1.27	1.15	2.42	0.50	0.90	1.40	0.28	0.51	0.79
Demold/A	0.79	0.75	1.53	0.60	0.90	1.50	0.18	0.27	0.45
Demold/A	1.12	1.1	2.22	0.60	1.10	1.70	0.28	0.52	0.80
Demold/A	0.33	0.25	0.58	0.80	0.90	1.70	0.44	0.49	0.93
Demold/A	1.56	1.49	3.05	0.90	1.00	1.90	0.76	0.85	1.61

Table 15 Continued: Worker-specific TDI Exposure Data and Urine TDA Levels
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Demold/A	1.94	2.09	4.04	0.90	3.10	4.00	0.69	2.37	3.06
Demold/A	1.51	1.47	2.98	1.00	2.60	3.60	0.56	1.47	2.03
Demold/B	2.13	1.76	3.89	0.60	1.50	2.10	0.46	1.15	1.61
Demold/B	0.89	0.89	1.77	0.60	2.10	2.70	0.27	0.93	1.20
JOB/LOCATION	**TDI EXPOSURE DATA[1]**			**URINE TDA DATA[2] NO VOLUME CORRECTION**			**URINE TDA DATA[3] CREATININE VOLUME CORRECTION**		
	2,4-TDI	**2,6-TDI**	**TOTAL TDI**	**2,4-TDA**	**2,6-TDA**	**TOTAL TDA**	**2,4-TDA**	**2,6-TDA**	**TOTAL TDA**
Demold/B	0.88	0.9	1.78	0.70	2.70	3.40	0.31	1.21	1.52
Demold/B	1.18	1.07	2.25	0.70	2.80	3.50	0.81	3.26	4.07
Demold/B	1.82	1.49	3.32	0.80	2.50	3.30	0.51	1.59	2.10
Demold/B	4.3	3.77	8.07	1.70	6.00	7.70	1.33	4.69	6.02
Forklift Operator/A	0.06*	0.06	0.12	1.30	0.25*	1.55	1.78	0.34	2.12
Forklift Operator/B	0.72	0.6	1.32	0.20*	0.60	0.80	0.40	1.20	1.60
Forklift Operator/B	0.09	0.08	0.16	0.40	0.25*	0.65	0.21	0.13	0.34
Forklift Operator/Warehouse	0.06*	0.02*	0.09	0.20*	0.25*	0.45	0.08	0.10	0.18
Forklift Operator/Warehouse	0.16	0.12	0.27	0.20*	0.25*	0.45	0.13	0.17	0.30
Forklift Operator/Warehouse	0.13	0.11	0.24	0.20*	0.25*	0.45	0.57	0.71	1.28
Forklift Operator/Warehouse	0.06*	0.04	0.1	0.20*	0.25*	0.45	0.15	0.19	0.34
Insert/A	0.61	0.55	1.16	0.20*	0.25*	0.45	0.23	0.29	0.52
Insert/A	1.6	1.19	2.79	0.20*	0.70	0.90	0.74	2.59	3.33
Insert/A	1.52	1.22	2.74	0.50	1.30	1.80	0.18	0.47	0.65

Table 15 Continued: Worker-specific TDI Exposure Data and Urine TDA Levels
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Insert/A	0.63	0.51	1.14	0.70	0.80	1.50	0.33	0.38	0.71
Insert/A	0.17	0.19	0.36	0.70	1.10	1.80	0.20	0.31	0.51
Insert/A	0.66	0.5	1.16	1.00	1.00	2.00	0.48	0.48	0.96
Insert/A	1.15	1.04	2.19	1.10	1.70	2.80	0.34	0.52	0.86
Insert/A	0.77	0.58	1.36	9.70	7.30	17.00	2.88	2.17	5.05
Insert/B	1.03	0.66	1.69	0.20*	0.25*	0.45	0.11	0.13	0.24
Insert/B	1.64	1.26	2.9	0.20*	0.25*	0.45	0.49	0.61	1.10
Insert/B	1.9	1.47	3.37	0.20*	0.70	0.90	0.63	2.19	2.82
Insert/B	0.22	0.22	0.43	0.80	0.60	1.40	0.31	0.24	0.55
JOB/LOCATION	**TDI EXPOSURE DATA[1]**			**URINE TDA DATA[2] NO VOLUME CORRECTION**			**URINE TDA DATA[3] CREATININE VOLUME CORRECTION**		
	2,4-TDI	**2,6-TDI**	**TOTAL TDI**	**2,4-TDA**	**2,6-TDA**	**TOTAL TDA**	**2,4-TDA**	**2,6-TDA**	**TOTAL TDA**
Insert/B	0.68	0.63	1.31	1.10	6.90	8.00	0.40	2.54	2.94
Insert/B	0.23	0.21	0.44	1.30	1.40	2.70	0.88	0.95	1.83
Insert/B	0.6	0.52	1.12	1.30	2.00	3.30	0.78	1.20	1.98
Insert/B	2.39	1.87	4.26	1.70	3.20	4.90	1.00	1.88	2.88
Mechanic/Maintenance	0.06*	0.02*	0.08	0.20*	0.25*	0.45	0.12	0.14	0.26
Mechanic/Maintenance	0.85	0.51	1.36	0.70	1.00	1.70	0.44	0.63	1.07
Mechanic/Maintenance	3.37	2.47	5.84	2.00	1.30	3.30	1.48	0.96	2.44
Mechanic/Maintenance	0.72	0.53	1.24	3.30	1.80	5.10	1.29	0.70	1.99
Repair/A	1.24	0.9	2.14	0.20*	0.70	0.90	0.29	1.00	1.29

Table 15 Continued: Worker-specific TDI Exposure Data and Urine TDA Levels
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

Repair/A	0.42	0.33	0.75	0.40	0.50	0.90	0.26	0.32	0.58
Repair/B	0.19	0.13	0.32	0.60	1.00	1.60	0.61	1.01	1.62
Repair/B	0.16	0.14	0.31	2.00	4.90	6.90	1.20	2.93	4.13
Repair/Bagging/B	0.37	0.27	0.63	0.20*	4.00	4.20	0.18	3.60	3.78
Sort/A	0.16	0.19	0.35	0.50	1.00	1.50	0.30	0.60	0.90
Sort/A	0.13	0.19	0.31	0.70	1.50	2.20	0.38	0.82	1.20
Sort/B	0.19	0.21	0.4	1.50	3.40	4.90	1.09	2.48	3.57
Sort/B	0.25	0.27	0.51	3.90	5.20	9.10	1.18	1.58	2.76
Sort/Trim/B	0.32	0.36	0.68	2.00	6.60	8.60	1.12	3.69	4.81
Technician	0.11	0.05	0.16	0.20*	0.25*	0.45	0.18	0.22	0.40
Technician	0.42	0.35	0.77	0.60	0.60	1.20	0.24	0.24	0.48
Trim/A	0.66	0.51	1.17	0.20*	0.90	1.10	0.11	0.49	0.60
Trim/A	0.2	0.21	0.41	0.20*	1.10	1.30	0.07	0.37	0.44
Trim/A	0.16	0.16	0.31	0.50	0.90	1.40	0.25	0.45	0.70
JOB/LOCATION	**TDI EXPOSURE DATA[1]**			**URINE TDA DATA[2] NO VOLUME CORRECTION**			**URINE TDA DATA[3] CREATININE VOLUME CORRECTION**		
	2,4-TDI	**2,6-TDI**	**TOTAL TDI**	**2,4-TDA**	**2,6-TDA**	**TOTAL TDA**	**2,4-TDA**	**2,6-TDA**	**TOTAL TDA**
Trim/Repair/A	0.29	0.27	0.55	0.90	2.50	3.40	0.42	1.16	1.58
Trim/Sort/B	0.58	0.46	1.04	0.50	1.40	1.90	0.30	0.84	1.14
Utility/A	0.65	0.48	1.13	0.20*	0.25*	0.45	0.51	0.64	1.15
Utility/B	2.1	1.62	3.71	0.20*	0.25*	0.45	0.54	0.68	1.22

Table 15 Continued: Worker-specific TDI Exposure Data and Urine TDA Levels
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

JOB/LOCATION	TDI EXPOSURE DATA[1]			URINE TDA DATA[2] NO VOLUME CORRECTION			URINE TDA DATA[3] CREATININE VOLUME CORRECTION		
Utility/B	0.37	0.29	0.66	0.80	1.20	2.00	1.16	1.74	2.90
Utility/Warehouse	0.06*	0.02*	0.08	1.60	1.70	3.30	1.34	1.43	2.77

[1] TDI exposure data are in micrograms of analyte per cubic meter of air.
[2] Urine TDA data are in micrograms per liter. No volume correction method has been applied to the data.
[3] Urine TDA data expressed using the creatinine volume correction method. Data are in micrograms per gram of creatinine.
* Indicates that the TDI exposure concentration or urine TDA concentration was below the MDC or LOD, respectively.

Table 16: Summary Statistics for the TDI and TDA Comparison Data
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801

JOB CATEGORY	n[1]	MEAN TDI EXPOSURE[2] (RANGE OF EXPOSURES)			MEAN URINE TDA, NO VOLUME CORRECTION[3] (RANGE OF TDA LEVELS)			MEAN URINE TDA, CREATININE CORRECTION[4] (RANGE OF TDA LEVELS)		
		2,4-TDI	2,6-TDI	TOTAL TDI	2,4-TDA	2,6-TDA	TOTAL TDA	2,4-TDA	2,6-TDA	TOTAL TDA
ALL DATA	**80**	**0.79** (0.06-4.30)	**0.68** (0.02-3.77)	**1.47** (0.08-8.07)	**0.83** (0.20-9.70)	**1.46** (0.25-7.30)	**2.29** (0.45-17.0)	**0.54** (0.07-2.88)	**0.99** (0.10-4.69)	**1.53** (0.18-6.02)
DEMOLD	16	1.45 (0.33-4.30)	1.36 (0.25-3.77)	2.81 (0.58-8.07)	0.72 (0.20-1.7)	1.89 (0.25-6.0)	2.61 (0.45-7.7)	0.48 (0.14-1.33)	1.29 (0.17-4.69)	1.77 (0.31-6.02)
INSERT	15	1.19 (0.17-2.63)	0.98 (0.19-2.39)	2.17 (0.36-5.02)	1.37 (0.20-9.7)	1.63 (0.25-7.3)	3.01 (0.45-17.0)	0.69 (0.11-2.88)	1.05 (0.13-2.59)	1.74 (0.24-5.05)
FORKLIFT OPERATOR	7	0.18 (0.06-0.72)	0.15 (0.02-0.60)	0.33 (0.09-1.32)	0.39 (0.20-1.3)	0.30 (0.25-0.6)	0.69 (0.45-1.55)	0.47 (0.08-178)	0.41 (0.10-1.20)	0.88 (0.18-2.12)
POST-CRUSHER	31	0.48 (0.06-2.10)	0.40 (0.02-1.62)	0.89 (0.08-3.71)	0.73 (0.20-3.9)	1.71 (0.25-6.9)	2.44 (0.45-9.1)	0.53 (0.07-1.34)	1.12 (0.16-3.69)	1.65 (0.41-4.81)
MECHANIC	5	1.08 (0.06-3.37)	0.78 (0.02-2.47)	1.86 (0.08-5.84)	1.36 (0.20-3.3)	0.99 (0.25-1.8)	2.35 (0.45-5.1)	0.71 (0.12-1.48)	0.53 (0.14-0.96)	1.25 (0.26-2.44)
ADMINISTRATIVE	6	0.14 (0.06-0.27)	0.09 (0.04-0.15)	0.23 (0.10-0.42)	0.33 (0.20-1.0)	0.41 (0.25-1.2)	0.74 (0.45-2.2)	0.31 (0.11-0.65)	0.38 (0.14-0.81)	0.69 (0.25-1.46)

[1] n - sample size, number of exposure measurements in the given "job category."
[2] 2,4-, 2,6-, and total TDI exposure data are in micrograms per cubic meter of air. The "range of exposures" (data in parentheses) are the minimum and maximum exposure measurements for each job category.
[3] Urine TDA levels are in micrograms per liter of urine. No volume correction was applied to these data. The "range of exposures" (data in parentheses) are the minimum and maximum exposure measurements for each job category.
[4] The creatinine volume correction was applied to these data. The urine TDA levels are in micrograms per gram of creatinine. The "range of exposures" (data in parentheses) are the minimum and maximum exposure measurements for each job category.

Table 17
Summary of Formaldehyde Sampling Typical Operations
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801
May 24, 1999

Personal Breathing Zone Samples			
Job Title	Sampling Time	Sample Volume[1]	Formaldehyde Concentration[2]
Line B Demold	0733-1454	44.1	.055
Line A Demold	0758-1451	41.3	.061
Line B Insert	0718-1441	44.3	.057
Line A Demold	0805-1528	44.3	.070
Line A Insert	0742-1442	42.0	.070
Occupational exposure limits	NIOSH REL		.016 (LFC)[4]
	ACGIH TLV		0.3 C [3]
	OSHA PEL		0.75
Area Samples			
Sample Location	Sampling Time	Sample Volume	Formaldehyde Concentration
Above Line B passive exhaust vent	0655-1440	46.5	.065
Inside Quality Lab	0648-1435	46.7	.034
Storage Area- North end of plant	0645-1445	48.0	.052

Table 17 Continued
Summary of Formaldehyde Sampling Typical Operations
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801
May 24, 1999

Above poly N/E tank manway	0702-1437	45.5	.050
Background sample- collected outside	0643-1447	48.4	.002

[1] Sample volumes are expressed in liters of air.
[2] Concentrations are expressed in parts per million (ppm).
[3] "C" denotes a ceiling concentration value which should not be exceeded during any part of the working exposure.
[4] "LFC" denotes Lowest Feasible Concentration. The .016 ppm exposure limit is not a health-based standard, rather it was derived from the analytical detection limits at the time the REL was issued.

Table 18
Summary of Formaldehyde Sampling During Cold Blast Operations
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801
May 22, 1999

Personal Breathing Zone Samples			
Job Title	Sampling Time	Sample Volume[1]	Formaldehyde Concentration[2]
Mold cleaner	0743-1442	42.0	.021
Mold cleaner	0802-1435	39.2	.056
Mold cleaner	0737-1444	42.7	.017
Mold cleaner	0742-1445	42.3	.014
Occupational exposure limits	NIOSH REL		.016 (LFC)[4]
	ACGIH TLV		0.3 C [3]
	OSHA PEL		0.75
Area Samples			
Sample location	Sampling Time	Sample Volume	Formaldehyde Concentration
On table in blasting area	0748-1448	42.0	.012
Above molds entering the blasting area	0747-1448	42.1	.017
Above Poly N/E tank manway	0757-1439	40.2	.020
Above passive exhaust opening above B-line oven	0823-1440	37.7	.013

Table 18 Continued
Summary of Formaldehyde Sampling During Cold Blast Operations
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801
May 22, 1999

Storage area - north end of the plant	0812-1437	38.5	.013
Background sample collected outside	0733-1442	42.9	.003

[1] Sample volumes are expressed in liters of air.
[2] Concentrations are expressed in parts per million (ppm).
[3] "C" denotes a ceiling concentration value which should not be exceeded during any part of the working exposure.
[4] "LFC" denotes Lowest Feasible Concentration. The .016 ppm exposure limit is not a health-based standard, rather it was derived from the analytical detection limits at the time the REL was issued.

Table 19
Sampling Results for Total Hydrocarbons (TH)
Woodbridge Corporation
Brodhead Wisconsin
HETA 98-0011-2801
March 4, 1999

Personal Air Samples				
Job Description	Sampling Time	Sample Volume[1]	TH Concentration[2]	
B-line insert	0745-1446	42.1	10.	
B-line insert	0742-1445	42.3	9.2	
B-line insert	0740-1446	42.6	8.0	
B-line insert	0747-1445	41.8	3.1	
A-line demold/wax applicator	0800-1459	41.5	13	
A-line demold/wax applicator	0800-1459	41.9	6.7	
A-line demold/wax applicator	0801-1448	40.9	4.7	
Occupational exposure limits	NIOSH REL		350	
	ACGIH TLV		525	
	OSHA PEL		2000	
Area Air Samples				
Sample Location	Sampling Time	Sample Volume	Concentration	
A-line wax application booth	0802-1112	19.0	190	190
	1114-1456	22.2	190	
B-line between wax application booth and insert area	0745-1447	42.2	11	

[1] Sample volumes expressed in liters

[2] Concentration expressed in milligrams of hydrocarbons per cubic meter of air (mg/m^3) as an 8-hour time-weighted average. Exposure during un-sampled portion of work shift was assumed to equal exposure during sampled portion of shift.

Table 20
Sampling Results forBis (2-dimethylaminoethyl) ether (DMAEE)
Woodbridge Corporation
Brodhead Wisconsin
HETA 98-0011-2801
May 24, 1999

Personal Breathing Zone Samples			
Job Title	Sampling Time	Sample Volume[1]	DMAEE Concentration[2]
A-Line Insert	0744-1448	441	0.14* [4]
A-Line Insert	0748-1441	405	0.13*
A-Line Demold	0755-1453	410	0.18*
A-Line Demold	0802-1448	418	0.21*
B-Line Insert	0724-1442	439	0.19*
B-Line Insert	0716-1444	457	0.18*
B-Line Insert	0722-1442	449	0.17*
B-Line Demold	0735-1455	436	0.41*
B-Line Demold	0729-1448	439	0.33*
Occupational exposure limits	NIOSH REL		LFC[3]
	ACGIH TLV		0.33
	OSHA PEL		LFC
Area Samples			
Sample location	Sampling Time	Sample Volume	DMAEE Concentration
Above B-line passive exhaust vent	0655-1440	493	0.21*
Inside Quality Lab	0648-1435	48	0.11

Table 20: Continued
Sampling Results for Bis (2-dimethylaminoethyl) ether (DMAEE)
Woodbridge Corporation
Brodhead, Wisconsin
HETA 98-0011-2801
May 24, 1999

Storage Area- North end of plant	0645-1445	499	0.09
Near B-line pourhead	0818-1455	421	0.14*
Mixing Area on top of control panel	0818-1435	381	0.14
B-line Demold area	0730-1453	452	0.03*
Above poly N/E tank manway	0702-1437	467	0.14*
Background/Outside	0643-1447	499	<.002

[1] Sample volumes expressed in liters
[2] Concentration expressed in milligrams of DMAEE per cubic meter of air (mg/m^3) as an 8-hour time weighted average. Exposure during un-sampled portion of work shift was assumed to equal exposure during sampled portion of shift.
[3] "LFC" means lowest feasible concentration
[4] "*" indicates samples for which analyte breakthrough on to the back-up section exceeded 10%.

For Information on Other
Occupational Safety and Health Concerns

Call NIOSH at:
1–800–35–NIOSH (356–4674)
or visit the NIOSH Web site at:
www.cdc.gov/niosh

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