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**HEALTH HAZARD EVALUATION AND
TECHNICAL ASSISTANCE REPORT**

HETA 91-0354-2532

**South Dade Disposal Site
Goulds, Florida**

September 1995

Preface

The Hazard Evaluations and Technical Assistance Branch 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 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 and 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.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) 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 the National Institute for Occupational Safety and Health.

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SUMMARY

In October 1990, the National Institute for Occupational Safety and Health (NIOSH) received a request from the American Federation of State, County, and Municipal Employees (AFSCME) to conduct a Health Hazard Evaluation (HHE) at the Metro Dade Solid Waste Managements' South Dade Disposal Site located in Goulds, Florida. This request was prompted because of employee concerns of workplace exposures related to waste shredding operations. Some employees had experienced rashes, eye problems, and respiratory difficulty, which they believed might be related to the workplace. In November 1990, two NIOSH representatives, an industrial hygienist and an occupational health nurse, conducted an initial site visit at the disposal site.

In August 1991, NIOSH received a second request for an HHE at the disposal site from the State of Florida, Department of Labor and Employment Security, Division of Safety, Bureau of Consultation and Enforcement. NIOSH was asked to conduct a comprehensive survey because of expressed concerns about working conditions at the South Dade site, in particular the Shredding Facility. In February 1992, NIOSH representatives conducted a second site visit at the disposal site. After that visit, it was determined that the field investigation should be conducted during the summer months, when temperatures and relative humidity levels were expected to be elevated.

An industrial hygiene and medical investigation was initiated during the week of August 17, 1992, with a scheduled completion date of August 28, 1992. The study protocol was designed to focus on maintenance activities during the first survey week and shredding activities during the second survey week.

Three days after the initiation of the study, during the evening of August 23, Hurricane Andrew arrived. The eye of the hurricane passed over the facility and destroyed most of the buildings at the facility. The tipping floor and shredder facility, however, received minimal damage. Due to the devastation of the facility and the surrounding community, the study was terminated. After the hurricane, the majority of the peak flow devices were never recovered. Employees were individually notified of their chest x-ray and spirometry results. Both the industrial hygiene and medical aspects of the study were disrupted by the hurricane. Bioaerosol determinations were not completed. Because of the hurricane and incomplete data results, conclusions from the investigations are limited.

Personal breathing zone concentrations of total welding fume ranged from 0.33 to 22.1 milligrams per cubic meter (mg/m^3). The mean concentration was $4.6 \text{ mg}/\text{m}^3$, with a standard deviation of $7.1 \text{ mg}/\text{m}^3$. Time-weighted average (TWA) concentrations in three of nine

samples (33%) collected on welders exceeded the Occupational Safety and Health Administration (OSHA) Permissible Exposure Level (PEL) of 5 mg/m^3 , with one being over four times the PEL.

Personal exposures to chromium ranged from 1 - $81 \text{ } \mu\text{g/m}^3$. The mean chromium exposure concentration of the samples collected was $21.5 \text{ } \mu\text{g/m}^3$, with a standard deviation of $26 \text{ } \mu\text{g/m}^3$. TWA concentration in all 15 samples collected on welders and maintenance personnel equaled, and 13 exceeded, the NIOSH Recommend Exposure Limit (REL) for chromium VI of $1 \text{ } \mu\text{g/m}^3$. Five of those samples were 20 times, and one 80 times higher than the NIOSH REL. None of the chromium exposure concentrations exceeded the OSHA PEL for chromium VI of $100 \text{ } \mu\text{g/m}^3$.

Nickel was detected in 10 of 15 samples collected on both welders and support personnel. Personal exposure concentrations to nickel ranged from not detected (ND) to $16 \text{ } \mu\text{g/m}^3$. The mean nickel exposure concentration was $7 \text{ } \mu\text{g/m}^3$, with a standard deviation of $5 \text{ } \mu\text{g/m}^3$. One nickel exposure concentration exceeded the NIOSH REL of $15 \text{ } \mu\text{g/m}^3$. None of the nickel exposures exceeded the OSHA PEL of $1000 \text{ } \mu\text{g/m}^3$. Lead was detected on two samples at $2.7 \text{ } \mu\text{g/m}^3$ on each sample. The lead exposures measured were below exposure criteria.

One carbon monoxide sample collected on a welder exceeded the American Conference of Governmental Industrial Hygienists' (ACGIH) Threshold Limit Value (TLV) of 25 parts per million (ppm) for a TWA personal exposure. None of the carbon monoxide samples collected exceeded the NIOSH REL of 35 ppm or the OSHA PEL of 50 ppm.

Sulfur dioxide was detected on 11 of 18 samples collected on both welders and support personnel. Personal breathing zone samples were collected on workers engaged in welding operations and support activities. Personal exposure concentrations for the samples collected on welders ranged from ND to 3.66 ppm, with a mean exposure concentration of 1.5 ppm. Personal exposure concentrations for those samples collected on support personnel ranged from ND to 1.5 ppm, with a mean exposure of 0.46 ppm. Two samples collected on welders exceeded the NIOSH REL and the ACGIH TLV of 2 ppm for a TWA exposure. However, none of the samples collected exceeded the OSHA PEL of 5 ppm.

Nitrogen dioxide was detected on 17 of 18 samples collected on both welders and support personnel. Exposure concentrations to nitrogen dioxide ranged from ND to 0.41 ppm. None of the samples collected for nitrogen dioxide exceeded any occupational exposure criteria.

Forty-eight workers completed standardized symptom questionnaires. Eleven workers mentioned that their chest felt tight or their breathing became difficult. Six of those workers associated the chest tightness with entering the worksite, one worker developed the chest tightness in 1-3 hours after leaving the worksite, and four workers developed the chest tightness in 3-8 hours after leaving the worksite.

Seven had chest tightness with headaches, muscle aches, and/or nausea/diarrhea symptoms consistent with occupational inflammatory lung disease (OILD). Of the 11 workers reporting chest tightness, 3 were welders, 4 worked in an area of low dust exposure, and 4 worked in an area of high dust exposure.

Forty-eight workers completed one session of spirometry, but due to the hurricane only 7 workers completed cross-shift spirometry. There were no forced expiratory volume in one second (FEV₁) declines observed in the cross-shift studies. Four workers (8%) had mild obstructive patterns, and another worker (2%) had a mild restrictive pattern. None of the radiographs had signs of pneumoconiosis.

On the basis of the data obtained, the investigators have concluded that during this investigation, welders had the greatest potential to be overexposed to total welding fume, chromium, and nickel. The lack of engineering controls to remove welding fumes and gases from the workers' breathing zone greatly contributed to their exposures.

Keywords: SIC 4953 (Refuse Systems), solid waste, welding, carbon monoxide, chromium, nickel, sulfur dioxide, nitrogen dioxide, spirometry, x-ray.

INTRODUCTION

In October 1990, the National Institute for Occupational Safety and Health (NIOSH) received a request from the American Federation of State, County, and Municipal Employees (AFSCME) to conduct a Health Hazard Evaluation (HHE) at the Metro Dade Solid Waste Management's South Dade Disposal Site located in Goulds, Florida. This request was prompted because of employee concerns of workplace exposures during shredding operations. Some employees had experienced rashes, eye problems, and respiratory difficulty which they felt may be related to the workplace. In November 1990, two NIOSH representatives, an industrial hygienist, and an occupational health nurse conducted an initial site visit at the disposal site. After that visit, it was decided that a NIOSH investigation was warranted.

While preparing to conduct that investigation, NIOSH investigators became aware that the initial request made by AFSCME was invalid under the Occupational Safety and Health Act of 1970. In March 1991, a letter was sent to the AFSCME and MDSWM outlining the legal issues, along with recommendations for correcting workplace deficiencies observed during the initial site visit. A copy of that letter was also sent to the Florida State Health Department.

In August 1991, NIOSH received a second request for an HHE at the disposal site from the State of Florida, Department of Labor and Employment Security, Division of Safety, Bureau of Consultation and Enforcement. NIOSH was asked to conduct a comprehensive survey because of expressed concerns about working conditions at the South Dade site, in particular the Shredding Facility. In February 1992, NIOSH representatives conducted a second site visit at the disposal site and determined that to evaluate a "worst case" environment for microorganisms, the best time to conduct the field investigation would be during the summer months, when temperatures and relative humidity levels were expected to be high.

An industrial hygiene and medical investigation was initiated during the week of August 17, 1992, with a scheduled completion of August 28, 1992. The study protocol was designed to focus on maintenance activities during the first survey week and shredding activities during the second survey week.

Three days after the initiation of the study, during the evening of August 23, Hurricane Andrew arrived. The eye of the hurricane passed over the facility and destroyed most of the buildings at the facility. The tipping floor and shredder facility, however, received minimal damage. Due to the devastation of the facility and the surrounding community, the study was terminated. After the hurricane, the majority of the peak flow devices were never recovered. Employees were individually notified of their chest x-ray and spirometry results. Both the industrial hygiene and medical aspects of the study were disrupted by the hurricane. Bioaerosol determinations were not completed. Because of the hurricane and incomplete data results, conclusions from the investigations are limited.

No additional follow-up activities are planned by NIOSH, since much of the landfill site was devastated or destroyed by the hurricane. In addition, Dade County officials indicated that the shredding of solid waste was being phased out due to the recycling policies of the county. It was evident at the time of the field survey that the volume of solid waste was noticeably reduced from the initial site visit in November 1990.

PROCESS DESCRIPTION

The South Dade Disposal Site is located in Goulds, Florida, approximately 15 miles south of Miami. The shredder and landfill facilities were in operation by 1981 and were designed to accept municipal solid waste such as household garbage, construction debris, and old tires. Debris such as asbestos, dead animals, and medical waste are separated manually and land filled in specific areas.

Waste from the county was unloaded inside the shredding facility daily except for Sundays and may have remained on the tipping floor from Saturday evening till Monday morning. The waste was initially dumped onto the tipping floor, where bulldozers were used to push it forward in the building to accommodate more trash. During the shredding process, bulldozers advanced the trash into one of three open pits at the front of the floor. These pits had a conveyor system that carried the trash up an incline and dumped into a bin. From the bin the trash was funneled onto a rotary hammer that spun at 900 rpm and pulverized the debris.

An attendant stood next to each open pit on the tipping floor and manually removed large items (mattresses, lumber, etc). The attendants are provided with safety glasses, hearing protection, and a disposable dust mask for respiratory protection.

The tipping floor and the shredders are separated by an approximately 12 foot high large retaining wall. During the process of shredding, no employees are permitted behind the wall. This wall protects employees on the tipping floor from the debris that may explode in the shredder (propane tanks, etc). Attendants are located between the retaining wall on one side, and a mountain of trash on the other side. Air flows from the shredders towards the conveyor pits and was visibly dusty.

Once it is shredded, a conveyor system then carries the debris outside the building to towers where it is deposited into dump trucks. Loading of the trucks is monitored by an operator who sits in a control tower and communicates using a video system. The shredded debris is driven a quarter of a mile to the landfill and dumped, and then spread on the landfill by bulldozers.

The facility has three parallel shredding systems that operate off the tipping floor. Each system includes a conveyor tower and loading tower.

In the process of shredding the trash, the rotary metal hammers wear down and require daily welding to balance and resurface the hammers. Thus two crews are involved in the shredding

process. One crew performs maintenance of the hammers and shredder machinery, and the other crew performs the waste shredding. Each crew has its own bargaining unit with management. The maintenance welders and electricians are in the general unit, and the shredder operators, waste attendants, and equipment operators are in the waste unit.

The facility operates in three shifts each day. The morning shift begins at 5:00 a.m. and continues through 1:00 p.m. This shift repairs and maintains equipment to prepare for the actual shredding operations. The crew includes welders and welders' helpers. Others workers perform housekeeping such as sweeping under the shredders and cleaning the tipping floor of trash that has fallen out of the conveyor system and shredder.

The afternoon shift starts at 1:00 p.m., when the actual shredding operation begins and continues until 12:00 a.m. This crew typically includes 24 equipment operators involved in operating heavy equipment on the tipping floor and in the landfill; 3 designated truck drivers who haul the trash to the landfill; and 12 waste attendants involved in removing the larger debris on the tipping floor and in clean up around the shredders.

BACKGROUND

The working environment of solid waste facilities is variable, being affected by changes in the quantity and quality of refuse, the weather, and the season. Household garbage in the waste stream is a source of Gram-negative bacteria. In the summer, temperature and humidity are favorable to the proliferation of bacteria and other microorganisms.⁽¹⁾

Previous investigations have been conducted of workers at waste handling facilities. Environmental studies have identified bacteria, fungi, and endotoxin in waste handling facilities, with cough, asthma, and organic dust toxic syndrome (ODTS) in exposed workers.^(2,3,4) A study at a waste shredding plant in Denmark reported that 9 of 15 exposed workers had symptoms of eye irritation, coughing at night, chills, and fever. In this study, 8 of the 9 symptomatic workers exhibited daily peak expiratory flow variation of more than 100 liters per minute (l/min). Three workers had suspect cases of ODTS. Immunologic studies did not define a single etiologic agent. After having left the work environment for up to 2 years, 6 workers still noted respiratory symptoms.

The Oregon Department of Human Resources investigated illnesses at a compost plant and concluded that respiratory symptoms of wheezing, phlegm, and cough, along with muscle pain and rash, were significantly associated with working in the picking room (room where trash was manually sorted into recyclables). Workers in the picking room were 6 times more likely to report wheezing, 4 times more likely to report phlegm, and 3.5 times more likely to have developed cough since beginning work at the plant, in comparison to employees who did not work in the picking room most of the time.⁽⁵⁾

METHODS

Industrial Hygiene

Due to diminished tonnage during the NIOSH survey, only one shredder was in operation. As a result, maintenance activities associated with shredding, including welding operations, were also diminished. However, personal breathing zone air samples were collected during the limited welding operations. Each of the four welders wore personal breathing zone samples for total welding fume, elemental metals, carbon monoxide (CO), nitrogen dioxide (NO₂), nitric oxide (NO), and sulfur dioxide (SO₂). In addition, other support maintenance personnel who were not actively involved in welding were monitored to determine their passive exposures from welding operations. The sampling methods utilized during this investigation are outlined below and are summarized in Table 1.

Total Welding Fume

Personal breathing zone air samples for total welding fume exposure, based on total mass, were collected on pre-weighed, 37 mm, 5.0 micrometer (µm) pore size polyvinyl chloride filters housed in two piece polystyrene cassettes. During sampling, each cassette was connected by flexible tubing to a personal sampling pump operated at a flow rate of 2.0 liters per minute (lpm). The samples were analyzed for total welding fume according to NIOSH Analytical Methods.⁽⁶⁾ Each sample was analyzed by post weighing the sample filters to determine the total collected mass. The limit of detection reported for the analysis was 0.02 mg, which equates to a minimum detectable concentration of 0.04 mg/m³, assuming a sampling volume of 480 liters.

Elemental Metals

Personal breathing zone air samples for determining elemental metals exposures were collected on 37 mm, 0.8 µm pore size cellulose ester membrane filters housed in two piece polystyrene cassettes. During sampling, each cassette was connected by flexible tubing to a personal sampling pump operated at a flow rate of 1.0 lpm. Samples were analyzed according to NIOSH Analytical Method 7300 for specific metals and oxides, such as iron, manganese, chromium, nickel, copper, zinc, and cadmium.

Analysis consisted of first transferring each sample filter from the polystyrene sampling cassette to a 125 milliliter (mℓ) Phillips beaker. To each beaker 1 mℓ of perchloric acid and 4 mℓ of nitric acid were added and the sample placed on a hot plate and heated to approximately 150° C to digest the filter matrix. Sample volume was reduced to approximately 0.5 mℓ. Samples were then quantitatively transferred to 10 mℓ volumetric flasks and analyzed using a Thermo Jarrell Ash ICAP61 inductively coupled plasma emission spectrometer controlled by an NEC personal computer. The limit of detection reported for chromium was 0.5 micrograms per sample (µg/sample), 1.0 µg/sample for lead, and 0.5 µg/sample for nickel. Based on those Limit of Detections (LODs), the minimum detectable concentrations, assuming a 480 liter sample, for chromium, lead, and nickel was 0.001mg/m³, 0.002 mg/m³, and 0.001mg/m³, respectively.

Carbon Monoxide

Air samples for the estimation of carbon monoxide (CO) exposures were collected using Dräger diffusion detector tubes (Catalog No. 67 33191, National Dräger, Pittsburgh, Pennsylvania). These tubes were used to determine personal exposures to CO. These tubes operate on the diffusion processes in gases (Fick's Law of Diffusion). Once the tube is exposed to air containing CO, the CO molecules in the air enter the tube due to the effect of diffusion processes in gases. Therefore, a sampling pump is not required for the measurement. The tube contains a yellow indicating layer that reacts with CO to change to a grayish black. Concentration of CO, in parts per million (ppm), is calculated by dividing the length of the discoloration, scaled in ppm-hours, by the time in hours that the tube was exposed. The detection range of this sampling method is 6 to 75 ppm for an 8-hour sampling duration. The accuracy for this method, as reported by the manufacturer, is ± 25%.

Sulfur Dioxide

Air samples for personal beathing zone exposures to sulfur dioxide (SO₂) were collected using Dräger detector tubes (Catalog No. 81 01091, National Dräger, Pittsburgh, Pennsylvania). This detector tube, similar to the CO tube, also operates on a diffusion process. The indicating layer changes from a bluish violet to pale yellow when exposed to SO₂. Concentration of SO₂, is calculated by dividing the length of the discoloration, scaled in ppm-hours, by the time in hours that the tube was exposed. The detection range of this sampling method is 0.7 to 19 ppm for an

8-hour sampling duration. The accuracy for this method, as reported by the manufacturer, is \pm 25%.

Oxides of Nitrogen

Air samples for the estimation of personal breathing zone exposures to nitrogen dioxide (NO₂) were collected using Palmes passive dosimeters (NIOSH Method 6700). The principle of operation of the dosimeters is that NO₂ will diffuse through a tube at a rate proportional to its concentration (Fick's Law of Diffusion) and will react immediately onto a collection medium. The medium is then analyzed for NO₂ collected.

The sampler consists of a cylindrical section of rigid plastic tubing of accurately known dimensions (with a length-to-cross section area ratio of 0.1). Reagent (triethanolamine) coated metal screens are enclosed on one end by a plastic cap. The plastic cap that covers the other end is removed to sample the NO₂ in the surrounding air. During analysis, a sulfanilamide-phosphoric acid NEDA solution is added to the dosimeter. The collected NO₂ is desorbed and a red color complex is formed. This solution is then analyzed by spectrophotometry and the NO₂ concentration determined.

Medical Investigation

Questionnaire

A standardized questionnaire was administered to study participants. Demographic information, current employment status including job title and work area, and smoking status were obtained. Information was requested regarding symptoms of cough, phlegm, wheezing, past respiratory disease, chest tightness, exertional dyspnea, chills, fever, headache, muscle ache, nausea, and diarrhea. Additional questions about nasal, eye, and dermatologic symptoms were asked. The temporal relationship of these symptoms to entering or leaving the worksite was determined.

Spirometry

Spirometry was performed using a dry rolling-seal spirometer interfaced to a dedicated computer. At least five maximal expiratory maneuvers were recorded for each person. All values were corrected to BTPS (body temperature, ambient pressure, saturated with water vapor). The largest forced vital capacity (FVC), and forced expiratory volume in one second (FEV₁) were the parameters selected for analysis, regardless of the curves on which they occurred. Testing procedures conformed to the American Thoracic Society's recommendations for spirometry.⁽⁷⁾ Predicted values were calculated using the Knudson reference equations.⁽⁸⁾ Predicted values for African-Americans were determined by multiplying the value predicted by the Knudson equation by 0.85.⁽⁹⁾ Test results were compared to the 95th percentile lower limit of normal (LLN) values obtained from Knudson's reference equations to identify participants with abnormal spirometry patterns of obstruction and restriction.⁽⁸⁾ Five percent of the general adult population will have

predicted values that fall below the normal range, or LLN, while 95% will have predicted values above the lower limit.

Using this comparison, obstructive and restrictive lung disease patterns are defined as:

Obstruction: Observed ratio of FEV₁ / FVC% below the LLN.

Restriction: Observed FVC below the LLN; and FEV₁ / FVC% above the LLN.

The criteria for interpretation of the level of severity for obstruction and restriction, as assessed by spirometry, is based on the NIOSH classification scheme (available upon request from the Division of Respiratory Disease Studies, Morgantown, West Virginia). For those persons with values below the LLN, the criteria are:

Classification	Obstruction (FEV₁/FVC x 100)	Restriction (% Predicted FVC)
Mild	>60	> 65
Moderate	≥ 45 to ≤ 60	≥ 51 to ≤ 65
Severe	< 45	< 51

Cross-shift spirometry was used to document acute airway response. Spirometry was to be performed pre and post-shift on the last day of the participant's work week and again on the first day of the following work week. However because of Hurricane Andrew, only limited cross-shift spirometry data was available.

Peak Flow Monitoring

Although peak flow monitoring was attempted on these workers, no results are available. Due to the disruption caused by Hurricane Andrew, the study was cut short and many of the peak flow meters and data were either lost or not returned by workers.

Posterior-Anterior (PA) Chest X-rays

Each PA chest x-ray was taken on a full size (14 x 17 inch) film and read independently by two NIOSH-certified pneumoconiosis B Readers who, without knowledge of the participant's age, occupation, or smoking history, classified the films according to the 1980 Guidelines for the use of ILO International Classification of Radiographs of Pneumoconioses.⁽¹⁰⁾ A chest radiograph was defined as positive for (that is, consistent with) pneumoconiosis if each of the two B Readers classified small opacity profusion as 1/0 or greater. In the event of disagreement between the two readers, a third reading was obtained and the median reading was utilized.

EVALUATION CRITERIA & TOXICOLOGY

General Guidelines

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 increase 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)⁽¹¹⁾, (2) the American Conference of Governmental Industrial Hygienists' (ACGIH) Threshold Limit Values (TLVs)⁽¹²⁾, and (3) the U.S. Department of Labor, OSHA Permissible Exposure Limits (PELs).⁽¹³⁾ In July 1992, the 11th Circuit Court of Appeals vacated the 1989 OSHA PEL Air Contaminants Standard. OSHA is currently enforcing the 1971 standards which are listed in the current Code of Federal Regulations; however, some states operating their own OSHA approved job safety and health programs continue to enforce the 1989 limits. NIOSH encourages employers to follow the 1989 OSHA limits, the NIOSH RELs, the ACGIH TLVs, or whichever are the more protective criterion. The OSHA PELs reflect the feasibility of controlling exposures in various industries where the agents are used, whereas NIOSH RELs are based primarily on concerns relating to the prevention of occupational disease. It should be noted when reviewing this report that employers are legally required to meet those levels specified by an OSHA standard and that the OSHA PELs included in this report reflect the 1971 values.

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

For the substances monitored during this survey, a brief toxicology description and current environmental exposure criteria are outlined below and are summarized in Table 2. This section also describes the possible toxicological and physiological effects from exposure to substances

monitored during this survey. These effects are described so workers will be familiar with the symptoms and health consequences of overexposure.

Toxicology and Exposure Criteria

Particulates, not otherwise classified

Often the chemical composition of the airborne particulate does not have an established occupational health exposure criterion. It has been the convention to apply a generic exposure criterion in such cases. Formerly referred to as nuisance dust, the preferred terminology for the non-specific particulate ACGIH TLV criterion is now "*Particulates, not otherwise classified (n.o.c.)*," [or "*not otherwise regulated*" (n.o.r.) for the OSHA PEL].

Excessive concentration of dusts in the workroom air may seriously reduce visibility; may cause unpleasant deposits in the eyes, ears, and nasal passages; or cause injury to the skin or mucous membranes by chemical or mechanical action per se or by the rigorous skin cleansing procedures necessary for their removal.⁽¹⁴⁾

The OSHA PEL for total particulate, n.o.r., is 15.0 mg/m³ and 5.0 mg/m³ for the respirable fraction, determined as 8-hour averages. The ACGIH recommended TLV for exposure to a particulate, n.o.c., is 10.0 mg/m³ (total dust, 8-hour TWA). These are generic criteria for airborne dusts which do not produce significant organic disease or toxic effect when exposures are kept under reasonable control. These criteria are not appropriate for dusts that have a biologic effect and may not be appropriate for evaluating shredding operations.

Total Welding Fume and Elemental Metals

Total welding fume cannot be classified simply since the composition and quantity of the welding fume, and therefore the potential health effects, are dependent upon the alloy being welded, the base metal, the electrode used, and the fluxing agents. During this evaluation, the predominant form of weld was hard facing using a carbon electrode (AWS 5.20 E71T-1). Iron oxide fume is the major metallic constituent emitted in this welding process, although chromium, nickel, manganese oxide, silicon dioxide, and fluoride are also likely to be present since the electrode contains these compounds. Inhalation of iron oxide may cause a “benign” pneumoconiosis known as siderosis, which may cause functional alterations in the lung.⁽¹⁵⁾

The current OSHA PEL and ACGIH TLV for welding fume (total particulate) is 5 mg/m³ averaged over an 8-hour work shift. The individual constituents that are likely to be present should also be measured to determine whether the specific OSHA PELs or ACGIH TLVs are exceeded. NIOSH considers welding fume to be a potential occupational carcinogen and recommends that exposures to all welding be reduced to the lowest feasible levels using state-of-the-art engineering controls and work practices.

Chromium

The dust from chromium metal can be oxidized to a soluble chromium (VI) compound. Samples collected during this survey were assumed to be chromium trioxide fume.

The toxicity and solubility of chromium compounds that contain chromium in the Cr²⁺, Cr³⁺, or Cr⁶⁺ valence state vary greatly, but those that contain chromium VI (Cr⁶⁺) are of the greatest health concern. Chromium VI compounds include lead chromate and zinc chromate pigments, chromic acid, and soluble compounds such as those used in chromium plating. Some chromium VI compounds are severe irritants of the respiratory tract and skin, and some (including chromates) have been found to cause lung cancer in exposed workers. Allergic dermatitis is one of the most common effects of chromium toxicity among exposed workers.⁽¹⁴⁾

The ACGIH Biological Exposure Index (BEI) for chromium VI are a 10 µg/g of creatinine increase during the work shift, and 30 µg/g of creatinine when measured in exposed workers at the end of the workweek.⁽¹⁶⁾ These recommended BEIs apply only to operations where water soluble chromium VI fume is present. The BEIs represent levels that are likely to be found in biological samples collected from healthy workers who have inhalation exposure to water soluble chromium VI at the current TLV-TWA of 50 µg/m³. The NIOSH REL-Ceiling for chromates, based on its designation as a potential occupational carcinogen, is 1 µg/m³. The OSHA PEL for a 15 minute ceiling exposure to chromium VI is 100 µg/m³.

Nickel

Inorganic nickel compounds are suspected of causing lung and nasal cancers, based on the mortality experience of nickel refinery workers. Occupational exposure to nickel occurs from working with compounds, solutions, or metals containing nickel that can become airborne or can be splashed on the skin or in the eyes. Nickel fumes are respiratory irritants and may cause pneumonitis. Skin contact may cause an allergic skin rash known as "nickel itch." NIOSH considers nickel an occupational carcinogen. Nickel compounds have been associated with cancer of the paranasal and lung.⁽¹⁷⁾

The ACGIH TLV for elemental nickel is 1000 $\mu\text{g}/\text{m}^3$ for a TWA exposure, with a notice of intended change to 50 $\mu\text{g}/\text{m}^3$. The current OSHA PEL for occupational exposure to nickel is being enforced at the transitional level of 1000 $\mu\text{g}/\text{m}^3$ (which is identical to the 1989 Air Contaminants Standard). The NIOSH REL for nickel is 15 $\mu\text{g}/\text{m}^3$ as a TWA for up to 10 hours per day during a 40-hour week.

Lead

Lead is ubiquitous in U.S. urban environments due to the widespread use of lead compounds in industry, gasoline, and paints during the past century. Exposure to lead occurs via inhalation of dust and fume, and ingestion through contact with lead-contaminated hands, food, cigarettes, and clothing. Absorbed lead accumulates in the body in the soft tissues and bones. Lead is stored in bones for decades and may cause health effects long after exposure as it is slowly released in the body.

Symptoms of lead exposure include weakness, excessive tiredness, irritability, constipation, anorexia, abdominal discomfort (colic), fine tremors, and "wrist drop."^(14,18,19) Overexposure to lead may also result in damage to the kidneys, anemia, high blood pressure, infertility and reduced sex drive in both sexes, and impotence. An individual's blood lead level (BLL) is a good indication of recent exposure to, and current absorption of lead.⁽²⁰⁾ The frequency and severity of symptoms associated with lead exposure generally increase with the BLL.

The overall geometric mean BLL for the U.S. adult population (ages 20-74 yrs) declined significantly between 1976 and 1991, from 13.1 to 3.0 micrograms per deciliter of blood ($\mu\text{g}/\text{dL}$)--this decline is most likely due primarily to the reduction of lead in gasoline. More than 90% of adults now have a BLL of <10 $\mu\text{g}/\text{dL}$, and more than 98% have a BLL <15 $\mu\text{g}/\text{dL}$.⁽²¹⁾

Under the OSHA general industry lead standard (29 CFR 1910.1025), the PEL for airborne exposure to lead is 50 $\mu\text{g}/\text{m}^3$ (8-hour TWA).⁽²²⁾ The standard requires lowering the PEL for shifts exceeding 8 hours, medical monitoring for employees exposed to airborne lead at or above the action level of 30 $\mu\text{g}/\text{m}^3$ (8-hour TWA), medical removal of employees whose average BLL is 50 $\mu\text{g}/\text{dL}$ or greater, and economic protection for medically removed workers. Medically removed workers cannot return to jobs involving lead exposure until their BLL is below 40 $\mu\text{g}/\text{dL}$. The OSHA interim final rule for lead in the construction industry (29 CFR 1926.62)

provides an equivalent level of protection to construction workers. ACGIH has proposed a TLV for lead of $150 \mu\text{g}/\text{m}^3$ (8-hour TWA), with a notice of intended change to $50 \mu\text{g}/\text{m}^3$.

ACGIH recommends worker BLLs be controlled at or below $20 \mu\text{g}/\text{dL}$, and designation of lead as an animal carcinogen.⁽¹²⁾ The U.S. Public Health Service has established a goal, by the year 2000, to eliminate all occupational exposures that result in BLLs greater than $25 \mu\text{g}/\text{dL}$.⁽²³⁾

The occupational exposure criteria (above) are not protective for all the known health effects of lead. For example, studies have found neurological symptoms in workers with BLLs of 40 to $60 \mu\text{g}/\text{dL}$, and decreased fertility in men at BLLs as low as $40 \mu\text{g}/\text{dL}$. BLLs are associated with increases in blood pressure, with no apparent threshold through less than $10 \mu\text{g}/\text{dL}$. Fetal exposure to lead is associated with reduced gestational age, birth weight, and early mental development with maternal BLLs as low as 10 to $15 \mu\text{g}/\text{dL}$.⁽²⁴⁾ Men and women who are planning on having children should limit their exposure to lead.

Carbon Monoxide

Carbon monoxide (CO) is a colorless, odorless gas, lighter than air. It is produced whenever incomplete combustion of carbon-containing compounds occurs. Typical environmental sources of CO exposure, to name a few, are poorly vented heating systems, automobile exhaust, and cigarette smoke. The combination of incomplete combustion and inadequate venting often results in overexposure. The danger of this gas derives from its affinity for the hemoglobin of red blood cells, which is 300 times that of oxygen. The hazard of exposure to CO is compounded by the insidiousness with which high concentrations of carboxyhemoglobin (CO-Hb) can be obtained without marked symptoms. Intermittent exposures are not cumulative in effect. Symptoms occur more acutely with higher concentrations of CO.⁽¹⁴⁾

The NIOSH REL for CO is 35 ppm for an 8-hour TWA exposure, with a ceiling limit of 200 ppm which should not be exceeded. The NIOSH REL of 35 ppm is designed to protect workers from health effects associated with CO-Hb levels in excess of 5%. The ACGIH recommends an 8-hour TWA TLV of 25 ppm. The OSHA PEL for CO is 50 ppm for an 8-hour TWA exposure.

Sulfur Dioxide

Sulfur dioxide gas is a severe irritant of the eyes, mucous membranes, and skin. Its irritant properties are due to the rapidity with which it forms sulfurous acid on contact with moist membranes. In combination with certain particulate matter and/or oxidants, the effects may be markedly increased. Approximately 90% of all sulfur dioxide inhaled is absorbed in the upper respiratory passages, where most effects occur. High concentrations of sulfur dioxide may produce respiratory paralysis and pulmonary edema. Exposure to concentrations of 10 to 50 ppm can cause irritation to the eyes and nose, runny nose, choking, cough, nosebleeds, and sometimes, reflex bronchoconstriction with increased pulmonary resistance.⁽²⁵⁾ NIOSH and ACGIH have set an exposure criteria for sulfur dioxide at 2 ppm for an 8-hour TWA exposure. The OSHA PEL is 5 ppm for an 8-hour TWA exposure.

Oxides of Nitrogen

Nitric oxide changes into nitrogen dioxide in air. Nitrogen dioxide is more toxic than nitric oxide and may cause severe breathing difficulties that may be delayed in onset. Nitrogen dioxide gas is a respiratory irritant; it causes pulmonary edema and rarely, among survivors, residual lung damage. Brief exposure of humans to concentrations of about 250 ppm caused cough, production of mucoid or frothy sputum, and increasing yspnea. The effects expected in humans from exposure to nitrogen dioxide for 60 minutes are: 25 ppm, respiratory irritation and chest pain; 50 ppm, pulmonary edema with possible subacute or chronic lesions in the lungs; 100 ppm, pulmonary edema and death.⁽¹⁴⁾

The NIOSH REL for nitrogen dioxide is a 15 minute short-term exposure limit (STEL) of 1 ppm. The OSHA PEL for nitrogen dioxide is 5 ppm for a ceiling exposure. The ACGIH TLV for an 8-hour exposure to nitrogen dioxide is 3 ppm.

Health Effects

Occupational Dermatitis

Despite numerous protective mechanisms, the skin is particularly vulnerable to environmental injuries and diseases. Over the last decade, skin diseases have accounted for a disproportionately large percentage of all occupational illnesses, ranging from 24% to 37%.⁽²⁶⁾

Dermatological conditions other than injuries are usually the result of sustained or cumulative exposures and usually involve long intervals between exposure and occurrence of disease. These conditions include contact dermatitis, infection, acne, and skin cancer. Contact dermatitis makes up the vast majority (93.8%) of all occupational skin disease (OSD), occurring most often (88%) on the hands.⁽²⁷⁾

Occupational dermatoses may be produced either by irritant or by allergic contact sensitivity reactions. Irritants alter the chemistry of the skin. This alteration may cause itching, redness, inflammation, discomfort due to dryness, and pain related to fissures and ulcers. Quaternary ammonium salts, ethanol, ethanolamine, phosphoric acid, and iodine are all skin irritants.⁽¹⁴⁾ Certain irritants may also act as sensitizers. Initial skin contact with the substance may not produce irritation, but after repeated or extended exposure some people may develop an allergic reaction termed allergic contact dermatitis.⁽²⁸⁾ Approximately 80% of occupational contact dermatitis cases are due to non-immunologic irritant contact dermatitis, whereas 20% are attributable to allergic etiologies.⁽¹⁴⁾

Frequent causes of work-related irritant contact dermatitis are water, soaps, and detergents. Also, occlusion of a substance against the skin, such as a chemical trapped beneath a glove, combined with frictional forces can accelerate cutaneous absorption of a compound.⁽²⁹⁾

Environmental factors in the workplace also play a role. If it is hot and humid, workers are less likely to wear protective clothing. Additionally, they are more vulnerable to sweating, which can solubilize particulate matter, enhancing its penetration into the skin. Perspiration can leach out allergens from certain materials.⁽³⁰⁾ The most common causes of occupational dermatitis are dust, sweating, exposure to water, soap, external irritants, temperature effects, and contact with animals.⁽³¹⁾

Occupational Inflammatory Lung Diseases (OILD)

It is well recognized that inhalation of substances in the workplace can result in inflammatory and immunologic response to the foreign material. Several conditions may be categorized as OILD with diagnostic criteria for each being primarily a clinical decision.

Occupational Asthma

Asthma is a disease characterized by intermittent respiratory symptoms (shortness of breath, chest tightness, wheezing, and cough) and reversible or variable airflow obstruction.⁽³²⁾

Occupational asthma is characterized by variable airflow obstruction, related to exposure in the workplace environment to airborne dusts, gases, vapors, or fumes.⁽³³⁾ The greatest number of occupational agents causing asthma have known or suspected allergic properties. Organic high molecular weight compounds induce an immunologic response by producing specific IgE antibodies. These include animal and plant proteins, insects, and biological enzymes. Inhalation of the allergen induces an early, late, or biphasic asthmatic reaction, due to inflammatory mediators released in the airway, with resulting edema, inflammation, and airflow limitation.⁽³⁴⁾ Affected persons may be asymptomatic for prolonged periods, except when exposed to a specific sensitizing agent. Variable airflow obstruction can be documented by cross-shift spirometry or periodic peak flow measurement.

Organic Dust Toxic Syndrome (ODTS)

ODTS is a respiratory illness that may follow initial exposures to heavy concentrations of organic dust. Workers are typically exposed when handling contaminated organic materials.

Development of the syndrome does not require a previous exposure or sensitization. Prevalence rates may reach 30-40% or more of the exposed workers. ODTS is thought to be causally related to inhalation of microbial products, e.g., bacterial endotoxins. The syndrome is characterized by fever and flu-like symptoms such as general weakness, headache, chills, body aches, and cough occurring 4 to 12 hours after exposure.⁽³⁵⁾ Typically lung infiltrates are not seen on the chest x-ray. Symptoms usually recur on re-exposure to the organic material. Overall, ODTS is thought to be self-limiting and has not been reported to result in permanent physiological derangements.⁽³⁶⁾

Hypersensitivity Pneumonitis (HP)

The inhalation of aerosolized organic materials can also lead to the development of respiratory symptoms and clinical findings of hypersensitivity pneumonitis (HP). Persistent lung damage can result when inflammatory cells in the lung become sensitized and stimulated by the inhaled material. The lung reacts with the development of granulomas and may progress to scarring of the lung architecture. HP differs from ODTS in that it requires this prior sensitization before symptoms become manifest.

Workers with acute hypersensitivity pneumonitis also typically develop 4 to 8 hours after inhaling the offending agent, with flu-like symptoms of fever, muscle aches, and at times, headaches. Dyspnea is the most common respiratory symptom, and cough and chest tightness may also be present. Workers with subacute HP experience similar but sometimes less severe complaints, although the dyspnea may not completely resolve between episodes of exposure. Workers with chronic HP may never experience episodes of fever or dyspnea, but will note progressive worsening of exertional dyspnea, fatigue, and weight loss.

Chest x-ray findings in workers with HP are variable and are influenced by the severity of an acute episode and the timing of the film. During the acute episode, ill-defined patchy lung infiltrates are common. In chronic disease, diffuse fibrosis and even honeycombing may be seen, with upper lobes being predominantly involved.⁽³⁷⁾

Metal Fume Fever

Metal fume fever is an acute, self-limited, flu-like illness, which is characterized by malaise, muscle aches, and fever developing after the inhalation of metal oxides.⁽³⁸⁾ There are usually few respiratory symptoms and little or no chest x-ray or functional abnormalities. Symptoms typically begin several hours after exposure to the metal oxides, and subside spontaneously. The exact pathogenesis of metal fume fever is poorly understood. In some instances an allergic

mechanism may be involved.⁽³⁹⁾ There is a striking resemblance between metal fume fever and ODTS, which occurs after heavy exposure to organic dust.⁽⁴⁰⁾

Mucous Membrane Irritation

Irritation of the eye, nose and throat can occur after exposure to many substances, and has been associated with work-related asthmatic symptoms. Nasal and eye symptoms may result from direct irritation or from development of immunologic sensitization to dusts or chemicals. The latency period of work exposure can range from months to years prior to onset of respiratory symptoms.⁽⁴¹⁾ In animal handlers, rhinitis is the most common manifestation of allergy.⁽⁴²⁾ In Western Red Cedar asthma, some workers experienced rhinorrhea several weeks before the onset of the respiratory symptoms.⁽⁴³⁾ The onset of mucous membrane irritation may suggest the development of sensitization to the exposure.

RESULTS

Industrial Hygiene

Total Welding Fume Exposures

The results of the total welding fume analyses for samples collected during welding operations are shown in Table 3. A total of nine personal breathing zone air samples were collected over a 3 day period on three welders. Exposure concentrations to total welding fume ranged from 0.33 to 22.1 mg/m³. The mean exposure concentration for total welding fume was 4.6 mg/m³ with a standard deviation of 7.1 mg/m³. Three samples (33%) exceeded the OSHA PEL of 5 mg/m³, with one sample being over four times the PEL.

An additional 11 personal breathing zone samples for total welding fume were collected on five employees not specifically engaged in welding, but whom were occasionally in close proximity to welding operations. Those results are also displayed in Table 3. Exposure concentrations to total welding fume for these support personnel ranged from 0.16 to 0.77 mg/m³. The mean exposure concentration was 0.40 mg/m³. None of the samples collected on the support personnel exceeded the OSHA PEL for total welding fume exposure.

Elemental Metals Exposures

The results of the elemental metal analyses for samples collected during welding and support operations are shown in Table 4. Nine personal breathing zone air samples were collected over a 3 day period on three employees engaged in welding operations. An additional six personal breathing zone samples were collected on maintenance personnel. The predominate metal identified on all samples was iron.

Other metals of significance identified on the samples collected on welders were chromium and nickel. Chromium was identified on all samples collected. Personal exposures to chromium ranged from 1 - 81 $\mu\text{g}/\text{m}^3$. The mean chromium exposure concentration of the samples collected was 21.5 $\mu\text{g}/\text{m}^3$, with a standard deviation of 26 $\mu\text{g}/\text{m}^3$. TWA concentration in all 15 samples collected on welders and maintenance personnel equaled, and 13 exceeded, the NIOSH Recommend Exposure Limit (REL) for chromium VI of 1 $\mu\text{g}/\text{m}^3$. Five of those samples were 20 times, and one 80 times higher than the NIOSH REL. None of the chromium exposure concentrations exceeded the OSHA PEL for chromium VI of 100 $\mu\text{g}/\text{m}^3$.

Nickel was detected in 10 of 15 samples collected on both welders and support personnel. Personal exposure concentrations to nickel ranged from not detected (ND) to 16 $\mu\text{g}/\text{m}^3$. The mean nickel exposure concentration was 7 $\mu\text{g}/\text{m}^3$, with a standard deviation of 5 $\mu\text{g}/\text{m}^3$. One nickel exposure concentration exceeded the NIOSH REL of 15 $\mu\text{g}/\text{m}^3$. None of the nickel exposures exceeded the OSHA PEL of 1000 $\mu\text{g}/\text{m}^3$.

Lead was detected on two samples collected on welders. Exposure concentrations on each sample were 2.7 $\mu\text{g}/\text{m}^3$. No lead exposures concentrations were above exposure criteria.

Carbon Monoxide

Results for the carbon monoxide samples collected during this survey are shown in Table 5. Personal breathing zone samples were collected on workers engaged in welding operations and support activities. Personal exposure concentrations for the samples collected on welders ranged from not detected (ND) to 30.3 ppm. The mean exposure concentration was 7.9 ppm. Personal exposure concentrations for the samples collected on support personnel ranged from ND to 3.7 ppm, with a mean exposure of 2.2 ppm. Only one sample collected exceeded the ACGIH TLV of 25 ppm for a TWA exposure to carbon monoxide.

Nitrogen dioxide

Results for the nitrogen dioxide samples collected during this survey are also shown in Table 5. Personal breathing zone samples were collected on workers engaged in welding operations and support activities. Personal exposure concentrations for the samples collected on welders ranged from ND to 0.41 ppm. The mean nitrogen dioxide exposure concentration for welders was 0.08 ppm. Personal exposure concentrations for the samples collected on support personnel ranged from 0.01 to 0.05 ppm, with a mean exposure of 0.03 ppm. None of the samples collected for nitrogen dioxide exceeded any exposure criteria.

Sulfur Dioxide

Results for the sulfur dioxide samples collected during this survey are also shown in Table 5. Personal breathing zone samples were collected on workers engaged in welding operations and

support activities. Personal exposure concentrations for the samples collected on welders ranged from ND to 3.66 ppm, with a mean exposure concentration of 1.5 ppm. Personal exposure concentrations for those samples collected on support personnel ranged from ND to 1.5 ppm, with a mean exposure of 0.46 ppm. Two samples collected on welders exceeded the NIOSH REL and the ACGIH TLV of 2 ppm for a TWA exposure. However, none of the samples collected exceeded the OSHA PEL of 5 ppm.

Medical Results

Questionnaire

A total of 48 male workers participated in the NIOSH medical evaluation. The median age of workers who participated was 46 years with a range of 23-64 years. The prevalence of current cigarette smoking was 25%. Current smokers had smoked a median of 20 pack-years (a pack-year is equivalent to smoking an average of 20 cigarettes per day for a year). Forty-two percent of participants reported that they were former smokers, having smoked a median of 17 pack-years. The remaining 33% of participants had never smoked. The median tenure of the participants was 5 years, with a range of 0-19 years. Tenure was calculated by subtracting date of hire from the interview date, rounded off to the nearest year. Because information on breaks in service was not included, tenure will be overestimated in anyone who was not continuously employed at the facility.

Individuals who participated were assigned to one of three exposure categories based on visual workplace observations of high or low dust exposure and involvement in the welding operation. The high dust exposure (HD) group worked on the shredding process, in housekeeping around the shredders, or on the landfill. The low dust exposure (LD) group included supervisors, workers involved in video-monitoring the truck loading operations, or in enclosed cab equipment. The welding (W) exposure group comprised welders and welders' helpers. The overall distribution of exposure categories were 15% (7/48) for welders, 40% (19/48) high dust exposure, and 46% (22/48) low dust exposure.

Responses to respiratory and mucous membrane irritation (nose, eye, skin) questions were categorized by exposure category and are displayed in Table 6.

Nasal Symptoms

Nineteen workers stated they experienced nasal stuffiness, with 11 indicating the onset of the nasal symptoms after beginning work at the facility. Of these 11 workers, one was a welder, 6 were in the low dust exposure, and 4 were in the high dust exposure category. Ten of the 19 symptomatic workers reported improvement in their symptoms away from the worksite

Dermatologic Symptoms

Of 13 workers who reported dermatologic symptoms; 9 indicated the skin problems began after starting the job at this facility. Of those nine workers, two were welders, five were from the low dust exposure, and two were from the high dust exposure category. Four of the 13 symptomatic workers mentioned improvement in symptoms away from work.

Eye symptoms

Twenty-five workers experienced eye symptoms. Fourteen stated that the symptoms began after starting work at this facility. Of those 14 workers, 3 were welders, 4 were from the low dust category, and 7 were from the high dust category. Seventeen of the 13 symptomatic workers reported improvement in symptoms away from the worksite.

Chest Tightness

Eleven workers mentioned that their chest felt tight or their breathing became difficult. Six of those workers reported the chest tightness began soon after entering the worksite, one worker developed the chest tightness 1-3 hours after leaving the worksite, and in four workers the chest tightness developed 3-8 hours after leaving the worksite.

Seven had chest tightness with headaches, muscle aches, and/or nausea/diarrhea symptoms consistent with occupational inflammatory lung disease (OILD). Of these 11 workers reporting chest tightness, 3 were welders, 4 worked in a low dust exposure, and 4 worked in a high dust exposure.

Nine of the 11 workers who reported chest tightness stated they also experienced mucous membrane (nasal) irritation. One worker who experienced chest tightness 3-8 hours after exposure denied mucous membrane symptoms but noted chest wheezing and whistling. He did not experience constitutional symptoms but reported childhood asthma. He was a non-smoker and had been employed 5 months prior to the study in a low dust exposure area. The other worker with chest tightness, but no nasal symptoms, also mentioned symptoms of wheezing and whistling in the chest. He had been employed for 5 months in a high dust exposed area.

Spirometry

Forty-eight workers completed one session of spirometry (Table 7), but only seven workers completed cross-shift spirometry. There were no FEV₁ declines observed in the cross-shift studies. Five of the 48 workers tested (10%) had abnormal spirometry. Four workers (8%) had mild obstructive patterns, one worker (2%) had a mild restrictive pattern.

Radiographic

None of the 48 chest x-rays taken on participants revealed pneumoconiosis. Two films revealed upper lung zone granulomas thought to suggest tuberculosis. Letters notifying employees of the possibility of tuberculosis were sent suggesting they review findings with their physicians; a copy of these letters were sent to the local health department. Both of these workers were involved in tipping floor/high biological dust exposure jobs.

DISCUSSION AND CONCLUSIONS

Three days after the initiation of the study, during the evening of August 23, Hurricane Andrew arrived. The eye of the hurricane passed over the facility and destroyed most of the buildings at the facility. The tipping floor and shredder facility, however, received minimal damage. Due to the devastation of the facility and the surrounding community, the study was terminated. After the hurricane, the majority of the peak flow devices were never recovered. Employees were individually notified of their chest x-ray and spirometry results. Both the industrial hygiene and medical aspects of the study were disrupted by the hurricane. Bioaerosol determinations were not completed. Because of the hurricane and incomplete data results, conclusions from the investigations are limited.

For example, cross-shift spirometry was only obtained on seven workers. None of these were in the group with chest tightness. Peak flow data were not available from any of the workers.

Three of the seven welders reported chest tightness and delayed systemic symptoms consistent with OILD that may represent a manifestation of metal fume fever. Fume exposures were noted to exceed recommended limits. Organic dust exposure may have occurred during welding in the shredder hoods.

Eleven of the 48 workers who completed a questionnaire complained of respiratory symptoms that were consistent with OILD. Whether these workers experienced Occupational Asthma, HP, ODTD, or Metal Fume Fever could not be determined with the data collected.

Since much of the worksite was destroyed by Hurricane Andrew, it is difficult to provide specific recommendations. Clearly, the potential exists for overexposure in this setting. Therefore, it is recommended that the County conduct a comprehensive evaluation of other similar operations within its jurisdiction and apply engineering controls and personal protective equipment where necessary.

REFERENCES

1. Rahkonen P, Ettala M, Loikkanen I. [1987]. Working conditions and hygiene at sanitary landfills in Finland. *Ann. Occup. Hyg.* Vol. 31, No. 4A, pp. 505-513.
2. Sigsgaard T, Bach B, Malmros P. [1990]. Respiratory impairment among workers in a garbage handling plant. *American Journal of Industrial Medicine* 17:92-93.
3. Malmros P., Sigsgaard T., Bach B. [1992]. Occupational health problems due to garbage sorting. *Waste Manage. Res.* 10:227-234.
4. Duce G. et.al. [1976]. Exposure of trash collectors to bacteria. *Sozial- und Praventivmedizin* 21(4):136-138.
5. Oregon Department of Human Resources [1991]. Report of illness investigation at Riedel Oregon Compost Company, Inc. Plant. Health Division, Office of Epidemiology & Health Statistics.
6. NIOSH [1984]. NIOSH manual of analytical methods. Eller PM, ed., 3rd rev. ed. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 84-100.
7. American Thoracic Society [1987]. Standardization of spirometry-1987 update. *Am Rev Resp Dis* 136:1285-1298.
8. Knudson RJ, Lebowitz MD, Holberg CJ, Burrows B [1983]. Changes in the normal maximal expiratory flow-volume curve with growth and aging. *American Review of Respiratory Disease* 127:725-734.
9. Lanese RR, Keller MD, Foley MF, Underwood EH [1978]. Differences in pulmonary function tests among whites, blacks, and american indians in a textile company. *Journal of Occupational Medicine* 20:39-44.
10. International Labour Office [1980]: Guidelines for the use of ILO international classification of radiographs of pneumoconiosis. Revised Ed. 1980. Geneva, Switzerland: International Labour Office, 1980 (International Labour Office: occupational safety and health series No. 22, Rev 80).

11. NIOSH [1992]. Recommendations for occupational safety and health: compendium of policy documents and statements. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 92-100.
12. ACGIH [1994]. 1994-1995 Threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienist.
13. CFR [1993] . Code of Federal regulations. Occupational Safety and Health Administration: air contaminants; final rule. Washington, DC: U.S. Government Printing Office, Office of the Federal Register.
14. Proctor NH, Hughes JP, et al (eds) [1991]. Chemical hazards of the workplace, 3rd ed. Van Norstrand Reinhold, New York.
15. NIOSH [1988]. Criteria for a recommended standard: occupational exposure to welding, brazing and thermal cutting. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control; National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 88-110.
16. ACGIH [1991]. Chromium (VI), water soluble fume. In: Documentation of the threshold limit values and biological exposure indices, Sixth ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, Inc, BEI-69.
17. NIOSH [1988]. NIOSH testimony on the Occupational Safety and Health Administration's proposed rule on air contaminants, August 1, 1998, OSHA Docket No. H-020, 29 CFR Part 1910. NIOSH policy statements. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health.
18. Hernberg S, et al [1988]. Lead and its compounds. In: Occupational medicine. 2nd ed. Chicago, IL: Year Book Medical Publishers.
19. Landrigan PJ, et al [1985]. Body lead burden: summary of epidemiological data on its relation to environmental sources and toxic effects. In: Dietary and environmental lead: human health effects. Amsterdam: Elsevier Science Publishers.
20. NIOSH [1978]. Occupational exposure to inorganic lead. Cincinnati, OH: U.S. Department of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHEW (NIOSH) Publication No. 78-158.

21. Pirkle JL, et al [1994]. The decline in blood lead levels in the United States, the National Health and Nutrition Examination Surveys (NHANES). *JAMA*, 272:284-291.
22. CFR. Code of Federal Regulations [1992]. OSHA lead standard. 29 CFR, Part 1910.1025. Washington, DC: U.S. Government Printing Office, Federal Register.
23. DHHS [1990]. Healthy people 2000: national health promotion and disease objectives. Washington, DC: U.S. Department of Health and Human Services, Public Health Service, DHHS Publication No. (PHS) 91-50212.
24. ATSDR [1990]. Toxicological profile for lead. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. DHHS (ATSDR) Publication No. TP-88/17.
25. NIOSH [1977]. Occupational diseases: a guide to their recognition. Revised Ed. Cincinnati, OH: U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, DHEW (NIOSH) Publication No. 77-181.
26. Bureau of Labor Statistics [1991]. Occupational injuries and illnesses in the United States by industry. Washington, DC: U.S. Department of Labor, 1988, Bulletin 2366.
27. Emmett E [1987]. Occupational dermatoses. In Fitzpatrick TB, Eisen AZ, Wolff K, et al (eds). *Dermatology in General Medicine*, 3rd ed, vol 1. New York: McGraw-Hill, pp 1567-1575.
28. Levy BS, Wegman DH [1983]. Occupational health recognizing and preventing work-related disease, 2nd ed. Boston, MA: Little, Brown, and Company.
29. Lammintausta K, Maiback HI [1990]. Contact dermatitis due to irritation. In Adams RM (ed): *Occupational Skin Disease*, 2nd ed. Philadelphia, PA: WB Saunders, pp 1-25.
30. Stewart L.A. [1992]. Occupational Contact Dermatitis. *Occupational Asthma and Allergies, Immunology and Allergy Clinics of North America*. Vol 12. No. 4., pp. 831-846.
31. Terr A.L. [1986]. The Atopic Worker. *Clinical Reviews in Allergy*, 54(3): 267-288.
32. Balmes JR [1991]. Surveillance for occupational asthma. *Occupational Medicine: State of the Art Reviews* 6:(1)101-110, January-March.
33. Newman-Taylor AJ [1980]. Occupational asthma. *Thorax* 35:241-245.

34. Chan-Yeung M, Lam S. [1990]. Evidence for mucosal inflammation occupational asthma. *Clinical and Experimental Allergy*, vol 20, pp 1-5.
35. NIOSH [1994]. NIOSH Alert: requests for assistance in preventing organic dust toxic syndrome. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication 94-102.
36. doPioico GA. [1986]. Report on Diseases (Diseases associated with the agricultural environment). *American Journal of Industrial Medicine* 10:261-265.
37. Parker JE, Siegel PD, Lewis DM . [1994]. Indoor air quality and non-IgE mediated immunologic respiratory disease. *Immunology and allergy clinics of North America*, Vol 14., No. 3 pp. 591-605.
38. Blanc P, Wong H, Berstein MS, Boushey HA. [1991]. An experimental human model of metal fume fever. *Annals of Internal Medicine*. Vol 114, No. 11 pp 930-936.
39. Farrell FJ. [1987]. Angioedema and urticaria as acute and late phase reactions to zinc fume exposure, with associated metal fume fever-like symptoms. *Am J Ind Med* 1987, 12, 331-337.
40. Nemery B. [1990]. Metal toxicity and the respiratory tract. *Eur Respir. J.* Vol 3, pp. 202-219.
41. Musk A.W., Venables K.M., Crook B., Nunn A.J., Hawkins R., Crook G.D.W., Graneek G.J., Tee R.D., Farrer N., Johnson D.A., Gordon D.J., Darbyshire J.H., Newman-Taylor A.J. [1989]. Respiratory symptoms, lung function, and sensitisation to flour in a British bakery. *Br. J. Ind. Med.* 46:636-642.
42. Newman-Taylor AJ., Gordon S. Laboratory animal and insect allergy. In "Asthma in the Workplace." Ed. by I. Leonard Bernstein et. al. pp. 399-414.
43. Chan-Yeung M., Barton G., MacLean L., Grzybowski S [1973]. Occupational asthma and rhinitis due to Western red cedar (*Thuja plicata*). *Am Rev Respir Dis* 108:1094-1102.

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1. Bureau of Consultation and Enforcement, Division of Safety, Department of Labor and Employment Security, State of Florida
2. Metropolitan Dade County, Department of Solid Waste Management
3. AFSCME Locals 1363 and 3290
4. Metropolitan Dade County, General Services Administration
5. Dade County Department of Public Health, Department of HRS
6. OSHA, Region IV

Table 1.
Air Sampling and Analytical Methods

Analyte	LOD per sample	Sampling and analytical methods
Welding Fume	0.02mg	Sample collected on pre-weighed, 37mm, 5.0 micron pore size filter at a flow rate of 2.0 liters per minute. Gravimetric analysis using NIOSH Analytical Method 0500.
Metals	0.1-10 µg ^A	Samples collected on 37mm, 0.8 micron pore size cellulose ester membrane filters in clear cassette holders with a flow rate of 1.0 lpm. Analysis for 30 elements by inductively coupled argon plasma, atomic emission spectroscopy--NIOSH Method 7300. ^B
Carbon Monoxide	6 ppm (8 hrs)	Direct-reading time-weighted average measurements were made with Dräger Carbon Monoxide 50/a-D diffusion tubes.
Nitrogen Dioxide	0.01 µg	Samples collected with diffusion tubes (Palmer tube with three triethanolamine-treated screens), analysis by visible absorption spectrophotometry--NIOSH Method 6700.
Sulfur Dioxide	0.7ppm (8 hrs)	Direct-reading time-weighted average measurements were made with Dräger Sulfur Dioxide 5/a-D diffusion tubes.

NOTES:

^A LOQ varied with analyte. Specific minimum quantifiable concentrations are reported with results.

^B Analytes were Ag, Al, As, Ba, Be, Ca, Cd, Co, Cr, Cu, Fe, La, Li, Mg, Mn, Mo, Na, Ni, P, Pb, Pt, Se, Sr, Te, Ti, Tl, V, Y, Zn, Zr.

Table 2.
Summary of Selected Occupational Exposure Limits and Health Effects

Substance (units)	NIOSH REL-TWA	OSHA PEL-TWA	ACGIH TLV-TWA	Primary Health Effects*
Welding Fume(NOC) (mg/m ³)	LFL	5	5	Iron oxide fume is the major metallic constituent emitted in this welding process, although chromium, nickel, manganese oxide, silicon dioxide, and fluoride are also likely to be present since the electrode contains these compounds. Inhalation of iron oxide may cause a benign pneumoconiosis known as siderosis, which may cause functional alterations in the lung.
Chromium VI (µg/m ³) (water-soluble)	1	100 (C) as CrO ₃	50	Respiratory system cancer. Some chromium VI (Cr ⁶⁺) compounds are severe irritants of the skin and respiratory systems, and cause sensitization dermatitis, kidney damage, asthma, and pulmonary edema.
Lead (µg/m ³)	<100	50	150	Weakness, irritability, gastrointestinal disturbance, reproductive and central nervous system effects, developmental effects, neuromuscular dysfunction, kidney damage.
Nickel, metal (µg/m ³)	15	1000	50 ^A	Sensitization dermatitis, asthma, pneumoconiosis, cancer of the lung, sinus, and nasal passages.
Carbon Monoxide (ppm)	35	50	25	The danger of this gas derives from its affinity for the hemoglobin of red blood cells, which is 300 times that of oxygen. The hazard of exposure to CO is compounded by the insidiousness with which high concentrations of carboxyhemoglobin (CO-Hb) can be obtained without marked symptoms.
Sulfur Dioxide (ppm)	2	5	2	Sulfur dioxide gas is a severe irritant of the eyes, mucous membranes, and skin. Its irritant properties are due to the rapidity with which it forms sulfurous acid on contact with moist membranes.
Nitrogen Dioxide (ppm)	1(STEL)	5 (C)	3	Respiratory irritation, severe exposures cause pulmonary edema and death.

* = Source: *Proctor and Hughes' Chemical Hazards of the Workplace, 3rd ed.*

C = Ceiling limit.

^A = Notice of intended change.

LFL = Lowest feasible limit.

Table 3.
Personal Sampling Results for Total Welding Fume

Job	Sample Number	Volume (m³)	Exposure (mg/m³)	Date
Welder	92429	0.85	0.35	19AUG92
Welder	92417	0.85	6.82	19AUG92
Welder	92425	0.86	7.29	19AUG92
Maint/Repair	92431	0.85	0.61	19AUG92
Welder	92428	0.82	0.33	20AUG92
Welder	92420	0.8	1.63	20AUG92
Welder	92418	0.79	22.13	20AUG92
Maint/Repair	92413	0.79	0.52	20AUG92
Maint/Repair	92415	0.8	0.35	20AUG92
Maint/Repair	92419	0.81	0.46	20AUG92
Maint/Repair	92427	0.8	0.23	20AUG92
Maint/Repair	92421	0.79	0.29	20AUG92
Welder	92450	0.73	0.39	21AUG92
Welder	92443	0.84	0.65	21AUG92
Welder	92444	0.68	2.12	21AUG92
Maint/Repair	92448	0.85	0.31	21AUG92
Maint/Repair	92414	0.74	0.47	21AUG92
Maint/Repair	92454	0.74	0.77	21AUG92
Maint/Repair	92412	0.85	0.16	21AUG92
Maint/Repair	92451	0.79	0.23	21AUG92
Minimum Detectable Concentration (MDC)			0.04 mg/m ³	

Table 4.
Personal Sampling Results of Elemental Metals Exposure ($\mu\text{g}/\text{m}^3$)

Job	Sample Number	Collection Date	Sample Vol. (m^3)	Chromium	Nickel	Lead
Welder	20751	19AUG92	0.72	20	5	ND
Welder	20754	19AUG92	0.73	81	16	3
Welder	20754	19AUG92	0.73	20	14	ND
Maint/Repair	20749	19AUG92	0.73	2	ND	ND
Welder	20757	20AUG92	0.67	60	13	3
Welder	20729	20AUG92	0.62	1	ND	ND
Welder	20761	20AUG92	0.7	19	6	ND
Welder	20742	20AUG92	0.68	20	4	ND
Maint/Repair	20750	20AUG92	0.68	2	ND	ND
Maint/Repair	20735	20AUG92	0.67	2	ND	ND
Maint/Repair	20738	20AUG92	0.64	10	1	ND
Welder	20730	21AUG92	0.72	10	2	ND
Welder	20744	21AUG92	0.58	17	5	ND
Maint/Repair	20748	21AUG92	0.63	10	1	ND
Maint/Repair	20759	21AUG92	0.72	1	ND	ND
Minimum Detectable Concentration (MDC)				1	1	2

ND = Not Detected

Table 5.**Personal Sampling Results of TWA Gas Exposures**

Job	Nitrogen Dioxide (ppm)	Carbon Monoxide (ppm)	Sulfur Dioxide (ppm)	Collection Date
Welder	0.06	14.1	0.98	19AUG92
Welder	0.02	1.39	0.69	19AUG92
Welder	0.03	ND	ND	19AUG92
Maint/Repair	0.03	3.51	0.7	19AUG92
Welder	0.41	30.3	1.52	20AUG92
Welder	ND	ND	ND	20AUG92
Welder	0.006	3.66	3.66	20AUG92
Welder	0.06	4.48	2.98	20AUG92
Maint/Repair	0.04	ND	ND	20AUG92
Maint/Repair	0.03	3.78	1.5	20AUG92
Maint/Repair	0.03	3.68	0.73	20AUG92
Welder	0.02	0.71	0.71	21AUG92
Welder	0.04	8.72	1.74	21AUG92
Maint/Repair	0.01	3.64	0.72	21AUG92
Maint/Repair	0.05	ND	ND	21AUG92
Maint/Repair	0.03	3.55	ND	21AUG92
Maint/Repair	0.04	ND	ND	21AUG92
Maint/Repair	0.02	ND	ND	21AUG92

ND = Not Detected

Table 6.
Reported Symptom Prevalence for Current Workers

	Welders		High Dust		Low Dust		Total Group	
	N=7*		N=19		N=22		N=48	
	<i>No.</i>	<i>%</i>	<i>No.</i>	<i>%</i>	<i>No.</i>	<i>%</i>	<i>No.</i>	<i>%</i>
Chronic cough	3	43	3	16	1	5	7	15
Chronic phlegm	2	29	3	16	4	18	9	19
Exertional dyspnea	1	14	1	5	-	-	2	4
Asthma -Doctor Dx	1	14	-	-	1	5	2	4
Symptoms of HP	3	43	2	11	2	9	7	15
Nasal Symptoms	3	43	6	32	10	45	19	40
Eye symptoms	4	57	9	47	12	55	25	52
Skin Symptoms	2	29	2	11	9	41	13	27
Chest Tightness	3	43	4	21	4	18	11	23
Wheeze	3	43	4	21	6	27	13	27
Asthma-symptoms	1	14	1	5	-	-	2	4

* N = number of workers

Table 7.
Pulmonary Function Test Results

SPIROMETRY MEASUREMENT	Welders (n=7*)		High Dust (n=19)		Low Dust (n=22)		TOTAL (n=48)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
FVC (liters)	4.02	0.51	4.27	0.72	4.44	0.92	4.3	0.79
% Predicted FVC	89.1	11.3	98.5	13.7	97.0	12.2	96.4	12.8
FEV₁	3.3	0.4	3.45	0.66	3.48	0.77	3.44	0.7
% Predicted FEV₁	88.2	8.8	96.1	13.7	92.8	15.1	93.4	13.8
FEV₁/FVC (%)	81.6	5.3	80.6	5.9	78.1	5.9	79.6	5.9

* N = number of workers

