This Health Hazard Evaluation (HHE) report and any recommendations made herein are for the specific facility evaluated and may not be universally applicable. Any recommendations made are not to be considered as final statements of NIOSH policy or of any agency or individual involved. Additional HHE reports are available at http://www.cdc.gov/niosh/hhe/reports

HETA 94-0122-2578 Bath Iron Works Corporation Bath, Maine

David C. Sylvain, M.S., CIH

PREFACE

The Hazard Evaluations and Technical Assistance Branch of 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 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.

The Hazard Evaluations and Technical Assistance Branch 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 the National Institute for Occupational Safety and Health.

ACKNOWLEDGMENTS AND AVAILABILITY OF REPORT

This report was prepared by David C. Sylvain, M.S., CIH, of the Hazard Evaluations and Technical Assistance Branch, Division of Surveillance, Hazard Evaluations and Field Studies (DSHEFS). Field assistance was provided by Mark F. Greskevitch and methods for the analysis of steel shot were developed by Mark Millson. Desktop publishing by Pat Lovell.

Copies of this report have been sent to employee and management representatives at BIW and the OSHA Regional Office. This report is not copyrighted and may be freely reproduced. Single copies will be available for a period of three years from the date of this report. To expedite your request, include a self-addressed mailing label along with your written request to:

NIOSH Publications Office 4676 Columbia Parkway Cincinnati, Ohio 45226 800-356-4674

After this time, copies may be purchased from the National Technical Information Service (NTIS) at 5825 Port Royal Road, Springfield, Virginia 22161. Information regarding the NTIS stock number may be obtained from the NIOSH Publications Office at the Cincinnati address.

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.

Health Hazard Evaluation Report 94-0122-2578 Bath Iron Works Corporation Bath, Maine May 1996

David C. Sylvain, M.S., CIH

SUMMARY

In December 1993, the National Institute for Occupational Safety and Health (NIOSH) received a Health Hazard Evaluation (HHE) request from Bath Iron Works Corporation (BIW) Occupational Safety and Health Department, Bath, Maine, to evaluate the accuracy of personal air monitoring conducted during abrasive blasting in confined spaces. The BIW industrial hygiene staff provided information indicating that abrasive grit particles enter environmental air sampling cassettes, resulting in an overestimation of worker exposure to inhalable airborne metals.

On March 31, 1994, an opening conference was held and a walk-through evaluation of the abrasive blasting operation was conducted. On July 19-21, 1994, 15 personal breathing zone (PBZ) air samples were collected during manual abrasive blasting in confined spaces. A second sample, collected concurrently with each PBZ sample, was obtained behind each blaster's head to determine if sample placement had an effect on reducing the amount of grit entering the cassette. In addition to analysis for lead and selected metals, a visual assessment of the grit in the cassettes was made. On February 28-March 2, 1995, ten sets of side-by-side PBZ air samples were collected during abrasive blasting inside a 7' x 4' x 2' steel tank. Each set consisted of a sample using a standard closed-face cassette; a closed-face cassette fitted with a metal guard to shield the inlet from high-velocity grit ("grit guard"); and a sample using a 10mm nylon cyclone at a flow rate of 300cc/minute. Prior to sample analysis, the steel grit was separated from the filters and dust in the closed-face cassettes; the fractions were analyzed separately.

Although only low levels of lead and other contaminants were present in the base-metal, surface coating, and steel grit, the relatively large mass of grit that entered the air sampling cassettes resulted in an overestimation of the airborne lead concentration. Evaluation of air samples collected during this HHE indicates that conventional air sampling methods over estimate concentrations of lead and other inhalable airborne contaminants present in abrasive blasting environments. Use of the metal guard, and locating sampling cassettes behind the head (or body) were not reliable methods for preventing grit from entering cassettes. Grit did not penetrate most of the cyclones in the test tank; however, cyclones would not be useful under actual blasting conditions, because they would be inverted as the blasters climbed through confined spaces. Neither grit guards or placement of several samples appear to effectively control entrance of abrasive blasting grit.

Conventional sampling and analytical methods do not provide an accurate estimate of airborne concentrations of lead and other contaminants present during abrasive blasting in small, confined spaces. Abrasive grit enters the cassette inlet due its high velocity (inertia), resulting in an overestimation of actual airborne contaminant concentrations. Neither grit guards or placement of personal samples appear to effectively control entrance of abrasion blasting grits.

Keywords: SIC 3731 (Ship building and repairing), abrasive blasting, air sampling, elemental metals, lead.

TABLE OF CONTENTS

Preface ii
Acknowledgments and Availability of Report ii
Summaryiii
Introduction
Background 2
Methods3Air Samples3Bulk Samples4
Evaluation Criteria4Lead5Lead in surface dust and soil6Lead - childhood exposure6Cadmium6Chromium8
Results
Discussion
Conclusions 11
References 11

INTRODUCTION

In December 1993, the National Institute for Occupational Safety and Health (NIOSH) received a Health Hazard Evaluation (HHE) request from Bath Iron Works Corporation (BIW) Occupational Safety and Health Department, Bath, Maine. This was a request for assistance in evaluating the accuracy of sampling methods used to collect personal air samples during abrasive blasting in confined spaces. The BIW Industrial Hygiene Division had collected sampling data which indicated that large, high-velocity grit particles enter sampling cassettes, resulting in an overestimation of worker exposure to lead.

An accurate estimate of lead exposure is needed to select appropriate personal protective equipment, conduct appropriate medical monitoring, and implement appropriate administrative controls. The Occupational Safety and Health Administration (OSHA) lead standard (29 CFR 1910.1025) requires that controls be implemented if personal exposure monitoring indicates that employees are exposed to lead concentrations which exceed the OSHA action level or permissible exposure limit. The lead standard prescribes an acceptable level of accuracy for the sampling and analytical method used to evaluate workplace exposure to lead. The NIOSH Manual of Analytical Methods, OSHA compliance directives, and OSHA instructions provide guidance on various sampling issues, such as calibration of sampling pumps, and sampling flow rate; however, no guidance is provided to address the issue of large, noninhalable particles found in samples obtained during abrasive blasting in confined spaces. Since there is no acceptable method for removing blasting grit from sampling cassettes prior to sample analysis, the grit is included in the analysis. Even a small quantity of steel grit can result in an overestimate of the concentration of inhalable lead particulate.

On March 31, 1994, an opening conference was held and a walk-through evaluation of the abrasive

blasting operation was conducted. Air sampling was conducted during subsequent site visits on July 19 - 21, 1994, and February 28 - March 2, 1995.

BACKGROUND

BIW is a large shipyard on the coast of Maine which employed approximately 9,000 workers at the time of this evaluation. Operations at BIW are primarily involved with the construction of U.S. Navy Aegis destroyers. The destroyers are constructed in large sections known as units, which undergo a sequential construction process at various locations throughout the shipyard. When the hull, decks, bulkheads, tanks, and compartments of a unit have been constructed from steel plate, the unit is moved into one of two blast buildings (Blast-1 and Blast-2) where it undergoes manual abrasive blasting to remove scale, rust, and zinc-based preconstruction primer. When structural work is completed, the unit is transported to the ways where it is hoisted into place, and assembled with other units. Construction continues upon the ways, as the ship is fitted-out in preparation for launching.

Mild and alloy steel plate is used in the construction; no leaded steel is used. The abrasive blasting material is angular steel grit which is collected and reused. The floor of each blast building consists of a steel grate which allows used grit to be collected by a mechanical sweeper system which returns the grit to a classifier where fines are removed. Fines include rust, paint, base-steel, and disintegrated grit. Grit that is deposited in the unit during blasting is removed by vacuuming. After passing through a classifier, the grit is returned to the grit pots for reuse. Grit is periodically replenished with virgin grit, as disintegrated grit is removed by the Blast-1 is equipped with two classifier. classifiers, and Blast-2 has one classifier. General mechanical ventilation is provided in each blast building.

Each blast building is equipped with two grit pots, each of which serves eight blasters. Since all surfaces must be prepared for painting, the blasters have to climb over and around baffles, stiffeners, and other obstacles to reach the tanks, bilges, and other remote locations within the units. When blasting in tight spaces, the blaster and air monitoring equipment are exposed to grit that rebounds off work surfaces.

Blasters wear personal protective equipment (ppe) which includes gloves, boots, coveralls (taped at the boots), and an MSA[®] continuous-flow supplied-air blast hood. Facilities provided at the site include separate lockers for street and work clothes, and mandatory showers. Blasters are required to participate in a medical monitoring program, which includes physical examinations and blood-lead monitoring.

METHODS

Air Samples

This evaluation consisted of observation of work practices (including the use of ppe), and environmental sampling in the blast buildings. On July 19-21, 1994, 15 personal breathing zone (PBZ) air samples were collected during abrasive blasting inside of two units. A second sample, collected concurrently with the PBZ sample, was obtained behind each blaster's head. Each sample was obtained using a battery-powered sampling pump to draw air through a 37 millimeter (mm) diameter, 0.8 micrometer (um) pore-size cellulose ester membrane (MCE) filter mounted in a closedface cassette. The pumps were operated at a nominal flow rate of 2.0 liters per minute (lpm), and were calibrated before and after sampling to ensure that the desired flow rate was maintained throughout the sampling period. Air samples were analyzed for lead, and selected elemental metals, according to NIOSH Method 7300 modified for microwave digestion and standard matrix matching of samples. Analysis was performed using a Thermo Jarrell Ash ICAP-61

inductively coupled plasma (ICP) emission spectrometer controlled by a Digital DEC Station 333c personal computer. Analysis for lead was repeated according to NIOSH Method 7105 (atomic absorption spectroscopy). In addition, the analyst provided a visual assessment of the quantity of grit in the sampling cassettes, and noted whether there was an appreciable difference between the amount of grit in PBZ and behindthe-head samples.

On February 28-March 2, 1995, ten sets of sideby-side PBZ air samples were collected during abrasive blasting inside of a 7' x 4' x 2' steel tank. Each set of samples consisted of one sample obtained on a 37 mm diameter, 0.8 µm pore-size MCE filter mounted in a closed-face cassette: a second sample was collected using a standard closed-face cassette which was fitted with a "grit guard"; and a third sample was collected on a MCE filter using a 10 millimeter nylon cyclone. The pumps that were used to collect samples in closed-face cassettes were operated at a nominal flow rate of 2.0 lpm. Cyclone samples were obtained at a nominal flow rate of 300 cubic centimeters per minute (cc/m) in an attempt to collect a thoracic cut, i.e., particulate which would be deposited within the airways and gas-exchange region of the lung. Each sampling pump was calibrated before and after sampling to ensure that the desired flow rate was maintained throughout the sampling period.

Prior to analysis, closed-face cassettes were opened by the analyst, and the steel grit particles were separated from the dust in each sample using a rubber-coated magnet. Deionized water was used to rinse residual dust from the shot into a beaker containing the filter. The steel grit was scraped into a separate beaker; and the magnet was rinsed with 10 percent nitric acid. Air samples (including cyclone samples) were analyzed for lead, and selected elemental metals according to NIOSH Method 7300.

Bulk Samples

On July 20 and 21, 1994, bulk samples of used abrasive blasting grit, fines, paint (primer), and base metal (steel) were collected. Grit was obtained from the bilge of the unit in Blast-1, and from the grit pots in both blast buildings. A sample of fines was obtained from classifiers in each building. Samples of weldable primer and steel plate were provided by the BIW laboratory. Bulk samples were analyzed for lead, cadmium, and arsenic according to NIOSH Method 7300.

During the 1995 site visit, one sample each of virgin and used abrasive blasting grit was collected. Five replicate aliquots of each grit sample were weighed and then placed in 15 ml graduated centrifuge tubes. Ten ml of five percent nitric acid was added to each sample which was then sonicated for 15 minutes. After two hours, the leachates were decanted into separate containers. To each grit residue, 0.5 ml of a 4% nitric acid-1% perchloric acid solution was added, and the samples were allowed to stand at room temperature until complete dissolution had occurred. The samples were then diluted with de-ionized water to 10.0 ml final volume, and were analyzed by ICP-AES.

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 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 $(RELs)^1$, (2) the American Conference of Governmental Industrial Hygienists' (ACGIH) Threshold Limit Values $(TLVs^{TM})^2$, and (3) the U.S. Department of Labor, OSHA Permissible Exposure Limits (PELs)³. 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 time-weighted average (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.

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."^{4, 5, 6} 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.⁷ 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 (μ g/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 μ g/dL, and more than 98% have a BLL <15 μ g/dL.⁸

Under the OSHA general industry lead standard (29 CFR 1910.1025), the PEL for airborne exposure to lead is $50 \mu g/m^3$ (8-hour TWA).³ 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 μ g/m³ (8-hour TWA), medical removal of employees whose average BLL is 50 µg/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 $40 \,\mu g/dL$. 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 adopted a TLV for lead of 50 μ g/m³ (8-hour TWA), with worker BLLs to be controlled to at or below $30 \mu g/dL$, and designation of lead as an animal carcinogen.² 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 μ g/dL.⁹

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 $60 \mu g/dL$, and decreased fertility in men at BLLs as low as 40 $\mu g/dL$. BLLs are associated with increases in blood pressure, with no apparent threshold through less than 10 $\mu g/dL$. Fetal exposure to lead is associated with reduced gestational age, birth weight, and early mental development with maternal BLLs as low as 10 to $15 \mu g/dL$.¹⁰ Men and women who are planning on having children should limit their exposure to lead.

In homes with a family member occupationally exposed to lead, care must be taken to prevent "take home" of lead, that is, lead carried into the home on clothing, skin, and hair, and in vehicles. High BLLs in resident children, and elevated concentrations of lead in house dust, have been found in homes of workers employed in industries associated with high lead exposure.¹¹ Particular effort should be made to ensure that children of persons who work in areas of high lead exposure receive a BLL test.

Lead in surface dust and soil

Lead-contaminated surface dust and soil represent potential sources of lead exposure, particularly for young children. This may occur either by direct hand-to-mouth contact, or indirectly from hand-tomouth contact with contaminated clothing, cigarettes, or food. Previous studies have found a significant correlation between resident children's BLLs and house dust lead levels.¹² There is currently no federal standard which provides a permissible limit for lead contamination of surfaces in occupational settings. As required by Section 403 of the Toxic Substances Control Act (TSCA), EPA is in the process of developing a rule to address hazards from lead-contaminated dust and soil in and around homes.

EPA currently recommends the following clearance levels for surface lead loading be met after residential lead abatement or interim control activities: uncarpeted floors, 100 micrograms per square foot (μ g/ft²); interior window sills, 500 μ g/ft², and window wells, 800 μ g/ft^{2.13} These levels have been established as achievable through lead abatement and interim control activities, and they are not based on projected health effects associated with specific surface dust levels.

EPA currently recommends a strategy of scaled responses to soil lead contamination, depending upon lead concentrations and site-specific factors. When lead concentrations exceed 400 ppm in bare soil, EPA recommends further evaluation and exposure reduction activities be undertaken, appropriate to the site-specific level of risk. If soil lead concentrations exceed 5000 ppm, EPA recommends permanent abatement of contaminated soil.¹³

Lead-childhood exposure

The adverse effects of lead on children and fetuses include decreases in intelligence and brain development, developmental delays, behavioral disturbances, decreased stature, anemia, decreased gestational weight and age, and miscarriage or stillbirth. Lead exposure is especially devastating to fetuses and young children due to potentially irreversible toxic effects on the developing brain and nervous system.¹⁰

No threshold has been identified for the harmful effects of lead in children; the Centers for Disease Control and Prevention (CDC) currently recommends a multitier approach to defining and preventing childhood lead poisoning, based on BLL screening.¹⁴ The BLLs and corresponding actions which CDC has recommended are: ≥ 10 $\mu g/dL$, community prevention activities; ≥ 15 µg/dL, individual case management including nutritional and educational interventions and more frequent screening; $\geq 20 \ \mu g/dL$, medical evaluation, environmental investigation and Additionally, environmental remediation. investigation and remediation are recommended for BLLs of 15-19, if such levels persist.

Overall, U.S. population blood lead levels have declined since 1976. A recent national survey found that the geometric mean BLL for children ages 1-11 ranged from 2.5-4.1 μ g/dL, with the highest mean BLL among children aged 1-2 years.¹⁵ However, it was estimated from the survey that 8.9% of U.S. children under 6 years, or about 1.7 million children, have elevated BLLs ($\geq 10 \mu$ g/dL).

Cadmium

Cadmium is a toxic heavy metal used in the manufacture of batteries, pigments, and jewelry, and as a neutron absorber in nuclear reactors. Cadmium may enter the body either by ingestion (swallowing) or by inhalation (breathing) of cadmium metal or oxide. Approximately 6 to 10% of ingested cadmium and 15 to 30% of inhaled cadmium is absorbed into the body.¹⁶ Cadmium is transported from the site of absorption by the red blood cells and plasma. It is deposited in organs throughout the body, but major depositions occur in the liver and kidneys. Under normal conditions, the kidneys accumulate

the greatest concentrations of cadmium. Cadmium is excreted from the body only very slowly, and thus accumulates in the cortex of the kidneys over a lifetime. The blood cadmium concentration is the best biological indicator of recent cadmium exposure and absorption.^{17,18} The normal blood cadmium level is below 0.7 µg/dL. Chronic cadmium exposure can be assessed by measuring the cadmium content of the kidneys through the technique of neutron activation analysis.¹⁹ The urine cadmium concentration, although used widely as an index of exposure, is primarily an indicator of cadmium-induced kidney damage; the urine cadmium concentration does not ordinarily begin to increase until after injury has occurred to the kidneys.²⁰

Acute inhalation exposure to cadmium can cause pneumonia or pulmonary edema,²¹ as well as liver and kidney damage.²² Ingestion of toxic quantities can produce nausea, vomiting, and diarrhea. Exposure to an airborne concentration of 50 mg/m³ cadmium dust is considered immediately dangerous to life.

Occupational exposure to cadmium is usually chronic. Chronic occupational exposure to cadmium can produce toxic effects, including emphysema of the lungs and chronic kidney disease.¹⁶ Kidney disease, resulting from cadmium exposure, is a principal concern.¹⁶ Although much remains to be learned about the development of kidney disease in persons exposed to cadmium, the process appears to be a gradual one.²⁰ Also, the process is dose-related; persons with greatest lifetime absorption of cadmium are at greatest risk of kidney disease (nephropathy). The initial signs of cadmium nephropathy are subtle--affected workers will usually have no symptoms in the early stages, and their kidney function test results may still be within the broad range of normal, although their test results will tend over time to move toward the high end of the normal range.

Because the kidney has an enormous reserve capacity, results of the usual renal function

tests--blood urea nitrogen (BUN), serum creatinine, and serum uric acid--will not become frankly abnormal until one-third to one-half of kidney function has been destroyed.²³ For that reason, more sensitive screening tests of renal function have been sought. These include measurement of serum concentrations of 1,2,5-dihydoxy vitamin D (which may be decreased),²⁴ and measurement of urine concentrations of cadmium and of protein beta-2-microglobulin (both of which are reported to increase in persons with kidney damage caused Also, aminoaciduria, renal by cadmium).²⁵ glycosuria, or hyperphosphaturia may develop.

When any of these test results are abnormal in a person exposed to cadmium, or even when two or more test results are in the high normal range, there exists a possibility of kidney damage. In that circumstance, more complete evaluation of the worker by a kidney disease specialist is required.

Occupational cadmium exposure has been associated with cancer of the prostate gland.²⁶ In 1976, a NIOSH study of workers in a cadmium production factory provided limited evidence that occupational cadmium exposure may be associated with lung cancer.²⁷ Subsequent to the 1976 report, the cohort was expanded from the original 292 workers to 602 white males who had worked at least six months between 1940 and 1969 in the cadmium production area of the cadmium smelting plant²⁸ In this study, the mortality from cancer of the respiratory tract was significantly greater in the entire cohort than would have been expected from rates in the general U.S. population. This study found that lung cancer mortality increased with increasing cumulative exposure to cadmium.

Under the OSHA general industry cadmium standard (29 CFR 1910.1027), the PEL for airborne exposure to cadmium is 5 μ g/m³, calculated as an 8-hour TWA. The standard requires medical monitoring for employees exposed to airborne cadmium at or above the

action level of 2.5 μ g/m³ (8-hour TWA); and requires medical removal of employees based upon the physician's determination. The OSHA final rule for cadmium in the construction industry (29 CFR 1926.63) provides an equivalent level of protection for construction workers.²⁹ ACGIH has adopted a TLV for cadmium of 0.01 μ g/m³ (8hour TWA) for inhalable (total) dust/particulate; and 0.002 μ g/m³ for the respirable fraction. NIOSH classifies cadmium as a potential occupational carcinogen, and recommends that occupational exposures be reduced to the "lowest feasible level" (LFL) because it is not possible to establish thresholds for carcinogens which will protect 100% of the population.

Chromium

Chromium (Cr) exists in a variety of chemical forms, and toxicity varies among the different forms. For example, elemental chromium is relatively non-toxic.³⁰ The PEL for chromium metal is an 8-hour TWA concentration of 1 mg/m³. The ACGIH TLV, and NIOSH REL for chromium metal, and chromium (II) and chromium (III) compounds, is 0.5 mg/m³.

Other chromium compounds may cause skin irritation, sensitization, and allergic dermatitis. In the hexavalent form (Cr(VI)), Cr compounds are corrosive, and possibly carcinogenic. Until recently, the less water-soluble Cr(VI) forms were considered carcinogenic while the water-soluble forms were not considered carcinogenic. Recent epidemiological evidence indicates carcinogenicity among workers exposed to soluble Cr(VI) compounds.^{31,32,33,34,35} Based on this evidence, NIOSH recommends that all Cr(VI) compounds be considered as potential carcinogens.

RESULTS

Air samples collected on July 19 - 21, 1994, were analyzed for inorganic arsenic, lead, and cadmium according to NIOSH Method 7300 (inductively coupled plasma-atomic emission spectroscopy). Due to high iron content in the samples, numerous dilutions were required to eliminate spectral saturation interferences. As a consequence, the analytical limits of detection and quantitation were increased up to forty times the limits listed for this method, thus rendering this analysis insensitive and unreliable. Analysis for lead and elemental chromium was repeated according to NIOSH Methods 7105 (atomic absorption spectroscopy) and 7300 respectively. Analysis for elemental chromium was initiated by the laboratory due to high chromium concentrations detected during the first analysis. Results of the repeat analysis are presented in Table 1. For sideby-side samples where the quantity of grit in each cassette could be visually assessed, analysis of the air sample filters (and loose grit) revealed that the cassette containing the greatest amount of grit generated results indicating the highest air contaminant concentration. The exception to this observation in sample set P12 - BH12, appears to be due to the marked difference between the sample volumes for P12 and BH12 (212 L vs. 4.82 L).

Analysis of bulk samples of used grit collected during the July 1994 visit revealed concentrations of elemental lead, cadmium, and arsenic in the range of 43 to 50 μ g/g (Table 2; samples K23 -K25). The lead content of classifier fines appeared to be somewhat higher than in the grit; while the arsenic concentration in the fines was less than that in grit samples. Forty to 97 μ g/g lead was detected in paint and base-metal samples. No arsenic was detected in paint or base-metal samples; cadmium concentrations in these samples were less than was found in grit.

Results of air samples collected on February 28 through March 2, 1995, are presented in Table 3.

Eight samples exceeded the OSHA PEL for lead when the steel grit fraction was included in the calculation of the 8-hour TWA concentration. Four of the 8 samples exceeded the OSHA PEL for elemental chromium when the steel grit fraction was included. None of the results exceeded the PELs if the contribution of the grit fraction was not included. The minimum quantifiable concentration (MOC) for all of the cadmium analyses was greater than the OSHA PEL; therefore, cadmium results could not be evaluated in terms of the OSHA cadmium standard, 29 CFR 1910.1027. Similarly, the MQC for arsenic analyses was greater than the REL and/or PEL; and, as a consequence, the air sampling conducted for arsenic during this visit could not be evaluated with respect to these limits.

Analysis of surface dust, extracted from bulk samples of abrasive blasting grit, determined that lead and cadmium concentrations in the dust were below the analytical limit of quantitation (LOQ) (Table 4). Dissolution and analysis of virgin grit (after extraction of surface dust) detected average lead and cadmium concentrations of $115 \,\mu$ g/g, and $15.4 \,\mu$ g/g respectively. Dissolution and analysis of used grit determined the average lead and cadmium content of used grit to be 90.8 μ g/g and 24.5 μ g/g.

DISCUSSION

In the absence of guidelines for the analysis of non-inhalable particles, all dust and grit is digested, analyzed, and reported as airborne contaminant(s): in this case, lead and selected elemental metals. Analysis of side-by-side air samples obtained during manual abrasive blasting in July 1994, appears to support the supposition that analysis of steel grit found in sample cassettes contributes substantially to sampling results: as expected, the analysis revealed that the cassette which contained the greater quantity of grit generated a higher estimate of airborne lead concentration than did the corresponding sample. This observation was based on a visual comparison of the quantity of grit inside of paired cassettes in relation to analytical results. This analysis did not attempt to distinguish between particulate that was collected on the filter, dust on the surface of the grit, and the contaminants which comprise the steel grit itself. Figure 1 shows an open cassette with grit that accumulated during abrasive blasting sampling.

Bulk samples had similar lead concentrations in the base metals, zinc-based primer, and used grit. The highest concentration of lead $(97\mu g/g)$ was in steel used to construct internal stiffeners, which comprise little of the surface area within the units. Although the composition of the grit was similar to that of the base metals and primer, much more grit entered the sample cassettes during sampling than did primer or base metals. Thus, the factor which appeared to have the greatest impact on sample results was the amount of grit in the cassettes, and the lead contamination contributed by the grit.

The intent of paired samples (PBZ and behind the head), was to obtain a qualitative assessment of the effect of "shielding" the cassette from rebounding grit. The PBZ sample contained more grit in five sets; the behind-the-head sample contained more grit in two sets; and the amount of grit was determined to be essentially the same in one sample set. These results indicate that locating the cassette behind the head (or body) is not a reliable method of preventing rebounding grit from entering sampling cassettes. The space within tanks, bilges, etc. is extremely small, and grit enters the cassettes regardless of location.

Difficulties encountered during sampling included equipment and sampling train damage/failure; and sample overloading. The sampling train was often damaged as the workers moved over, under, and around obstacles within the units. Damage included broken cassettes, torn Tygon tubing, and detached pump belt clips. Due to the inaccessibility of the work areas, it was not possible to change cassettes to prevent cassettes from becoming overloaded with grit.

During the 1995 visit, sampling was conducted inside of a tank that the BIW Industrial Hygiene Division had constructed for the purpose of collecting sampling data. The tank provided a relatively uniform, controlled environment in which sampling could be conducted with minimal sample loss. The tank was constructed of the same grade of steel as the units, and was primed with the same zinc-based primer prior to blasting. Unlike actual spaces within the units, this tank allowed the blaster to remain in an upright position, and allowed for collection of samples using nylon cyclones. Cyclones could not be used because they would be inverted as the blasters climbed through the units.

Most cyclone sampling results approximated the filter and dust results that were obtained using closed-face cassettes. Grit did not penetrate most cyclones; however, sample 3C contained only steel grit and no dust. The absence of dust in this sample was due to failure of the sampling pump after 7 minutes of sampling. Although the pumps were operated at 300cc/min in an attempt to collect a thoracic cut, it is not known if this size range was actually collected. Thus, it is not possible to evaluate the performance of the cyclones in the test tank. However, if cyclones performed satisfactorily in a test environment, they could not be used in the units.

Side-by-side sampling was conducted using a conventional closed-face cassette; a conventional closed-face cassette fitted with a "grit guard;" and a 10mm nylon cyclone operated at a flow rate of 300cc/minute. (A grit guard is a metal guard, developed by BIW, that clips onto a closed-face cassette to shield the inlet from high-velocity abrasive grit.) Because the grit in the cassettes was not weighed, the effectiveness of the grit guard could not be ascertained directly. Using lead-content (μ g/sample) as a benchmark, 8 of 10 sample sets had less grit in the cassette when fitted with the grit guard than in the conventional closed-face cassette. However, in three of these

sample sets, chromium analysis produced contradictory results.

Prior to the second round of sampling, attempts were made to identify a size-selective sampler that could be used in the presence of high-velocity grit in the blast environment. When an appropriate size-selective sampler could not be identified, a method for separating grit from the samples was proposed. The initial method specified the use of a 100 μ m sieve to separate large particles from filters and dust. However, when none of the grit would pass through the sieve, the use of the sieve was discontinued, and a rubber-coated magnet was used to remove steel grit. Grit, similar to the grit shown in figure 1, was removed from the samples using the magnet.

Separation of grit from filters and dust provided further evidence that steel grit in the cassettes produced results which overestimated the actual concentrations of airborne lead and other metals. In 18 of the 21 samples where lead was present, only the grit fraction produced results above the LOQ. In eight samples, the 8-hour TWA concentration exceeded the lead PEL when grit was included; however, results were below the LOQ when the grit fraction was not included in the calculation.

Analysis of bulk samples of virgin and used grit indicated that it is the grit, not dust on the surface of the grit, that contributes the greatest amount of lead and other metals. Analysis of dust, which was extracted (removed) from the surface of used and virgin grit, revealed that lead and cadmium concentrations in the dust were below the LOQ (10 μ g/g). The composition of used and virgin grit appeared to differ with respect to lead, cadmium and chromium. It is possible that these samples represent grit from different manufacturers, or grit from different lots.

CONCLUSIONS

The current NIOSH sampling and analytical method(s) does not accurately represent worker exposure to lead and other elements during abrasive blasting in small, confined spaces. This inaccuracy arises as the result of abrasive blasting grit which enters the cassette inlet due to its high velocity (inertia), rather than being collected on the filter as an airborne contaminant in an air sample of known volume. In the absence of guidelines for the analysis and interpretation of non-inhalable particle sampling data, all dust and grit is digested, analyzed, and reported as an airborne contaminant (in this case, lead and The sampling conducted elemental metals). during this HHE indicates that accumulations of steel grit in sampling cassettes can produce results which overestimate the concentration of inhalable lead particulate. Sampling conducted during this HHE points to the need for a sampling analytical method that can provide an accurate estimate of airborne exposure to lead and other elements during abrasive blasting.

REFERENCES

1. 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.

2. ACGIH [1995]. 1995-1996 threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

3. Code of Federal Regulations [1993]. 29 CFR 1910 Washington, DC: U.S. Government Printing Office, Federal Register.

4. Hernberg S, et al. [1988]. Lead and its compounds. In: Occupational medicine. 2nd ed. Chicago, IL: Year Book Medical Publishers.

5. 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.

6. Proctor NH, Hughes JP, Fischman ML [1991]. Lead. In: Chemical hazards of the workplace. 3rd ed. Philadelphia, PA: J.B. Lippincott Company, Philadelphia, pp 353-357.

7. 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.

8. 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.

9. 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.

10. 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.

11. Grandjean P and Bach E [1986]. Indirect exposures: the significance of bystanders at work and at home. Am Ind Hyg Assoc J 47(12):819-

824.

12. Farfel MR and Chisholm JJ [1990]. Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. American Jour of Pub Health, 80:10, 1240-1245.

13. EPA [1994]. Guidance on residential leadbased paint, lead-contaminated dust, and leadcontaminated soil. Washington, DC: U.S. Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances. Memorandum from Lynn Goldman, Assistant Administrator, July 14, 1994.

14. CDC [1991]. Preventing lead poisoning in young children: a statement by the Centers for Disease Control - October 1991. Atlanta, GA: U.S. Department of Health and Human Service, Public Health Service, Centers for Disease Control.

15. Brody DJ, et al. [1994]. Blood lead levels in the US population, phase 1 on the third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). JAMA 272:277-283.

16. Webb M: Cadmium. Br Med Bull 1975;31:246-50.

17. Lauwerys R, Roels H, Regniers M, et al. Significance of cadmium concentration in blood and in urine in workers exposed to cadmium. Environ Res 1979;20:375-91.

18. Kjellstrom T. Exposure and accumulation of cadmium in populations from Japan, the United States, and Sweden. Environ Health Perspect 1979;28:169-97.

19. Ellis KJ, Vartsky D, Zanai I, et al. Cadmium: in vivo measurement in smokers and nonsmokers. Science 1979;205:323-25.

20. Friberg L, Piscator M, Nordberg GF, et al.

Cadmium in the environment. Cleveland: CRC Press, 1974.

21. Baker EL, Peterson WA, Holtz J, Coleman C, Landrigan PJ. Subacute cadmium intoxication in jewelry workers: an evaluation of diagnostic procedures. Arch Environ Health 1979;34:173-77.

22. Scott R, Paterson PJ, Mills EA, et al. Clinical and biochemical abnormalities in coppersmiths exposed to cadmium. Lancet 1976;2:396-98.

23. Page LB, Culver PJ. A syllabus of laboratory examinations in clinical diagnosis. Cambridge: Harvard University Press, 1962.

24. Rosen JF, Chesney R, Hamstra A, DeLuca H, Mahaffey K. Reduction in 1,25dihydroxyvitamin D in children with increased lead absorption. N Engl J Med 1980;302:1128-31.

25. Kazantzis G. Renal tubular dysfunction and abnormalities of calcium metabolism in cadmium workers. Environ Health Perspect 1979;28:155-59.

26. Piscator M. Role of cadmium in carcinogenesis with special reference to cancer of the prostate. Environ Health Perspect 1981;40:107-20.

27. Lemen RA, Lee JS, Wagoner JK, et al. Cancer mortality among cadmium production workers. Ann NY Acad Sci 1976;271:273-79.

28. Thum MJ, Schnorr TM, Smith AB, Halperin WE, Lemen RA. Mortality among cadmium production workers - an update. Journal of the Nat Cancer Inst 1985;2:325-333.

29. Code of Federal Regulations [1993]. 29 CFR 1926 Washington, DC: U.S. Government Printing Office, Federal Register. 30. Proctor NH, Hughes JP, Fischman ML [1988]. Chemical hazards of the workplace, 2nd ed. Philadelphia, PA: J.B. Lippincott.

31. Blair A, Mason TJ [1980]. Cancer mortality in the united States counties with electroplating industries. Arch Environ Health 35(2):92-94.

32. Franchini I, Magnani F, Mutti A [1983]. Mortality experience among chrome plating workers: initial findings. Scand J Work Environ Health 9:247-252.

33. Royle H [1975]. Toxicity of chromic acid in the chromium plating industry. Environ Res 10:39-53.

34. Silverstein M, Mirer F. Kotelchuck D, Silverstein B, Bennett M [1981]. Mortality among workers in a die-casting and electroplating plant. Scand J Work Environ Health 7 (suppl. 4):156-165.

35.Sorahan T, Burges DCL, Waterhouse JAH [1987]. A mortality study of nickel\chromium platers. Br J Ind Med 44:250-258.

sample number	sample period (minutes)	sample volume (liters)	Pb (mg/m ³)	Cr (mg/m ³)	grit
P1	123	245	0.0045	1.1	
BH1	malfunction				
P2	116	230	0.043	10.	P2>BH2
BH2	109	218	0.0046	0.26	
P3	139	267	0.012	0.17	
BH3	125	206	0.028	8.3	BH3>P3
P4	49	109	0.12	16.	
BH4	48	107	0.24	42.	BH4>P4
P5	45	104	0.11	7.6	P5>BH5
BH5	45	110	0.072	0.49	
P6	11	23.6	(0.003)	(0.03)	
BH6	83	195	0.033	0.14	BH6>P6
P7	malfunction				
BH7	58	140	0.031	1.9	
P9	75	167	0.090	3.4	P9=BH9
BH9	75	167	0.066	1.6	
P10	75	173	0.098	7.5	P10>BH10
BH10	75	184	0.060	1.7	
P11	75	179	0.030	3.5	P11>BH11
BH11	75	176	0.017	0.40	
P12	87	212	0.90	12.	P12>BH12
BH12	2	4.82	7.1	100.	
P13	127	283	0.064	1.3	
BH13	malfunction				
P14	80	184	0.14	7.1	
BH14	malfunction				
P15	46	99.8	0.30	34.	P15>BH15
BH15	83	195	0.092	2.8	
P16	82	204	0.020	1.8	P16>BH16
BH16	82	197	0.011	0.13	

Table 1. Personal Air Sampling. Bath Iron Works, July 19-21, 1994

 mg/m^3 = milligrams of contaminant per cubic meter of air

() = Value is between the minimum detectable concentration (MDC) and minimum quantifiable concentration (MQC). For Pb analysis of this sample, this range is 0.002 - 0.0064 mg/m3; for Cr, 0.01 - 0.04 mg/m3.

> = greater than.

P = Sampling cassette was located in the personal breathing zone.

BH = Sampling cassette was located behind the blaster's head.

#	material	building/location	corresponding air samples	Concentration (µg/g)			
				Pb	Cd	As	
K-23	grit	Blast-2/bilge	P1, P2, P3, BH2, BH3	46.	45.	43.	
K-24	grit	Blast-1/grit pot	P13, P14, BH13, BH14	50.	44.	47.	
K-25	grit	Blast-2/grit pot	P11, P12, BH11, BH12	45.	44.	44.	
K-26	fines	Blast-1/classifier	P13, P14, BH13, BH14	71.	39.	38.	
K-27	fines	Blast-2/classifier	P11, P12, BH11, BH12	72.	50.	31.	
L28	primer ¹	all surfaces	all	54.	2.2	<0.5	
S29	steel ²	internal stiffeners	all	97.	4.3	<0.6	
S30	steel ²	decks, bulkheads	all	40.	2.7	<0.5	

Table 2. Bulk Samples, Elemental Analysis. Bath Iron Works (HETA 94-0122), July 19-20, 1994

= Micrograms of contaminant per gram of bulk sample. Liquid sample of weldable primer. µg/g

1.

Sample of the same grade of steel comprising the surfaces being blasted. 2.

sample number ¹	sample period (minutes)	sample volume (liters)	Filter & Dust (mg/m ³)					Steel Grit ^{2,3} (mg/m ³)			
			Pb	Cd	As	Cr	Fe	Pb	Cd	Cr	Fe
1A	85	173	nq	nq^7	nq ⁵	0.027	16.9	0.665 ⁸	nq ⁷	3.28	>200.
1B	84	172	nq	nq ⁷	nq ⁵	0.017	10.7	0.605 ⁸	nq ⁷	7.62 ⁸	>200.
1C	86	26.6	nq	nq ⁷	nq ⁷	0.29	18.7				
2A	59	120	nq	nq ⁷	nq ⁷	0.033	18.3	0.359	nq ⁷	5.57	>200.
2B	14	28.7	nq	nq ⁷	nq ⁷	0.08	46.7	0.320	nq ⁷	4.46	>300.
2C	57	17.1	nq	nq ⁷	nq ⁷	0.11	55.5				
3A	46	93.8	nq	nq ⁷	nq ⁷	0.10	65.5	0.854 ⁸	nq ⁷	12.38	>300.
3B	47	94.9	nq	nq ⁷	nq ⁷	0.047	27.2	1.8	nq ⁷	3.97	>300.
3C	7	2.14						5.75 ⁸	nq ⁷	75.0 ⁸	>3000.
4A	63	127	nq	nq ⁷	nq ⁵	0.016	12.1	0.119	nq ⁷	1.24	>80.
4B	22	45.3	nq	nq ⁷	nq ⁷	0.035	31.3	nq	nq ⁷	nq	>200.
4C	63	19.2	nq	nq ⁷	nq ⁷	0.130	77.3				
5A	83	173	nq	nq ⁷	nq ⁵	0.044	34.	0.3478	nq ⁷	4.9	>200.
5B	83	171	nq	nq ⁷	nq ⁵	0.063	38.	0.022	nq ⁷	0.191	>60.
5C	85	25.6	nq	nq ⁷	nq ⁷	0.051	29.				
6A	68	141	nq	nq ⁷	nq ⁵	0.018	11.9	0.16	nq ⁷	2.11	>70.
6B	68	137	nq	nq ⁷	nq ⁵	0.055	31.	0.017	nq ⁷	0.244	>70.
6C	68	20.7	nq	nq ⁷	nq ⁷	nq	26.8				
7A	62	128	nq	nq ⁷	nq ⁵	0.011	10.	0.138	nq ⁷	1.59	>80.
7B	46	94.8	nq	nq ⁷	nq ⁷	0.021	17.2	0.261	nq ⁷	3.15	>100.
7C	42	12.8	nq	nq ⁷	nq ⁷	nq	10.4				

Table 3. Personal Air Sampling. Bath Iron Works (HETA 94-0122), February 28-March 2, 1995.

sample number ¹	sample period (minutes)	sample volume (liters)	Filter & Dust (mg/m ³)									
			Pb	Cd	As	Cr	Fe	Pb	Cd	Cr	Fe	
8A	68	139	nq	nq^7	nq ⁵	0.029	18.	0.264	nq ⁷	3.71	>70.	
8B	69	140	nq	nq ⁷	nq ⁵	0.066	39.6	0.019	nq^7	0.173	>70.	
8C	68	20.8	nq	nq ⁷	nq ⁷	0.063	28.3					
9A	55	110	nq	nq ⁷	nq ⁷	0.044	23.	0.945 ⁸	nq ⁷	10.3 ⁸	>300.	
$9B^4$	66	134	0.057	nq ⁷	nq ⁵	0.557	>96.3	0.057	nq ⁷	0.426	>70.	
9C	55	17.0	0.059	nq ⁷	nq ⁷	0.270	95.8					
10A	5	10.3	nq ⁷	nq ⁷	nq ⁷	0.330	240.	8.98 ⁸	nq ⁷	6.98	>3000.	
$10B^4$	65	132	0.045	nq^7	nq ⁵	0.564	>97.7	0.239	\mathbf{nq}^7	3.82	>200.	
10C	5	1.52	nq ⁷	nq ⁷	nq ⁷	nq ⁵	28.2					

Table 3. Personal Air Sampling. Bath Iron Works (HETA 94-0122), February 28-March 2, 1995.

 $mg/m^3 =$ milligrams of contaminant per cubic meter of air

- nq = none quantified. Analytical results are reported in terms of the minimum quantifiable concentration (MQC). The MQC for samples with numbers ending in "A" or "B" ranges from 0.007 to 0.5 mg/m³ for cadmium in the steel grit fraction; and 0.0069 to 0.12 mg/m³ for all other analyses. The MQC for cyclone samples ("C" samples) ranges from 0.02 to 0.4 mg/m³ for lead analyses; and from 0.045 to 0.79 mg/m³ for all other analyses.
- 1. Ten sets of side-by-side samples were obtained: samples identified with numbers ending in "A" were obtained using a closed-face cassette in the breathing zone; "B" samples were obtained using a closed-face cassette fitted with a "grit guard"; and "C" samples were obtained using a 10 mm nylon cyclone operated at a nominal flow rate of 300 cc/minute.
- 2. Arsenic analysis could not be accomplished on the steel grit sample solutions due to spectral interference caused by high iron content.
- 3. Steel grit did not penetrate most cyclones; however, sample 3C contained only large particles of grit and no dust.
- 4. A piece of steel grit may have been imbedded in the filter.
- 5. $MQC \ge REL$
- $6. \qquad MQC \ge PEL$
- 7. $MQC \ge both (REL, PEL)$
- 8. The OSHA PEL is exceeded when this value is included in the calculation of the 8-hour time-weighted average concentration.

Sample ¹		Concentration (µg/g)					
		Pb	Cd	Cr	Fe		
Virgin grit							
K1-A	extracted surface dust dissolved grit	<10.0 126.	<10.0 15.6	30.2 698.	42100 >150000		
K1-B	extracted surface dust dissolved grit	<10.0 109.	<10.0 15.8	62.5 745.	8183 >150000		
K1-C	extracted surface dust dissolved grit	<10.0 125.	<10.0 16.3	29.5 802.	48400 >150000		
K1-D	extracted surface dust dissolved grit	<10.0 118.	<10.0 15.0	36.3 655.	47900 >150000		
K1-E	extracted surface dust dissolved grit	<10.0 96.0	<10.0 14.3	25.5 606.	49800 >150000		
Used grit							
K2-A	extracted surface dust dissolved grit	<10.0 93.7	<10.0 20.9	51.4 1150	45600 >150000		
K2-B	extracted surface dust dissolved grit	<10.0 95.3	<10.0 22.7	38.0 1040	41300 >150000		
K2-C	extracted surface dust dissolved grit	<10.0 88.4	<10.0 21.4	69.1 1230	46400 >150000		
K2-D	extracted surface dust dissolved grit	<10.0 94.6	<10.0 33.0	49.7 1480	39300 >150000		
К2-Е	extracted surface dust dissolved grit	<10.0 82.0	<10.0 24.6	80.9 1330	56600 >150000		

Table 4. Bulk Samples, Elemental Analysis. Bath Iron Works (HETA 94-0122), March 2, 1995

 $\mu g/g {=}$ Micrograms of contaminant per gram of bulk sample.

1. Samples K1 and K2 were divided into five replicate aliquots identified as A through E.

NIOSH (REL) OSHA (PEL) Substance (mg/m^3) (mg/m^3) C 0.002, (Ca) 0.01 Arsenic (As) 0.005 Cadmium (Cd) (Ca) Chromium metal (Cr) 0.5 1. Iron Oxide (Fe2O3) 5. 10. (as Fe) Lead (Pb) 0.1 0.05

Table 5. Occupational Exposure Criteria. Bath Iron Works (HETA 94-0122)

All exposure criteria expressed as Time-Weighted Average (TWA) concentrations unless otherwise indicated. $mg/m^3 =$ milligrams of contaminant per cubic meter of air.

C = 15 minute ceiling.

Ca = Potential occupational carcinogen. NIOSH recommends that occupational exposure be limited to the lowest feasible concentration.

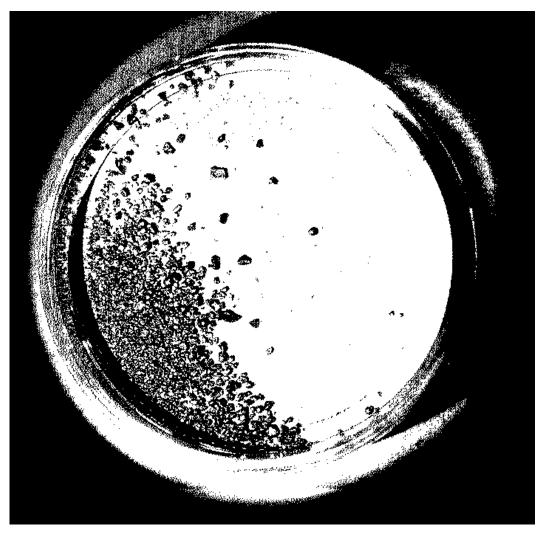


Figure 1. Photograph of steel grit inside an open 37mm cassette. This sample was collected by BIW during previous air monitoring.

- 1. 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.
- ACGIH [1995]. 1995-1996 threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.
- 3. Code of Federal Regulations [1993]. 29 CFR 1910 Washington, DC: U.S. Government Printing Office, Federal Register.
- 4. Hernberg S, et al. [1988]. Lead and its compounds. In: Occupational medicine. 2nd ed. Chicago, IL: Year Book Medical Publishers.
- 5. 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.

- 6. Proctor NH, Hughes JP, Fischman ML [1991]. Lead. In: Chemical hazards of the workplace. 3rd ed. Philadelphia, PA: J.B. Lippincott Company, Philadelphia, pp 353-357.
- 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.
- 8. 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.
- 9. 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.
- 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.
- 11. Grandjean P and Bach E [1986]. Indirect exposures: the significance of bystanders at work and at home. Am Ind Hyg Assoc J 47(12):819-824.
- 12. Farfel MR and Chisholm JJ [1990]. Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. American Jour of Pub Health, 80:10, 1240-1245.
- 13. EPA [1994]. Guidance on residential lead-based paint, lead-contaminated dust, and leadcontaminated soil. Washington, DC: U.S. Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances. Memorandum from Lynn Goldman, Assistant Administrator, July, 14, 1994.
- 14. CDC [1991]. Preventing lead poisoning in young children: a statement by the Centers for Disease Control October 1991. Atlanta, GA: U.S. Department of Health and Human Service, Public Health Service, Centers for Disease Control.
- 15. Brody, DJ, et al. [1994]. Blood lead levels in the US population, phase 1 on the third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). JAMA 272: 277-283.
- 16. Webb M: Cadmium. Br Med Bull 1975;31:246-50.
- 17. Lauwerys R, Roels H, Regniers M, et al. Significance of cadmium concentration in blood and in urine in workers exposed to cadmium. Environ Res 1979;20:375-91.
- 18. Kjellstrom T. Exposure and accumulation of cadmium in populations from Japan, the United States, and Sweden. Environ Health Perspect 1979;28:169-97.
- 19. Ellis KJ, Vartsky D, Zanzi I, et al. Cadmium: in vivo measurement in smokers and nonsmokers. Science 1979;205:323-25.
- 20. Friberg L, Piscator M, Nordberg GF, et al. Cadmium in the environment. Cleveland: CRC Press, 1974.

- 21. Baker EL, Peterson WA, Holtz J, Coleman C, Landrigan PJ. Subacute cadmium intoxication in jewelry workers: an evaluation of diagnostic procedures. Arch Environ Health 1979;34:173-77.
- 22. Scott R, Paterson PJ, Mills EA, et al. Clinical and biochemical abnormalities in coppersmiths exposed to cadmium. Lancet 1976;2:396-98.
- 23. Page LB, Culver PJ. A syllabus of laboratory examinations in clinical diagnosis. Cambridge: Harvard University Press, 1962.
- 24. Rosen JF, Chesney R, Hamstra A, DeLuca H, Mahaffey K. Reduction in 1,25-dihydroxyvitamin D in children with increased lead absorption. N Engl J Med 1980;302:1128-31.
- 25. Kazantzis G. Renal tubular dysfunction and abnormalities of calcium metabolism in cadmium workers. Environ Health Perspect 1979;28:155-59.
- 26. Piscator M. Role of cadmium in carcinogenesis with special reference to cancer of the prostate. Environ Health Perspect 1981;40:107-20.
- 27. Lemen RA, Lee JS, Wagoner JK, et al. Cancer mortality among cadmium production workers. Ann NY Acad Sci 1976;271:273-79.
- 28. Thun MJ, Schnorr TM, Smith AB, Halperin WE, Lemen RA. Mortality among cadmium production workers an update. Journal of the Nat Cancer Inst 1985; 2:325-333.
- 29. Code of Federal Regulations [1993]. 29 CFR 1926 Washington, DC: U.S. Government Printing Office, Federal Register.
- 30. Proctor NH, Hughes JP, Fischman ML [1988]. Chemical hazards of the workplace, 2nd ed. Philadelphia, PA: J. B. Lippincott.
- 31. Blair A, Mason TJ [1980]. Cancer mortality in the United States counties with metal electroplating industries. Arch Environ Health 35(2):92-94.
- 32. Franchini I, Magnani F, Mutti A [1983]. Mortality experience among chrome plating workers: initial findings. Scand J Work Environ Health 9:247-252.
- 33. Royle H [1975]. Toxicity of chromic acid in the chromium plating industry. Environ Res 10:39-53.
- 34. Silverstein M, Mirer F, Kotelchuck D, Silverstein B, Bennett M [1981]. Mortality among workers in a die-casting and electroplating plant. Scand J Work Environ Health 7 (suppl. 4):156-165.
- 35. Sorahan T, Burges DCL, Waterhouse JAH [1987]. A mortality study of nickel/chromium platers. Br J Ind Med 44:250-258.



Delivering on the Nation's promise: Safety and health at work for all people Through research and prevention