NIOSH HEALTH HAZARD EVALUATION REPORT

HETA #2004-0169-2982
U.S. Magnesium
Rowley, Utah

October 2005

DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control and Prevention
National Institute for Occupational Safety and Health

PREFACE

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

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

ACKNOWLEDGMENTS AND AVAILABILITY OF REPORT

This report was prepared by Bruce P. Bernard, M.D., M.P.H., Eun A. Kim, M.D., Ph.D. and Eric J. Esswein, M.S.P.H., C.I.H. of HETAB, Division of Surveillance, Hazard Evaluations and Field Studies (DSHEFS). Field assistance was provided by Robert E. McCleery, M.S.P.H., C.I.H., Charles Mueller, M.S. Analytical support was provided by Donald G. Patterson Jr., Wayman E. Turner, CDC/NCEH Laboratory; Data Chem Latoratories. Desktop publishing was performed by Patricia McGraw and Shawna Watts. Editorial assistance was provided by Ellen Galloway.

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

Highlights of the NIOSH Health Hazard Evaluation

Evaluation of Chlorinated Hydrocarbons at a Magnesium Processing Plant

The National Institute for Occupational Safety and Health (NIOSH) received a request for a health hazard evaluation (HHE) at U.S. Magnesium in Rowley, Utah from the management, United Steel Workers of America, and the Environmental Protection Agency (EPA). The requestors wanted to find out the extent of exposure to carbon tetrachloride (CCl_4), chlorine (Cl_2), and chlorinated hydrocarbons, including dibenzo-p-dioxins (dioxins), chlorinated dibenzofurans (furans), polychlorinated biphenyls (PCBs), and hexachlorobenzene (HCB) during the processing of magnesium. NIOSH investigators conducted the investigation in May, August, and November 2004.

What NIOSH Did

- Interviewed 30 employees about their work-related exposures.
- Measured blood levels of dioxins, furans, PCBs, HCB, and liver function in 30 employees.
- Measured levels of dioxins, furans, PCBs, HCB, carbon tetrachloride, and Cl_2 in air.
- Assessed how workers' activities relate to exposures.

What NIOSH Found

- Workers had measurable dioxins, furans, PCBs, and HCB in their blood. However, the blood levels were lower than those associated with observable health problems.
- One chemical, 2,3,7,8-TCDD, a potential carcinogen, was found in low levels in workers' blood.
- Chlorine levels in air exceeded OSHA standards.
- Most workers reported respiratory irritation from exposure to chlorine gas.
- HCB levels in air exceeded recommended levels.
- Many workers' hands and workplace surfaces had HCB on them.
- The reactor building and electrolytics areas had the highest levels of chemical exposure.

What U.S. Magnesium Managers Can Do

- Implement engineering controls to reduce exposure to chemicals in air.
- Control chlorine gas exposures.
- Require respirators in the reactor building and electrolytics areas until engineering controls are in place.
- Use vacuuming instead of blowing compressed air to clean the electrolytic cells.
- Evaluate silica exposures during removal and relining of the bricks in the launders.
- Teach workers about risks of working with chlorinated hydrocarbons and importance of washing hands.
- Start a quantitative respirator fit testing program. Use NIOSH-approved combination cartridges and respirators.
- Ensure that workers change out of work clothes before leaving the workplace.
- Launder the workers' supplied work clothes.

What U.S. Magnesium Employees Can Do

- Attend training sessions on chlorinated hydrocarbon exposures to learn about the risks of working with them.
- Wash hands with soap before eating, drinking, or smoking, and after bathroom breaks.
- Use the supplied work clothes and avoid taking the used work clothes home.
- Shave every day if you wear a respirator; otherwise your respirator won't fit well, and will not protect you.
- Wear your respirator in the reactor building and electrolytics area.
- Wear two pairs of nitrile gloves if you clean out anode dust boxes.
- Tell your supervisor if you have respiratory symptoms, such as shortness of breath, wheezing, or persistent cough.

What To Do For More Information:
We encourage you to read the full report. If you would like a copy, either ask your health and safety representative to make you a copy or call 1-513-841-4252 and ask for HETA Report #2004-0169-2982

Health Hazard Evaluation Report 2004-0169-2982
U.S. Magnesium
Rowley, Utah
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SUMMARY

In March 2004, the National Institute for Occupational Safety and Health (NIOSH) received a joint request for a Health Hazard Evaluation (HHE) from the management of U.S. Magnesium LLC, the United Steel Workers of America, and the Environmental Protection Agency (EPA). The requesters asked NIOSH to assess employee exposures to chlorinated hydrocarbons (CHCs), including chlorinated dibenzo-p-dioxins (PCDDs), chlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs), certain pesticides (e.g., hexachlorobenzene [HCB]), and chlorine, all generated as byproducts during magnesium production at the U.S. Magnesium plant in Rowley, Utah.

NIOSH investigators conducted a walk-through survey of the facility in May 2004, and made return visits in August and November 2004. The HHE involved quantitative exposure assessments, employee medical and occupational history questionnaires, and biological monitoring. NIOSH investigators determined blood levels of CHCs in a sample of longest-tenured workers in specific areas where CHCs were likely generated.

Several measured chlorine exposures exceeded the NIOSH ceiling Recommended Exposure Level (REL) of 0.5 part per million (ppm) with peak exposures between 10 and 50 ppm. Carbon tetrachloride (CCl_4), known to be generated in the production process, was measured by personal breathing zone (PBZ) samples, and exposures were mostly very low. Hand wipes and surface samples found evidence of HCB (from 0.14 to 3.5 micrograms [µg]) and indicated the potential for dermal exposure. Of forty-two PBZ and two area air samples collected for HCB and PCBs, five exceeded the exposure criterion (REL). Full-shift air sampling results for HCB ranged from 0.096 micrograms per cubic meter (µg/m^3) for a sample collected on a foundry operator to 5.3 µg/m^3 for a maintenance helper working on the sixth floor reactor. In total, 5 of 42 PBZ samples (or approximately 12% of the total sample set) exceeded or were very close to an adjusted Threshold Limit Value for HCB. Air sample results indicate that certain congeners of PCB were present in workplace air above the minimum detection concentration (MDC). Bulk sample results indicated the presence of HCB at 250 micrograms per gram (µg/gr) and three congeners of PCB in collected dust.

Nine of the twelve dioxin-like PCB congeners with assigned TEFs (dioxin-like PCB congeners have been assigned 2,3,7,8-TCDD Toxicity Equivalency Factors [TEFs], indicating their toxicity relative to 2,3,7,8-TCDD, which itself has been assigned a TEF of 1.0) were detected, including congener numbers 77, 105, 114, 123, 126, 156, 167, 169, and 180. Congener 20, which does not have an assigned TEF, was also

detected. The 10 congeners that were detected ranged from trace (between the limit of detection [LOD] and limit of quantification [LOQ]) to 2 µg/sample for congener #209. Since only the Arochlor series of PCB have occupational exposure criteria, these results indicate that certain congeners of PCB are present in workplace air above the MDC; however, the meaning in terms of health risks is unclear.

Of the 30 workers interviewed, 60% reported headaches and 80% reported having had acute upper respiratory symptoms from exposure to chlorine gas at some time during their employment. The blood sample results of the workers revealed that levels of PCDDs and PCDFs were well below levels reported in association with observable health effects. We compared the 30 workers' average blood levels for CHC using the World Health Organization-toxic equivalency quotient (WHO-TEQ $_{98}$) and found the average level in the 30 workers was higher than the level found in the general population. The workers' mean blood level of 2,3,7,8-TCDD was well below the level found to be associated with observable health effects in all published studies. However, 2,3,7,8-TCDD and PCBs are considered to be potential human carcinogens, and a no-risk threshold for human exposures does not exist. The blood HCB levels were higher than the general population, but studies have not found observable clinical health problems at these levels.

NIOSH investigators conclude that biological and environmental monitoring results show evidence of work-related exposure to dioxins, dibenzofurans, PCBs, HCB, and chlorine. Because health-based biological exposure indices are not available for all of these compounds (even less information is available for the combination of exposures), health risk consequences are unclear.

Industrial hygiene monitoring results found the areas of the plant with the greatest risks for HCB and PCB exposures were the reactor building and the electrolytics area. Reactor maintenance workers, particularly less experienced workers, had the highest exposures. Respirator use throughout the areas evaluated was observed to be sporadic at best. Sampling results found peak chlorine exposures at greater than 10 and up to 50 ppm, which represents a serious risk for unprotected workers.

NIOSH investigators determined that an occupational health hazard due to exposures to chlorinated hydrocarbons including chlorinated dibenzo-p-dioxins (PCDDs), chlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs); certain pesticides (e.g., hexachlorobenzene [HCB]); and chlorine existed for workers at U.S. Magnesium. Recommendations for controlling workplace exposures include identifying fugitive emissions and inadequately controlled processes, controlling emissions by engineering controls, enhancing local exhaust ventilation, improving housekeeping, and enforcing the use of personal protective equipment. Additional recommendations are included at the end of this report.

Keywords: Primary Smelting and Refining of Nonferrous Metals, Magnesium, Chlorine, Chlorinated hydrocarbons, TCDD, dioxins, chlorinated dibenzo-p-dioxins, PCDDs, dibenzofurans, PCDFs, polychlorinated biphenyls, PCBs, hexachlorobenzene, HCB, industrial hygiene, biological monitoring. SIC 3339; NAICS 331419

Table of Contents

INTRODUCTION

In March 2004, NIOSH received a request for a Health Hazard Evaluation (HHE) from the management of U.S. Magnesium LLC (U.S. Magnesium) in Rowley, Utah, the United Steel Workers of America, and the U.S. Environmental Protection Agency (EPA). These organizations expressed concerns about exposure to the following substances: (1) chlorinated hydrocarbons (CHCs) including chlorinated dibenzo-p-dioxins (PCDDs), chlorinated dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs), and certain pesticides such as hexachlorobenzene (HCB) generated as byproducts during magnesium processing and (2) chlorine exposure. CHCs are produced unintentionally at U.S. Magnesium during industrial processes involving extraction, concentration, and electrolytic production of magnesium metal from water taken from the Great Salt Lake. The finding by EPA of chlorinated hydrocarbons in soil outside the perimeter of the U.S. Magnesium facility prompted the request for a NIOSH HHE. When inhaled or ingested over long periods of time CHCs can cause increased body burdens and possibly lead to serious health problems. As foreign substances, CHCs are not metabolized through normal metabolic pathways; they tend to remain in human tissues for extended periods.

The objectives of this HHE were as follows:

1. To determine the blood levels of CHCs in a sample of workers who had the longest tenure at U.S. Magnesium and worked in the areas where CHCs were likely generated. If biological monitoring results indicated CHC concentrations at levels that could be associated with observable health effects, a larger study would be recommended for the entire workforce within the plant.
2. To quantitatively assess specific exposures in the plant including chlorine, HCB, and PCBs and to evaluate risks and pathways for these exposures.
3. To develop health and safety recommendations to mitigate employee exposures in the plant.

NIOSH investigators made an initial site visit on May 24, 2004, to conduct a walk-through survey and worker interviews. On August 9–14, 2004, medical interviews and blood testing were done on 30 selected workers for CHCs and alpha glutathione transaminase (alpha GST). Individual worker notification letters to participants and summary letters to union and management representatives concerning the results of the blood tests were sent on October 19, 2004 (Appendix B). In November 2004, industrial hygiene exposure assessments were made. Two interim letters dated December 21, 2004, and February 2, 2005, were sent to the HHE requestors. These letters contained summary biological and industrial hygiene results and recommendations to U.S. Magnesium management to address occupational health and safety issues and control exposures. This final report integrates previous results and findings and includes additional results and conclusions

BACKGROUND

Process description

The facility specializes in the manufacture and supply of magnesium ingots, magnesium recycling services, and chemical byproducts (chlorine, ferric chloride, ferrous chloride, calcium chloride, hydrochloric acid). Approximately 400 workers were employed at the time of the NIOSH HHE, working in a variety of millwork, chemical processing, and foundry operations where magnesium has been produced since 1972. The plant's throughput is reported to be 43,000 metric tons of magnesium per year. The production facilities encompass over 80,000 acres, and consist of a vast solar pond system, a series of local feed stock holding reservoirs, chemical processing zones, a foundry, support facilities, and transportation systems.

Magnesium metal is produced by an electrolytic process in which magnesium chloride is decomposed in an electrolytic bath (Appendix A). The source of raw materials for the manufacture of magnesium is water "mined" or extracted from the Great Salt Lake, which contains a high concentration of salts including magnesium chloride ($MgCl_2$).

The $MgCl_2$ is concentrated in brine produced by solar evaporation of lake water. The brine is moved through holding ponds and desulfated by the reaction of the brine with calcium chloride ($CaCl_2$.) Boron is removed from the desulfinated brine using a kerosene-decanol extractant. Deboronated brine is mixed with a ferrous chloride solution, and pumped to a spray dryer. More than 80% of the typical product coming from the spray dryers is $MgCl_2$ powder, which is used as a primary feedstock for melt/reactor cells.

Spray-dried $MgCl_2$ contains impurities including magnesium oxide (MgO), water, bromide, and sulfate. Oxygen and chlorine are added to the melt process to scavenge various oxides and impurities. Iron compounds catalyze the introduction of chloride into the melt for faster reactions. A coal coke mixture (also used to scavenge oxygen) is added to the spray dry powder feedstock. The CHCs are formed de novo from the point where the addition of the coal coke provides a carbon substrate to the spray dry powder containing the $MgCl_2$ and chloride. Two feed systems and four melt/reactor trains comprise the primary production process. Initial chlorination using chlorine gas occurs in the refractory-lined, electrically heated melt cells. Product from the melt cells flows through a covered refractory-lined launder system and associated reactor cells for final chlorination. Engineering controls for off-gas products from the melt/reactor cells include local exhaust ventilation in the form of a dedicated off-gas extraction system.

$MgCl_2$ salt is transported in vacuum trucks to the electrolytic cells for final separation. Two electrolytic cell lines are currently in operation. The cells are refractory-lined steel wells that contain the molten $MgCl_2$ salt. Two electrodes, a positively charged graphite anode and a negatively charged steel cathode, separate the magnesium and chlorine. The cell bath or electrolyte consists of 10%–20% magnesium chlorine and 25%–40% sodium chloride. When a direct current passes through the molten electrolyte, magnesium ions move toward the negatively charged cathode and deposit magnesium metal, while chloride ions move toward the positively charged anode and form chlorine gas that bubbles at the anode surface. To replenish the $MgCl_2$ within the reactor building, melted $MgCl_2$ is added to the cells four times per day. A sample from each cell is analyzed each day to determine the amount of $MgCl_2$ each cell will require over 24 hours. Molten magnesium metal is removed from cells twice per day by vacuum suction into a mobile pressure vessel, which is then transported and discharged into the cast house crucibles. Chlorine is continuously removed from the anode through a header system and goes to a chlorine plant. If the draft drops too low, a portion of the chlorine flow is bypassed away from the chlorine plant and is emitted by the main stack through the cathode ventilation system. The cells are periodically rebuilt by the service mechanical rebuild crews when the cell's components start to deteriorate, which lowers the performance efficiency of the cells. At the cast house, magnesium metal is cast into the shapes and alloys.

METHODS

Medical

Selection of participants for blood testing

Workers with the longest tenure at the plant who worked in areas where CHC exposures were likely due to process area (e.g., cell rebuild, electrolytics) or job tasks (e.g., general and reactor maintenance) were selected for blood testing. After consulting with several dioxin experts,[1] we determined that obtaining blood from 30 workers would be sufficient to verify CHC levels among workers at the plant. When

determining which areas to sample, we considered results of previous biological monitoring in 2002 for HCB that showed workers in cell rebuild, electrolytics, general, and reactor maintenance had higher HCB blood levels compared to employees in other departments.

Fifty-two of 123 hourly workers were selected from cell rebuild, electrolytics, general maintenance, and reactor departments. The initial list was larger than 30 to ensure that a sufficient number of workers would be available. Forty-three of the 52 employees agreed to participate on the dates scheduled. Thirty employees were selected serially from the final list to participate in the study. One worker refused to participate, so we added one replacement from the list of 43. Of the 30 who participated in blood testing, 11 were from cell rebuild, eight from electrolytics and cell service, five from reactor maintenance, and six from general maintenance.

Medical interviews

Study participants were interviewed regarding age, smoking status, alcohol use, height, and weight. Participants also were asked about past medical history, health symptoms experienced at work, and possible exposures outside of work that might affect serum CHC concentrations. Questions included previous work in jobs with potential exposure to CHCs (including waste incineration, reclamation or hazardous waste work, work with transformers or capacitors, and herbicide manufacturing), military experience in Vietnam, and consumption of fish caught in local rivers. The interview form is attached as Appendix B.

Serum collection and analysis

Blood was drawn for analysis of: 1) CHCs, including PCDDs, PCDFs, coplanar PCBs, and ortho-substituted PCBs; 2) HCB; and 3) alpha-GST. Alpha-GST was measured as an indicator of liver injury, which can occur with exposure to CHCs.

A NIOSH phlebotomist drew 40 milliliters of blood from each study participant. NIOSH personnel stored the blood samples on site, and then shipped them to the CDC National Center for Environmental Health (NCEH) laboratories in Atlanta, Georgia, for analysis. Fasting was not a requirement for participation in the study, so results were lipid adjusted to account for non-fasting lipid levels.[2] Seven polychlorinated PCDDs, 10 PCDFs, three non-ortho substituted or coplanar PCBs, 36 ortho-substituted PCBs, and HCB were measured in serum by high-resolution gas chromatography/isotope-dilution high-resolution mass spectrometry (HRGC/ID-HRMS). The concentration of alpha-GST in serum was measured using a commercially available, enzyme-linked immunoassay kit.[3] Workers were notified of their individual results (Appendix C).

Industrial hygiene

Personal breathing zone (PBZ) air samples, area air samples, and bulk samples were collected on November 1–4, 2004, to determine risks for occupational exposures to HCB, PCBs, and carbon tetrachloride (CCl_4). Potential exposures to CCl_4 were investigated because some generation of this chemical was believed to occur during the manufacturing processes. To evaluate risks for dermal exposures to HCB, employee hand wipe samples were collected from U.S. Magnesium employees and surface wipe samples were collected from lunchroom tabletops.

Full-shift PBZ exposure assessments were conducted for employees working in the reactor building, the electrolytics area, general and reactor maintenance, and the cast house (foundry). Employees working on two different shifts (0700–1500 hours and 1200–2400 hours) were sampled. Sampling trains were calibrated using a Dry Cal® DC Lite primary flow calibrator manufactured by BIOS International. Calibration was conducted before the sampling trains were placed on the workers and immediately after the sampling trains were removed when the work shift ended. Samplers were secured in the employees' breathing zones by attaching the sampler to the work uniform

lapel or, in the case of the chlorine monitors, to the front pocket of the employees' coveralls. Sampling trains were checked throughout the day for correct positioning and to ensure that the sampling pumps and the monitors functioned correctly. Sampling devices were left on during rest breaks and at lunch because employees did not leave the general plant area during these times. All air samples were refrigerated each night after the work shift, and chain of custody was ensured throughout the investigation. All industrial hygiene samples were hand-delivered to the NIOSH contract analytical laboratory in Salt Lake City, Utah, on the last day of the site visit.

Exposures to CCl_4 were sampled using coconut shell charcoal tubes (100/50 milligrams, SKC Inc. Lot #2000) connected to SKC Pocket Pumps. Sampling trains were calibrated to a flow rate of either 50 or 100 cubic centimeters per minute. Analysis was by gas chromatography and flame ionization detection according to NIOSH Manual of Analytical Methods (NMAM) Method 1003 (Halogenated Hydrocarbons).[4]

Airborne concentrations of HCB and PCBs were measured using polyurethane foam (PUF) sorbent tubes (SKC Cat. # 226-92) according to EPA Method TO-10A.[5] All samples were collected at a flow rate of 5 liters per minute (Lpm) using Leland Legacy high volume sampling pumps (pump flow range capacity was 5–15 Lpm). After the shift ended, the PUF samplers were removed from the workers, wrapped in aluminum foil, and then placed in the original factory package (an airtight jar with a tight-fitting screw top lid). At the laboratory, the PUF sorbents were removed from the sampler and extracted continuously for 16 hours with 300 mL of 5% diethyl ether/hexane in a Soxhlet extractor. Each extract was concentrated in a K-D apparatus and adjusted to final volume with hexane. The final extract for each sample was analyzed by gas chromatography (GC) configured with dual electron capture detectors. Each sample was analyzed for HCB, Arochlor 1242 and 1254 series, and the following individual PCB congeners: 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, 170, 180, 189, 190, and 209.

A bulk sample of greenish particulate was collected during cleanout of an off-gas header from the electrolytics floor of Building 1. This work procedure is referred to as "pigging a header" because a small steel block (acting as a small plow, or resembling a pig's snout) is dragged through the rectangular header to scrape and remove accumulations of settled particulates inside the off-gas header. A 3.25 gram portion of dust was collected during the cleaning process and analyzed for the presence of HCB and PCBs using the Soxhlet extraction/KD/GC method described above for the PUF air samples. The sample was also analyzed for various metals using inductively coupled plasma emission spectrometry according to a modified NIOSH Method 7303.[6]

Hand wipe and surface wipe samples were collected using pre-extracted 4"x 4" gauze pads (Eagle Picher, Miami, OK. Lot No. G 4089010, QA Level 1) that had been wetted with reagent grade ethanol. The gauze pads were quality assured to be free of a wide variety of semi-volatile, inorganic, and pesticide analytes including HCB. Employees were asked to remove the wipe from a storage container and wipe the palms and backs of their hands using firm pressure for 30 seconds. Surface wipe samples were collected using disposable plastic 10 x 10 centimeter sampling templates that were secured to the desired sampling surface. The wipes had been slightly moistened with 1 milliliter of reagent grade ethanol that was poured onto the wipe to allow the alcohol to be absorbed for several hours. All samples were collected using firm pressure to wipe horizontally across the masked sample area, then vertically, then wiping horizontally again. After sampling was conducted, the pads were folded with the soiled side inwards, and the wipe was placed back into the sample collection and storage container. NIOSH investigators donned a new pair of nitrile gloves after collection of each wipe sample. No standard sampling or analytical method is available for HCB on gauze

wipes, so the samples were analyzed using gas chromatography and a combination of the methods and conditions listed in EPA Method TO-10A and NIOSH Method 5602 with some modifications.[7]

Exposures to chlorine gas were measured using two models of Gas Alert Extreme real-time direct-reading chlorine monitors (BW Technologies, Calgary, AB, Canada). One monitor had data logging capabilities and one model did not; it was configured as direct read-out only. The monitors were factory fresh and had been calibrated for response to chlorine gas in a range of 1–50 ppm. The monitors were configured to take a measurement every 5 seconds. Three U.S. Magnesium employees wore the monitors for their full work shifts on November 3–4, 2004.

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 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 becomes available.

Introduction: exposure criteria

The primary sources of environmental evaluation criteria for the workplace are: (1) NIOSH Recommended Exposure Limits (RELs),[8] (2) the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs),[9] and (3) the U.S. Department of Labor, Occupational Safety and Health Administration (OSHA) Permissible Exposure Limits (PELs).[10] Employers are encouraged to follow the OSHA limits, the NIOSH RELs, the ACGIH TLVs, or whichever is the more protective criterion. The Utah Labor Commission under Utah Administrative Code R614-6 recognizes the ACGIH TLVs as the applicable occupational exposure criteria for industrial workplaces such as U.S. Magnesium that are classified as hot metallurgical operations.[11] However, state-based OSHA plans are required to have a program in place that is as protective as the Federal OSHA criteria, therefore Federal OSHA criteria such as PELs also apply in the State of Utah.

OSHA requires an employer to furnish employees a place of employment that is free from recognized hazards that are causing or are likely to cause death or serious physical harm [Occupational Safety and Health Act of 1970, Public Law 91-596, sec. 5(a)(1)]. However, employers should understand that not all hazardous chemicals have specific OSHA exposure limits such as PELs and short-term exposure limits (STELs). An employer is still required by OSHA to protect its employees from hazards, even in the absence of a specific OSHA PEL.

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 STEL or ceiling values that are intended to supplement the TWA where there are recognized toxic effects from higher exposures

over the short-term. An example of a chemical with different criteria is chlorine gas, for which there are TWA, STEL and ceiling occupational health criteria.

Unusual work schedules

Occupational exposure criteria are based on "standard" work shifts that are 8 hours (or in some cases up to 10 hours) in length. Work shifts for certain employees at U.S. Magnesium are longer than a standard 8-hour shift. Some U.S. Magnesium employees work 12-hour work shifts for 3 consecutive days, then are off for 3 days, then return for 3 days of 12-hour shifts. This longer work schedule equates to a 48-hour workweek (four 12-hour shifts) rather than a standard 40-hour week. Because of the longer numbers of hours worked per week for some employees, occupational exposure criteria need to be modified. The rationale for adjusting occupational exposure criteria for longer work shifts is to ensure, as much as possible, that persons working longer shifts are placed at no greater risk of exposure, injury, or discomfort than those who work standard 8-hour days and a 40-hour workweek.[12] The Brief and Scala model referenced in the ACGIH TLV booklet is intended to address workdays that exceed 8 hours or workweeks that exceed 40 hours.[9] The Brief and Scala model uses the following formula:

$(40 \div h) \times (168 - h) \div 128)$ = TLV reduction factor, where h = hours worked per workweek.

HHE, exposure criteria are compared to the adjusted RELs for those employees whose job shift requires them to work four 12-hour shifts in a workweek.

Chlorine (Cl_2)

Chlorine (Cl_2) gas is a greenish-yellow gas with a characteristic irritating odor. Exposure to Cl_2 gas can cause severe irritation of the eyes and respiratory tract, resulting in tearing, runny nose, sneezing, choking, and chest pain. Breathing difficulty with delayed onset can also occur. Severe exposure to Cl_2 can result in pulmonary edema that can be fatal. Mucous membrane and eye irritation has been reported to occur at levels as low as 0.2–2 ppm.[13,14] The ACGIH TLV for Cl_2 is 0.5 ppm as a TWA and 1 ppm as a TLV-STEL. The ACGIH TLV is based on evidence indicating that exposures to 1 ppm result in annoying nose, throat, and eye symptoms in exposed workers.[9] The NIOSH REL for chlorine is 0.5 ppm as a 15-minute ceiling concentration; the criterion is based on severe eye, mucous membrane, and skin irritation.[15] The federal OSHA PEL for Cl_2 is 1 ppm as a ceiling limit.[10] The NIOSH Immediately Dangerous to Life and Health (IDLH) concentration for Cl_2 is 10 ppm.[16]

Carbon tetrachloride (CCl_4)

Carbon tetrachloride (CCl_4) is a colorless organic chlorinated solvent that was historically used as a solvent for oils, lacquers and varnishes, as a degreasing agent, and as a fumigant for grains. CCl_4 was once widely used as a dry cleaning agent but has since been replaced with less toxic chlorinated solvents. Exposures to CCl_4 in sufficient doses can cause central nervous system depression and kidney and liver damage. There are reports of industrial workers affected by CCl_4 inhalation exposures at concentrations between 25 and 30 ppm; the most common symptoms were headache, nausea, vomiting, and dizziness. Animal studies have suggested that CCl_4 causes liver cancer, but the studies indicated this occurred after liver necrosis and fibrosis, suggesting that CCl_4 is not a direct liver carcinogen.[17,18,19]

The ACGIH recommends a TLV-TWA of 5 ppm (20 mg/m^3) based on reviews of rodent, primate, and human studies indicating that the liver was the most sensitive target organ for CCl_4 toxicity and that significant liver toxicity was not observed at doses of 10–20 mg/kg of body weight or airborne exposures at less than 10 ppm. A TLV-STEL of 10 ppm is also recommended by the ACGIH with notes that the criterion may not be protective for workers who consume alcoholic beverages or have pre-existing liver disease or compromised liver function.[20]

CCl_4 has been designated an A2 classification by ACGIH, a suspected human carcinogen.[20]

NIOSH lists CCl_4 as a potential occupational carcinogen.[21] Federal OSHA has determined that a TWA limit is more appropriate than a short-term exposure limit for CCl_4 because low-level exposure to CCl_4 presents a chronic, rather than an acute, health hazard. The federal OSHA exposure criterion of 12.6 mg/m^3 (2 ppm) is intended to protect workers from chronic exposures capable of causing cancer.[22] The NIOSH REL for CCl_4 is numerically the same as the OSHA PEL, however the NIOSH criterion is based on a 60-minute STEL, and is intended to protect against liver cancer.[8]

Polychlorinated biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are chlorinated aromatic hydrocarbons that were manufactured in the United States from 1929 to 1977, and marketed under the trade name Aroclor.[23] PCBs found wide use because they were heat stable; resistant to chemical oxidation, acids, bases, and other chemical agents; and stable to oxidation and hydrolysis in industrial use.[24] PCBs were used in hundreds of industrial and commercial applications including heat transfer, electrical, and many other applications. Although no longer manufactured, PCBs can still be found *de novo* because they are formed in incineration processes.

Several recent studies have been published about the toxicity, human health effects, and body burdens of PCBs.[25,26,27,28,29,30,31] Agency for Toxic Substances and Disease Registry (ATSDR)[32] and United States Environmental Protection Agency (USEPA)[33] have recently updated and summarized the environmental and animal data regarding PCB exposure. PCBs have been shown to cause cancer and non-cancerous health effects in animals. Recent studies raise concerns that PCBs are associated with cancer, [34] neurobehavioral effects[35] and abnormal thyroid[36] and immune function[37] in humans, but these studies have not shown consistent results. Several mortality studies[38,39,40] conducted in occupationally exposed groups in the late 1970s and 1980s showed elevated rates of various cancers in exposed groups. However, in certain follow-up studies or updates of cohorts,[41,42,] results have found that the increases of specific cancers were not consistent between studies, or the elevations did not remain consistent over time. Three studies[43,44,45] found increases in malignant melanoma among worker cohorts; however, dose-response relationships were not seen, and confounding exposures to other chemicals and sunlight were not accounted for. These studies had small sample sizes, brief follow-up periods, and were not amenable to dose-response analysis. Along with a lack of consistency among the epidemiologic studies of the occupationally exposed, a lack of consistency and clinically apparent illness in situations with high PCB exposures has been found. Chloracne has been observed in studies of workers in Italy,[46] but not among workers in Australia,[47] Finland,[48] or the United States.[49,50,51,52] Chloracne[53,54] was seen in Japan and Taiwan from ingestion of cooking oil contaminated by a PCB, PCDFs, and polychlorinated quaterphenyls. Weak positive correlations between serum PCB level, and serum aspartate aminotransferase (SGOT) level,[44] serum gamma-glutamyltranspeptidase (GGTP) level[41,43,45,47] and plasma triglycerides[44,55,56] have been reported. Correlations between plasma triglycerides[57] and GGTP[58] have also been found among community residents with low-level PCB exposures.

The International Agency for Research on Cancer (IARC) has concluded that the evidence for PCB carcinogenicity to animals and to humans is limited. In February 1986, NIOSH reiterated its previous recommendation that exposure to PCB in the workplace not exceed 1 $\mu g/m^3$ (based upon the recommended sampling and analytical method in use at the time), determined as a TWA for up to a 10-hour workday, 40-hour workweek.[59] In 1971, based on the 1968 ACGIH TLVs, OSHA promulgated its permissible exposure limits of 1 mg/m^3 for airborne chlorodiphenyl products (PCB) containing 42% chlorine and 0.5 mg/m^3 for chlorodiphenyl products containing 54% chlorine, determined as 8-hour TWA

concentrations (29 CFR 1910.1000). The TLVs, which have remained unchanged at 1.0 and 0.5 mg/m^3, are based on the prevention of (non-carcinogenic) systemic toxicity.[9] The OSHA PEL and the ACGIH TLV values include a "skin" notation, which refers to the potential contribution to overall exposure by the cutaneous route, including the mucous membranes and eyes, by either airborne or direct skin contact with PCB. The World Health Organization (WHO) has assigned 12 PCB congeners toxicity equivalency factors (TEFs), indicating their toxicity relative to the 2,3,7,8 isomer of TCDD, which itself has been assigned a TEF of 1.0). Table 1 lists TEFs for the 12 PCB isomers that have been assigned TEFs and that were included as part of the PCBs requested for analysis in the samples collected during this HHE.

2,3,7,8-TCDD

In 2003, the National Academy of Sciences (NAS) Committee to Review the Health Effects in Vietnam Veterans of Exposure **t**o Herbicides published its fourth biennial update on chlorinated hydrocarbons and their health effects.[60] The Committee found a positive association between certain chlorinated hydrocarbons (particularly focusing on 2,3,7,8-TCDD) and several health outcomes for which chance, bias, and confounding could be ruled out with reasonable confidence. These included chronic lymphocytic leukemia, soft-tissue sarcoma, non-Hodgkin's lymphoma, Hodgkin's disease, and chloracne. The NAS Committee also identified those health outcomes in which limited or suggestive evidence of an association existed. These included respiratory cancer (of lung and bronchus, larynx, and trachea), prostate cancer, multiple myeloma, acute and subacute transient peripheral neuropathy, porphyria cutanea tarda, Type 2 diabetes, and spina bifida in the children of Vietnam veterans exposed to herbicides.

Chloracne

The most commonly observed effect from 2,3,7,8-TCDD exposure in humans has been chloracne.[60] Chloracne is a skin condition characteristic of high-exposure of 2,3,7,8-TCDD[61] which appears shortly after chemical exposure, not after a long latency period. Although it doesn't respond to acne treatments, it usually regresses over time once exposure stops.

There is little human data from which to determine the threshold level of 2,3,7,8-TCDD at which chloracne occurs, or who is at greatest risk for developing chloracne. Most reports related to chloracne found levels higher than hundreds or thousands of picograms per gram (pg/g) lipid of blood 2,3,7,8-TCDD. (For PCDDs and PCDFs, the units used are picogram per gram lipid, which is equal to values given in ppt [parts per trillion.]) Lansing et al.[62] reported chloracne at dioxin levels ranging from 163 to 1,935 pg/g lipid. Mocarelli et al.[63] described chloracne in persons present in zone A of the reactor during the Seveso incident. These persons had very high serum 2,3,7,8-TCDD levels ranging from 820 to 56,000 pg/g lipid measured within 1 year of the incident. Schecter et al.[64] reported chloracne occurring in levels as low as a mean of 185 pg/g lipid in workers exposed to dioxins at a chemical factory. On the other hand, in the Ranch Hand study of Vietnam War veterans there was no increase in the number of cases of chloracne in the range of 5.2–59.1 pg/g lipid.[65] Chloracne was not found among Missouri residents who resided in dioxin-contaminated homes[66,67] examined 10 years after exposure. It is important to note that although chloracne is characteristic of dioxin exposure, it is not a marker of exposure to dioxins.

Risk for diabetes

Some studies have suggested a relationship between exposure to 2,3,7,8-TCDD and the development of Type 2 diabetes. Type 2 diabetes accounts for about 90% of cases of primary diabetes (i.e., not secondary to a known disease or condition, such as pancreatitis). Onset of Type 2 diabetes rarely occurs before 30 years of age, but incidence increases steadily with age thereafter. It is generally accepted that the main risk factors for Type 2 diabetes include age (older people are at higher risk), obesity, central fat deposition, a history of gestational diabetes

(diabetes during pregnancy), physical inactivity, ethnicity (for example, prevalence is greater in blacks and Hispanics), and perhaps most important, a family history of Type 2 diabetes.

Recently, in the NAS update[60], the NAS Committee found there was inadequate or insufficient information to determine whether an association exists between exposure to 2,3,7,8 TCDD and diabetes. They reached this conclusion after re-examining the association between TCDD and diabetes from a combined analysis of data from the Ranch Hand study and a NIOSH study of 2,3,7,8-TCDD-exposed workers at chemical plants in New Jersey and Missouri.[68] The known predictors of diabetes risk—family history, physical inactivity, and obesity—continue to greatly outweigh any suggested increased risk posed by exposure to dioxins.

Mortality from diabetes was assessed in the NIOSH and IARC occupational cohorts and among Seveso, Italy adult residents exposed to dioxins in certain areas (zones A, B, and R) after a 1976 industrial incident.[69,70,71] In the NIOSH cohort,[69] mortality due to diabetes was slightly, but not statistically significantly, elevated when diabetes was considered as an underlying cause of death. In the subset of workers in the IARC cohort[70] exposed to 2,3,7,8-TCDD or chlorophenols, there were modest, but not statistically significant elevations in the risk of death from diabetes. In the Seveso cohort,[71] modest, statistically significant elevations in mortality from diabetes were observed only in females of zones B, but not in the other zones or in males.

In July 2005, the Department of Defense released its latest report of the Air Force Health Study,[72] a 20-year epidemiologic investigation into the health effects of exposure to herbicides used during the Vietnam War, primarily 2,3,7,8 TCDD. This report summarizes data from the final examinations conducted in 2002. The Ranch Hand cohort was stratified according to three dioxin exposure categories: background, low, and high. The background category comprised Ranch Hands with 1987 2,3,7,8-TCDD levels of 10 ppt or less. (Note that the 2,3,7,8-TCDD levels of all U.S. Magnesium workers tested were below 4.5 ppt, which would place them in the background range in this study.) The remaining Ranch Hands (with 2,3,7,8-TCDD levels above 10 ppt) were separated into low and high categories by the median of their estimated initial dioxin levels. The risk of diabetes requiring insulin control was increased in the Ranch Hand high dioxin category. The data suggested that as dioxin levels rise, the incidence and severity of Type 2 diabetes increase, and the time to disease onset decreases.

Risk for cancer

With regards to cancer mortality risk, the NIOSH re-examination of the cancer literature found that workers exposed to dioxins have a slightly greater risk of dying from all types of cancer than the U.S. general population.[68] For the NIOSH study cohort, the update found that the standardized mortality ratio (SMR) for all cancers combined was 1.13 (95% CI: 1.02-1.25). Recently, EPA[73] reviewed several cohort studies related to cancer and dioxin exposure. From this review, the large U.S. study by NIOSH[74] and its update,[68] as well as the Dutch cohort study,[75,76] are considered to be the most important studies in the field of TCDD cancer epidemiology.

In an update of the IARC study,[77] the study group was increased to 26,976 workers by adding the NIOSH study and four plants in Germany. The authors reported that among those exposed to phenoxy herbicides containing 2,3,7,8-TCDD, mortality from malignant neoplasms was slightly but statistically significantly elevated (SMR = 1.12, 95% CI: 1.04-1.21).

Mackie et al.[78] reported that there was no safe dose for cancer threshold for dioxin in their re-analysis of EPA data. NIOSH recommends that 2,3,7,8-TCDD be regarded as a potential occupational carcinogen and that occupational exposure to 2,3,7,8-TCDD be controlled to the lowest feasible concentration.

Blood level of HCB

Controversy exists concerning the association of specific observable human health effects and inhalation exposure to HCB. The research results on humans who inhale HCB have been inconsistent. While test results on animals show that inhaling HCB has been linked to liver, kidney, and thyroid cancer, the results from human studies aren't definite regarding the risk of cancer. However, IARC considers HCB to be reasonably anticipated to be a carcinogen.[79]

There is a clear association between human health effects and the ingestion of HCB, based on continuing studies of a small cohort from an incident in Turkey in the 1950s, in which people ingested grain contaminated with HCB.[80] These people developed acute liver problems and nerve symptoms, such as tremors, convulsions, muscle weakness, and abnormal feelings in their hands and feet. Upon follow-up years after their initial exposure, some people showed certain changes in their blood cells when tested for immunologic function. Even so, most of the immunologic changes for the people with high exposure to HCB were still within the normal range.

Another study[81] investigated worker health status related to HCB exposures in the manufacture of chlorinated solvents. This study examined inhalational exposure and not ingestion. Results found no instances of porphyria cutanea tarda (a skin disease marked by blisters, lumps, and fragile skin) or increased urinary porphyrins, as was found in the Turkish incident noted above.

A recent report found that a level greater than 63 ppb of blood HCB was related to increases in urinary excretion of coproporphyrins.[82] Daniel et al.[83] reported a reduction in interferon with greater than 2.75 ppb of blood HCB, and Queiroz et al.[84,85]reported that HCB at levels of 3.84 ppb (0.1–16) was associated with impaired neutrophilic chemotaxis and cytolytic activity, and increases in immunoglobinlin levels of IgG, IgA, and IgM. The small number of study subjects, the lack of a dose-response relationship between exposure and outcomes, and non-specific health outcomes weaken most of these studies.

Neither OSHA nor NIOSH has occupational exposure criteria for HCB. The ACGIH TLV for HCB is 0.002 mg/m^3 (2 $\mu g/m^3$) as a TWA. The TLV is based on oral exposure (ingestion) scenarios and extrapolations from animals to humans based on what is believed to be a no adverse effects level in humans. ACGIH has assigned HCB an A3 notation, i.e., "confirmed animal carcinogen with unknown relevance to humans." ACGIH has assigned HCB a skin notation based on the ability to penetrate intact human skin in significant quantities.[9]

PCDDs, PCDFs, PCBs and HCB

Exposures to PCDDs, PCDFs, and PCBs are generally expressed in terms of 2,3,7,8-TCDD toxicity equivalents. As mentioned above, because of the many different isomers of chlorinated dioxin and "dioxin-like" compounds, each with varying levels of toxicity, the Toxicity Equivalent Quotient (TEQ) system was developed. With the TEQ system, each isomer is assigned an equivalency factor that reflects its toxicity relative to 2,3,7,8-TCDD. The latest revised weighting factors, TEFs, were proposed in 1998 by the WHO.[86]

For comparison of individual PCDDs, PCDFs, PCBs, and HCB blood concentrations, we used the 95th percentile values in the U.S. general population from the *Second national report on human exposure to environmental chemicals* (Table 2).[87] This report presents biological monitoring data for the non-institutionalized, civilian U.S. population over the 2-year period 1999–2000. Chemicals and their metabolites were measured in blood and urine samples from selected participants in the National Health and Nutrition Examination Survey (NHANES)[88] conducted by CDC's National Center for Health Statistics. We compared the 95th percentile for 6 PCDDs, 9 PCDFs, 3 coplanar PCBs and 22 ortho-PCBs with the results of 30 U.S. Magnesium workers. To assess the TEQ-WHO_{98} values of U.S. Magnesium workers, we used age-specific TEQ values reported by Patterson et al (2004).[89] In the Patterson et al. paper, the

$TEQ\text{-}WHO_{98}$ values were calculated without ortho-PCBs. The values of PCDDs, PCDFs, PCBs, and HCB used in this report are adjusted with blood lipid contents. For PCDDs and PCDFs, the units used are picogram/g lipid (pg/g lipid), which is equal to values given in ppt. For the PCBs and HCB, the values are given in nanogram/g lipid (ng/g lipid), which is equal to ppb.

Statistical methods

Statistical method for biological monitoring data

For each compound, we calculated an imputed value when that compound was not detected, i.e., was below the limit of detection (LOD).[90] The method for calculating the imputed value differed according to the percentage of nondetectable results. When the compound was not detected in more than 50% of the samples, a value of one-half the LOD or MDC was assigned. This technique was used for:

tetrachlorodibenzofuran (2,3,7,8-TCDF)
hexachlorodibenzofuran (1,2,3,7,8,9-HxCDF)
tetrachlorobyphenyl (3,4,4',5-TCB)
2,3,3',4,4'-Pentachlorobiphenyl (PCB 105)
2,3',4,4',5,5'-Hexachlorobiphenyl (PCB 167)
2,3,3',4,4',5'-Hexachlorobiphenyl (PCB 157)
2,3,3',4,4',5,5'-Heptachlorobiphenyl (PCB 189)
2,3',4,4'-Tetrachlorobiphenyl (PCB 66)
2,2',3,3',5,5',6-Heptachlorobiphenyl (PCB 178)
2,2',3,4,4',5',6-Heptachlorobiphenyl (PCB 183)
2,2',3,3',4',5,6-Heptachlorobiphenyl (PCB 177)
2,2',3,3',4,5,5'-Heptachlorobiphenyl (PCB 172)
2,2',3,3',4,4',5,6-Octachlorobiphenyl (PCB195)
2,2',3,3',4,4',5,5',6-Nonachlorobiphenyl (PCB 206)
2,2',5-Trichlorobiphenyl (PCB 18)
2,4,4'-Trichlorobiphenyl (PCB 28)
2,2',5,5'-Tetrachlorobiphenyl (PCB 52)
2,2',4,5'-Tetrachlorobiphenyl (PCB 49)
2,2',3,5'-Tetrachlorobiphenyl (PCB 44)
2,2',4,5,5'-Pentachlorobiphenyl (PCB 101)
2,2',3,4,5'-Pentachlorobiphenyl (PCB 87)
2,3,3',4',6-Pentachlorobiphenyl (PCB 110)
2,2',3,5,5',6-Hexachlorobiphenyl (PCB 151)
2,2',3,4',5',6-Hexachlorobiphenyl (PCB 149)
2,2',3,3',4,4'-Hexachlorobiphenyl (PCB 128)

When the compound was detected in at least 50% of the samples, the imputed value is equal to the MDC divided by the square root of 2. When the imputed value was less than the median value for each exposure group it was retained; if it was greater than the median, it was considered too imprecise and the median value was used. This method was used for:

tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD)
pentachlorodibenzo-p-dioxin (1,2,3,7,8-PeCDD)
hexachlorodibenzo-p-dioxin (1,2,3,4,7,8-HxCDD; 1,2,3,7,8,9-HxCDD)
pentachlorodibenzofuran (2,3,4,7,8-PeCDF)
hexachlorodibenzofuran (2,3,4,6,7,8-HxCDF)
heptachlorodibenzofuran (1,2,3,4,7,8,9-HpCDF)
2,3',4,4',5-Pentachlorobiphenyl (PCB 118)
2,3,3',4,4',5-Hexachlorobiphenyl (PCB 156)
2,4,4',5-Tetrachlorobiphenyl (PCB 74)
2,2',4,4',5-Pentachlorobiphenyl (PCB 99)
2,2',3,4',5,5'-Hexachlorobiphenyl (PCB 146)
2,2',3,4',5,5',6-Heptachlorobiphenyl (PCB 187)
2,2',3,3',4,4',5-Heptachlorobiphenyl (PCB 170)
2,2',3,3',4,5',6,6'-Octachlorobiphenyl (PCB 201)
2,2',3,3',4,4',5,6'-Octachlorobiphenyl (PCB 190-203)
2,2',3,3',4,4',5,5'-Octachlorobiphenyl (PCB 194)
2,2',3,3',4,4',5,5',6,6'-Decachlorobiphenyl (PCB 209)

When the compound could not be reported because of QA/QC procedures, it was assigned a value of zero. This method was used for 3,3',4,4'-Tetrachlorobiphenyl (PCB 77).

To ensure that the imputation method did not bias our findings, statistical analyses were repeated by replacing non-detectable values with zero. The conclusions were unchanged.

Differences between individual congeners (the different CHC compounds) by work departments were examined. Because PCDD and PCDF levels were log-normally distributed, the median levels are reported. The Kruskal-Wallis test was used to determine the statistical significance of group differences. A p-value ≤ 0.05 was considered statistically significant.

In addition to conducting a statistical analysis for each congener individually, the total TEQ based on TEF-WHO_{98}[86] was calculated. Total TEQ and the portion of the TEQ contributed by PCDDs and PCDFs were examined.

Statistical method for industrial hygiene data

Statistical techniques were used to describe frequency distributions and summary measures and to investigate differences by work area. Work areas and numbers of samples included the reactor (n=15), electrolytics (n=21), and the foundry (cast house, n=7). One-way analysis of variance and two-sample t-tests were used to determine whether PBZ levels of HCB and PCB differed for the reactor, electrolytics, and foundry areas of the plant. In addition, correlations between HCB and PCB levels were evaluated using the Pearson correlation coefficient (r). Because the distributions of HCB and PCB were skewed to the right, results were log-transformed for statistical analyses.

RESULTS

Medical

The average age and working duration of the 30 workers interviewed was 50.3 (40–59) years and 26.9 (19.9–31.4) years, respectively. The average age varied significantly by work area (Table 3). More than half (53%) of the workers' BMI (body mass index) was higher than 30 kg/m^2 (an indication of obesity, which may be related to diabetes risk). There were no significant differences in BMI between work areas, working duration, and proportion of smokers or drinkers. Arthritis the most common medical disorder was reported by 12 (40%) participants. [Table 4]

In the interim letter dated February 2, 2005, we stated that five employees had reported a history of diabetes. Since that time, we noted an error in our transcription of the questionnaire data – four employees reported a history of diabetes (not five as stated in the letter), with three employees reporting having taken medication for it; two reported a history of hypoglycemia. Regarding irritative symptoms, 25 (83%) of the 30 workers reported throat and eye irritation or cough at work. Headache and shortness of breath with exertion were also reported by more than 60% of the workers. There were no important differences by work areas in non-occupational exposures related to CHCs, e.g., fish consumption, Vietnam experience, hazardous waste work, etc.

Twenty five of thirty workers reported being exposed to fugitive chlorine gas while performing work tasks. Three workers in the electrolytic department reported chlorine exposure daily.

Blood PCDDs/PCDFs/PCBs

Among the 30 workers, the mean of the TEQ-WHO_{98} was 37.3. For the workers 55–59 years old, the mean of TEQ was 53.2 (Table 5). Table 6 gives TEQ results by work area, showing a significant difference between maintenance workers and cell brick workers. Twelve workers (40%) had a TEQ higher than the 95th percentile of the U.S. general population (one cell brick worker, four electrolytic workers, four maintenance workers, and three reactor workers). Overall, the mean of PCDFs (23.7 TEQ) was higher than the mean of PCDDs (11.6 TEQ), and PCBs (5.5 TEQ) (Table 7). The mean of PCDDs was highest in the electrolytic department (13.1 TEQ), and the mean of PCDFs was highest in the maintenance department (47.6 TEQ). Table 8 shows that the mean of 2,3,7,8 TCDD was 1.28 pg/g lipid. Among the OCDDs, the highest mean concentration was for 1,2,3,4,6,7,8,9-OCDD (281.50 pg/g lipid); among the PCDFs the highest mean concentration was for 1,2,3,4,6,7,8-HpCDF (192.49 pg/g lipid). Among the PCBs, 3,3',4,4',5,5'-HxCB (18.37 pg/g lipid) had the highest concentration.

In all 30 workers, levels of two PCDDs, 1,2,3,7,8,9-HxCDD and 1,2,3,6,7,8-HxCDD, were higher than the 95th percentile of the U.S. general population. Similarly, levels of four PCDFs, 2,3,4,7,8-PeCDF, 1,2,3,4,7,8-HxCDF, 1,2,3,7,8,9-HxCDF, and 1,2,3,4,6,7,8-HpCDF,

were higher than the 95th percentile of the U.S. general population (Table 9).

Hexachlorobenzene

The mean of lipid adjusted and whole blood HCB from the 30 workers was 891 ng/g lipid (range: 253.0 – 6790.0), and 0.7 (range: 0.2 –3.4) ng/g lipid, respectively. Workers in maintenance (mean = 2465.13 ng/g lipid) had statistically significantly higher HCB blood concentrations than workers in the other departments (Table 10).

Blood alpha glutathione S-transferase

The mean alpha GST was 4.5 (range: 2.1 – 9.4) µg/L. All the workers' levels were lower than the reference value (0–12 µg/l). All departments showed similar levels (Table 11).

Industrial hygiene

Chlorine gas (CL_2)

Two Assistant Melt Cell Operators (AROs) wore real-time Cl_2 monitors for their full shifts (1200–2400 hrs.) on November 3, 2004, while they worked in the reactor building. The TWA for one ARO during his shift was 0.5 ppm, which exceeded the adjusted TLV for Cl_2 of 0.39 ppm. The datalogger also recorded 618 measurements during the worker's 12-hour shift that exceeded the PEL of 1 ppm. Peak exposures between 10 and 20 ppm were also recorded numerous times during the worker's shift. The highest peaks of Cl_2 were 40 ppm (occurred twice) and $\geq$ 50 ppm (one peak) which was recorded for a period of 5 seconds at 23:32 hrs (see Figures 1 and 3). Results from the same monitor indicated that TLV STEL concentrations for Cl_2 were exceeded during four periods between 16:13 and 23:48 hrs (see Figure 2). The employee wore a half mask air-purifying respirator (APR) with dual chlorine cartridges during his work shift. On the same day, workplace concentrations of Cl_2 for the second ARO were read from the other real-time Cl_2 monitor but these data were not downloadable.

On November 4, 2004, another ARO wore the datalogging monitor while working in Buildings 1 and 2 of the electrolytics area (see Figures 4 and 5). The data logger was not placed on the worker after the start of the shift. Results indicated the worker's TWA was 0.1 ppm, and the STEL was 0.6 ppm, which exceeded the REL. The highest concentration recorded was 7.7 ppm at 1417 hrs. There were 44 discrete measurements that exceeded the Federal OSHA ceiling limit of 1 ppm. This worker wore a half mask APR configured with cartridges.

Carbon tetrachloride (CCl_4)

Thirteen full-shift PBZ samples for CCl_4 were collected from workers in maintenance, electrolytics, and the reactor building (see Table 12). All samples with the exception of one were not detected (ND) or were at trace concentrations (laboratory values reported between the LOD and the LOQ that are trace concentrations; considered by NIOSH to be semi-quantitative values.) The single sample that was above the LOD was collected on a vacuum wagon operator working in the electrolytics area. It had a TWA value of 0.18 mg/m^3 (0.03 ppm), which is well below the adjusted ACGIH TLV of 31.5 mg/m^3 (5 ppm), and below the Federal OSHA criterion of 12.6 mg/m^3 (2 ppm).

Hand and table top wipe samples for hexachlorobenzene (HCB)

Fifteen hand wipe samples and nine surface wipe samples were collected (see Table 13). Four of the hand wipe samples were less than the LOD of 0.03 µg per wipe, four were reported at trace concentrations (between 0.03 and 0.09 µg per wipe), and seven had quantitative values for HCB from 0.14 to 3.5 µg HCB per wipe.

Five of the surface wipe samples from lunchroom tables were reported as non-detected (ND). One sample was reported at trace and three samples had quantifiable concentrations of HCB in a range of 0.092 to 0.18 µg HCB per wipe.

Bulk sample for HCB, PCBs, and metals

A bulk sample was collected during cleanout of the Building 4 off-gas collection header. The sample was analyzed for HCB, Arochlors 1242 and 1254, and PCB congeners. Analytical results indicated the presence of HCB and three congeners of PCB. Results were reported in units of microgram/gram (µg/gr) as follows: HCB–250; PCB #114–1.3; PCB #157– 4.8; PCB #209–230 µg/gr. The following metals were also detected on a µg/gram basis: aluminum–2900; arsenic–30; calcium–51,000; cadmium–7.5; chromium–30; copper–31; iron–53,000; lithium–24,000; magnesium–64,000; manganese–150; nickel–9.6; phosphorus–2,700; sodium–56,000; titanium–1,400; vanadium–35; yttrium–trace (between 0.2 and 0.6 µg/gr); zinc–9.7; zirconium–14. The following metals were not detected: antimony, beryllium, cobalt, molybdenum, lead, platinum, selenium, tin, silver, tellurium, and thallium.

Hexachlorobenzene (HCB)

Forty-two PBZ and two area samples were collected for HCB (Table 14). All PBZ and area samples were above the MDC of 0.0036 µg/m^3. Personal samples ranged from 0.096 µg/m^3 for the full-shift sample collected on a foundry operator to 5.3 µg/m^3 for a maintenance helper working on the sixth floor reactor.

The two area samples (USM 17 and USM 20) were collected in electrolytics and on the sixth floor of the reactor building. Both samples were below the ACGIH TLV at 0.092 and 0.56 µg/m^3, respectively. Two PBZ samples exceeded the unadjusted ACGIH criterion of 2 µg/m^3 and three samples approached this criterion. The samples exceeding the unadjusted TLV (USM 10 and 57) were collected on the same worker, a reactor maintenance helper on the sixth floor reactor building on November 1, 2004, and November 4, 2004. The results were 2.3 and 5.3 µg/m^3, respectively. Three samples approached the unadjusted TLV and were collected from a reactor maintenance worker working on the high energy scrubber pump on the ground floor (USM 2, 1.5 µg/m^3), an assistant cell service operator working in electrolytics (USM 5, 1.4 µg/m^3), and a vacuum wagon operator in electrolytics (USM 26, 1.6 µg/m^3). Adjusting the TLV (following the same calculations as for chlorine) provides a value of 1.6 µg/m^3 as the exposure criterion. Considering the adjusted TLV, three samples exceeded or were at this value (USM 10 and 57 and USM 26). Two samples were just below the adjusted value TLV (USM 2 and 4). In total, 5 of 42 PBZ samples (approximately 12% of the total sample set) exceeded or were very close to the adjusted TLV for HCB.

Polychlorinated biphenyls (PCBs)

Since PCBs were sampled simultaneously with HCB, forty-two PBZ and two area samples were collected (Table 15). Neither Arochlor series, 1242 nor 1254, were detected to an LOD of 0.10 µg/sample or a MDC of 0.0036 µg/m^3. Nine of the twelve dioxin-like PCB congeners with assigned TEFs were detected, including congener numbers 77, 105, 114, 123, 126, 156, 167, 169, and 180. Congener #20, which does not have an assigned TEF, was also detected. The 10 congeners that were detected ranged from trace (between the LOD and LOQ) to 2 µg/sample for congener #209. Because only the Arochlor series of PCB has occupational exposure criteria, these results indicate that certain congeners of PCB are present in workplace air above the MDC. The 12 congeners with TEFs are considered to have 2,3,7,8-TCDD dioxin-like properties. The most commonly detected congener was #126, which was detected in 23 samples, followed by #209, which was detected in 21 samples. Other congeners that were detected (fewer than five times in all samples) included congener numbers 77, 105, 114, 123, 167, 180, and 169. The congener that was detected most often above the MDC was #209, which was detected in 14 samples in a range of 0.019 to 0.55 µg /m^3. The sample with the greatest concentration of all congeners of PCBs was a sample collected on a vacuum wagon operator working in electrolytics. Field notes taken during the HHE reveal that this worker was adding carbon to the cells during a portion of his shift. This same worker's sample for HCB was at the adjusted

TLV for HCB (1.6 µg /m^3) for the full shift, indicating that if the worker was not consistently using adequate respiratory protection (a properly-fitting facepiece and a combination cartridge) he was exposed to HCB and four congeners of PCB during the work shift.

Comparisons of HCB concentrations in the plant, based on area

Results for HCB indicated that concentrations were highest in the reactor area (n=15, GM = 0.68 µg/m^3) followed by electrolytics (n=21, GM = 0.38 µg/m^3) and were lowest in the foundry (n=7, GM = 0.21 µg/m^3). HCB concentrations were found to be significantly higher in the reactor compared to those in electrolytics (p=0.03). Significantly greater concentrations of HCB were found in the reactor compared to the foundry (p=0.01). No statistically significant differences were determined for HCB concentrations comparing electrolytics and the foundry (p=0.11).

Results for total PCBs indicate that geometric mean concentrations were highest in the electrolytics (0.026 µg/m^3), followed by reactor (0.014 µg/m^3), and lowest in the foundry (0.0087 µg/m^3). No statistically significant differences in GM concentrations for total PCBs were found for samples collected in the reactor compared to electrolytics (p=0.26), the reactor compared with the foundry areas (p=0.50) or electrolytics compared with the foundry (p=0.08).

Associations between HCB and PCB concentrations

Possible correlations between HCB and total PCB concentrations by areas of the plant were investigated. Log-transformed HCB concentrations were significantly and positively correlated with the log-transformed PCB concentrations (r = 0.36, p=.02). When higher HCB concentrations were seen on air samples, correspondingly higher PCB concentrations were also detected. Differences were found for concentrations of HCB in at least two of the three areas of the plant, but this was not the case for total PCBs.

DISCUSSION

Biological monitoring results

This investigation revealed detectable concentrations of PCDDs, PCDFs, and PCBs in the blood of all 30 workers selected for the study. For 12 of the 30 workers, TEQs (varying by percentage of individual congeners) were in excess of the U.S. general population levels. However, all biological measures of exposures were below the levels reported in published epidemiologic studies to be related to observable health effects. Because health-based biological exposure indices are not available for these compounds, the long-term health significance of these exposures is unclear.

The HCB levels of the 30 workers were higher than those found in the general U.S. population. Although the levels of HCB in the blood of U.S. Magnesium workers were higher than the general U.S. population levels (a concentration that is below the laboratory limit of detection, or essentially a non-detectable concentration), these levels have not been associated with observable clinical health problems in epidemiologic studies. The presence of detectable HCB in the blood of the 30 workers tested is likely from occupational exposures at U.S. Magnesium.

Similarity with other magnesium study

From these results, we find the profile of the 30 U.S. Magnesium workers' PCDDs and PCDFs to be similar to the profile reported in the Norwegian magnesium production environment,[91] and to be higher than the general population (which had no occupational exposure). OCDFs were found to be present as major contributors to the total TEQ among the 30 workers tested. Generally, OCDF is a minor component (and often not detected) in human samples. In a Norwegian magnesium facility with a similar process to U.S. Magnesium, PCDFs were reported higher than PCDDs, and

HpOCF and OCDF were the main congeners. A significant increase was found in the concentrations of some of the congeners, mainly PCDFs, in the nine workers studied in the Norwegian magnesium facility as compared with the control group.

Needman et al.[92] reported that for most general population studies 2,3,4,6,7,8 HxCDF, 1,2,3,7,8,9 HxCDF, 1,2,3,4,7,8,9 HpCDF, and OCDF were lower than the limit of detection. In the *Second National Report on Human Exposure to Environmental Chemicals*, most PCDFs of the U.S. general population were lower than the LOD except 2,3,4,7,8-PeCDF and 1,2,3,4,6,7,8-HpCDF.

Blood test results by department

Workers performing maintenance had the highest TEQ and the highest levels for PCDFs and HCB. Levels of PCDDs and PCBs were highest in the electrolytic department. The blood test results for PCDDs, PCDFs, and PCBs reflect accumulated or chronic exposures over many years. These compounds are not metabolized and excreted and can remain in the body for many years — they have a half-life of about 7.5 years. The half-life of blood HCB is reported to be 1–2 years. The job classification was by current work assignment during the time the study was conducted, so that exposure is not necessarily associated with the current job title, but may reflect exposure from work over several years.

Industrial hygiene exposure assessments

NIOSH investigators understand from review of previous reports that previous industrial hygiene investigations at U.S. Magnesium did not detect HCB or PCBs in workplace air. There may be several reasons for this discrepancy from our sampling results. The first reason is the larger sample air volumes achieved in this HHE due to using the latest technology in personal sampling pumps. The sampling pumps were capable of collecting samples for a full 12-hour shift at a constant volume. This allowed collection of a larger volume sample, thus allowing for a lower analytical limit of detection. We anticipated that large sample volumes would be necessary to calculate low MDCs for the chlorinated hydrocarbon analytes of interest, especially HCB. A second reason is the exposure assessment sampling strategy, which was designed to include as many workers in as many locations as possible. This sampling strategy was also designed to cover several work shifts, and many days of consecutive sampling during the investigation. Sampling results from previous surveys may not have been representative of exposures.

Workplace observations

While in the plant, NIOSH investigators observed many hazards. Some of these observations were discussed at a closing conference on November 4, 2004. Examples of safety hazards identified and corrective actions taken include the following: a large machine bolt was found wedged in the metal grating of a stair landing on the sixth floor reactor building. This was seen as a possible hazard to employees who might be walking or working below if the bolt loosened from foot traffic across the grating. NIOSH investigators brought this problem to the attention of the Safety Manager, who dealt with the hazard immediately. Another example concerned two emergency eye-wash stations in the north end of the fourth and sixth floors of the reactor building that were not in working order, apparently due to lack of water pressure. This was brought to the attention of the Safety Manager for resolution.

We noted warnings on the packaging information of the REFCO refractory material that free silica in the form of quartz and cristobalite was present. This material is refractory clay used to line the launders from the reactor to the holding tanks through which molten $MgCl_2$ salt flows. We witnessed employees using jackhammers to break out the old refractory in the launders. We observed considerable amounts of airborne dust generated during this process. It is likely that airborne crystalline silica is generated during this activity. Consequently, inhalation hazards to respirable

crystalline silica are likely to be present for employees not wearing respirators, or for those wearing respirators without particulate filtering capability.

We observed that Kaowool™ ceramic blanketing material was used as a thermal cover for the launders and used on the reactors to cover and presumably contain emissions where thermocouples were inserted. Kaowool™ is a porous material, and did not appear to contain or prevent discharge of a visible reddish-brown smoke (the composition of which is unknown at this time).

We also observed a worker removing and stacking molded ingots at the casting machine, which raised concerns about the degree and amount of bending and twisting required for the task of loading ingots onto pallets. NIOSH believes there is sufficient evidence for an association between low back pain and twisting and bending.

NIOSH investigators frequently observed maintenance and reactor workers with respirators worn around their necks, but not covering their nose and mouth while working on the sixth floor and on other floors of the reactor building. The north entry door on the sixth floor of the reactor building was clearly posted as a respirator use area at the time of the NIOSH HHE, but this was not actively enforced.

Interpretation of IH sampling data

The highest concentrations of HCB were detected in the reactor area followed by the electrolytics area. HCB area concentrations were lowest in the foundry. Statistically significant differences in HCB concentrations were found for workers in the reactor compared to workers in electrolytics and for workers in the reactor compared to those in the foundry.

Concentrations of PCB were highest in the electrolytics area (similar to the blood sampling results) followed by the reactor area; concentrations were lowest in the foundry. While there were quantitative differences in total PCBs, they were not statistically significant for any of the work areas.

HCB was detected on the skin of some employees and on various surfaces within the plant by hand wipe results. HCB has a skin notation, so the presence of even low concentrations suggests a hazard. Because sample collection efficiency is unknown for the method that was used, the results must be interpreted with some degree of caution.

Positive and statistically significant correlations were found between concentrations of HCB and PCBs. Provided that process operations do not change the positive correlation between HCB and PCB may suggest that sampling for HCB could be used as a surrogate for PCBs. This relationship between HCB and PCB needs further investigation, and could be explored after implementing ventilation engineering control options.

Reducing exposures

Our findings indicate that industrial processes in the workplace at U.S. Magnesium are not sufficiently controlled to prevent workplace overexposures to numerous chemicals. To reduce exposures, an appropriate hierarchy of controls including the use of engineering controls (such as local exhaust ventilation); administrative or managerial controls (such as ensuring employee adherence to occupational health and safety policies and procedures), and the proper use of personal protective equipment (respirators, etc.) are needed.

CONCLUSIONS

Our evaluation provides evidence of work-related exposure to dioxins, dibenzofurans, PCBs, and HCB. Although some exposure levels were above those found in the U.S. population, they were below levels associated with observable health effects. However, 2,3,7,8 TCDD and PCBs are considered by NIOSH to be potential human carcinogens; and there is no known threshold for human

exposures. Therefore, although levels are low, the presence of 2,3,7,8-TCDD, PCB, and other chemicals in workers' blood is sufficient reason to limit workplace exposures. Collecting additional blood samples on workers in other areas of the plant would not contribute to our further understanding of the CHC exposures at U.S. Magnesium or the effects of past exposures.

This study focused on exposure assessment and was not set up to determine the health status of the participants. However, in our interviews with the U.S. Magnesium workers, none reported a history of chloracne, nor had evidence of facial or extremity chloracne on examination during the week that we were on site. All 30 workers denied a history of liver or gastrointestinal problems. The results of the enzyme immunoassay for liver function were all within the normal range, providing no evidence of impairment to the liver. Four workers reported diabetes; two others mentioned a past history of hypoglycemia. Because our investigation was not designed to examine the rate of diseases in this population, and it was not a random sample, we are unable to state whether the number of cases of diabetes in this small group was unusual.

The area of the plant with the greatest risks for HCB and PCB exposures was determined by industrial hygiene sampling to be the reactor building and the electrolytics areas. Blood PCB levels were higher in the electrolytics area workers as well. However, although exposures were greater in some areas than in others, exposures do seem to be occurring throughout the plant. Fugitive emissions from chlorine and chlorinated hydrocarbons have been documented in several areas during the manufacturing processes.

For HCB, the specific work practices with the greatest risks for these exposures are unknown, but industrial hygiene sampling results suggest that workers performing reactor maintenance, and perhaps work being done by less experienced workers (as found by the NIOSH industrial hygiene sampling results), may lead to greater risk for exposures. It is not uncommon for the less experienced industrial worker to be at greater risk of exposure. This can be attributed to many factors, including longer time required to perform work tasks, hence longer time in an area of potentially high exposures.

We noted that the majority of workers reported exposures to chlorine gas and upper airway irritation during their employment at U.S. Magnesium. This is consistent with the industrial hygiene data demonstrating a significant degree of risk for Cl_2 overexposures. Considering the concentrations of the chlorine exposures that were measured, it is not surprising that many of the workers reported respiratory irritation.

Engineering controls should be the primary means of controlling exposures. However, until those controls are in place, respirators will be required in those areas identified as posing exposure risks. While employees wearing well-fitting and appropriately configured half mask respirators can be adequately protected from Cl_2, as well as CHC exposures, air-purifying respirators are not designed to protect wearers to Cl_2 concentrations of 10 ppm, a concentration that was measured on numerous occasions and that represents a serious inhalation hazard. The magnitude and unpredictability of Cl_2 atmospheres in ranges of 10–50 ppm must be viewed not only with respect to occupational exposure criteria such as TWA and STEL values, but also must consider the NIOSH IDLH concentration, which is defined as 10 ppm as a 30-minute exposure.[16] While the discrete peak exposures that were recorded were not 30 minutes in length (and therefore technically could not considered to be IDLH), the fact that concentrations of exposures to Cl_2 at this level occurred with some regularity indicates the severity of the hazard.

Due to the unpredictability of such high concentrations of Cl_2, workers at U.S. Magnesium who are not wearing their respirators correctly, or have beard stubble that interferes with the seal of the mask, or especially those who are not wearing them at all, are at

serious risk for Cl_2 overexposures that could result in acute and chronic adverse health effects involving the lungs, eyes, and upper respiratory system. The areas of the plant with the greatest risks for Cl_2 exposures appear to be the reactor building and the electrolytics areas; however, because the exact location(s) of these releases are not known, it is possible that these risks are present plant-wide.

RECOMMENDATIONS

The following recommendations were included in the NIOSH interim letters dated December 21, 2004, and February 2, 2005. Appendix D includes a copy of the letter from U.S. Magnesium to the EPA regarding the implementation plan for these recommendations.

Engineering controls and exposure assessment

1) U.S. Magnesium managers, the union, and the workers should:
    a. Identify all potential areas of exposures to dioxins, dibenzofurans, PCBs, HCB, and Cl_2.
    b. Evaluate the effectiveness of current engineering controls intended to reduce fugitive emissions.
    c. Implement appropriate local exhaust ventilation and general dilution ventilation engineering control measures.
    d. Initially focus on the reactor and electrolytics areas, but also include the foundry.
    e. Work together to assess work practices and tasks.
2) Investigate the electrolytics area to determine if vacuum wagon operators (who remain with their vehicles for most of the shift) have increased risks for overexposures to HCB and Cl_2, compared to cell service operators (or others) who add carbon to cells, service the leveling wells, or whose work practices require close work around the cells and potential exposures to cell emissions.
3) Consider the need for additional posting of mandatory respirator use areas on other floors of the reactor building and electrolytics area, unless the source(s) of the chlorinated hydrocarbon and Cl_2 emissions can be confirmed and repaired.
4) Use air monitoring results from full-shift PBZ sampling as the metric that determines if posting of additional areas is required.
5) Conduct additional PBZ air monitoring if the process operations changes, to determine whether exposure risks have changed.
6) Conduct full-shift sampling on employees in the reactor building to evaluate exposures to respirable quartz and cristobalite during removal and relining of the launders. Conduct representative PBZ exposure assessments for employees working in areas of the building where dusts are reasonably expected to migrate (i.e., PBZ samples for workers on the floors above or working in adjacent areas, and representative area samples in the area.).
7) Repair emergency eye wash stations on the 4^{th} and 6^{th} floors of the reactor building, then conduct maintenance on a regular schedule. In the interim, install portable eye wash stations in these locations if repairs are expected to be lengthy because of the time required to obtain repair parts, or additional plumbing requirements.
8) Use a palletizer and a pallet jack, or other methods, to reduce the degree of bending and required to transfer ingots from the cast machine to the pallet.

Work practices

9) Do not use compressed air to clean accumulated dust off the tops of the electrolytic cells. This results in resuspension of particulates into the workplace increasing inhalation hazards for workers in the area. Consider other dust removal methods such as HEPA vacuuming.

Administrative

10) Train workers about CHCs and the routes of exposure (including inhalation, ingestion, and possibly dermal exposure).

11) Train workers in practices to control or limit potential exposures, including careful hand hygiene especially before eating, drinking, smoking, or bathroom breaks.

Personal protective equipment

12) Enforce entire sixth floor of the reactor building as a mandatory respirator use area until adequate engineering controls are put into place.
13) Regarding work clothes:
    a. Enforce the policy on providing and laundering work clothes and laundering work clothes on site.
    b. Prevent workers from taking work clothing home, to avoid taking home potentially contaminated clothes.
    c. Inform individuals who launder used work clothing of the potential hazards (and the control measures necessary) of exposures to chlorinated hydrocarbons including dioxins, furans, PCBs, and HCB.
14) Institute a quantitative fit test program for all employees who wear respirators, due to the potential for uncontrolled and unpredictable releases of Cl_2 gas. This recommendation is based on the NIOSH Guide to Industrial Respiratory Protection that states: "quantitative fit testing is recommended when face piece leakage must be minimized for work in highly toxic atmospheres or those immediately dangerous to life or health."
15) Use NIOSH-approved combination cartridges (Cl_2, acid gas and particulate) in respirators to prevent exposures to fume, aerosols and respirable particulates which are generated and present in the plant in addition to Cl_2 gas. The use of NIOSH-approved particulate arrestance pre-filters (e.g., N-95 or greater) in combination with Cl_2 cartridges is also suggested as an interim measure if problems arise with use of combination cartridges that may interfere with employee use of protective face shields.
16) Remind employees who wear respirators of the importance of carefully shaving each day before coming to work. NIOSH investigators observed notable beard stubble on numerous workers, which can dramatically compromise the fit of negative pressure air-purifying respirators.
17) Workers who clean out anode dust boxes should wear two pairs of nitrile gloves during this operation. We observed workers who conducted this operation with torn gloves. Wearing two pairs of gloves offers additional protection and allows the worker to doff the most contaminated glove layer first, then remove other personal protective clothing and respiratory protective equipment, minimizing cross contamination to respirator face pieces, safety glasses, and work uniforms worn underneath the outer Tyvek® (or other brand) protective coverall.

Medical practices

18) Although there is continuing potential exposure to CHCs, including PCBs, dioxins, dibenzofurans, we do not believe that further blood testing of these compounds for surveillance purposes is warranted.

The NIOSH investigation was not designed to examine the scope of diseases among workers at U.S. Magnesium. The selection of workers involved in the blood testing was to determine levels of CHCs among 30 long term workers in areas thought to have high exposure to CHCs. It was not a random sample. If there are concerns about long-term disease states among the workers (which is a separate issue from CHC exposure), a more thorough, prospective epidemiologic investigation would need to take place.

REFERENCES

1. Personal communication with Hryhorczuk D. University of Illinois; Calvert G, NIOSH, Piacitelli L. NIOSH, Steenland K, Emory University.

2. Akins Jr., Waldrep K, Bernert JT [1989]. The estimation of total serum lipids by a completely

enzymatic summation method. Clin Chem Acta 184:219-226.

3. High sensitivity Alpha GST EIA; Biotrin International, Dublin, Ireland.

4. NIOSH [2003]. NIOSH manual of analytical methods. 4th ed. Hydrocarbons, halogenated, method 1003. Cincinnati, OH. DHHS (NIOSH) Publication No. 94-113.

5. http://www.epa.gov/ttn/amtic/files/ambient/airtox/to-10ar.pdf (accessed September 29, 2004).

6. NIOSH [2003]. NIOSH manual of analytical methods. 4th ed. Elements by ICP, method 7303. Cincinnati, OH. DHHS (NIOSH) Publication No. 94-113.

7. NIOSH [2003]. NIOSH manual of analytical methods. 4th ed. Chlorinated and Organonitrogen Herbicides, method 5602. Cincinnati, OH. DHHS (NIOSH) Publication No. 94-113.

8. NIOSH [1992]. Recommendations for occupational safety and health: compendium of policy documents and statements. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 92-100.

9. ACGIH [2005]. 2005 TLVs and BEIs: threshold limit values for chemical substances and physical agents. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

10. CFR [1997]. 29 CFR 1910.1000. Code of Federal Regulations. Washington, DC: U.S. Government Printing Office, Office of the Federal Register.

11. http://www.rules.utah.gov/publicat/coder614/r614-006.htm#T7.

12. Anderson ME, MacNoughton MG, [1987]. Adjusting exposure limits for long and short exposure periods using a physiological pharmacokinetics model. Am Ind Hyg Assoc J 48:335-343.

13. NIOSH [1978]. NIOSH/OSHA occupational health guidelines for chemical hazards (chlorine). Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 81-123.

14. Hathaway GJ. Proctor NH, Hughes JP, Fischman ML [1991]. Proctor and Hughes' Chemical Hazards of the Workplace, 3rd ed. Van Nostrand Reinhold, NY. p. 153.

15. NIOSH [2004]. NIOSH pocket guide to chemical hazards. 4th ed. NIOSH Publications Cincinnati, OH. DHHS (NIOSH) Publication NO. 97-140.

16. NIOSH [1996]. Chlorine Immediately Dangerous to Life and Health Documentation. http://www.cdc/gov/niosh/idlh/7782505.html accessed 12/14/04

17. Agency for Toxic Substances and Disease Registry. [2003]. Draft Toxicological Profile for Carbon Tetrachloride CAS'# 56-23-5; September.

18. Carbon Tetrachloride [1999] International Programme on Chemical Safety (IPCS), United Nations Environment Programme, the International Labour Organisation/World Health Organization, Environmental Health Criteria 208:1-5.

19. EPA [1984] Health assessment document for carbon tetrachloride. US Environmental Protection Agency (EPA), Environmental Criteria and Assessment Office, Cincinnati, Ohio.

20. ACGIH [2001]. American Conference of Governmental Industrial Hygienists (ACGIH).

Documentation of Threshold Limit Values for Chemical Substances, Carbon Tetrachloride. ACGIH, Cincinnati, Ohio.

21. http://www.cdc.gov/niosh/topics/cancer/ [2005].

22. http://www.cdc.gov/niosh/pel88/56-23.html *OSHA comments from the January 19, 1989 Final Rule on Air Contaminants Project extracted from 54FR2332 et. seq.* Accessed 30 November, 2004.

23. Ballschmitter K, Rappe C, Buser HR. [1989] Chemical properties, analytical methods, and environmental levels of PCBs, PCTs, PCNs, and PBBs. InL Kimbrough RD, JensenAA, eds., Halogenated Biphenyls, terphenyls, naphthalenes, dibenzodioxins, and related products. AdsterdamL Elsevier Biomedical Press, L47-342. 20.

24. Ballschmitter K, Rappe C, Buser HR. [1989] Chemical properties, analytical methods, and environmental levels of PCBs, PCTs, PCNs, and PBBs. InL Kimbrough RD, JensenAA, eds., Halogenated Biphenyls, terphenyls, naphthalenes, dibenzodioxins, and related products. AdsterdamL Elsevier Biomedical Press, L47-342. 20.

25. Environmental Protection Administration. [2005] Polychlorinated Biphenyls. http://www.epa.gov/opptintr/pcb.. 2005.

26. Kimbrough RD and Krouskas CA [2003] Human exposure to poychlorinated biphenyls and health effects. Toxic Rev 22 (4) 217-32.

27. Kimbrough RD.[1995] Polychlorinated biphenyls (PCBs) and human health an update. Crit Rev Toxicol: 25: 133-163.

28. Cogliano VJ.[1998] Assessing the cancer risk from environmental PCBs. Environmental Health Perspectives 1998 Jun;106(6):317-23.

29. Schantz SK, Widholm JJ, Rice DC. [2003] Effects of PCB exposure on neuropsychological function in children. Environ Health Perspectives; 111: 1-20.

30. Ross G. [2004] The public health implications of polychlorinated biphenyls (PCBs) in the environment. Ecotoxicol Environ Saf. Nov; 59(3):275-91.

31. Longnecker MP, Rogan WJ, Lucier G. [1997] The human health effects of DDT (dichlorodiohenyltrichloroethane) and PCBs (polychlorinated biphenyls) and an overview of organochlorines in public health. Annual Rev Public Health 18:221-44.

32. U.S. Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Diseases Registry. [2000] Toxic profile for polychlorinated biphenyls (update). Nov. NTIS PB 2000 10827.

33. U.S. Environmental Protection Agency [1996] PCBs: Cancer dose-response assessment and application to environmental mixtures. National Center for Environmental Assessment Office of Research and Development EPA/600/P-96/001F.

34. Mallin K, McCann K, D'Aloisio A, Freels S, Piorkowski J, Dimos J, Persky V. [2004] Cohort mortality study of capacitor manufacturing workers, 1944-2000. J Occup Environ Med. Jun; 46(6):565-76.

35. Gray KA, Klebanoff MA, Brock JW, Zhou H, Darden R, Needham L, Longnecker MP. [2005] In utero exposure to background levels of polychlorinated biphenyls and cognitive functioning among school-age children. Am J Epidemiol. Jul 1; 162(1):17-26.

36. Arisawa K, Takeda H, Mikasa H. [2005] Background exposure to PCDDs/PCDFs/PCBs and its potential health effects: a review of epidemiologic studies. J Med Invest Feb;52(1-2):10-21.

37. Schoenroth L, Chan S, Fritzler M. [2004] Autoantibodies and levels of polychlorinated biphenyls in persons living near a hazardous

waste treatment facility. J Investig Med. Apr;52(3):170-6.

38. Brown DP, Jones J. [1981] Mortality and industrial hygiene of workers exposed to PCBs Arch Environ Health; 36:120-9.

39. Bertazzi PA, Riboldi L, Pesatori A, et al. [1987] Cancer Mortality of capacitor manufacturing workers Am J Ind Med; 11: 165-76.

40. Taylor PR. [1988] The health effects of PCBs (doctoral thesis). Boston, Mass. Harvard School of Public Health.

41. Kimbrough RD, Doemland ML, LeVois ME. [1999] Mortality in male and female capacitor workers exposed to polychlorinated biphenylsl J Occ Env Med; 41: 161-71.

42. Kimbrough RD, Doemland ML, Mandel JS. [2003]. A mortality update of male and female capacitor workers exposed to chlorinated biphenyls. J Occ Env Med; 45:271-82.

43. Sinks T, Steele G, Smith A,, et al. [1992] Mortality among workers exposed to polychlorinated biphenyls. Amer J Epi; 136:389-98.

44. Bahn AK, Grover P, Rosenwaike, I et al. [1977] Melanoma after exposure to PCBs. [letter] NEJM; 295:450.

45. Loomis D, Browning SR, Schenk AP, et al. [1997]. Cancer mortality among electric utility workers exposed to polychlorinated biphenyls. Occ Env Med 1997; 54: 720-8.

46. Maroni M, Colombi A. Arbosti G, Cantoni S, Foa V [1981]. Occupational exposure to polychlorinated biphenyls in electrical workers, II. Health effects. Br J Ind Med 38:55.

47. Ouw KH, Simpson GR, Siyali DS [1976]. The use and health effects of Aroclor 1242, a polychlorinated biphenyl in the electrical industry. Arch Environ Health 31:189.

48. Karppanen E, Kolho L [1973]. The concentration of PCB in human blood and adipose tissue in three different research groups. In: PCB Conference II, Stockholm, 1972. National Swedish Environmental Protection Board, 124.

49. Smith AB, Schloemer J, Lowry LK, et al [1982]. Metabolic and health consequences of occupational exposure to polychlorinated biphenyls (PCBs). Br J Ind Med 39:361.

50. Fischbein A, Woolf MS, Lilis R, Thornton J, Selikoff IJ [1979]. Clinical findings among PCB_exposed capacitor manufacturing workers. Ann NY Acad Sci 320:703.

51. Chase KH, Wong O, Thomas D, Berney BW, Simon RK [1982]. Clinical and metabolic abnormalities associated with occupational exposure to polychlorinated biphenyls (PCBs). J Occup Med 24:109.

52. NIOSH [1984]. Health hazard evaluation report no. HETA 80_007_1520. Cincinnati, OH: National Institute for Occupational Safety and Health.

53. M, Yoshimura T, Matsuzaka J, Yamaguchi A [1972]. Epidemiologic study on Yusho, a poisoning caused by ingestion of rice oil contaminated with commercial brand of polychlorinated biphenyl. Environ Health Perspect 1:119.

54. Wong CK [1981]. PCB poisoning special issue. Clinical Medicine (Taipei), Volume 7, no. 1.

55. Lawton RW, Sack BT, Ross MR, Feingold J [1981]. Studies of employees occupationally exposed to PCBs. General Electric Research and Development Center, Schenectady.

56. Crow KD [1970]. Chloracne: a critical review including a comparison of two series of cases of acne from chloronaphthalene and pitch fumes. Trans St John's Hosp. Dermatol Soc 56:79.

57. Baker EL Jr., Landrigan PJ, Glueck CJ [1980]. Metabolic consequences of exposure to polychlorinated biphenyls in sewage sludge. Am J Epid 112:553.

58. Kreiss K, Zack MM, Kimbrough RD, Needhan LL, Smrek AL, Jones BT [1981]. Association of blood pressure and polychlorinated biphenyl levels. JAMA 245:2505.

59. NIOSH [1986]. Current intelligence bulletin 45: polychlorinated biphenyls (PCB's): potential health hazards from electrical fires or failures. Cincinnati, OH: National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 86-111.

60. Veterans and Agent Orange: Update 2002; [2003] Committee to Review the Health Effects in Vietnam Veterans of Exposure to Herbicides (Fourth Biennial Update) National Academy of Sciences. Washington, D.C.

61. Sweeney MH, Calvert GM, Egeland GA, Fingerhut MA, Halperin WE, Piacitelli LA. [1997-8] Review and update of the results of the NIOSH medical study of workers exposed to chemicals contaminated with 2,3,7,8-tetrachlorodibenzodioxin. Teratog Carcinog Mutagen;17(4-5):241-7.

62. Lansing P-J, Korff R. [1994] Blood levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin and -globulins in a follow-up investigation of employees with chloracne. J Dermatol Sci.:91-95.

63. Mocarelli, P; Needham LL; Marocchi A; et al. [1991] Serum concentrations of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and test results from selected residents of Seveso, Italy. J Toxicol Environ Health 32:357-366.

64. Schecter A, McGee H, Stanley J, et al. [1993] Chlorinated dioxin, dibenzofuran, coplanar, mono-ortho, and di-ortho substituted PCB congener levels in blood and semen of Michigan Vietnam veterans compared with levels in Vietnamese exposed to Agent Orange. Chemosphere;27(1-3):241-252.

65. Needham LL, Patterson DG Jr., Houk VN. [1991] Levels of TCDD in selected human populations and their relevance to human risk assessment. Banbury Report 35: Biological basis for risk assessment of dioxins and related compounds. Cold Spring Harbor Press, p 229-247.

66. Hoffman, RE; Stehr-Green, PA; Webb, KB; et al. [1986] Health effects of long-term exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. JAMA 255:2031-2038.

67. Webb, KB; Evans, RG; Knudsen, DP; et al. [1989] Medical evaluation of subjects with known body levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin. J Toxicol Environ Health; 28:183-193.

68. Steenland K, Calvert G, Ketchum N, Michalek J. [2001]. Dioxin and diabetes mellitus: an analysis of the combined NIOSH and Ranch Hand data. Occup Environ Med; 58(10):641-8.

69. Steenland K, Piacitelli L, Deddens J, Fingerhut M, Chang L. [1999]. Cancer, heart disease, and diabetes in workers exposed to 2,3,7,8, TCDD. J Natl Cancer inst. 91(9):779-86.

70. Vena J; Boffetta P; Becher H; et al. [1998]. Exposure to dioxin and non-neoplastic mortality in the expanded IARC international cohort study of phenoxy herbicide and chlorophenol production workers and sprayers. Environ Health Perspect 106 Suppl 2:645-653.

71. Pesatori, AC; Zocchetti, C; Guercilena, S; et al. [1998] Dioxin exposure and non-malignant health effects: a mortality study. Occup Environ Med; 55:126-131.

72. USAF [2005] An epidemiologic investigation of health effects in Air Force Personnel following exposure to herbicides: 2002 follow-up examination results May 2002-March 2005. Contract F41624-01-C-1012.

73. USEPA [2000] Part III: Integrated summary and risk characterization for 2,3,7,8-TCDD and related compounds. September 2000, SAB Review Draft. EPA/600/P-00/001Bg.

74. Fingerhut MA, Halperin WE, Marlow DA, et al. [1991] Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. New Engl J Med 1991; 324:212-218.

75. Hooiveld, M; Heederik, D. [1996] Preliminary results of the second follow-up of a Dutch cohort occupationally exposed to phenoxy herbicides, chlorophenols, and contaminants. Organohalogen Compounds;30:185-189.

76. Hooiveld M; Heederik DJ J; Kogevinas M; et al.[1998] Second follow-up of a Dutch cohort occupationally exposed to phenoxy herbicides, chlorophenols, and contaminants. Am J Epidemiol;147(9):891-901.

77. Kogevinas, M; Becher, H; Benn, T; et al. [1997] Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxin. An expanded and updated international cohort study. Am J Epidemiol;145 (12): 1061-1075.

78. Mackie D; Liu J; Loh, Y, Thomas V. [2003] No evidence of dioxin cancer threshold. Environmental Health Perspectives;111(9):1145-1147

79. International Agency for Research on Cancer (IARC). [1997]. Polychlorinated dibenzo-para-dioxins and polychlorinated dibenzofurans. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol. 69. Lyon, France.

80. Peters HA, Cripps DJ, Lambrecht RW, Erturk E, Morris CR, Bryan GT. [1986] IARC, History and geography of hexachlorobenzene poisoning in southeastern Turkey. Sci Publ;(77):131-2.

81. Currier MF. [1980]. et.al., Hexachlorobenzene blood levels and the health status of men employed in the manufacture of chlorinated solvents. J. Toxicol. Enviorn. Hlth. 6:367-377.

82. Sala M, Sunyer J, Otero R, Santiago-Silva M, Ozalla D, Herrero C, To-Figueras J, Kogevinas M, Anto JM, Camps C, Grimalt J. [1999] Health effects of chronic high exposure to hexachlorobenzene in a general population sample. Arch Environ Health;54(2):102-09.

83. Daniel V, Huber W, Bauer K, Suesal C, Conradt C, Opelz G. [2001] Associations of blood levels of PCB, HCHS, and HCB with numbers of lymphocyte subpopulations, in vitro lymphocyte response, plasma cytokine levels, and immunoglobulin autoantibodies. Environ Health Perspect; 9(2):173-178.

84. Queiroz ML, Bincoletto C, Perlingeiro RC, Souza CA, Toledo H. [1997] Defective neutrophil function in workers occupationally exposed to hexachlorobenzene. Hum Exp Toxicol.;16(6):322-326.

85. Queiroz ML, Bincoletto C, Perlingeiro RC, Quadros MR, Souza CA. [1998] Immunoglobulin levels in workers exposed to hexachlorobenzene. Hum Exp Toxicol; 17(3):172-175.

86. Van Den Berg, M. et al. Toxic equivalent factors (TEFs) for PCBsm PCDDs, PCFS for humans and wildlife. *Environmental Health Perspectives*, 106:775-792 (1998).

87. CDC, NCEH [2003] Second National Report on Human Exposure to Environmental Chemicals. NCEH Pub. No. 02-0716 Revised 2003.

88. National Center for Health Statistics. Plan and operation of the fourth National Health and Nutrition Examination Survey, 1999–2000. National Center for Health Statistics (NCHS).

89. Patterson Jr. DG, Patterson D, Canady R, Wong LY, Lee R, Turner W, Caudill S, Needham L, Henderson A.[2004] Age Specific Dioxin TEQ Reference Range. Organohalogen Compounds; 66:2878-2883.

90. Hornung RW, Reed LD. Estimation of average concentration in the presence of nondetectable values. Appl Occ Environ Hyg 1990; 5:46-51.

91. Rappe C. Dioxin, Patterns and source identification. Chemosphere 1994: 348: 63-75.

92. Needham LL, Patterson DG Jr., Burse VW, et al. [1996] Reference range data for assessing exposure to selected environmental toxicants. Toxicology and Industrial Health 1996;2(3/4):507-513.

Table 1
HETA 2004-0169-2982
U.S. Magnesium Corporation, Rowley, Utah
WHO Toxicity Equivalent Factors for Dioxin-like Polychlorinated Biphenyls

Congener #	IUPAC* Chlorobiphenyl Isomer Prefix	TEFs–WHO_{98}† (Humans and Mammals)
77	3,3',4,4'-tetra	0.001
81	3,4,4',5 –tetra	0.001
105	2,3,3',4,4'-penta	0.001
114	2,3,4,4',5-penta	0.005
118	2,3',4,4'5-penta	0.001
123	2,3',4,4'5'-penta	0.001
126	3,3',4,4'5-penta	0.1
156	2,3,3',4,4',5-hexa	0.005
157	2,3,3',4,4',5'-hexa	0.005
167	2,3',4,4',5,5'-hexa	0.00001
169	3,3',4,4',5,5'-hexa	0.01
170	2,2',3,3',4,4',5,-hepta	No TEF assigned
180	2,2',3,4,4',5,5'-hepta	No TEF assigned
189	2,3,3',4,4'5,5'-hepta	0.0001

*IUPAC is the abbreviation for International Union of Pure and Applied Chemists
†WHO Toxicity Equivalent Factors

Table 2
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
Reference value of TEQ* for the U.S general population

Age group (years)	N	Mean†	Std	Median†	P75†	P90†	P95†	Min†	Max†
30 – 44	199	11.8	6.9	9.8	16.6	21.1	23.2	0.2	50.4
45 – 59	160	16.9	9.6	14.9	22.3	29.5	32.8	0.8	55.4

***TEQ = toxic equivalency quotient.** The TEQ is a shorthand method for comparing the toxicity of types or mixtures of dioxins to the toxicity of the compound 2,3,7,8-TCDD.
†Units: pg/g lipid (parts per trillion)

Table 3
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
Characteristics of the Participants

	Total (n=30)	Cell Brick (n=11)	Electrolytic (n=8)	Maintenance (n=6)	Reactor (n=5)
Age (Years)	50.3 (40-59)	48.2* (42-56)	54.6* (50-59)	50.5 (40-58)	47.8* (46-51)
BMI (kg/m^2)	30.4 (20.3-42.6)	31.2 (26.9-42.6)	30.0 (20.3-35.0)	30.8 (27.6-40.7)	28.4 (22.4-33.0)
Working duration	26.9 (19.9-31.4)	26.9 (22.9-30.3)	26.4 (19.9-31.4)	25.5 (20.0-29.1)	24.4 (20.3-26.7)
Fish consumption† (times/week) Mean frequency (times/week)	0.5 (0-3.0)	0.7 (0-3.0)	0.4 (0-1.0)	0.4 (0-1.0)	0.5 (0-1.0)
Number of workers	9	3	2	2	2
Ever smoked cigarettes	7	1	2	2	2
Drink alcohol (yes)	15	5	3	4	3
Heat‡	7	3	1	1	2

*P<0.05

†Number of workers who ate fish more than one time / week

‡Number of workers using coal or wood heating in their homes

Table 4
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
Frequency of Past Medical Disorders

Medical Condition	**Total** (n=30)
Arthritis	12
Respiratory	5
Respiratory Allergy	5
Gout	5
Diabetes	5
Cardiovascular disorder	4
Gastrointestinal	6
Hypoglycemia	2
Psychological	2
Acne	2
Blackheads	1

Table 5
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
TEQ* of U.S. Magnesium by age group

Age	Tenure (years)	N	Mean	Min	Max
40 - 44	22.1 (20.0-25.0)	3	20.95	13.8	34.6
45 - 49	26.6 (20.2-29.3)	10	28.9	17.1	58.6
50 - 54	26.7 (20.7-30.3)	10	37.4	12.0	89.5
55-59	27.2 (19.9-31.4)	7	53.2	26.1	147
Total	26.1 (19.9-31.4)	30	37.3	12.0	147

Min: Minimum, Max: Maximum

*****TEQ-WHO_{98} = toxic equivalency quotient. The TEQ is a shorthand method for comparing the toxicity of types or mixtures of dioxins to the toxicity of the compound 2,3,7,8-TCDD.

Table 6
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
TEQ* by Department

Department	No.	Age (Years)	TEQ-WHO_{98}*	
			Mean	Range
Cell Brick	11	48.2	23.0	13.8 - 48.5
Electrolytic	8	54.6	35.5	12.0 - 57.1
Maintenance	6	50.5	63.8	24.6 - 146.8
Reactor	5	47.8	39.7	26.7 - 55.4
Total	30	50.3	37.3	12.0 – 146.7

*****TEQ-WHO_{98} = toxic equivalency quotient, World Health Organization 1998.

Table 7
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
Means of PCDDs, PCDFs and PCBs by Department

	PCDDs		PCDFs		PCBs*	
	pg/g lipid*	TEQ†	pg/g lipid	TEQ	ng/g lipid‡	TEQ
Cell Brick	287.1 ± 109.7	8.7 ± 3.2	217.8 ± 205.9	12.7 ± 6.6	13.4 ± 1.3	4.8 ± 1.3
Electrolytic	536.3 ± 312.5	13.1 ± 6.2	614.2 ± 270.9	20.3 ± 8.8	17.2 ± 1.3	6.1 ± 1.4
Maintenance	348.7 ± 68.4	12.7 ± 6.6	707.7 ± 679.3	47.6 ± 42.2	15.0 ± 1.4	5.5 ± 1.4
Reactor	320.2 ± 183.7	13.1 ± 3.9	340.9 ± 153.6	24.6 ± 8.3	15.2 ± 1.4	6.1 ± 1.5
Total	371.4 ± 209.6	11.6 ± 5	442 ± 398.5	23.7 ± 22.7	15.0 ± 13.1	5.5 ± 1.4

*Sum of congeners (pg/g lipid unit)

† TEQ: toxic equivalency factor

‡Sum of PCB congeners (ng/g lipid unit), including the coplanar PCBs and Ortho-PCBs having greater than 0 TEQ

Table 8
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
Mean of OCDDs, OCDFs, and PCBs

	Mean	Minimum	Maximum
OCDDs (pg/g lipid)			
2,3,7,8-TCDD	1.28	< LOD	4.40
1,2,3,7,8-PeCDD	5.22	< LOD	10.00
1,2,3,4,7,8-HxCDD	3.22	< LOD	9.60
1,2,3,7,8,9-HxCDD	37.45	19.20	63.00
1,2,3,6,7,8-HxCDD	6.80	< LOD	16.80
1,2,3,4,6,7,8-HpCDD	35.90	8.10	82.70
1,2,3,4,6,7,8,9-OCDD	281.50	106.00	1060.00
OCDFs (pg/g lipid)			
2,3,7,8-TCDF	0.61	< LOD	2.90
1,2,3,7,8-PeCDF	4.97	< LOD	42.50
2,3,4,7,8-PeCDF	20.20	5.90	86.2
1,2,3,4,7,8-HxCDF	51.30	11.40	257.00
1,2,3,7,8,9-HxCDF	51.27	10.20	322.00
1,2,3,6,7,8-HxCDF	0.26	< LOD	3.11
2,3,4,6,7,8-HxCDF	6.80	< LOD	30.20
1,2,3,4,6,7,8-HpCDF	192.49	33.70	951.00
1,2,3,4,7,8,9-HpCDF	13.87	< LOD	68.50
1,2,3,4,6,7,8,9-OCDF	97.89	8.60	440.00
PCBs			
Coplanar (pg/g lipid)			
3,4,4',5-TCB (PCB 81)	0.70	< LOD	8.30
3,3',4,4',5-PeCB (PCB 126)	18.05	9.10	42.30
3,3',4,4',5,5'-HxCB (PCB 169)	18.37	4.80	38.60
Ortho PCBs (ng/g lipid			
2,3',4,4',5-PeCB (PCB 118)	4.17	< LOD	10.60
2,3,3',4,4'-PeCB (PCB 105)	< LOD	< LOD	< LOD
2,3',4,4',5,5'-HxCB (PCB 167)	< LOD	< LOD	< LOD
2,3,3',4,4',5-HxCB (PCB 156)	4.26	< LOD	14.90
2,3,3',4,4',5'-HxCB (PCB 157)	0.11	< LOD	3.30
2,3,3',4,4',5,5'-HpCB (PCB 189)	< LOD	< LOD	< LOD

Table 9
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
Contribution of Individual OCDDs, OCDFs, and PCBs to total TEQ Proportion of the each Congener Higher than 95th Percentile of U.S. general population

		>95th percentile
OCDDs	**Percent of TEQ**	Number (percent) of workers
2,3,7,8-TCDD	5.1 (1.2 - 10.6)	0
1,2,3,7,8-PeCDD	7.6 (1.5 - 15.2)	14 (47%)
1,2,3,4,7,8-HxCDD	0.9 (0 - 2.4)	NA
1,2,3,7,8,9-HxCDD	11 (4 - 19.2)	30 (100%)
1,2,3,6,7,8-HxCDD	2 (0.6 - 4)	9 (30%)
1,2,3,4,6,7,8-HpCDD	1.1 (0.2 - 2.4)	NA
1,2,3,4,6,7,8,9-OCDD	0.9 (0.1 - 2.2)	1 (3.3%)
OCDFs		
2,3,7,8-TCDF	0.3 (0.1 - 0.8)	0
1,2,3,7,8-PeCDF	0.6 (0.2 - 1.8)	8 (26.7%)
2,3,4,7,8-PeCDF	24.5 (17.2 - 33.4)	30 (100%)
1,2,3,4,7,8-HxCDF	11.6 (6.6 - 18.6)	30 (100%)
1,2,3,7,8,9-HxCDF	11.1 (6.8 - 21.3)	30 (100%)
1,2,3,6,7,8-HxCDF	0.6 (0.1 - 2.1)	5 (16.7%)
2,3,4,6,7,8-HxCDF	1.7 (0.6 - 2.9)	18 (60%)
1,2,3,4,6,7,8-HpCDF	4.7 (1.4 - 14.5)	30 (100%)
1,2,3,4,7,8,9-HpCDF	0.3 (0.1 - 1.1)	NA
1,2,3,4,6,7,8,9-OCDF	0.3 (0 - 3.6)	26 (86.7%)
PCBs		
3,4,4',5-TCB (PCB 81)	-	0
3,3',4,4',5-PeCB (PCB 126)	5.4 (1.5 - 12)	0
3,3',4,4',5,5'-HxCB (PCB 169)	0.5 (0.2 - 0.9)	0
2,3',4,4',5-PeCB (PCB 118)	1.5 (0.3 - 3.4)	0
2,3,3',4,4'-PeCB (PCB 105)	0.5 (0.1 - 1.5)	0
2,3',4,4',5,5'-HxCB (PCB 167)	0 (0 - 0.2)	0
2,3,3',4,4',5-HxCB (PCB 156)	6.8 (1.9 - 14.5)	0
2,3,3',4,4',5'-HxCB (PCB 157)	2.5 (0.7 - 7.5)	0
2,3,3',4,4',5,5'-HpCB (PCB 189)	0.5 (0.1 - 1.5)	0

Table 10
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
Hexachlorobenzene Blood Levels

Department	N	Method	Mean	Minimum	Maximum
Cell Brick	11	Lipid adjusted[1]	591.70	252.90	2179.83
		Whole blood[2]	0.5	0.2	1.6
Electrolytic	8	Lipid adjusted	778.99	316.08	1629.45
		Whole blood	0.5	0.2	1.1
Maintenance	6	Lipid adjusted	2465.13	824.68	6788.60
		Whole blood	1.7	0.4	3.4
Reactor	5	Lipid adjusted	801.91	472.01	1980.29
		Whole blood	0.7	0.3	1.5
Total	30	Lipid adjusted	891.1	253.0	6790.0
		Whole blood	0.7	0.2	3.4

[1]ng/g lipid (ppb). [2]µg/ liter (ppb)

Table11
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
August 2004
Blood Alpha glutathione S-transferase level (µg/L by Work Area

Department	Mean	Minimum	Maximum
Cell Brick	4.5	2.1	9.4
Electrolytic	4.6	2.7	8.8
Maintenance	4.5	3.1	8.8
Reactor	4.5	2.7	8.2
Total	4.5	2.1	9.4

Table 12
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
Carbon Tetrachloride Industrial Hygiene Measurements
November 2004

Sample No.	Date of sample /shift	Job Title /Location	Carbon Tetrachloride (mg/sample)	Sample Volume (Liters)	Results (mg/m^3)
USM 14	11/1/04, 0700-1500	Jour. Millwright, Electrolytics	ND[1]	42.7	ND
USM 15	11/1/04, 0700-1500	Asst. Cell Service Operator Electrolytics	Trace[2]	15.3	Trace
USM 36	11/1/04	n/a, unmarked FB	ND	0	ND
USM 37 +USM 40 *	11/2/04, 12a-12p	Melt Cell Operator, Reactor off gas scrubber	ND	34.8	ND
USM 38-FB	11/2/04	n/a, sample was FB	ND	0	ND
USM 39	11/2/04 12a-12p	Reactor building, area sample, located above 2C reactor	ND	31.9	ND
USM 47 +USM 51*	11/3/04 12a-12p	Area sample in reactor building, east side of 3D reactor	ND	70.0	ND
USM 48 +	11/3/04 12a-12p	Cell Building Operator, Electrolytics, smutting #1 holding cell	Trace	39.7	Trace
USM 49	11/3/04 12a-12p	Vacuum Wagon Operator, Electrolytics	0.0071	37.9	0.18
USM 61	11/4/04 0700-1500	Maintenance, reactor, welding on 3C launder	Trace	32.8	Trace
USM 62	11/4/04 0700-1500	Maintenance, reactor, welding on 3C launder	Trace	23.5	Trace
USM 63	11/4/04 0700-1500	Cell Service Operator, Electrolytics, pigging header	ND	32.5	ND
USM 64	11/4/04 0700-1500	Asst. Cell Service Operator, Electrolytics, checking leveling wells	Trace	23.9	Trace
USM 65-FB	11/4/04 0700-1500	n/a, sample was FB	ND	0	ND

Notes
[1]ND = not detected
[2]trace = quantity reported between the limit of detection (LOD) and limit of quantitation (LOQ)
LOD = 0.001 millligrams per sample
LOQ = 0.004 millligrams per sample
Minimum detectable concentration was 0.03 mg/m^3
FB = Field blank
* samples were changed during the sampling period and analytical results and sample volumes combined

Table 13
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
November 1-4, 2004
Wipe samples for Hexachlorobenzene

Sample No.	Date of sample / shift	Job Title /Location	Hexachlorobenzene (µg/sample)
343144	11/1/04, 0700 -1500	Cell Service Operator, Electrolytics, Bldg 1	Trace
343145	11/1/04, 0700 -1500	Surface area sample, electrolytics/foundry lunchroom, NW corner table	0.095
343146	11/4/04, 0700 -1500	Asst. Cell Service Operator, Electrolytics (driving forklift)	3.5
343147	11/4/04, 0700 -1500	Asst. Cell Service Operator, Electrolytics (pigging header, post hand wash)	1.3
343148	11/1/04, 0700 -1500	Surface area sample, electrolytics/foundry lunchroom, SW corner table	0.18
343149	11/4/04, 0700 -1500	Surface area sample, Cell service lunchroom, rectangular (blue laminate) table	ND
343150	11/1/04, 0700 -1500	Surface area sample, reactor building, 4th floor lunch room, rectangle table	ND
343151	11/1/04, 0700 -1500	General Maintenance, millwright, reactor building	0.14
343152	11/4/04, 0700 -1500	Surface area sample, brick house lunchroom, round (laminate) table	ND
343153	11/1/04, 0700 -1500	Asst. Cell Service Operator, Electrolytics, Bldg 1	ND
343154	11/1/04, 0700 -1500	Surface area sample, reactor building, 4th floor lunch room, round table	ND
343155	11/1/04, 0700 -1500	Asst. Cell Service Operator, Electrolytics	Trace
343156	11/1/04, 0700 -1500	Reactor Maintenance, High energy scrubber pump	0.20
343157	11/4/04, 0700 -1500	Cell Service Operator, Electrolytics, pigging header, other tasks	0.48
343158	11/2/04, 12a -12p	Surface area sample, reactor building, 4th floor lunch room, round table	ND
343159	11/3/04, 12a -12p	Vacuum Wagon Operator Electrolytics	ND
343160	11/1/04, 0700 -1500	General Maintenance, doing PMs on reactor and cells	2.4
343161	11/3/04, 12a -12p	Asst Melt Cell Operator, Reactor building	ND
343162	11/4/04, 0700 -1500	Asst. Cell Service Operator, (pigging header, other tasks) post hand wash	Trace
343163	11/2/04, 12a -12p	Surface area sample, lunch room, Electrolytics, plastic table closest to vending machines	0.092
343164	11/3/04, 12a -12p	Vacuum Wagon Operator	Trace
343165	11/3/04, 12a -12p	Asst Melt Cell Operator, Reactor	ND

		building	
343166	11/4/04, 0700 -1500	Surface area sample, Cell service lunchroom, wooden table	Trace
343167	11/4/04, 0700 -1500	Cell Service Operator, (pigging header, other tasks) post hand wash	1.6
343248-FB		Field blank	ND
343253-FB		Field blank	ND

Notes
µg = micrograms
Limit of Detection (LOD) = 0.03 µg per wipe
Limit of Quantitation (LOQ) 0.09 µg per wipe
Trace = laboratory were results reported between the LOD and LOQ
Area samples were collected using a 10 x 10 centimeter disposable masking template, total sample area = 100 cm^2

Table 14
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
November 1-4, 2004
PBZ sampling for Hexachlorobenzene (HCB)

Sample No.	Date of sample / shift	Job Title /Location	HCB (µg/sample)	Sample Vol. (Liters)	Results (mg/m^3)
USM 1	11/1/04, 0700-1500	ASO, Electrolytics	0.87	2210	0.00039
USM 2	11/1/04, 0700-1500	GMA, high energy scrubber pump, (outside for portion of shift)	3.4	2211	0.0015
USM 3	11/1/04, 0700-1500	Helper, Reactor Maintenance	2.7	2202	0.0012
USM 4	11/1/04, 0700-1500	ASO, Cast house, Potline, Anode Service	0.21	2214	0.000095
USM 5	11/1/04, 0700-1500	ASO Electrolytics	2.9	2116	0.0014
USM 6	11/1/04, 0700-1500	JMW, Electrolytics	0.44	1652	0.00026
USM 7	11/1/04, 0700-1500	LSO, Building 1	0.61	2108	0.00029
USM 8	11/1/04, 0700-1500	ASO Building 1	1.4	2142	0.00065
USM 9	11/1/04, 0700-1500	GMA, Reactor Building	1.1	2134	0.00051
USM 10	11/1/04 0700-1500	Helper, Reactor Maint. doing preventive maintenance	3.4	1459	0.0023
USM 11	11/1/04 0700-1500	JMW, Warehouse/Foundry	1.6	2287	0.00069
USM 12	11/1/04 0700-1500	GMA, Foundry	1.0	2037	0.00049
USM 13	11/1/04 0700-1500	GMA, gas scrubber, outside for part of shift	0.72	2196	0.00032
USM 16	11/2/04, 1200-2400	MRO, Foundry, #1 Cast Machine	0.43	3546	0.00012
USM 17	11/2/04, 1200-2400	Area sample, Electrolytics	0.26	2834	0.000092
USM 18	11/2/04, 1200-2400	VWO, Electrolytics	1.0	3006	0.00033
USM 19	11/2/04, 1200-2400	SVWO, Electolytics	0.71	3538	0.00020
USM 20	11/2/04, 1200-2400	Area, 6^{th} Floor Reactor Bldg. (above 2A and 2C reactors)	1.7	3075	0.00056
USM 21	11/2/04, 1200-2400	VWO, Electrolytics	0.69	3693	0.00018
USM 22	11/2/04, 1200-2400	ARO, Reactor, 6^{th} Floor	0.89	3596	0.00025
USM 23	11/2/04, 1200-2400	MRO, Foundry	0.35	3645	0.000096
USM 24	11/2/04, 1200-2400	DCO, Foundry	0.15	1925	0.000078
USM 25	11/2/04, 1200-2400	RAFO Reactor, Feed systems operator	2.4	2785	0.00086
USM 26	11/2/04, 1200-2400	VWO, Electrolytics	5.8	3608	0.0016
USM 27	11/2/04, 1200-2400	MCO, reactor area	1.2	3675	0.00033
USM 28	11/2/04, 1200-2400	SVWO, Electrolytics (4 hours in training, not in plant)	1.1	3086	0.00035
USM 29	11/2/04, 1200-2400	DCO, Foundry	0.25	3608	0.00069
USM 30	11/2/04, 1200-2400	SVWO, Electrolytics	1.64	3706	0.00044
USM 31	11/2/04, 1200-2400	VWO Electrolytics	0.85	3741	0.00023
USM 32-FB	11/1/04, 0700-1500	Field Blank	ND	0	< MDC
USM 33-FB	11/3/04 0700-1500	Field Blank	ND	0	<MDC
USM 34	11/2/04, 1200-2400	MCO, Stripper, holding cell	1.5	3624	0.00041
USM 35	11/2/04, 1200-2400	ARO, 6^{th} Floor, Reactor	0.82	3587	0.00023
USM 41	11/3/04, 1200-2400	VWO, Electrolytics	0.88	1515	0.00058
USM 42	11/2/04, 1200-2400	CBO, Electrolytics (smutting #1 holding cell)	0.58	3688	0.00015
USM 43	11/2/04, 1200-2400	CBO, Electrolytics (smutting #1 holding cell)	0.80	3653	0.00022
USM 44	11/2/04, 1200-2400	ARO, 6^{th} Floor, reactor	0.95	3512	0.00027
USM 45	11/2/04, 1200-2400	ARO, 6^{th} Floor reactor,	1.3	3368	0.00038
USM 46	11/2/04, 1200-2400	SVWO, Electrolytics	2.5	3721	0.00067

USM 50	11/2/04, 1200-2400	Field Blank	ND	0	<MDC
USM 52	11/4/04 0700-1500	GMA, 6th Floor Reactor	1.5	2211	0.00068
USM 53	11/4/04 0700-1500	ASO Electrolytics	0.59	2149	0.00027
USM 54	11/4/04 0700-1500	CSO Electrolytics	1.4	2199	0.00064
USM 55	11/4/04 0700-1500	SMA, welding, reactor 3C launder	2.6	2469	0.0010
USM 56	11/4/04 0700-1500	SMB, welding, reactor 3C launder	1.8	2436	0.00074
USM 57	11/4/04 0700-1500	GMA, Helper, 6th floor reactor	12	2244	0.0053
USM 58	11/4/04 0700-1500	ASO, Electrolytics	0.81	975	0.00083
USM 59	11/4/04 0700-1500	GMA, Reactor	1.9	2191	0.00086
USM 60-FB	11/4/04 0700-1500	Sample was inadvertently labeled as field blank but apparently was an actual field sample. No air volumes were available from sampling records, nor any employee identifers.	0.085	unknown	unknown

Notes
HCB Limit of Detection = 0.01 micrograms per sample
HCB Limit of Quantitation = 0.03 micrograms per sample
Minimum detectable concentration (MDC) was calculated to be 0.0000036 mg/m^3
FB = Field blank
Job Titles: MCO & ARO: Melt Cell Operator. and Asst. Operator.; CSO & ASO: Cell Service Operator. and Asst. Operator; CBO: Cell Building Operator, VWO and SVWO Vacuum Wagon Operator and Sr. Operator.;SMA-C Service Maintenance; GMA: General Maintenance Millwright; JMW Journeyman Millwright; DCO: Direct Chill Casting Operator; MRO Foundry Operator.; CMO: Casting Machine Operator.

Table 15
HETA 2004-0169-2982
U.S. Magnesium Corporation Rowley, Utah
November 1-4, 2004
Polychlorinated Biphenyls (PCBs)

Sample No.	Date of sample / shift	Job Title /Location	PCB µg/sample (congener #)	Sample Vol. (Liters)	Results (mg/m^3)
USM 1	11/1/04, 0700-1500	ASO, Electrolytics	0.87 (209)	2210	0.00004
USM 2	11/1/04, 0700-1500	GMA, High energy scrubber pump, outside for part of shift	ND	2211	<MDC
USM 3	11/1/04, 0700-1500	Helper, Reactor Maintenance	ND	2202	<MDC
USM 4	11/1/04, 0700-1500	ASO, Cast house, Electrolytics	Tr (209)	2214	Tr
USM 5	11/1/04, 0700-1500	ASO Electrolytics	0.2 (209)	2116	0.000095
USM 6	11/1/04, 0700-1500	JMW, Electrolytics	Tr (209)	1652	Tr
USM 7	11/1/04, 0700-1500	LSO, Building 1	Tr (126, 169) 0.046 (209	2108	Tr 0.000022
USM 8	11/1/04, 0700-1500	ASO Building 1	Tr (126) 0.16 (209)	2142	Tr 0.000075
USM 9	11/1/04, 0700-1500	GMA, Reactor Building	Tr (126)	2134	Tr
USM 10	11/1/04 0700-1500	Helper, Reactor Maint. , PMs	Tr (126)	1459	Tr
USM 11	11/1/04 0700-1500	JMW, Warehouse/Foundry	ND	2287	<MDC
USM 12	11/1/04 0700-1500	GMA, Foundry	0.038 (209)	2037	0.000019
USM 13	11/1/04 0700-1500	GMA, Gas scrubber, outside for part of shift	Tr (126)	2196	Tr
USM 16	11/2/04, 1200-2400	MRO, Foundry, #1 Cast Machine	Tr (126)	3546	Tr
USM 17	11/2/04, 1200-2400	Area sample, Electrolytics	ND	2834	<MDC
USM 18	11/2/04, 1200-2400	VWO, Electrolytics	0.045 (209)	3006	0.000015
USM 19	11/2/04, 1200-2400	SVWO, Electolytics	Tr (209)	3538	Tr
USM 20	11/2/04, 1200-2400	Area, 6th Floor Reactor Bldg. (above 2A and 2C reactors)	Tr (105)	3075	Tr
USM 21	11/2/04, 1200-2400	VWO, Electrolytics	ND	3693	<MDC
USM 22	11/2/04, 1200-2400	ARO, Reactor, 6th Floor	ND	3596	<MDC
USM 23	11/2/04, 1200-2400	MRO, Foundry	Tr (126)	3645	Tr
USM 24	11/2/04, 1200-2400	DCO, Foundry	0.036 (105) Tr (126)	1925	0.000018 Tr
USM 25	11/2/04, 1200-2400	RAFO Reactor, Feed systems oper.	Tr (126)	2785	Tr
USM 26	11/2/04, 1200-2400	VWO, Electrolytics	0.038 (126) 0.049 (156) 0.045 (167) 2.0 (209)	3608	0.00001 0.000014 0.000012 0.00055
USM 27	11/2/04, 1200-2400	MCO, Reactor area	Tr (126) 0.066 (169)	3675	Tr 0.000018
USM 28	11/2/04, 1200-2400	SVWO, Electrolytics (4 hours in training, not in plant)	0.043 (169)	3086	0.000014
USM 29	11/2/04, 1200-2400	DCO, Foundry	Tr (126)	3608	Tr
USM 30	11/2/04, 1200-2400	SVWO, Electrolytics	0.060 (209)	3706	0.000016
USM 31	11/2/04, 1200-2400	VWO, Electrolytics	ND	3741	<MDC
USM 32-FB	11/1/04, 0700-1500	Field Blank	ND	0	< MDC
USM 33-FB	11/3/04 0700-1500	Field Blank	ND	0	<MDC

USM 34	11/2/04, 1200-2400	MCO, Stripper, holding cell	Tr (209)	3624	Tr
USM 35	11/2/04, 1200-2400	ARO, 6th Floor, Reactor	ND	3587	<MDC
USM 41	11/3/04, 1200-2400	VWO, Electrolytics	Tr (126) Tr (156)	1515	Tr Tr
USM 42	11/2/04, 1200-2400	CBO, Smutting #1 holding cell	Tr (126) 0.25 (209)	3688	Tr 0.000067
USM 43	11/2/04, 1200-2400	CBO, Smutting #1 holding cell	Tr (126) Tr (209)	3653	Tr Tr
USM 44	11/2/04, 1200-2400	ARO, 6th Floor, reactor	ND	3512	<MDC
USM 45	11/2/04, 1200-2400	ARO, 6th Floor reactor,	Tr (209)	3368	Tr
USM 46	11/2/04, 1200-2400	SVWO, Electrolytics	0.043 (105) Tr (126) 0.22 (209)	3721	0.000012 Tr 0.000059
USM 50	11/2/04, 1200-2400	Field Blank	ND	0	<MDC
USM 52	11/4/04 0700-1500	GMA, 6th Floor Reactor	Tr (126) 0.70 (209)	2211	Tr 0.00032
USM 53	11/4/04 0700-1500	ASO Electrolytics	Tr (105) Tr (126) 0.25 (209)	2149	Tr Tr 0.00012
USM 54	11/4/04 0700-1500	CSO Electrolytics	Tr (126) 0.42 (209)	2199	Tr 0.00019
USM 55	11/4/04 0700-1500	SMA, Welding, reactor 3C launder	Tr (126) 0.035 (209)	2469	Tr 0.000014
USM 56	11/4/04 0700-1500	SMB, Welding, reactor 3C launder	1.8	2436	0.00074
USM 57	11/4/04 0700-1500	GMA, Helper, 6th floor reactor	Tr (114) Tr (126) 0.039 (209)	2244	Tr Tr 0.000017
USM 58	11/4/04 0700-1500	ASO, Electrolytics	Tr (123) Tr (126) 0.039 (209)	975	Tr
USM 59	11/4/04 0700-1500	GMA, Reactor	0.059 (77) Tr (126) Tr (209)	2191	0.000027 Tr Tr
USM 60-FB	11/4/04 0700-1500	Sample was inadvertently labeled as field blank but apparently was an actual field sample. No air volumes were available from sampling records, nor an employee identifer	0.065 (77) Tr (114) Tr (126) Tr (167) Tr (180)	unknown	unknown

Notes
Tr = trace amount
HCB Limit of Detection = 0.01 micrograms per sample
HCB Limit of Quantitation = 0.03 micrograms per sample
Minimum detectable concentration (MDC) was calculated to be 0.0000036 mg/m^3
FB = Field blank
Job Titles: MCO & ARO: Melt Cell Oper. and Asst. Oper.; CSO & ASO: Cell Service Oper. and Asst Oper; CBO: Cell Building VWO and SVWO Vacuum Wagon Oper and Sr. Oper.;SMA-C Service Maintenance; GMA: Gen'l Maintance Millwright; JMW Journeyman Millwright; DCO: Direct Chill Casting Oper; MRO Foundry Oper.; CMO: Casting Machine Oper.

Figure 1
HETA 2004-0169 US Magnesium
11/3/2004
Melt Cell Operator PBZ, Reactor Bldg. Peak and STEL

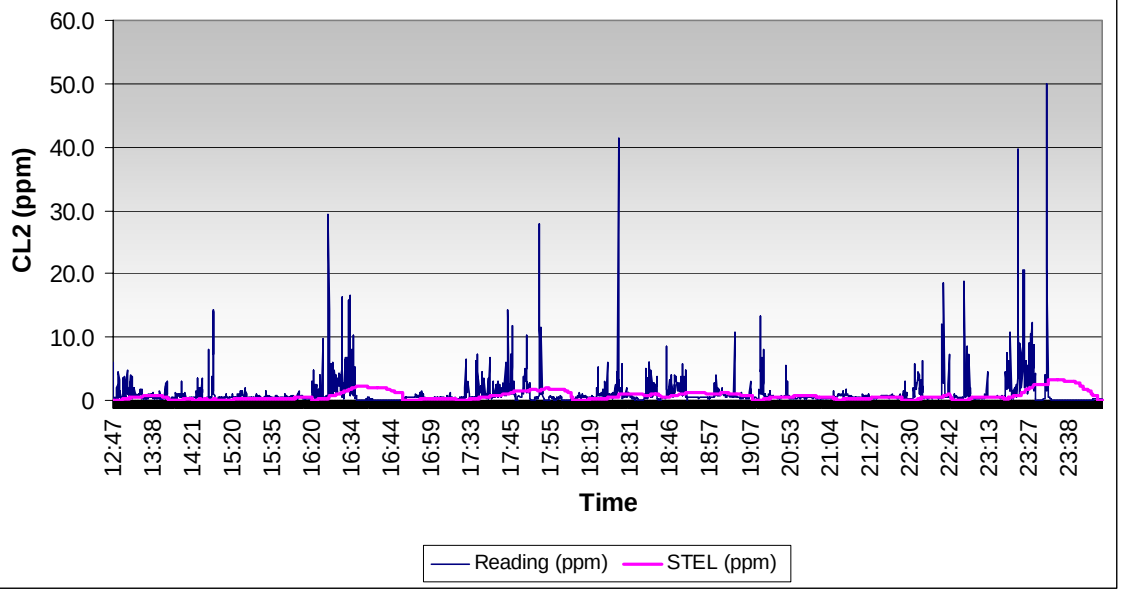


Figure 2
HETA 2004 0169 US Magnesium
11/3/04
Melt Cell Operator, PBZ Reactor Bldg. STEL concentrations

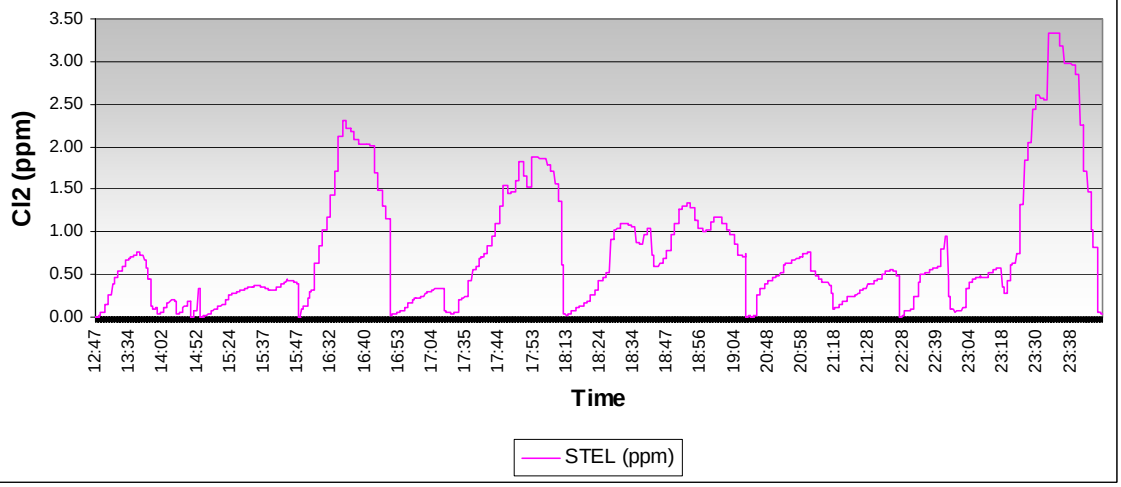


Figure 3
HETA 2004 US Magnesium 11/3/04 Melt Cell Operator PBZ Reactor Bldg.
STEL and TWA

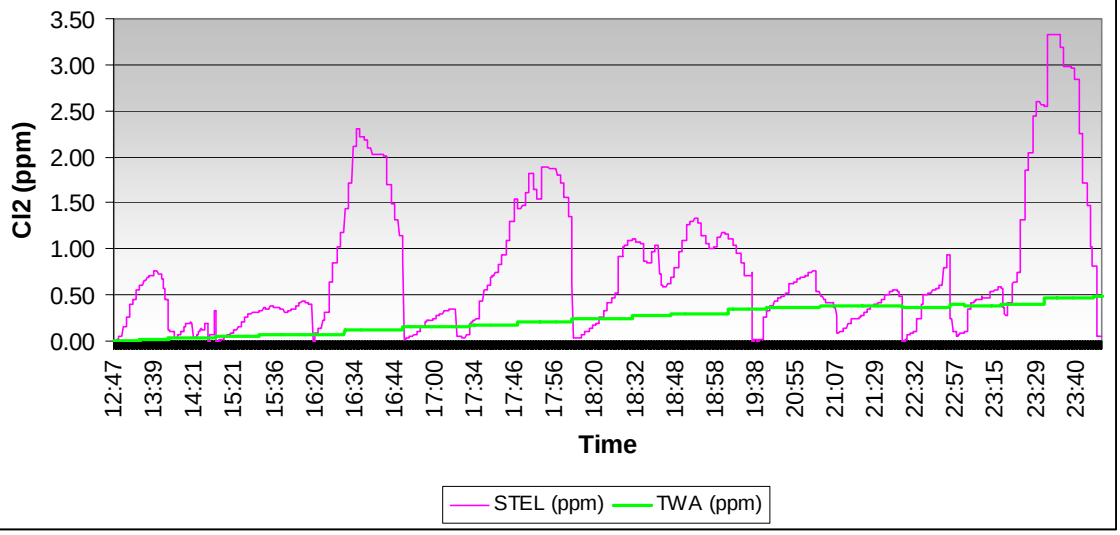


Figure 4
HETA 2004 -0169 US Magnesium 11/4/04
ASO Electrolytics, Peak and STEL concentrations

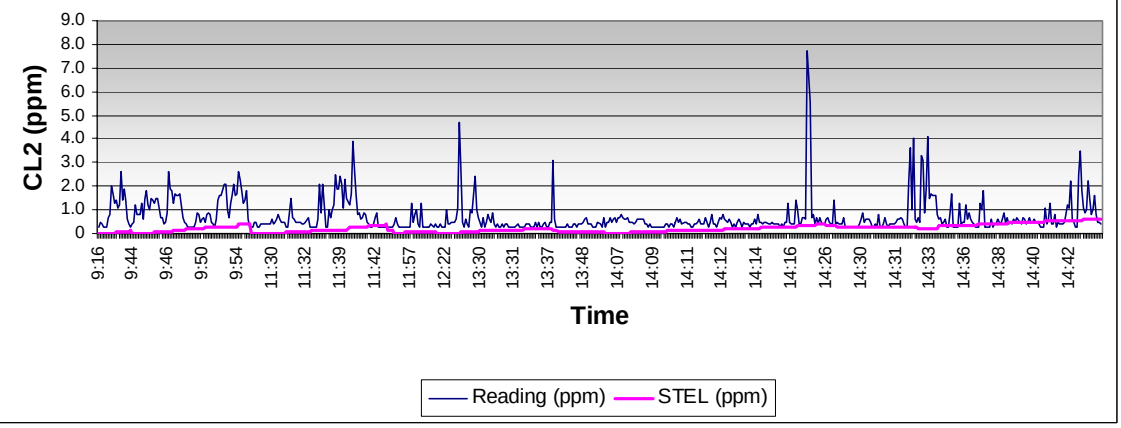
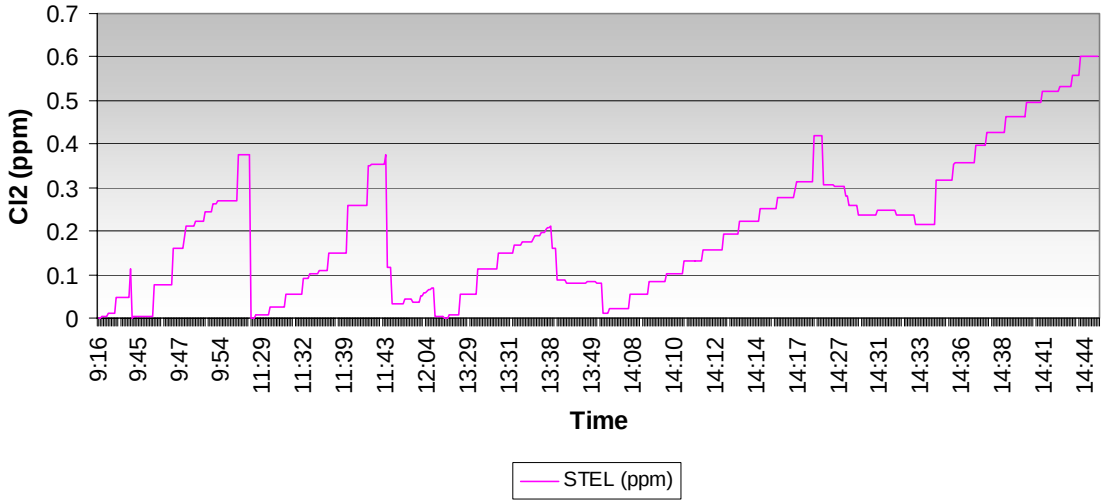
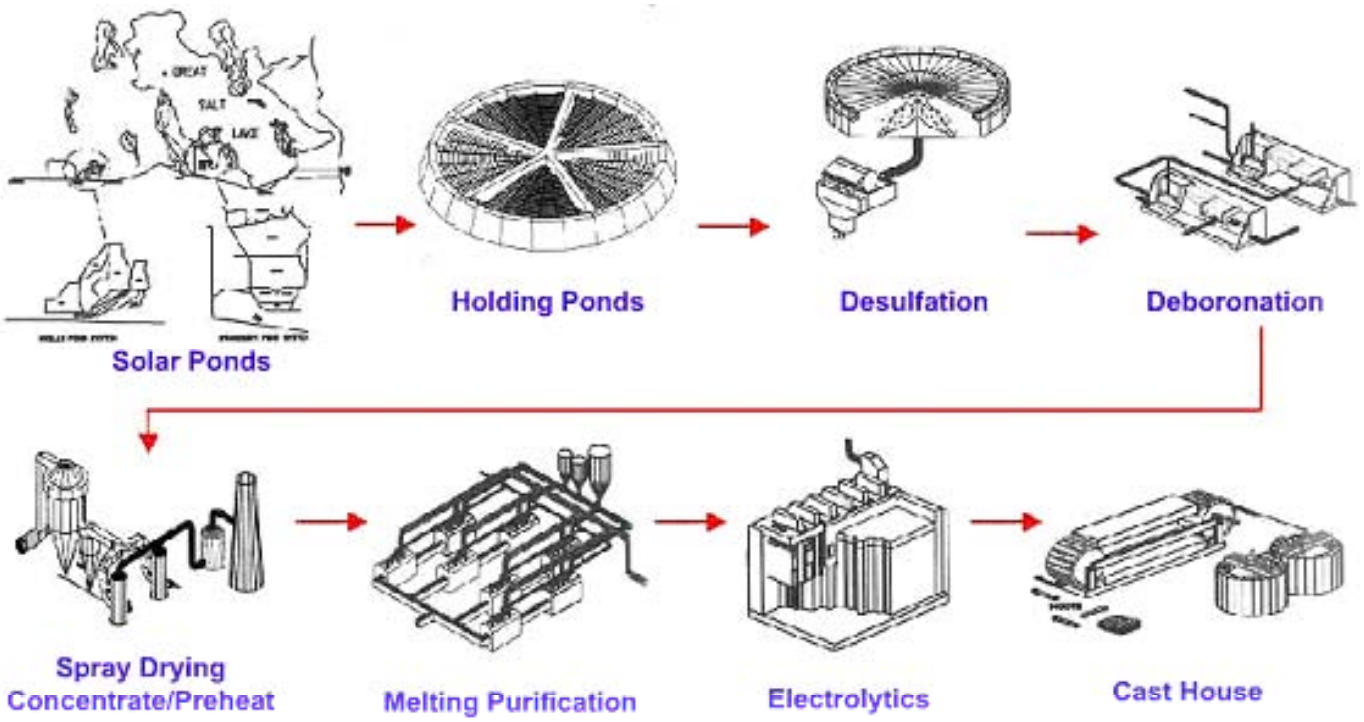


Figure 5
HETA 2004-0169 US Magnesium 11/4/04
ASO Electrolytics, STEL concentrations



Appendix A

Production Process of U.S. Magnesium



Appendix B
Interview Sheet (1 of 2)

DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service

HETA 2004-0169
US MAG Interview Sheet

National Institute for Occupational Safety and Health
Robert A. Taft Laboratories
4676 Columbia Parkway
Cincinnati OH 45226-1998

ID | Interview Date | Sampling Date | Schedule

Name | am | Birth date | AGE | years old
Race | **Gender** | **Address**
HIRE | **Job Date** | **Job Duration** | **Work duration** | Years
DEPT | **Work site** | **Job Code**
New department | **New Job**
Height | **SMOKE?** | **Drink or not?** | **Shift**
Weight | Start age | Drink freqency/wks | **Past Shift**
End age | Drink kinds
Duration yr | Drink amount(drinks)
Cigarette/day | Stop Drinking Date

OCCUP. HX **Current Job Date**

Any description for your Current Job In US MAG

Your past Job in US MAG

First Dept.
First Job
FIrstJob_date
2nd Dept.
2nd Job
2nd Job date
3rd Dept.
3rd Job
3rd Job date
4th Dept.
4th Job
4th Job date
5th Dept.
5th Job
5th Job date
6th Dept.
6th Job
6th Job date

Your Job Before US MAG

Your first Hiring Industry
Your Job
Begin date
End date
Your2nd Hiring Industr
Your Job
Begin date
End date
Your3rd Hiring Industry
Your Job
Begin date
End date
4th Hiring Industry
Your Job
Begin date
End date
5th Hiring Industry
Your Job
Begin date
End date
Other Exposure

pendi

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Interview Sheet (2 of 2)

DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service

HETA 2004-0169
US MAG Interview Sheet

National Institute for Occupational Safety and Health
Robert A. Taft Laboratories
4676 Columbia Parkway
Cincinnati OH 45226-1998

ID ☐ Interview Date ☐ Sampling Date ☐ ☐ ☐ ☐

Past Disorder Diagnosis Multiple choice

		Duration (month)			Duration (month)
Endo	☐			Arthritis ☐	
	☐			Thyroid ☐	
CVD	☐		Ds. Name		
PSY	☐		Ds. Name	Convulsion ☐	
PUL	☐		Ds. Name		
	☐				
GI GI	☐		Ds. Name	G-I regurg ☐	
Skin	☐		Ds. Name	Discoloration ☐	
	☐			Blackheads ☐	
ETC	☐		Ds. Name		

Medication Yes/No ☐ Medication for what disease

Medication name How long ? (months)

Time off due to any illness or injury related to work ? ☐

When? How many days?

Vetnam Experience

RANCHHAND ☐ Air Port ☐ Other ☐
CHE_CORPS ☐ Army ☐
Caught Fish ☐ Marine Corps ☐

Caught Fish consumption. Freq/week

Wood/Coal Heatg House now? ☐

Heating kind Wood ☐ Coal ☐

Dutation

Ever lived in wood/Coal Heating ? ☐

HeatK Kind Wood ☐ Coal ☐

Duration

Have you ever experience at work? Multiple choice

- ☐ Throat irritation
- ☐ Eye irritation
- ☐ Cough
- ☐ Headache
- ☐ Dyspnea
- ☐ Shortness of breath

Other Symptom

Respirator using due to Chlorine gas exposure ☐

Have you ever been "chlorine gassed"? ☐

Number of times per month you've been "chlorine gassed"

Number of times sought medical attention

BREAKFAST ☐

BREAKFAST Time

What did you eat for BREAKFAST?

LUNCH ☐

LunchTime

What did you eat for Lunch?

ANY EAT ☐

Any eat Time

What did you eat ?

MEDICATION Today ☐

Any other concern?

Appendix C
Worker Notification Letters

DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
National Institute for Occupational Safety and Health
Robert A. Taft Laboratories
4676 Columbia Parkway
Cincinnati OH 45226-1998

19 OCT 2004

Dear Mr. _____,

This letter pertains to the health hazard evaluation (HHE) that NIOSH conducted at U.S. Magnesium during the week of August 9, 2004. This packet will give you summary information about the blood tests that NIOSH investigators performed. We have sent individual notification letters to the participants with their own results.

This packet contains the following:
- A copy of the letter sent to the individual participants, but in the results tables for the dioxins, hexachlorobenzene, and alpha GST, we have given the average and range for the results of the 30 participants. In the letters to the participants, we gave them their actual individual results.
- A list of frequently asked questions about the effects of the chemicals we tested. We sent these to the workers as well.

The blood tests did not try to determine whether the workers had any specific health disorders, but determined the level of specific compounds in their blood. These pages will tell you the levels of specific dioxins and related compounds in the blood of the workers, and how they compare to levels that were found in other studies that measured these compounds.

We informed the workers that neither U.S. Magnesium management nor representatives from the Steel Worker's Union will receive their individual lab results. We told the workers that NIOSH will provide the combined results of all the workers tested (without names or information that would identify them personally) to U.S. Magnesium and the union as part of the HHE report. U.S. Magnesium will be required to post the final report, once they receive it, in a place accessible to all workers for 30 days.

We thanked the workers for participating in this survey. We believe that they have provided a service to their fellow workers by volunteering to help NIOSH find out the levels of dioxins and hexachlorobenzene that magnesium processing workers are exposed to in their work. With this information, NIOSH investigators will be able to make better recommendation to management and the Union to deal with these chemicals at U.S. Magnesium. We will send the interim letter further explaining the blood results soon.

If you have any questions about the enclosed information, please call me at 513-841-4589. Thank you.

Sincerely yours,

Bruce P. Bernard, M.D., M.P.H.
Medical Section Chief
Hazard Evaluations and Technical Assistance Branch
Division of Surveillance, Hazard Evaluations, and Field Studies

Blood Test Results from the NIOSH HHE

(August 2004)

1. Dioxins

To understand dioxin results it is important to keep in mind that dioxin is not just one chemical, but a family of chemicals.

We are giving you two blood test results on dioxin: 2,3,7,8-TCDD and WHO98-TEQ

2,3,7,8-TCDD This form of dioxin, 2,3,7,8-TCDD, is believed to be the most toxic.

WHO-TEQ 98 Dioxins are most often found in mixtures rather than as single compounds. The toxicity equivalence for dioxins and furans (WHO TEQ 98) is a shorthand method for comparing the toxicity of different types or mixtures of dioxins to the toxicity of the compound 2,3,7,8-TCDD.

Dioxins	2,3,7,8-TCDD in PPT*	WHO-TEQ 98 **
Average and Range of U.S. Magnesium (n=30)	1.28 (<LOD - 4.4)	32.9 (12-147)
Average dioxin level in U.S. Air Force personnel exposed to herbicides during the Vietnam War	12.4	
Average dioxin level in the U.S. general population		
age 30-44	Not detected	11.8
age 45-59	Not detected	16.9
Average dioxin level in workers from a magnesium plant in Norway (1995)	3.5	60

* parts per trillion ** This TEQ is from the World Health Organization 1998 (WHO 98) levels

The concentration of the dioxin 2,3,7,8 TCDD in your blood was similar to concentrations of this dioxin in the U.S. general population.

Your WHO-TEQ 98 level (the mixture of the dioxins and dioxin-like compounds) is higher than that found in the general population. The level of dioxin and dioxin-like substances, which are increased in your blood, show a similar pattern to workers studied in a magnesium plant in Norway in 1995. We believe that these elevated levels come from your exposure at work. Our testing does not tell us whether you have any specific health disorders due to these dioxin exposures.

The levels of dioxins found in the workers at U.S. Magnesium were well below the levels found by other scientists in workers who had an increased risk of heart disease, cancer, or diabetes. Your current blood level is not likely to cause the known dioxin health effects of chloracne (severe pimples on the face and body) or changes in liver function. A further explanation of what the results mean is included on the FAQ sheet.

Blood Test Results from the NIOSH HHE

(August 2004)

2. Hexachlorobenzene

Hexachlorobenzene (HCB)	HCB ppb*
Mean and range of U.S. magnesium workers (n=30) (Whole blood / lipid adjusted)	0.7 (0.2 - 3.4) / 891.1 (253 - 6790)
Average level of HCB in the U.S. general population	Not detected

* in parts per billion

Your blood sample results for HCB were higher than the level of HCB in the general population. We believe that the HCB comes from working at U.S. Magnesium. Our testing did not try to determine whether you had any health disorders due to HCB exposures; we were determining the level of this compound in your blood. However, currently there is not enough scientific information about the health effects from HCB exposure. An explanation of what the results might mean is included on the FAQ sheet.

3. Alpha glutathione S-transferase

Alpha glutathione S-transferase (GST)	GST
Mean and range of U.S. Magnesium Workers (n=30)	4.5 (2.1 - 9.4) microgram /liter
reference range	7.7 -11.8 microgram /liter

The Alpha glutathione S-transferase (alpha-GST) is an enzyme immunoassay test and is a sensitive indicator of liver cell function. We measured alpha-GST to give us an indication of whether workers have any liver injury. Results of the alpha-GST test from the blood samples of the 30 U.S. Magnesium workers did not show any indication of liver injury.

What do I do with these results?

You may want to share these results with your doctor because they indicate your exposure at work.

Although NIOSH investigators did not perform medical evaluations in this evaluation, we believe that periodic medical screening to detect and prevent heart disease, cancer, and diabetes is beneficial

The way to reduce your exposure and intake of dioxins and HCB at work is through workplace controls. We will make certain recommendations to the managers at U.S. Magnesium in our report to help ensure that your exposure level does not increase. Wearing a respirator at work may be helpful in keeping your dioxin level from rising. We will also address the right type of respirator and other personal protective equipment, as well as propose good work practices (including hand hygiene before eating, drinking, and smoking), which may be helpful in reducing exposures.

Appendix D
Letter from US Magnesium regarding U.S. Department of Justice, Civil Action Suit No. 2:01CV004B United States of America v. Magnesium Corporation of America et al, February 2005

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Re: United States of America v. Magnesium Corporation of America, et al, Civil Action No. 2:01CV004B

Ladies and Gentlemen:

This letter provides updating and clarifying information with respect to the worker health tasks in the settlement term sheet (January 6, 2005). In particular, the letter lists those near term worker health related measures identified in term sheet paragraphs 1-3 which US Magnesium LLC ("USM" or the "Company") agrees to implement immediately. Those items are as follows:

Respirators

1. The Company requires that respirators be worn full time on the Reactor Building sixth floor (except the office and stairwell), as recommended by NIOSH for chlorine protection. The mandatory use area may be altered in the future if chlorine emission control measures and area chlorine monitoring results demonstrate substantial improvement in chlorine exposure.

2. Combination cartridge respirators are available to employees who request them and can safely use them for assigned tasks (i.e., tasks which do not require face shields). The Company will require that respirators (combination cartridges or particulate filters) be worn in the Reactor and Electrolytics areas for tasks which involve exposure to process off gases. These tasks include, for example, adding carbon to electrolytic cells, cleaning off gas, removing anodes and work activities involving the Reactor off gas system**.**

3. Particulate filters in combination with chlorine cartridges will be required as an interim protection measure, as recommended by NIOSH, until the Company completes its review

of face shields which are compatible with combination cartridges. The Company has not yet found a face shield compatible with available combination cartridge respirators.

4. The Company will implement a quantitative fit test program for employee respirators after appropriate equipment can be obtained (estimated six months). The Company will also modify and formalize inspection programs for compliance with the facial hair policy.

Hygiene Training & Signage

5. The Company will complete in February 2005 a modified training program to improve basic hygiene awareness in lunch room and break areas. Training will be included in the annual safety training program.

6. The Company has installed fifty signs in lunch room and break areas to encourage improved hygiene. An additional thirty signs are on order and will be installed in four to six weeks.

Compressed Air Cleaning

7. The Company has eliminated use of compressed air cleaning of cells in Electrolytics.

Electrolytic Building Dust Containment.

The Company has isolated fugitive dust releases from Electrolytics to three basic activities: grizzly box dumping, anode pipe cleaning and drag chain maintenance. Dust containment plans are expected to be completed during consent decree negotiations. Some measures have already be implemented.

8. Grizzly Pits. The Company has installed fugitive dust release hoods on both the Building 1 and Building 4 grizzly pits, which are operated during anode dust removal activities. The Company will formalize its procedure for dumping of anode dust to insure the process is completed slowly to prevent unnecessary releases. The Company will also review the cleanup and decontamination policies to insure proper practices are implemented in these areas.

9. Anode Pipe Cleaning. The Company has modified the tools used in anode pipe cleaning to minimize fugitive releases and spills. The Company will formalize cleanup and decontamination procedures to insure proper practices are followed.

10. Drag Chain Maintenance. The Company will utilize spill containment and groundcover material to minimize releases of fugitive dust. Portable dust shelters will be utilized in applicable areas to minimize airborne losses of material. The Company will formalize cleanup and decontamination policies to insure proper practices are followed.

Reactor Fugitive Dust Release Containment

The Company has evaluated fugitive dust releases in the Reactor and has isolated releases to reactor cell launders, reactor cell exit hoods and melt cell quench tees. Corrective actions for these items include the following:

11. Reactor Cell Launders. The Company will design and install new covers on the launder systems to eliminate the use of Kaowool in favor of hard covers. (Kaowool may continue to be used as a gasket material). The Company will also design and install a fugitive collection system for the launder collection box, which currently has no ventilation.

12. Reactor Cell Exit Hoods. The Company will redesign the off gas piping for the exit hoods to facilitate operator cleaning on a regular basis, which will minimize releases caused by piping restrictions. The required operating maintenance practices will be formalized in the new procedure.

13. Melt Cell Quench Tees. Control of melt cell off gas suction can compromise dust releases from the quench tee due to pressure surges. The Company will initiate a study to review the adequacy of the current off gas control system and will modify controls that are problematic. The Company will also install a fugitive collection system for the off gas reamer shaft area to collect problem releases.

USM will incorporate into the draft consent decree the tasks identified in this letter. Thank you for your consideration.

Sincerely yours,

PARSONS BEHLE & LATIMER

David W. Tundermann

Appendix E

PCDDs, PCDFs, PCBs, and HCB Measurement

Seven PCDDs, 10 PCDFs, three non-ortho substituted or coplanar PCBs (cPCBs), 36 ortho-substituted PCBs, 13 persistent chlorinated pesticides and selected pesticide metabolites were measured in serum by high-resolution gas chromatography/isotope-dilution high-resolution mass spectrometry (HRGC/ID-HRMS).

Large volume serum samples (25 mL) analyzed for PCDDs/PCDFs/cPCBs and PCBs (10 mL) were spiked with 13C12-labeled internal standards and the analytes of interest were isolated in hexane using a C18 solid phase extraction (SPE) procedure which is followed by an automated (Fluid Management Systems Power-Prep) cleanup and enrichment procedure using multi-layered silica gel (acidic, basic, and neutral silica) and alumina columns coupled to AX-21 carbon columns. PCBs are isolated from the AX-21 carbon column in the forward direction with 1:1 Dichloromethane (DCM)/hexane and the PCDDs/PCDFs/cPCBs isolated in the reverse direction with toluene.

Small volume serum samples (<5 mL) analyzed for PCBs and pesticides are spiked with 13C12-labeled internal standards and the analytes of interest are isolated in hexane using a C18 SPE procedure followed by neutral silica and Florosil SPE columns. PCBs and pesticides are eluted from the Florosil column with hexane and 1:1 DCM/hexane.

PCDD/PCDD/cPCB, PCB congener and pesticide fractions were analyzed independently by HRGC/ID-HRMS. Samples are injected into a Hewlett-Packard 6890 gas chromatograph equipped with a DB-5ms capillary column (30 m x 0.25 mm x 0.25 µm film thickness) coupled to a Thermo Finnigan MAT95 XP mass spectrometer operated in EI mode using selected ion monitoring (SIM) at 10,000 resolving power. The concentration of each analyte is calculated from its linear calibration curve. For large volume samples, each analytical run consists of eight unknown serum samples, two method blanks, and two quality control samples. For small volume samples, each analytical run consists of eighteen unknown serum samples, two method blanks, and four quality control samples. After all data are reviewed using comprehensive quality assurance and quality control (QA/QC) procedures, the analytical results are reported on both a whole-weight and lipid-adjusted basis. Serum total lipids are calculated using an enzymatic "summation" method. Detection limits, on a whole-weight and lipid-adjusted basis, are reported for each sample, corrected for sample weight and analyte recovery.

Alpha glutathione S-transferase (alpha GST) Measurement

The normal range of alpha GST in humans is 0-12 µg/l. Levels can rise to about double the high range (20 µg/l) for chronic, low-level injury to the liver.[93,94] The concentration of alpha GST [54,55,95,96] in serum was measured using a commercially available, enzyme-linked immunoassay kit (High sensitivity Alpha GST EIA; Biotrin International, Dublin, Ireland). Standards of known concentrations (0, 62.5, 125, 250, 500, 1000, and 2000 µg /L) were included in the assay, and the concentrations of the samples and controls were calculated from the standard curve using linear regression (R^2=.999). The LOD for this particular assay is around 0.5 µg /L. Intra-assay CVs were 8.1 and 1.3%, at concentrations of 275 and 945 µg /L, respectively.

93. Rees GW, Trull AK, Doyle S. [1995] Evaluation of an enzyme-immunometric assay for serum alpha-glutathione S-transferase. Ann Clin Biochem;32:575-583.

94. Mulder TP, Court DA, Peters WH. [1999] Variability of glutathione S-transferase alpha in human liver and plasma. Clin Chem; 45(3):355-359.

95. Iwanaga Y, Komatsu H, Yokono S, Ogli K.[2000] Serum glutathione S-transferase alpha as a measure of hepatocellular function following prolonged anaesthesia with sevoflurane and halothane in paediatric patients. Paediatr Anaesth;10(4):395-398.

96. Suttner SW, Schmidt CC, Boldt J, Huttner I, Kumle B, Piper SN. [2000] Low-flow desflurane and sevoflurane anesthesia minimally affect hepatic integrity and function in elderly patients. Anesth Analg.;91(1):206-212.

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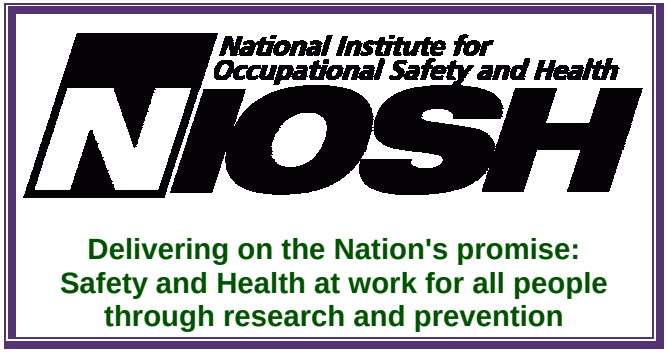

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