INTRODUCTION

On March 9, 1993, the National Institute for Occupational Safety and Health (NIOSH) received a request for a Health Hazard Evaluation from the New Jersey Department of Health (NJDOH). The request concerned possible employee exposure to lead and polychlorinated biphenyls (PCBs) from contaminated soil at Curcio Scrap Metal and Cirello Iron and Steel, two scrap metal dealers located next to each other in Saddle Brook, New Jersey. On June 21-22, 1993, a NIOSH medical investigator visited the facilities to evaluate the extent of the worker exposure to PCB and lead through biologic monitoring of employees. Participants were notified by letter of their blood test results on July 19, 1993.

BACKGROUND

The Curcio Scrap Metal (CSM) facility is involved in non-ferrous metal recycling, predominantly aluminum, but also copper and gold. Material is delivered by truck to the facility, where it is weighed and sorted. Aluminum is cut to fit into a compactor, then crushed into blocks and stored until it is sold to aluminum smelters. The work area is located indoors and contact with contaminated soil by employees during the course of their job appeared to be minimal.

Cirello Iron and Steel (CIS) is involved in ferrous metal recycling and is located on the unpaved (East) lot of the facility. Trucks carrying ferrous materials are weighed and a large electro-magnet is used to remove the material from the truck to the ground in the lot. The scrap is then cut, sorted, and removed to foundries for recycling. This worksite is outdoors, and is directly on the area contaminated with lead and PCBs.

The CIS/CSM site has been known to be contaminated with PCBs since 1982. According to the NJDOH, the site was first investigated on October 27, 1982, following a citizen's complaint that electrical transformers were being stored and cut up on the property. Environmental measurement by the New Jersey Department of Environmental Protection (NJDEP) revealed the presence of Aroclor 1260 (105 parts per million [ppm]) and Aroclor 1254 (47 ppm) in an oily black fluid found in a drainage ditch and several puddles on the site. The United States Environmental Protection Agency (EPA) conducted a Site Inspection in September 1984, and found PCBs, lead, copper, nickel, and trichloroethylene in soil samples. On May 15, 1985, an oil spill was discovered in a nearby pond and was traced back to CIS. Examination at the site of the spill at CIS revealed PCB concentrations ranging from 30 to 80 ppm. Another PCB spill occurred on August 8, 1989, at CIS. In this instance, Aroclor 1254 was measured in the soil in concentrations ranging from 400 ppm to 700 ppm. Soil contaminated from that spill was removed from site on August 14, 1989. In July 1987, the site was placed on EPA's National Priorities List (NPL) for clean-up. Soil remediation was started in September 1993.

Extensive soil sampling was conducted by the EPA in July 1989, and included 48 borings on the East, South, and West lots. Soil samples from the East lot revealed PCB concentrations ranging up to 6200 ppm for total PCB (Table 1). Volatile organic compounds (VOCS), including vinyl chloride, acetone, and trichloroethylene were also detected in the soil on the East lot. Further analysis of the soil samples for inorganic substances detected aluminum, arsenic, copper, lead, magnesium, mercury, and zinc. Lead was found in concentrations up to 39,300 ppm, which greatly exceeded the NJDEP Soil Action level range of 250 ppm to 1,000 ppm. The U.S. EPA conducted a risk assessment for the site in December 1990, and concluded that there was an appreciable risk to workers for the development of cancer. The U.S. EPA determined that soil remediation, including excavation with off-site incineration, should be undertaken because of the high concentration of contaminants in the soil and the theorized high risk of cancer associated with exposure to these levels.[1]

The NJDOH and the Agency for Toxic Substances and Disease Registry (ATSDR) conducted a "Public Health Assessment" of the site and in a draft, initial release concluded that the "Curcio Scrap Metal site is an urgent health threat to employees, scrap metal haulers and trespassers. They were exposed and are continuing to be exposed to sufficient levels of contaminated soil to possibly result in carcinogenic and non-carcinogenic effects." The NJDOH requested assistance from NIOSH to evaluate worker exposure.

As a result of the release of the ATSDR Public Health Assessment, the Occupational Safety and Health Administration (OSHA) inspected the CIS and CSM sites. Air samples were collected for lead, mercury, and PCBs. Only one lead level, 73.28 micrograms per cubic meter ($\mu g/m^3$), exceeded the OSHA Permissible Exposure Limit (PEL) of 50 $\mu g/m^3$. This level was recorded near a torch cutter at CIS and OSHA investigators believed that the lead originated from the material being cut. No other measured level was above 12 $\mu g/m^3$. Lead and PCB were detected in bulk and wipe samples taken throughout the site (Table 2). Lead levels ranged from non-detectable (ND) to 1.0% lead, and PCB levels ranged from ND to 265 ppm. No mercury or PCB was found in any air sample.

METHODS

This evaluation included a questionnaire survey and blood testing for lead and PCBs. All employees and management of both CIS and CSM were eligible to participate. Informed consent was obtained from all participants. The questionnaire consisted of a work history and questions relating to known risk factors for an increased blood lead level (BLL) and was administered on June 21, 1989. Blood sample collection was done in accordance with the Centers for Disease Control and Prevention (CDC) National Center for Environmental Health (NCEH) laboratory guidelines, using lead free needles and tubes for the BLL determination and acetone washed pipettes and Wheaton vials for the PCB analysis. Venous blood was obtained using a multiple-draw Vacutainer system. The blood was allowed to clot, and serum was separated, frozen, and shipped to the CDC NCEH laboratory in Atlanta, Georgia, for analysis. Tubes used for BLL analysis were drawn before the tubes used for PCB to minimize the possibility for contamination of the PCB sample. Five participants had a third tube of blood drawn as a quality control measure for BLL; it was submitted to the laboratory identified as if it were from another participant.

Blood samples were analyzed by the NCEH laboratory for both BLL and PCB. The BLLs were determined using the CDC graphite furnace atomic absorption method as used in the third National Health and Nutrition Examination Survey.[2] The PCB levels were determined by gas chromatography with an electron capture detector[3] and were reported as Aroclor 1260, the

predominant PCB in the samples. Results were reported to all study participants by mail on July 19, 1993.

EVALUATION CRITERIA

1. LEAD

Inhalation (breathing) of dust and fume, and ingestion (swallowing) resulting from hand-to-mouth contact with lead-contaminated food, cigarettes, clothing, or other objects are the major routes of worker exposure to lead. Once absorbed, lead accumulates in the soft tissues and bones, with the highest accumulation initially in the liver and kidneys.[4] Lead is stored in the bones for decades, and may cause toxic effects as it is slowly released over time. Overexposure to lead results in damage to the kidneys, gastrointestinal tract, peripheral and central nervous systems, and the blood-forming organs (bone marrow).

The frequency and severity of symptoms associated with lead exposure increase with increasing BLL. Signs or symptoms of acute lead intoxication include weakness, excessive tiredness, irritability, constipation, anorexia, abdominal discomfort, colic, anemia, high blood pressure, irritability or anxiety, fine tremors, pigmentation on the gums ("lead line"), and "wrist drop."[5,6]

An increase in an individual worker's BLL can mean that the worker is being overexposed to lead. While the BLL is a good indication of recent exposure to, and current absorption of, lead, it is not a reliable indication of the total body burden of lead.[7] Lead can accumulate in the body over time and produce health effects long after exposure has stopped. Long-term overexposure to lead may cause infertility in both sexes, fetal damage, chronic kidney disease (nephropathy), and anemia.

Under the OSHA standard regulating occupational exposure to inorganic lead in general industry, the PEL is 50 $\mu g/m^3$ as an 8-hour TWA.[8] The standard requires semi-annual monitoring of BLL for employees exposed to airborne lead at or above the Action Level of 30 $\mu g/m^3$ (8-hour TWA). Employees whose BLL is 40 µg/dl or greater must be retested every two months of employment. Medical removal of employees is required when an employee's average BLL is 50 µg/dl or greater over a six month period. A BLL of 60 µg/dl or greater, confirmed by retesting within two weeks, is an indication for immediate removal.

Recent studies suggest that there are adverse health effects at BLLs below the current evaluation criteria for occupational exposure. A number of studies have found neurological symptoms in workers with BLLs of 40 to 60 µg/dl. Hypertension in males has been associated with BLLs as low as 10 µg/dl.[9] Prenatal exposure to lead is associated with shorter gestational age, low birthweight, and delayed mental development at prenatal maternal BLLs as low as 10 to 15 µg/dl.[10]

In homes with a family member occupationally exposed to lead, lead dust may be carried home on clothing, skin, and hair, and in vehicles. High BLLs in resident children, and elevated concentrations of lead in the house dust, have been found in the homes of workers employed in industries associated with high lead exposure.[11] Both the CDC and the NJDOH recommend that the children of workers with lead poisoning, or who work in areas of high lead exposure, should be tested for lead exposure (BLL) by a qualified health-care provider.

In recognition of the health risks associated with exposure to lead, a goal for reducing occupational exposure was specified in *Healthy People 2000*, a recent statement of national consensus and U.S. Public Health Service policy for health promotion and disease prevention. The goal for workers exposed to lead is to eliminate, by the year 2000, all exposures that result in BLLs greater than 25 μg/dl.[12]

There are no Federal standards for occupational or childhood exposure to lead in soil. The CDC has previously stated (*Preventing Lead Poisoning in Young Children*--1985 edition) that soil concentrations exceeding 500-1,000 ppm appeared to cause increased BLLs in children. Based on this recommendation, the U.S. EPA Offices of Emergency and Remedial Response and Waste Programs Enforcement currently use an interim guideline for Superfund hazardous waste sites which specifies cleanup of soil to a total lead concentration in the range of 500 to 1000 ppm.[13]

2. POLYCHLORINATED BIPHENYLS (PCBs)

PCBs are chlorinated aromatic hydrocarbons that were manufactured in the United States from 1929 to 1977 and primarily marketed under the trade name Aroclor.[14] They found wide use because they are heat stable; resistant to chemical oxidation, acids, bases and other chemical agents; stable to oxidation and hydrolysis in industrial use; and have low solubility in water, low flammability and favorable dielectric properties. PCBs have been used commercially as insulating fluids for electrical equipment, hydraulic fluids, heat transfer fluids, lubricants, plasticizers, and components of surface coatings and inks.[15]

The different PCB mixtures marketed under different trade names are often characterized by a four-digit number. The first two digits denote the type of compound ("12" indicating biphenyl), and the latter two digits giving the weight percentage of chlorine.

Dietary PCB ingestion, the major source of population exposure, occurs especially through eating fish, but PCB residues are also found in milk, eggs, cheese and meat. PCB residues are detectable in various tissues without known occupational exposure to PCB. In past years, reported mean whole blood PCB levels ranged from 1.1 to 8.3 parts per billion (ppb),[16] with mean serum PCB levels usually ranging from 4 to 8 ppb and 95% of individuals having less than 20 ppb,[17] for persons without known occupational exposure. Mean serum PCB levels among workers in one capacitor manufacturing plant studied by NIOSH ranged from 111 to 546 ppb, or approximately 5 to 22 times the background level in the community. Mean serum PCB levels among workers in transformer maintenance and repair typically range from 12 to 51 ppb, considerably lower than among workers at capacitor manufacturing plants.[18]

PCB toxicity is complicated by the presence of highly toxic impurities, especially the polychlorinated dibenzofurans (PCDF),[19] which vary in amount depending on the manufacturer,[20] and percent chlorination,[21] and which are found in increased concentration after incomplete pyrolysis of the PCB.[22,23] Furthermore, different animal species, including humans, vary in their pattern of biologic response to PCB exposure.[24]

Two human epidemics of chloracne, "Yusho" and "Yu-cheng," from ingestion of cooking oil accidentally contaminated by a PCB heat-exchange fluid used in the oil's pasteurization, have been described in detail.[25,26] Although PCB was initially regarded as the etiologic agent in the Yusho study, analyses of the offending cooking oil demonstrated high levels of PCDF and polychlorinated quaterphenyls, as well as other unidentified chlorinated hydrocarbons, in addition to PCB.

The results of individual studies of PCB-exposed workers are remarkably consistent. Among the cross-sectional studies of the occupationally exposed, a lack of clinically apparent illness in situations with high PCB exposures seems to be the rule. Chloracne was observed in recent studies of workers in Italy,[27] but not among workers in Australia,[28] Finland,[29] or the United States.[18,30,31,32] Weak positive correlations between serum PCB level, and serum aspartate aminotransferase (SGOT) level,[28,29,30,31] serum gamma-glutamyltranspeptidase (GGTP) level,[18,28,31,32] and plasma triglycerides[18,33,34] have been reported. Correlations between plasma triglycerides[35] and GGTP[36] have also been found among community residents with low-level PCB exposures. Causality has not been imputed to PCBs in these cross-sectional studies.

The International Agency for Research on Cancer (IARC) has concluded that the evidence for PCBs carcinogenicity to animals and to humans is limited. IARC has reported that "Certain polychlorinated biphenyls are carcinogenic to mice and rats after their oral administration, producing benign and malignant liver neoplasms. Oral administration of polychlorinated biphenyls increased the incidence of liver neoplasms in rats previously exposed to N-nitrosodiethylamine."[37]

In a mortality study among workers at two capacitor manufacturing plants in the United States,[38] a greater than expected number of observed deaths from cancer of the liver and cancer of the rectum were noted. Neither increase was statistically significant for both study sites combined. However, in a recent update of this study,[39] with follow-up through 1982, the excess in liver/biliary tract cancer was statistically significant (5 observed versus 1.9 expected deaths); the rectum cancer was still elevated but not statistically significant. In this mortality study, the personal TWA airborne exposures in 1976 ranged from 24 to 393 $\mu g/m^3$ at one plant, and from 170 to 1260 $\mu g/m^3$ at the other. During the time period (1940-1976) when most of the workers were exposed, the levels were probably substantially higher. At one of the plants, the geometric mean serum levels in 1976 were 1470 ppb for 42% chlorinated biphenyls and 84 ppb for 54% chlorinated biphenyls.

In a mortality study among workers at a capacitor manufacturing plant in Italy,[40] males had a statistically significantly increased number of deaths from all neoplasms. When analyzed separately by organ system, death from neoplasms of the digestive organs and peritoneum (3 observed versus 0.88 expected) and from lymphatic and hematopoietic tissues (2 observed versus 0.46 expected) were elevated. This study was recently expanded to include vital status follow-up through 1982 for all workers with one week or more of employment.[41] In the updated results, there was a statistically significant excess in cancer among both females (12 observed versus 5.3 expected) and males (14 observed versus 7.6 expected). In both groups there were statistically non-significant excesses in lymphatic/hematopoietic cancer and a statistically significant excess in digestive tract cancer among males (6 observed versus 2.2 expected).

A retrospective cohort mortality study conducted by NIOSH[42] at an electrical equipment manufacturing plant using PCBs demonstrated a standardized mortality ratio for all malignant neoplasms below that expected when compared to the United States standard. A statistically significant increase in deaths from malignant melanoma and skin cancer, was observed (eight deaths observed compared with less than two deaths expected). In addition, there was an increase in the number of observed deaths from malignancies of the brain and central nervous system (five deaths observed compared with two deaths expected), though this increase is not statistically significant.

RESULTS

All 17 employees present at work on the days of the study participated. We could not get a blood sample from one individual at the worksite so he was not included in the analysis. Twelve participants worked at CSM and five worked at CIS. Thirteen employees were "white," one was "black," and three considered themselves "other" and wrote in "Hispanic." All of the CIS employees reported that they worked on the East lot 75% of the time or more, and all of the CSM employees reported that they worked on the East lot less than 25% of the time (and all but one of the CSM employees reported that they worked on the East lot less than 5% of the time). Seven employees (41%) reported that they smoked cigarettes, and all those who smoked also smoked at work. All employees reported washing their hands before lunch, and 13 (76%) reported washing four or more times a day. The employees ranged in age from 25 to 75, with a mean age of 49 years. Uniforms were provided to the employees and all employees except the secretary reported that they changed their clothes and shoes before they left work. According to management, respirators are presently provided to those employees cutting metals with a torch at both CIS and CSM, although four employees reported that they used a torch at work and did not use a respirator.

As shown in Table 3, BLLs ranged from 4.0 to 39.8 µg/dl (mean 19.9 µg/dl) and PCB levels ranged from <1 to 65.3 ppb (mean 7.5 ppb). BLLs in four (24%) of the participants, two from CIS and two from CSM, exceeded the Public Health Service goal of keeping BLL in lead-exposed workers below 25 µg/dl[12]. Differences in BLLs in paired samples were within the range of acceptable analytical variation. There was one individual whose PCB level was substantially higher than any other (65.3 ppm with the next highest PCB level being 13.2 ppb). His value was not included in the statistical analysis.

Statistical analysis involved use of t-tests to compare group BLL and serum PCB means with the dichotomous variables (where the employee worked, use of a torch at work [yes/no], use of a respirator [yes/no]), ANOVA for the categorical variables (time spent on the East lot and job category), and Pearson correlation coefficients for the continuous variables (age, the number of cigarettes smoked at work per day, and to determine if BLL was correlated with serum PCB). There was no statistically significant difference between BLL or PCB level and where the employee worked (CSM or CIS)(Table 4), use of a torch at work yes/no ($p=0.87$ for BLL, $p=.49$ for serum PCB), time spent on the East (unpaved) lot ($p=0.66$ for BLL; $p=0.79$ for serum PCB), age ($r=0.04$, $p=0.86$ for BLL; $r=0.26$, $p=0.32$ for PCB), or the use of a respirator ($p=0.57$ for BLL, $p=0.63$ for serum PCB). BLL was correlated with the number of cigarettes smoked at work ($r=0.62$, $p= 0.008$) but PCB level was not ($r=0.15$, $p=0.59$). The employees were divided into five categories for job duty based on their responses on the questionnaire. Employees who reported they were involved in sorting operations had the highest BLL levels and those who reported they were involved in either managerial or secretarial duties had the lowest levels (Tables 5,6) although the differences between all job duties were not statistically significant ($p=0.09$ for BLL and $p=0.76$ for PCB). Eating lunch in the work area was associated with higher levels of BLL and PCB (Tables 7,8). This difference was statistically significant for PCB level ($p=0.007$) but was not significant for BLL ($p=0.14$). The two workers with the highest serum PCB levels (13.2 and 65.3) both reported they ate their lunch in the work area. BLL was not correlated with serum PCB level ($r=0.32$, $p=.23$).

DISCUSSION

There was one participant who had a substantially higher serum PCB level (65.3 ppb) than any other. Contamination of the sample was considered as an explanation but the NCEH laboratory

reported that the analyte matched Aroclor 1260 well and was not associated with the randomness typically seen in contaminated samples. The employee was interviewed by telephone to determine whether he had other sources of PCB exposure or had different job duties than other employees. The interview did not reveal any other known source of PCB exposure although he reported that he was the only employee who regularly drank from an outside water spigot. No analysis of water from this spigot is available, but there is no reason to suspect that the water's source is different than that of water elsewhere on the site. Since the spigot is outdoors, it could be contaminated by soil. In any case, the employee has decided not to continue to drink from the spigot. This employee also reported on his questionnaire that he ate his lunch on the worksite. It may be possible that these practices increased his PCB intake through ingestion of contaminated water and contamination of his food while eating. However, it is hard to postulate a high PCB exposure at work that would not also result in a comparably high exposure to lead. The latter is not reflected in his BLL of 23. The worker declined an offer of a repeat serum PCB test. If his result is excluded from the analysis, the mean serum PCB for the remaining participants was 3.9 ppb.

This study demonstrated the absorption of lead from the occupational environment, with some BLLs in excess of PHS guidelines, without a readily identifiable industrial process involving lead. Airborne exposure above the OSHA PEL occurred for one worker involved in cutting lead-containing materials with a torch outdoors at CIS, while the other air measurements were well below the OSHA PEL of 50 $\mu g/m^3$. Although not in violation of the OSHA lead standard, BLLs were elevated at these worksites compared to non-occupationally exposed populations. The EPA estimated that the average BLL of the general population had declined to about 5 $\mu g/dl$ by 1990[43] and a study of non lead-exposed workers in New York City in 1990 revealed a mean BLL of 7.4 $\mu g/dl$.[44] Mean BLL at CIS was 21.7 $\mu g/dl$, and mean BLL at CSM was 19.2 $\mu g/dl$.

It was hypothesized that the workers at the CSM worksite would have lower BLLs than workers at the CIS worksite. CSM contained a paved floor so workers were not working directly on lead-contaminated soil. Still, there was no statistically significant difference between either BLL or serum PCB levels between the two worksites. This finding would suggest that either lead contamination was widespread throughout the facility, probably from wind-borne spread of contaminated soil, or there were other sources of lead exposure at CSM.

To evaluate wind-borne spread of the contaminated soil, it would be prudent to conduct environmental air sampling under dry, windy conditions. OSHA compliance officers reported that sampling was conducted on dry (not rainy) days. Whether this sampling was representative of worst-case exposures is unknown.

The lack of correlation between BLL and serum PCB was surprising since exposure to both compounds, assuming the primary source was soil on the site, would presumably be by similar routes, either ingestion or inhalation of contaminated particles. Possible reasons for this finding include:

1. There was another source of lead exposure besides the soil. This possibility would also explain the similar BLLs at CIS and CSM. It is possible that some of the aluminum recycled by CSM, which includes old gutter and aluminum siding, had been painted with lead-based paint, although management discounts this possibility. In this scenario, the workers may come in contact with lead during cutting (with or without a torch) and sorting operations. Air sampling conducted by OSHA measured a high lead level (above the PEL of 50 $\mu g/m^3$) related to the use of a torch in one of their samples. It is possible that this airborne lead associated with cutting further contaminated the worksite with lead and is a continuing source of lead exposure throughout the facility.

2. There was a difference between the environmental distribution of lead and PCB. The fine dust that is most likely to be wind-borne may contain relatively greater amounts of lead and relatively less PCB than the larger soil particles.

With the exception of one worker with a serum PCB of 65.3 ppb, serum PCB levels were not different from available general population levels,[17] and it is possible that, despite the contaminated soil, there was no measurable exposure for most workers to PCB at the site. This would account for lack of correlation between BLL and serum PCB. The worker with a level of 65.3 ppb may have had some unidentified exposure at the site, peculiar to him, a non-occupational exposure, or contamination of his blood sample. The workers with the two highest serum PCB levels, however, both reported that they ate their lunch in the work area and the possibility of ingestion of food contaminated with PCBs from the environment should be considered.

Elevated BLLs with comparatively low airborne lead exposure have been noted previously by other NIOSH researchers who concluded that the ingestion of lead-contaminated soil and, to a lesser extent, dust appear to be important routes of lead exposure at studied households, particularly among children.[45] Absorption of lead from dust and soil due to ingestion is usually found associated with children because they tend to ingest more soil than adults.[46] In another study of lead exposure from contaminated soil,[47] NIOSH investigators reported BLLs ranging from 4 to 13 µg/dl. Personal breathing zone air lead concentrations ranged from non-detectable to 7 $\mu g/m^3$ and soil levels ranged from 0.25% to 1.7%. These levels are lower than those found at CIS and CSM where personal air samples for lead, collected by OSHA, contained up to 73.3 $\mu g/m^3$ and soil levels, as determined by the EPA, ranged up to 3.9%. These higher environmental measurements were associated with higher BLLs at the CSM and CIS worksites.

CONCLUSIONS AND RECOMMENDATIONS

Blood analysis indicated that four workers had excessive exposures to lead, based on NJDOH and U.S. PHS guidelines of 25 µg/dl.[12] Usually, engineering controls are the initial, preferable method for controlling potential workplace exposures to harmful substances. Engineering controls are generally intended to control contaminants at their source. At CIS and CSM, a possible source of potential lead and PCB exposure is apparently the soil, although other sources must be considered. The correlation between BLL and the number of cigarettes smoked at work, and between serum PCB and eating lunch in the work area, suggests that hand to mouth contact may be a route of exposure at this worksite. Elimination of potential exposures to lead would require environmental cleanup of the site and preventive measures to avoid further contamination. While this has apparently has been started by the U.S. EPA, the following work practice controls are offered as prudent measures to improve work practices and decrease the potential for exposure to lead, PCBs and other hazardous substances.

1. Good personal hygiene practices can contribute to the control of worker exposure to lead and PCBs. The facility should be kept as clean as possible, and dust in the work areas should be controlled.

2. Workers need to understand the potential for exposing family members to lead and other toxic substances brought home on clothing, shoes, hair, etc. Other potentially hazardous substances should be identified and the workers made aware of their presence.

3. Workers should not eat, drink, or smoke in the work area except in designated, non-contaminated, areas and should wash their hands and face before eating, drinking or smoking. A dedicated lunchroom should be developed that is able to be isolated from the rest of the facility. Smoking should not be allowed indoors except in enclosed rooms, not used for other purposes, where the air is exhausted directly outdoors.

4. Outdoor faucets that are not protected from contamination by soil or process-related dust should not be used for drinking water.

Other recommendations regard the need for further evaluation.

1. Further industrial hygiene evaluations should be conducted by the NJDOH or the companies to further characterize work-related lead exposure since additional information is required to determine its source. This would include additional measurements of the cutting operations and analysis of the paint on painted materials to determine if lead is present. If lead exposure is occurring as part of the job duties, then engineering controls should be implemented to decrease the exposure.

2. Airborne lead concentrations should be measured during extremely windy, dry conditions to determine if respiratory protection is needed under these environmental conditions. If air monitoring indicates concentrations in excess of the OSHA PEL of 50 $\mu g/m^3$, respirators should be required on any windy dry day until exposure can be controlled by engineering methods. If respirators are required as an interim measure because of lead concentrations above the OSHA PEL, a respirator program that complies with the OSHA respiratory protection standard CFR 1910.134 and is consistent with the NIOSH Guide to Industrial Respiratory Protection should be implemented.

AUTHORSHIP AND ACKNOWLEDGEMENTS

Report Prepared by: Robert Malkin
Supervisory Epidemiologist
Medical Section

Hazard Evaluations and Technical Assistance Branch

Originating Office: Hazard Evaluations and Technical Assistance Branch

Technical Assistance by: Marian Coleman
Health Technician

Report Typed by: Tamme Ottlinger
Office Automation Assistant

Kathy Mitchell
Secretary

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1. New Jersey State Health Department
2. ATSDR
3. OSHA, Region II
4. Curcio Scrap Metal
5. Cirello Iron and Steel

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.

REFERENCES

1. Environmental Protection Agency (1991). Declaration Statement, Record of Decision, Curcio Scrap Metal.

2. Miller DT, Paschal DC, Gunter EW, Stroud PE, D'Angelo J [1987]. Determination of lead in blood using electrothermal atomic absorption spectrometry with a L'vov platform and matrix modifier Analyst, 112, pp. 1701-4.

3. Burse VW, Korver MP, Needham LL, Lapeza CR, Boozer EL, Head SL, Liddle JA, Bayse DD [1989]. Gas chromatographic determination of polychlorinated biphenyls (as aroclor 1254) in serum: collaborative study. J. Assoc. Off. Anal. Chem 72: 649-649.]

4. Code of Federal Regulations [1992]. OSHA lead standard. 29 CFR, Part 1910.1025. Washington, DC: U.S. Government Printing Office, Federal Register.

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

6. Proctor NH, Hughes JP, Fischman ML [1988]. Lead. In: Chemical hazards of the workplace. 2nd ed. Philadelphia, PA: J.B. Lippincott Company, Philadelphia, pp 294-298.

7. NIOSH [1978]. Occupational exposure to inorganic lead. Cincinnati, OH: U.S. Department of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHEW (NIOSH) Publication No. 78-158.

8. Code of Federal Regulations [1992]. OSHA lead standard. 29 CFR, Part 1910.1025. Washington, DC: U.S. Government Printing Office, Federal Register.

9. United States Environmental Protection Agency (1986). Air Quality Criteria for lead 87-12602, Volume 1.

10. Needleman HL, Schell A, Bellinger D, Leviton A, Allred E [1990]. The long term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. New Eng J Med 322(2)83-88.

11. Grandjean, P and Bach, E [1986]. Indirect exposures: the significance of bystanders at work and at home. Am. Ind. Hyg. Assoc. J. 47(12):819-824.

12. DHHS [1990]. Healthy people 2000: national health promotion and disease objectives. Washington, DC: U.S. Department of Health and Human Services, Public Health Service, DHHS Publication No. (PHS) 91-50212.

26. Wong CK [1981]. PCB poisoning special issue. Clinical Medicine (Taipei) 7:1.

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

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

29. Karppanen E, Kolho L. 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, 1973: 124.

30. Fischbein A, Woolf MS, Lilis R, Thornton J, Selikoff IJ. Clinical findings among PCB-exposed capacitor manufacturing workers. Ann NY Acad Sci 1979;320:703.

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

32. National Institute for Occupational Safety and Health. Health hazard evaluation report no. HETA 80-007-1520. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1984.

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

34. Crow KD. Chloracne: a critical review including a comparison of two chloronaphthalene and pitch fumes. Trans St John's Hosp. Dermatol Soc 1970;56:79.

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

36. Kreiss K, Zack MM, Kimbrough RD, Needham LL, Smrek AL, Jones BT. Association of blood pressure and polychlorinated biphenyl levels. JAMA 1981;245:2505.

37. International Agency for Research on Cancer. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Chemicals, Industrial Process, and Industries Associated with Cancer in Humans. IARC Monographs, Volumes 1 to 29. Supplement 4, Lyon, France, 1982: 218.

38. Brown DP, Jones M. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. Arch Environ Health 1981;36:120.

13. EPA [1989]. Memorandum, OSWER Directive #9355.4-02, Interim guidance on establishing oil lead cleanup levels at superfund sites. Office of Solid Waste and Emergency Response, United States Environmental Protection Agency, September 7, 1989.

Lloyd JW, Moore RM, Woolf BS, Stein HP [1076]. Polychlorinated biphenyls. J O Med 18:109.

15. Hutzinger O, Safe S, Zitko V [1974]. The chemistry of PCBs. Cleveland:The Chemical Rubber Company Press.

Landrigan PJ [1980]. General population exposure to halogenated biphenyls. In: Kimbrough RD, ed. Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins, and related products. Amsterdam: Elsevier/north Holland Biomedical Press, p.267.

17. Kreiss K [1985]. Studies on populations exposed to polychlorinated biphenyls. Env Hlth Perspectives 60:193-199.

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

Vos JG, Koeman JG [1970]. Comparative toxicologic study with polychlorinated biphenyls in chickens, with special reference to porphyrias, edema formation, lover necrosis, and tissue residues. Tox App Pharm 17:656.

20. Vos JG, Koeman JH van der Mass HL, ten Noever de Brauw MC, de Vos RH [1970]. Identification and toxicological evaluation of chlorinated dibenzofuran and chlorinated naphthalene in two commercial polychlorinated biphenyls. Fd Cosmet Toxicol 8:625.

Bowes GW, Mulvihill MJ, Simineti BRT, Burlingame AL, Risebrough RW [1975]. Identification of chlorinated dibenzofurans in American polychlorinated biphenyls. Nature 94:125.

Buser HR, Bosshadt HP, Rappe C [1978]. Formation of polychlorinated dibenzofurans (PCDFs) from the pyrolysis of PCBs. Chemosphere 7:109

23. Buser HR, Rappe C [1977]. Formation of polychlorinated dibenzofurans (PCDFs) from, the pyrolysis of individual PCB isomers. Chemosphere 8:157.

24. 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.

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

Brown DP. Mortality of workers exposed to PCBs. Arch Environ Health 1987;42:333-9

40. Bertazzi PA, Zocchetti C, Guercilena S, Foglia MD, Pesatori A, Riboldi L. Mortality study of male and female workers exposed to PCBs. Presented at the International Symposium on Prevention of Occupational Cancer, April, 1981, Helsinki, Finland.

Bertazzi PA, Riboldi L, Pesatori A, Radice L, Zocchetti C. Cancer mortality of capacitor manufacturing workers. Am J Industr Med 1987;11:165-76.

Sinks T, Smith AB, Steele GK. A retrospective cohort mortality study of workers at a capacitor plant utilizing polychlorinated biphenyls. Abstract, American Journal of Epidemiology 1990; 132:755-6.

USEPA [1991]. Strategy for reducing lead exposure. United States Environmental Protection Agency, Washington, D.C.

44. Malkin R, Brandt-Rauf P, Graziano J, Parides M [1992]. Blood lead levels in incinerator workers. Env Res 59:265-270.

NIOSH [1991]. Hazard evaluation and technical assistance report: Technical Assistance to the Jamaican Ministry of Health, Kingston, Jamaica. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, NIOSH Report No. HETA 87-371-1989.

46. Duggan MJ, Inskip MJ. Childhood exposure to lead in surface dust and soil: a community health problem. Public Health Rev 13:1-54, 1985.

47 NIOSH [1991]. Hazard evaluation and technical assistance report: Carbonnaire Company, Palmerton PA. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, NIOSH Report No. HETA 91-073-2165.

TABLE 1
Curcio Scrap Metal
Saddle Brook, New Jersey
HETA 93-0739
Range of PCB concentrations from soil
48 samples
July 1989
(EPA data)*

Aroclor 1242	ND (12 samples)→ 4500 ppm
Aroclor 1248	ND (all samples)
Aroclor 1254	ND (12 samples)→ 1700 ppm
Aroclor 1260	ND (37 Samples)→ 210 ppm

ND=not detectable
* United States Environmental Protection Administration [1991]. Declaration Statement, Record of Decision, Curcio Scrap Metal.

TABLE 2
Curcio Scrap Metal
Saddle Brook, New Jersey
HETA 93-0739

LEAD AND PCB RESULTS
OSHA DATA

LEAD RESULTS			
OCCUPATION	DATE	SAMPLE TYPE	RESULT
Torch Cutter	11/16/92	air- 505 minutes	12 µg/dl
Torch cutter	11/16/92	air-506 minutes	73.3 µg/dl
Shears cutter	11/16/92	air-500 minutes	2.7 µg/dl
press operator	11/16/92	air-472 minutes	11.9 µg/dl
next to metal compactor	11/16/92	bulk	0.1%
at shears cutter	11/16/92	bulk	0.04%
near press machine	12/2/92	bulk	1.0%
in Cirello locker room	12/2/92	bulk	0.6%
Bottom of torch cutter's street shoes	12/8/92	wipe	89 µg
Bottom of torch cutter's street shoes	12/8/92	wipe	91 µg
Bottom of press operator's street shoes	12/2/92	wipe	76 µg
PCB RESULTS			
Torch cutter	12/8/92	air-483 minutes	ND
Torch cutter	12/8/92	air-293 minutes	ND
next to metal compactor	11/16/92	bulk	81 ppm
shears cutter station	11/16/92	bulk	265 ppm
near tin press operator	12/8/92	bulk	82 ppm
in Cirello locker room	12/2/92	bulk	76 ppm
bottom of torch cutter's street shoes	12/8/92	wipe	ND
inside torch cutter's work gloves	12/8/92	wipe	ND
floor of curcio workroom	12/15/92	bulk	64 ppm

ND = not detectable
ppm = parts per million

Table 3
Curcio Scrap Metal
Saddle Brook, New Jersey
HETA 93-0739

BLL and Serum PCB levels
Curcio Scrap Metal
Cirello Iron and Steel
June 22, 1993.

#	BLL (second draw) [µg/dl]	Serum PCB [ppb]
1.	26.9	2.35
2.	24.6	1.64
3.	21.6	10.6
4.	15.5	1.6
5.	4.0	1.5
6.	30.4	5.6
7.	26.7	7.34
8.	39.8 (37.8)	2.34
9.	10.2	<1
10.	9.9	3.13
11.	20.4 (19.3)	2.06
12.	24.1	4.46
13.	23.0	65.3
14.	11.0 (10.9)	1.19
15.	19.6 (19.1)	<1
16.	24.2	13.2
17.	8.7 (8.6)	3.6

Table 4
Curcio Scrap Metal
Saddle Brook, New Jersey
HETA 93-0739

Mean BLL (μg/dl) and Serum PCB Levels (ppb)
June 22, 1993

	number	CIS	CSM	p
Mean BLL	17	21.7	19.2	0.58
Mean PCB	16*	5.1	3.3	0.39

* The outlier with a PCB level of 65.3 ppb was excluded from the analysis

Table 5
Curcio Scrap Metal
Saddle Brook, New Jersey
HETA 93-0739

Mean BLL (μg/dl) by Job Duty
June 22, 1993

JOB DUTY	N	Mean	Minimum	Maximum
any metal cutting	5	19.8	10.2	26.9
management/secretarial	3	10.7	4.0	19.4
sorting	4	29.1	23.0	38.8
press machine operator	3	18.0	9.9	24.2
vehicle operator	2	18.9	11.0	26.7

Table 6
Curcio Scrap Metal
Saddle Brook, New Jersey
HETA 93-0739

Mean Serum PCB (ppb) by Job Duty
June 22, 1993

JOB DUTY	N	Mean	Minimum	Maximum
any metal cutting	5	3.3	0.5	10.6
management/secretarial	3	1.9	0.5	3.6
sorting	3	1.7	2.3	5.6
press machine operator	3	6.1	2.1	13.2
vehicle operator	2	4.3	1.2	7.3

Table 7
Curcio Scrap Metal
Saddle Brook, New Jersey
HETA 93-0739

Minimum , Maximum, and Mean Serum PCB Levels (ppb) Depending on Where Lunch is Eaten
June 22, 1993

Location	Number	Minimum	Maximum	Mean
In the lunchroom	7	0.5	3.1	1.5
away from work	6	1.5	10.6	8.7
In the work area	2	13.2	65.3	39.25

Table 8
Curcio Scrap Metal
Saddle Brook, New Jersey
HETA 93-0739

Minimum , Maximum, and Mean BLL (µg/dl) Depending on Where Lunch is Eaten
June 22, 1993

Location	Number	Minimum	Maximum	Mean
In the lunchroom	7	9.9	19.9	14.3
away from work	6	4.0	26.9	21.3
In the work area	2	23.0	26.9	23.6