

Occupational and Environmental Lead and PCB Exposure at a Scrap Metal Dealer

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Blood lead levels (BPb) and serum polychlorinated biphenyl levels (PCB) were obtained from 17 employees at two adjacent scrap metal dealers. One facility was located outdoors, directly on top of soil known to be contaminated with lead and PCBs, and the other was located indoors with a concrete floor. BPbs ranged from 4.0 to 39.8 $\mu\text{g/dl}$ (mean 19.9 $\mu\text{g/dl}$, geometric mean 17.5 $\mu\text{g/dl}$) and PCB levels ranged from <1 to 65.3 ppb (mean 7.5 ppb). There was no significant difference in either BPb or serum PCB between the two sites. BPb was significantly correlated with the number of cigarettes smoked at work, and both BPb and serum PCB were significantly related to eating lunch outside the lunchroom, suggesting hand-to-mouth contact as a source of exposure. The lack of difference in BPb between employees of the two scrap metal dealers suggests an ongoing source of lead exposure at the sites, other than the soil.

INTRODUCTION

In 1993, the New Jersey Department of Health (NJDOH) asked the National Institute for Occupational Safety and Health (NIOSH) to evaluate worker exposure to lead and PCBs at two adjacent scrap metal companies. Because of the scrap metal operation, this site has been known, since 1982, to be contaminated with lead and PCBs. In July 1987, the site was placed on the Environmental Protection Agency's (EPA) National Priorities List (NPL) for cleanup. 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 and lead concentrations up to 39,300 ppm. Other contaminants included mercury and trichlo-

roethylene. The 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 associated with this contamination" (U.S. EPA, 1991).

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) 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 (from exposure to lead and PCBs) to possibly result in carcinogenic and noncarcinogenic effects" (ATSDR, 1992). As a result of the release of the ATSDR Public Health Assessment, the Occupational Safety and Health Administration (OSHA) inspected the sites. Full-shift, personal-breathing-zone air samples were collected for lead, mercury, and PCBs in November and December 1992. Only one lead level (out of seven samples) of 73.28 $\mu\text{g/m}^3$ exceeded the OSHA permissible exposure limit (PEL) of 50 $\mu\text{g/m}^3$ for lead. This level was recorded near a torch cutter and OSHA investigators believed that the lead originated from the material being cut. OSHA took six other full-shift samples for lead; four were below 6 $\mu\text{g/m}^3$ and two were 12 $\mu\text{g/m}^3$. Wipe samples for lead ranged from 28 to 140 $\mu\text{g}/100\text{ cm}^2$ and bulk samples ranged from 400 to 10,000 ppm. No PCB was found in any air or wipe samples taken by OSHA; bulk samples ranged from nondetectable to 265 ppm. These measurements were made at both companies located on the site.

The first facility (Company A) is involved in non-ferrous metal recycling, predominantly aluminum, but also copper and gold. The work area is located indoors and contact with contaminated soil by employees during their job seemed minimal. The sec-

ond facility (Company B) is involved in ferrous metal recycling and is located on an unpaved lot (known as the east lot) of the facility. This worksite is outdoors and is directly on the area contaminated with lead and PCBs. Because all previous studies measured only environmental levels of lead and PCBs, the primary purpose of this study, therefore, was to evaluate biologic markers of lead and PCB levels at these two companies to more accurately assess worker exposure. A secondary aim was to determine risk factors for lead and PCB exposure at these worksites.

METHODS

This evaluation included a questionnaire survey and blood testing for lead and PCB. All employees and management of both companies were asked to complete a self-administered questionnaire that addressed work history and risk factors for lead exposure including hobbies. Seven employees however spoke only Arabic or Polish. A translator from the NJDOH was provided for the Polish-speaking workers and a bilingual worker translated for the Arabic-speaking employees. Blood collection was done at the site on June 22, 1994. Collection and analysis of blood samples for lead and PCB content were done according to the Centers for Disease Control and Prevention (CDC) National Center for Environmental Health (NCEH) laboratory guidelines. BPbs were analyzed using graphite furnace atomic absorption (Miller *et al.*, 1987). Blood for serum PCB analysis was collected in anticoagulant free tubes and centrifuged, and serum was transferred and stored in hexane- and acetone-washed pipettes and serum storage bottles, provided by the NCEH lab. Serum was frozen and shipped with dry ice to the CDC laboratory. Analysis was performed using gas chromatography (Burse *et al.*, 1989). Tubes used for BPb 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 BPb; it was submitted to the laboratory identified as if it were from another participant.

Statistical analysis involved the following: (1) *t* tests to compare group BPb and serum PCB means with the dichotomous variables [where the employee worked, use of a torch at work (yes/no), where lunch was eaten (in the lunchroom or not)]; (2) ANOVA for the categorical variables (time spent on the east lot and job category); and (3) Pearson correlation coefficients for the continuous variables (age, the number of cigarettes smoked at work per day, and to determine if BPb was correlated with serum PCB).

To allow for comparison with the Third National Health and Nutrition Examination Survey (NHANES III) data, the geometric mean for BPb was calculated in the same fashion as Brody *et al.* (1994), by taking the antilog of the mean of \log_{10} BPbs.

RESULTS

All 17 employees present at work on the days of the study participated; 12 worked at Company A and 5 worked at Company B. BPbs ranged from 4.0 to 39.8 $\mu\text{g/dl}$ (mean 19.9 $\mu\text{g/dl}$, geometric mean 17.5 $\mu\text{g/dl}$) (Table 1). Two workers at Company A had BPbs greater than 30 $\mu\text{g/dl}$ and 3 had BPbs greater than 20 $\mu\text{g/dl}$ but less than 30 $\mu\text{g/dl}$. Four of the 5 workers at Company B had BPbs greater than 20 $\mu\text{g/dl}$, but none were greater than 30 $\mu\text{g/dl}$. Differences in BPbs in paired samples were within the range of acceptable analytical variation and no pair varied more than 6%. PCB levels ranged from <1 to 65.3 ppb (mean 7.5 ppb). Two PCB levels were below the level of quantification of 1 ppb and were assigned a value of 0.5 ppb. The PCB level of 1 individual was much higher than any other (65.3 ppm with the next highest PCB level being 13.2 ppb). This outlier was not included in the statistical analysis. PCB levels were not normally distributed and a log transformation was done as part of the analysis.

There was no statistically significant difference between mean BPb or PCB level and company (Table 1), use of a torch at work (yes/no) ($P = 0.87$ for BPb, $P = 0.49$ for serum PCB), or the time spent on the east (unpaved) lot ($P = 0.66$ for BPb; $P = 0.79$ for serum PCB). BPb was correlated with age ($r = 0.04$, $P = 0.86$ for BPb; $r = 0.26$, $P = 0.32$ for PCB). BPb 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 job categories based on their responses on the questionnaire. Employees who reported that they were involved in sorting operations had the highest BPb (mean = 29 $\mu\text{g/dl}$) and employees who reported that

TABLE 1
Mean BPb and Serum PCB Levels by Company,
June 22, 1993

	Number	Range	Company A (mean)	Company B (mean)	<i>P</i>
BPb ($\mu\text{g/dl}$)	17	4.0–39.8	19.2	21.7	0.58
PCB (ppb)	17	<1–65.3	3.9 ^a	5.1	0.39

^a The outlier with a PCB level of 65.3 ppb was excluded from determination of the mean.

they were press machine operators had the highest PCB levels (6 ppb). Those workers who reported that they were involved in either managerial or secretarial duties had the lowest levels (mean = 11 $\mu\text{g}/\text{dl}$ for BPb and 2 ppb for PCB), although the differences between all job duties were not statistically significant ($P = 0.09$ for BPb and $P = 0.76$ for PCB). Respondents did not report hobbies involving lead such as ceramics, craft work using leaded paints riflery, or others.

Eating lunch in the lunchroom was associated with lower blood levels of lead and PCB (Table 2) than eating lunch "not in the lunch room" and this difference was statistically significant for both PCB level and BPb ($P = 0.05$). However, the two workers with the highest serum PCB levels (13.2 and 65.3) both reported that they ate their lunch in the work area and were the only two workers who responded that way. BPb was not correlated with serum PCB level ($r = 0.32$, $P = .23$). There were not enough participants in the study to construct a multiple regression model and no variables were significant at the 0.15 level. Reanalyzing the PCB data using log-transformed values yielded the same results.

DISCUSSION

This study shows the absorption of lead from the occupational environment. BPbs in four (24%) of the participants, two from Company A and two from Company B, exceeded 25 $\mu\text{g}/\text{dl}$; a Public Health Service (PHS) goal for the year 2000 is for all workers exposed to lead to have a blood lead level less than 25 $\mu\text{g}/\text{dl}$ (DHHS, 1990). None exceeded 40 $\mu\text{g}/\text{dl}$, the level at which OSHA requires more frequent monitoring (29 CFR 1910.1025, 1989). Hypertension has been associated with BPbs of between 30 and 40 $\mu\text{g}/\text{dl}$ and possibly as low as 7 $\mu\text{g}/\text{dl}$ (U.S. EPA, 1986).

Most air measurements were well below the

TABLE 2
Minimum, Maximum, and Arithmetic Mean BPb and Serum PCB Levels Depending on Where Lunch Is Eaten, June 22, 1993

	Number	BPb ($\mu\text{g}/\text{dl}$)		
		Minimum	Maximum	Mean
In the lunchroom	6	9.9	19.9	14.3
Not in the lunchroom	8	4.0	26.9	21.9*
	Number	PCB Levels (ppb)		
		Minimum	Maximum	Mean
In the lunchroom	6	0.5	3.1	1.5
Not in the lunchroom	7	1.5	13.2	5.8*

* Statistically significant, $P < 0.05$.

OSHA PEL of 50 $\mu\text{g}/\text{m}^3$ except for one worker involved in cutting lead-containing materials with a torch. However, the association of higher BPb with the number of cigarettes smoked at work and the association of increased BPb and serum PCB with eating lunch not in the lunchroom suggest hand-to-mouth contact as a source of worker exposure.

Although not directly involved in working with lead, most workers at these worksites had higher BPbs than nonoccupationally exposed populations. The NHANES III assessment of blood lead levels in the general population, for the period 1988–1991, revealed a geometric mean blood lead level of 3.8 $\mu\text{g}/\text{dl}$ for males aged 20–49 (Brody *et al.*, 1994). The geometric mean for all workers in this study was 17.5 $\mu\text{g}/\text{dl}$. A study of non-lead-exposed workers in New York City in 1990 revealed an arithmetic mean BPb of 7.4 $\mu\text{g}/\text{dl}$ (Malkin *et al.*, 1992). The arithmetic mean BPb was 21.7 $\mu\text{g}/\text{dl}$ at Company B and 19.2 $\mu\text{g}/\text{dl}$ at Company A.

There was one participant who had a much 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 sample matched Aroclor 1260 well and did not have the random peaks in gas chromatographic analysis that are associated with contaminated samples. The employee was interviewed by telephone to determine whether he had the 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 (other jobs, eating fish from known contaminated waters), although he reported that he was the only employee who regularly drank from an outside water spigot. Water from this spigot was not analyzed, but there is no reason to suspect that the water's source is different from that of water elsewhere on the site. Since the spigot is outdoors, it could be contaminated by soil. 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 by ingesting PCB-contaminated soil while eating or drinking water from the potentially contaminated spigot. However, it is difficult to postulate a high PCB exposure at work that would not also result in a comparably high exposure to lead. The latter are not reflected in his BPb of 23. The worker declined an offer of a repeat serum PCB test. When his result is excluded from the analysis, the mean serum PCB for the remaining participants was 3.9 ppb, within the range expected for the general population.

Since Company A had a paved floor, workers were not working directly on lead-contaminated soil.

Therefore, if contaminated soil was the primary source of lead exposure, one might hypothesize that Company A workers would have significantly lower BPbs than Company B employees. There was no statistically significant difference between the companies with respect to either BPb or serum PCB levels between the two worksites. Reasons for this finding might include the following: (1) lead contamination was widespread throughout the facility, perhaps from wind-borne spread of contaminated soil; (2) there were other sources of lead exposure at Company A; or (3) there were other environmental sources of lead specific to Company A workers, such as lead-containing drinking water pipes in their homes.

The lack of correlation between BPb and serum PCB also suggests that soil was not the sole source of lead exposure since lead and PCB exposure would presumably occur by similar routes, either ingestion or inhalation of contaminated particles. Possible reasons for this lack of correlation include the following:

1. There was another source of lead exposure other than the soil, which would also explain the similar BPbs at Company A and Company B. It is possible that some aluminum recycled by Company A, 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 and sorting operations that might explain the higher BPbs in workers involved in sorting operations. Air sampling conducted by OSHA measured a high lead level (above the PEL of 50 $\mu\text{g}/\text{dl}$) related to the use of a torch in one of their samples. It is also 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 distribution of lead at both companies was more widespread than the distribution of PCB. OSHA sampling found that seven bulk samples for lead ranged from 400 to 10,000 ppm, while eight PCB bulk samples ranged from nondetectable to 265 ppm.

Except for one worker with a serum PCB of 65.3 ppb, serum PCB levels were not different from general population levels (Kreiss, 1985), and it is possible that, despite the contaminated soil, there was no measurable worker exposure to PCB at the site for most of the workers. The workers with the two highest serum PCB levels, however, both reported that

they ate their lunch in the work area so ingestion of food contaminated with PCBs from the environment may have occurred. The finding of elevated BPb without elevated serum PCB further suggests a source of lead exposure other than soil.

Limitations of this study include the lack of an unexposed group, the lack of environmental measurements to characterize the source of ongoing lead exposure from job duties at Company A, and the fact that many employees required translation to complete their questionnaire, possibly leading to exposure misclassification. Nevertheless, the worker BPbs indicate increased lead exposure in this cohort. Whether similar exposure to lead is occurring at other scrap metal facilities is unknown at this time, but further research is needed to determine the extent of lead exposure among workers in this occupation.

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REFERENCES

- 29 CFR 1910.1025 (1989). Code of Federal Regulations. U.S. Government Printing Office, Office of the Federal Register, Washington, DC.
- ATSDR (1992). Initial release—Public Health Assessment for Curcio Scrap Metal, Saddle Brook Township, New Jersey, CERCLIS No. NJD011717584, p. 18. Agency for Toxic Substances and Disease Registry, Atlanta, GA.
- Brody, D. J., Pirkle, J. L., Kramer, R. A., Flegal, K. M., Matte, T. M., Gunter, E. W., and Paschal, D. C. (1994). Blood lead levels in the U.S. population: Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES 111, 1988 to 1991). *JAMA* **272**(4), 277–283.
- Burse, V. W., Korver, M. P., Needham, L. L., Lapeza, C. R., Boozer, E. L., Head, S. L., Liddle, J. A., and Bayse, D. D. (1989). Gas chromatographic determination of polychlorinated biphenyls (as Aroclor 1254) in serum: Collaborative study. *J. Assoc. Off. Anal. Chem.* **72**, 649–659.
- DHHS (1990). Healthy People 2000: National Health Promotion and Disease Objectives, U.S. Department of Health and Human Services, Public Health Services, DHHS Publication No. (PHS) 91-50212. Washington, DC.
- Kreiss K. (1985). Studies on populations exposed to polychlorinated biphenyls. *Environ. Health Perspect.* **60**, 193–199.
- Malkin, R., Brandt-Rauf, P., Graziano, J., and Parides, M. (1992). Blood lead levels in incinerator workers. *Environ. Res.* **59**, 265–270.
- Miller, D. T., Paschal, D. C., Gunter, E. W., Stroud, P. E., and D'Angelo, J. (1987). Determination of lead in blood using electrothermal atomic absorption spectrometry with a L'vov platform and matrix modifier. *Analyst* **112**, 1701–1704.
- U.S. EPA (1986). Air Quality Criteria for Lead. United States Environmental Protection Agency, Washington, DC.
- U.S. EPA (1991). Declaration Statement, Record of Decision, Curcio Scrap Metal. United States Environmental Protection Agency, Region II: Hasbrouck Heights, New Jersey.