

TEMPORAL CHANGES IN BLOOD LEAD LEVELS OF HAZARDOUS WASTE WORKERS IN NEW JERSEY, 1984–1987

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Abstract. The blood lead levels of 568 New Jersey hazardous waste workers were determined at least once in the period 1984–1987. Workers almost always wore protective clothing in the field, which should have prevented exposure to lead. Therefore, despite their occupation, these workers are probably representative of the New Jersey suburban population in the mid-1980s. Blood lead levels ranged from 2.0–20.7 $\mu\text{g/dL}$, with an arithmetic mean value of 5.8 $\mu\text{g/dL}$, representing a decline from levels measured a decade earlier, but there was no clear change over the four years of the study. Blood lead levels increased with age of the worker up to age 60. Women showed lower levels than men in every age group.

1. Introduction

Lead is one of the best studied of toxic substances. It occurs naturally in the earth's crust; all populations are exposed to it, and it has no known biological function. The blood lead level is commonly used to assess recent exposure, but is less indicative of body burden. There have been recent studies of background blood lead levels in developed (Annest and Mahaffey, 1984; Watanabe, 1985; Elind *et al.*, 1986; Brockhaus *et al.*, 1988) and non-industrial regions (Hecker *et al.*, 1974; Piomelli *et al.*, 1980, Poole and Smythe, 1980), and there is a number of studies on adults and children with industrial or household exposure, particularly in proximity to smelters (Baker *et al.*, 1977). The consensus concerning the normal (or safe) blood lead level has shifted dramatically downward. For many years the toxic threshold level was considered to be 80 $\mu\text{g/dL}$ for the general public and 120 $\mu\text{g/dL}$ for industrial workers (Vaughn, 1973; Windeyer, 1972). Frank lead intoxication was identified with levels above 60 $\mu\text{g/dL}$ (Poulos, 1986), and acceptable limits were lowered to 30 $\mu\text{g/dL}$ (Ahmed, 1987), or 25 $\mu\text{g/dL}$ for children, with behavioral development studies promising to lower the level still further (CDC recommends < 10 $\mu\text{g/dL}$ for children) with 15 $\mu\text{g/dL}$ being the 'level of concern'. By contrast, in 1980 the 'normal' range in the United States was considered to be 15–25 $\mu\text{g/dL}$ (Piomelli *et al.*, 1980). Primitive man probably had a body burden of lead one-hundredth of that seen in people today (Patterson, 1965; Gerber, 1980). The quest for the background level of lead in humans, the irreducible minimum due to naturally occurring lead, resulted in studies of a variety of non-industrial populations. In dealing with low

level lead determinations it is critical to use methodologic procedures that are designed to avoid laboratory contamination (Poole and Smythe, 1980), and even today commercial laboratories have difficulties with the precision analysis of blood leads below 10 $\mu\text{g/dL}$ (Gochfeld *et al.*, unpublished QA/QC study).

Hecker *et al.* (1974) reported a mean value below 1 $\mu\text{g/dL}$ for Venezuelan Yanomamo Indians, compared with a mean of 14.6 for residents of Ann Arbor, Michigan. The methodology used was not standard and has been questioned (Poole and Smythe, 1980), but whether their 0.82 mean is valid or not, the results clearly indicated low lead levels in the Indian population. Children and adults in the Himalayan foothills averaged 3.4 $\mu\text{g/dL}$ (geometric mean; Piomelli *et al.*, 1980) with only 10% of values being greater than 10 $\mu\text{g/dL}$. They contrasted this with a study of New York school children by Piomelli *et al.* (1978), which found 0.1% with values $< 4 \mu\text{g/dL}$ and 95% $> 10 \mu\text{g/dL}$. At the same time Poole and Smythe (1980) reported a mean of 5.2 $\mu\text{g/dL}$ in children from Papua-New Guinea, with 14% of the values $< 3 \mu\text{g/dL}$ and 3% of values of $> 10 \mu\text{g/dL}$.

Major contributions to elevated lead levels in blood have included automotive emissions from leaded gasoline, leaded paint, soldered water pipes, occupational exposures, and foods. Workers with high potential exposure include smelters (Roberts, 1974; Mendes, 1977), jewelers (Ramakrishna, 1982), acetylene torch users (Cramer, 1974), workers in battery factories (Watson, 1978), potters (Koplan *et al.*, 1977), and workers in gunmetal foundries (Araki, 1986). The above exposures either occur from a point source (a specific factory or building), or from a diffuse source (i.e. food or leaded gasoline).

From 1984–1987 we included biological monitoring for lead in a medical surveillance program for hazardous waste workers employed by State and private agencies in New Jersey. These workers were involved in site inspections and remedial investigation/feasibility studies (RIFS), but not in clean-ups. Our objectives were to determine: (a) whether blood lead levels were higher than expected in the general population; (b) whether blood lead levels decreased over time, indicating a general reduction in ambient lead levels; (c) whether there were gender or age differences in lead levels; and (d) whether retested individuals showed a change in lead levels after working as hazardous waste workers.

The National Health and Nutrition Examination Survey (NHANES II) had documented a substantial decline in blood lead levels from 1976 to 1980 (Annest and Mahaffey, 1984). Our study investigated whether this decline continued in our workforce in the 1980s. Except for studies such as those by Goldwater and Hoover (1967) and Annest and Mahaffey (1984), most data on metal levels are obtained when high exposure has occurred. In the past two decades a major change in potential lead exposure has been the elimination of leaded gasoline, to which the NHANES report attributes a reduction in blood lead (Annest and Mahaffey, 1984).

Hazardous waste workers have a complicated potential exposure profile, because they can work on a number of sites for varying periods of time. Although a list of priority pollutants present will eventually be developed through an RIFS,

no such results exist to guide those workers who are initially surveying a site or performing the RIFS (Gochfeld and Burger, 1990). Thus, potential exposure is not necessarily known, either at the time workers are on the site, or at a later date. Therefore, depending on exposure potential, hazardous workers are required by federal law to undergo training, to wear protective clothing which should eliminate exposure, and to receive medical surveillance (OSHA, 1989). Thus, despite the potential for exposure, hazardous waste workers should not receive any occupational exposure and should not show elevated lead levels in their surveillance programs (Gochfeld, 1990).

2. Materials and Methods

The Robert Wood Johnson Medical School's Environmental and Occupational Health Clinical Center conducts a medical surveillance program for hazardous waste workers employed by New Jersey State Agencies and private employers. The program covers about 1000 workers, all of whom are classified according to their opportunity for exposure. Only individuals with high potential for exposure received biological monitoring of blood lead, and some of these individuals were tested annually. The purpose of the surveillance is explained to all participants and all receive copies and an explanation of the results. Virtually all participants complete informed consent forms, allowing the analysis of aggregate results.

Whole blood was drawn into heparinized, metal-free glass tubes. Specimens of 0.2 mL blood were diluted with 1.8 mL of Triton X-100 with the addition of a matrix modifier containing ammonium phosphate monobasic ($\text{NH}_4\text{H}_2\text{PO}_4$) and magnesium nitrate ($\text{Mg}(\text{NO}_3)_2$) (after Pruszkowska *et al.*, 1983).

Specimens were analyzed with stabilized platform, graphite furnace atomic absorption, using a Perkin-Elmer AA with an HGA-500 furnace and an AS-40 autosampler, reading at 283.3 nm wavelength. Each batch consisted of 20 samples, a spiked sample, a method blank, and three standards. If recovery of the spike was less than 85% or more than 115% the run was repeated. Values were not corrected for percentage recovery. Each digested sample was analyzed in duplicate and the mean value reported, provided that the discrepancy was less than 5%. We used a lower detection limit of 2.0 $\mu\text{g}/\text{dL}$. Twelve of the samples were independently confirmed by a commercial laboratory. The coefficient of variation on method replicates was 12%.

We used multiple regression techniques (S.A.S. PROC GLM) to determine those factors that entered the model which explained variations in blood lead levels (S.A.S., 1985). The procedure compensates for intercorrelation among variables. For the 135 individuals tested more than once, only the first test result is included in the analysis. We also used the nonparametric Kruskal-Wallis test to compare differences among age and sex groupings.

TABLE I
Model explanation variation in blood lead levels in New Jersey hazardous waste workers. For factors entering the model we give *F* values (level of significance).

Overall Model		
	<i>F</i>	6.77
	<i>r</i> ²	0.11
	<i>df</i>	9419
	<i>p</i>	0.0001
Factors entering		
	test year	NS
	age	14.6 (0.0002)
	sex	26.3 (0.0001)
	years of employment	NS

3. Results

The study population consisted of 568 workers (80% males). During the period 1984–1987, 35 of these had three tests and 100 had two tests. For the remainder only a single result was obtained, either as a baseline for individuals with low exposure or because of a change in their employment. The best multiple regression model explained 11% of the variability in blood lead levels according to sex and age, but not test year or years of employment in hazardous waste work (Table I). Overall blood lead levels averaged 5.8 $\mu\text{g/dL}$, and ranged from 2.0–20.7 $\mu\text{g/dL}$ (Table II). The median value was 5.3, and 95% of readings were below 11.3. Only two values (0.3%) exceeded 20 $\mu\text{g/dL}$. There was no decreasing time trend in blood lead levels from 1984 to 1987. Within this time period we had repeat tests one year apart for 135 subjects, and a third year's results on 35 of these. Considering only this subgroup, the mean values changed from 5.4 to 5.6 in one year. However, by the third test there was a slight but significant increase to 6.4 (Kruskal-Wallis $X^2 = 5.83$, $P = 0.015$).

Sex and age were both significant factors affecting variations in blood lead levels. Overall, males had significantly higher lead levels than females (Kruskal-Wallis $X^2 = 36.8$, $df = 1$, $P < 0.0001$, Table III). Females tended to have lower average blood lead levels in every age group (Table III). The sample sizes for certain groups were small, but suggest that, for both sexes, lead levels increased from the age of 20–60 years. The few males over 60 showed a slight decrease.

TABLE II
Arithmetic mean and blood lead levels (first test only) ($\mu\text{g/dL}$) by year for 568 New Jersey hazardous waste workers.

Year	N	PbB Mean (\pm SE)	Range
1984	63	6.06 ± 0.39	* 2.0 – 17.0
1985	208	5.79 ± 0.19	2.0 – 20.7
1986	190	5.94 ± 0.20	2.0 – 20.5
1987	107	5.64 ± 0.26	2.0 – 15.9
All years combined	568	5.81 ± 0.28	2.0 – 20.7

* 2.0 $\mu\text{g/dL}$ represents limit of detection.

TABLE III
Blood lead levels by age and sex in hazardous waste workers (1984–1987). Given are arithmetic means \pm standard error. (Only the first test result for any individual is included.)

Age	Male		Female	
	N	Mean	N	Mean
20–30	152	5.5 ± 0.3	86	3.8 ± 0.2
31–40	223	6.1 ± 0.2	22	4.2 ± 0.3
41–50	55	6.9 ± 0.7	4	5.0 ± 0.6
51–60	16	7.0 ± 0.2	3	6.2 ± 0.3
Over 60	7	5.4 ± 0.7	0	
Overall	453	6.15 ± 0.1	115	4.33 ± 0.2

4. Discussion

Prior to the 1980s most assessments of normal populations without industrial exposure throughout the world showed average blood lead levels between 7 (Peru) and 26 $\mu\text{g/dL}$ (Finland) (Goldwater and Hoover, 1967), or even lower levels in non-industrial nations, of < 1 $\mu\text{g/dL}$ (Hecker *et al.*, 1974) or 3–6 $\mu\text{g/dL}$ (Piomelli *et al.*, 1980; Poole and Smythe, 1980). In the United States, average lead levels in the general populations ranged from 9–17 $\mu\text{g/dL}$ (Mahaffey *et al.*, 1982; Annest *et al.*, 1983). Lead levels in exposed populations are considerably higher: often in excess of 50 $\mu\text{g/dL}$, ranging up to 400 $\mu\text{g/dL}$ (Vacca, 1986). The two groups of exposed subjects which have been studied extensively are children and workers suffering occupational exposure. Some more esoteric exposures include consumption of

home-made whiskey (moonshine) (Havelda *et al.*, 1980), and the application of lead-containing cosmetics in India or traditional Chinese medications (Lightfoote *et al.*, 1977).

Some of the highest occupational exposures reflected in mean blood lead have been found in acetylene torch workers (99 $\mu\text{g/dL}$, Sweden) (Cramer, 1974), smelter workers (93 $\mu\text{g/dL}$, Brazil) (Mendes, 1977), battery factory casters (81 $\mu\text{g/dL}$, Sudan) (Karim, 1986), auto traffic police (68 $\mu\text{g/dL}$, Egypt) (Aahmed, 1987), and bridge repair workers (Mehta *et al.*, 1990). High average lead levels are obtained when only highly exposed or ill patients are examined (Vacca, 1986). Most studies of occupationally exposed workers show average lead levels above 30 $\mu\text{g/dL}$, probably because workers are not studied unless there is evidence of excessive exposure.

The lead levels found in the present study generally averaged below 7 $\mu\text{g/dL}$ for all years and subsets (gender, age cohorts). Thus the hazardous waste workers examined in this study show evidence of low exposure, and are probably representative of an unexposed population. Even as an unexposed group, these levels are lower than those obtained in previous studies. Levels of 8 and 9 $\mu\text{g/dL}$ were reported for some segments of the United States' population (Annest *et al.*, 1983), and in West Germany (Brockhaus, 1983). Brockhaus *et al.* (1988) found geometric mean values between 5.5 $\mu\text{g/dL}$ (rural) and 7 $\mu\text{g/dL}$ (urban) for German children tested between 1982 and 1986. Watanabe *et al.* (1985) reported lower average lead levels of 3–5 $\mu\text{g/dL}$ for Japanese farmers. Elwood (1983) showed a 30% drop in blood lead in Welsh women from 1972 to 1982, with no change in lead in gasoline; their urban controls showed no increase of blood lead with age, and a decline after age 70. The low levels we found may represent a continuation of the blood lead level declines reported for the United States from 1970–1980 By Billick *et al.* (1979) and Annest (Annest *et al.*, 1983; Annest and Mahaffey, 1984). From 1976–1980, however, only 22% of people in the NHANES II had lead levels under 10 $\mu\text{g/dL}$, while 1.9% had lead levels over 30 $\mu\text{g/dL}$ (Mahaffey *et al.*, 1982). In our study 93% had levels below 10 $\mu\text{g/dL}$ and only 0.3% exceeded 20 $\mu\text{g/dL}$ (max = 20.7).

Information on gender differences in blood lead levels is contradictory. Several studies found no sex affect (Butt *et al.*, 1964; Gittleman and Eskenazi, 1983) while others reported higher blood levels in females (Haar, 1979) or in males (Watanabe, 1985; Annest, 1984; Mahaffey, 1982; Brockhaus, 1983; Fell, 1984). Brockhaus *et al.* (1988) summarized 14 studies, all of which showed higher blood levels in boys, while girls had higher levels in teeth and urine. In our study, blood levels in males were higher than for females, and ours is the second study of unexposed populations with very low levels where there was a sex difference. Watanabe *et al.* (1985) reported that female farmers (3 $\mu\text{g/dL}$) had lower levels than male farmers (5 $\mu\text{g/dL}$) in Japan. This is also consistent with the NHANES results (Mahaffey *et al.*, 1982; Annest *et al.*, 1983).

Likewise, age differences are often contradictory, but a picture emerges in

studies with large numbers of subjects (Annest and Mahaffey, 1984). Blood lead levels peak in 2–4 year olds (Annest and Mahaffey, 1984; Billick, 1979), then decrease throughout childhood (Annest and Mahaffey, 1984; Gittelman, 1983; Haar, 1979), remain stable (Ito, 1987) or slightly increase (Annest and Mahaffey, 1984) in young adults, and then increase steadily with age in adults (Annest and Mahaffey, 1984; Mahaffey *et al.*, 1982; Budnick, 1986), stabilizing beyond age 50. Sarter and Rondia (1980) found a continual increase from < 20 to > 70 years of age in an exposed population. In the present study, blood lead levels increased from 20–50 years, and decreased in the very small sample above age 60, consistent with earlier studies (Annest and Mahaffey, 1984). Although our over-60 group is small, the apparent decrease is interesting because these were active workers, not retirees, as was the case in previous studies.

Although the workforce as a whole showed no elevation of lead level, there was a slight increase with years of exposure as hazardous waste workers. Our statistical analysis did not show a significant effect of years of exposure, and we suggest that the increase reflects the overall age effect.

We conclude that this population has not had any significant occupational exposure to lead, and that lead levels reflect a New Jersey population with average blood lead levels below 6 $\mu\text{g}/\text{dL}$. We found no secular trend in the 1984–1987 period. In 1987, as a result of this study, we recommended the termination of routine biomonitoring for metals, thereby terminating the study itself (Gochfeld, 1990). With increasing concern over health effects of lower level lead exposure (ATSDR, 1988), it becomes increasingly important to understand the distribution of blood lead levels in different populations.

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