KELLOGG, IDAHO

PUBLISHED "WORK SUMMARY" MONOGRAPH, JANUARY 1976

J-5117

INCREASED LEAD ABSORPTION WITH ANEMIA AND SLOWED NERVE CONDUCTION IN CHILDREN NEAR A LEAD SMELTER

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cial status, or duration of
nalysis was undertaken by
ho had been selected for
ıe end of medical screening
ın, only 34 of the original
excluded any pair in which
r a change in his blood lead
vice versa). To augment this
data, we therefore chose
children who had no evident
l not changed, but who had
ıad been dropped from the
21 such pairs were formed.
lead and FEP levels, but

ısked whether their children
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level of activity according
h of six situations was
y) to 4 (severely hyperactive),
y, parents were asked to
rage, above average, or below
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dren living near a smelter
children in both groups. No
e groups in the results of
conduction velocity in the

55 control children was 54.2 m/sec, while mean velocity in the lead absorption group was 51.6 m/sec (Table 6).* This difference was statistically significant ($t = 2.58$, $p < 0.01$ by matched-pair one-tailed t-test).

Discussion

Increased absorption of lead, defined as a blood lead level ≥ 40 ug/100ml[17], was found in 98.8% of children living within 1.6 kilometers of the north Idaho smelter. To our knowledge, this is the highest prevalence of increased lead uptake ever recorded among children in a community. By contrast, only 1 of 89 children in the rural control area had levels ≥ 40 ug/100ml (mean 21.9 ug/100ml). This latter distribution was almost identical to that noted in previous studies of children with no known abnormal exposures to lead[27,28]. Proximity to the smelter was found here to explain 54.9% of the total variation in blood lead values.

Close correlations were found between blood lead levels and childrens' exposure to lead in air, soil, and dust; the closest correlation was with air. That finding might suggest that inhalation was the principal pathway of lead uptake near the Idaho smelter, and air lead levels near the smelter were certainly sufficient to have produced transpulmonary absorption. However, air lead exposure was computed only as a function of distance from the smelter and thus variations in individual exposure may have been eclipsed, and the correlation artefactually strengthened.

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* Nerve conduction data were also analyzed in the 34 original pairs (excluding the 21 "recombinants"). Results were 54.8 m/sec in the control group ($\pm$ S.E. 0.87) and 52.8 m/sec in the lead absorption group ($\pm$ S.E. 0.85). In these original groups, the difference in results was of borderline significance ($t = 1.69$, $p \approx 0.05$ by one-tailed, matched-pair, t-test).

Also, inhalation alone cannot have accounted for the disproportionate elevations in lead levels seen among the younger, poorer, and more highly oral children. Thus, it must be inferred that ingestion of lead particulates, often perhaps inadvertently[29], was a second important pathway of lead intake. In sum, then, these data indicate that the emissions of the Idaho smelter were the principal source of lead contamination in Shoshone County, and that inhalation and ingestion of lead particulates emitted by the smelter caused the widespread increased absorption of lead which was observed in children living in communities as far as 32 kilometers from the smelter.

The studies into the health effects of lead absorption which were conducted among children living near the Idaho smelter showed that FEP elevations and anemia were the principal hematologic consequences of lead uptake (Figure 3). These findings did not differ from those reported previously in children with increased lead intake from other sources[1]. A clear dose-response relationship was found between the severity of the hematologic abnormalities and the degree of lead absorption. There was no evidence for a threshold of effect.

The neurologic studies provided mild evidence for an association between lead intake and slowing of motor nerve conduction velocity. Although none of the children included in this analysis had clinical neurologic disease or frankly pathologic conduction velocities, there was observed in them a statistically significant dose-effect relation between conduction velocity and blood lead level (Figure 4). No threshold was evident in this effect. Conduction velocities in the control children were similar to those seen in apparently healthy children from previous studies[25]. The findings here are consistent with those among adults in which abnormal slowing of nerve conduction was found in relation to occupational lead exposure[31-33]. They are consistent also with a growing body of data

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children was not investigat

TABLE 1. Airborne Lead Leve
Shoshone County,

Year	W
1971	
1972	
1973	
1974 (Jan-Mar)	

* Arithmetic mean of 3 sam

which indicates that a variety of subtle abnormalities in neurologic and psychologic function may be found in children with absorption of lead from smelters[4,14,33] and from other sources[34-36]. It may be suspected that inhibition of sulfhydryl-containing enzymes in neurons and in red blood cells was the common mechanism underlying the neurologic and hematologic changes noted here[37]. The extent to which absorption of elements emitted by the smelter other than lead, such as cadmium, zinc, arsenic, mercury, or antimony, may have influenced these effects in the Idaho children was not investigated.

TABLE 1. Airborne Lead Levels (ug/m^3 air) by Distance from Smelter, Shoshone County, Idaho - 1971-1974

Year	Within 3.2 Kilometers*	55 Kilometers
1971	3.9	< 1.0
1972	8.6	< 1.0
1973	16.1	< 1.0
1974 (Jan-Mar)	13.2	< 1.0

* Arithmetic mean of 3 sampling sites at 1.4, 2.9, and 3.2 kilometers.

Table 2. Distribution of Blood Lead Levels in 1-9 Year-old Children, Shoshone County, Idaho - August, 1974

	SURVEY AREAS							
Blood Lead Range (ug/100ml)	1 (1.6 km)	2 (1.6-4.0 km)	3 (4.0-10.0 km)	4 (10.0-24.0 km)	5 (24.0-32.0 km)	Geologic Control	Rural Control (72 km)	TOTAL
20	0	1 (0.5)	1 (0.5)	4 (2.5)	12 (6.2)	1 (2.6)	34 (38.2)	53 (5.1)
20-39	2 (1.2%)	47 (23.6)	139 (72.8)	124 (76.1)	163 (84.0)	34 (87.2)	54 (60.7)	563 (53.8)
40-59	59 (34.3%)	109 (54.8)	50 (26.2)	33 (20.2)	16 (8.2)	4 (10.2)	1 (1.1)	272 (26.0)
60-79	73 (42.4%)	39 (19.6)	1 (0.5)	2 (1.2)	3 (1.5)			118 (11.3)
80-99	25 (14.5%)	3 (1.5)						28 (2.7)
100-164	13 (7.6%)							13 (1.2)
Total	172	199	191	163	194	39	89	1047
Mean Level	68.13	49.10	34.70	33.35	28.88	30.64	21.89	40.45
S.D.	19.92	13.20	8.18	8.60	8.40	6.63	6.20	18.79

Table 3. Lead content (ppm) in samples of interior and exterior dust and surface soil, and lead concentration by dry weight (%) in interior and experior paint. Shoshone County, Idaho - August, 1974

LEAD CONTENT

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Fig. 1 Airborne Lead Concentrations (Suspended Particulates) by Distance and Direction from Smelter, Shoshone County, Idaho, July 1974*

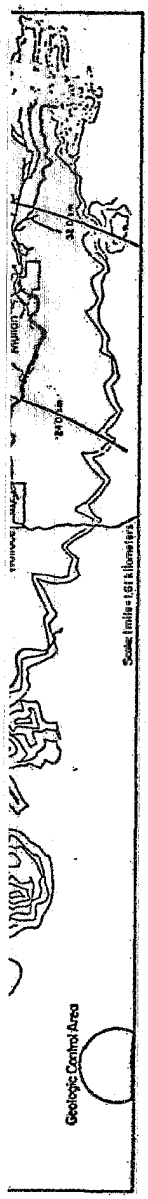


Fig. 3 FREE ERYTHROCYTE PROTOPORPHYRIN (FEP) VERSUS BLOOD LEAD LEVEL, SHOSHONE COUNTY, IDAHO, AUGUST 1974

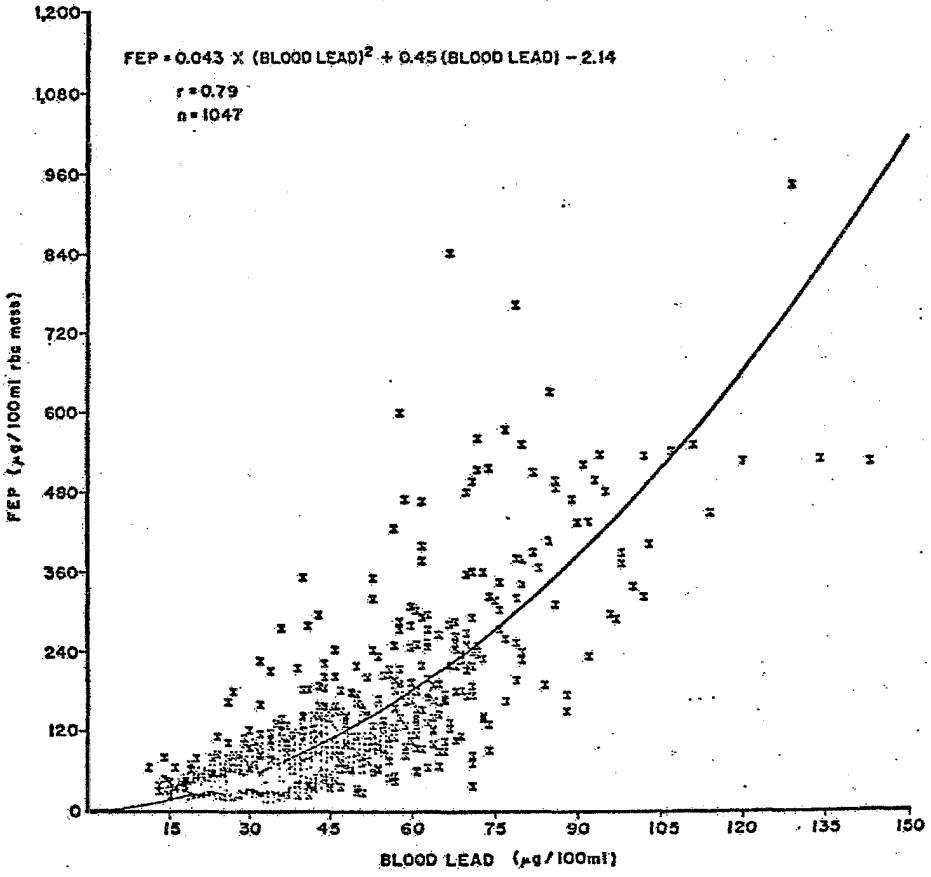
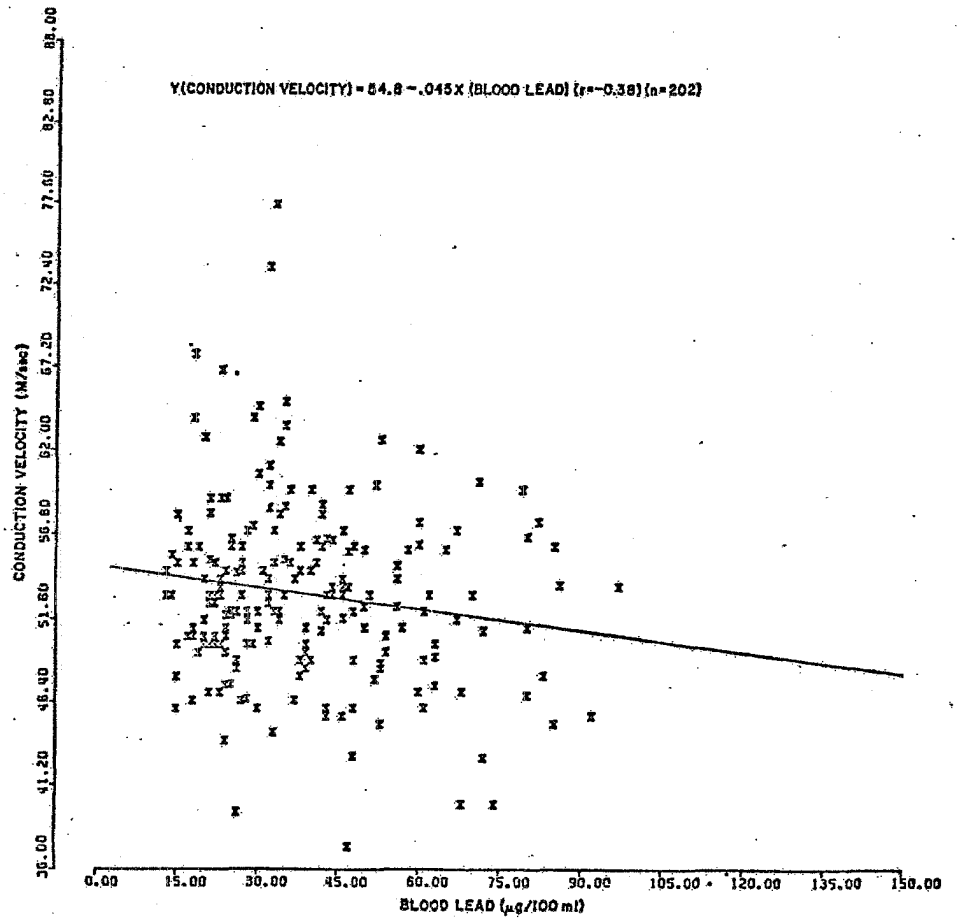


Fig. 4 PERONEAL NERVE CONDUCTION VELOCITY VERSUS BLOOD LEAD LEVEL, IDAHO, 1974



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A Discussion Concerning the Significance of Results for Children Tested in The Shoshone Project

P. S. Gartside, Asst. Professor, Division of Biostatistics, Dept. of Environmental Health, University of Cincinnati Medical Center

R. K. Panke, M.D., Member, Technical Steering Committee, Shoshone Project

Numerous data have been accumulated on a statistical sample of children who were tested in the Shoshone Lead Project. In particular, we wish to review the neurological studies by Landrigan, et al, and the intelligence testing by Gregory, et al.

The data compiled by the Landrigan and Gregory groups, if put in proper context, will add useful knowledge to the literature. However, it is important for the reader of this material to be aware that the matched pair technique in such a situation is not without pitfalls. Age differences, sex differences and SES point discrepancies in favor of the high Pb group could, in fact, explain many of the small differences found in the Landrigan data. As shown in Table I, other matched pairing and statistical analyses have been performed on these same two groups of children and little or no difference was found.

In particular, we would like to point out that nerve conduction velocities using Gregory's 50 more closely matched pairs were analyzed and no statistical significance was found (see Table II; $p = 0.11$ by one-tailed t-test). Furthermore, examination of nerve conduction velocities of all the children measured by Landrigan, et al, revealed that only six children had nerve conduction velocities below 45 meter/sec (nerve conduction velocity range = 37.5 m/sec – 43.0 m/sec, blood Pb range in Aug., '74 = 47 mg% – 74 mg%). Eight months after the initial nerve conduction velocity measurements, all six children who tested below 45 m/sec were retested using similar but different techniques at the Department of

Child Neurology, University of Washington School of Medicine. All six children were found to have nerve conduction velocities within the normal range* (NCV range = 45.1 m/sec - 72.0 m/sec, blood lead range 35 mg% - 62 mg%), thereby indicating reversible nerve conduction velocity depression, or possibly technique differences or recording error in data collection. Further collection of data is planned by the Department of Health and Welfare, State of Idaho.

Data are difficult to collect and analyze in a clinical industrial setting because of the large number of concomitant variables. Multiple regression analysis should be used wherever possible in future studies of this population in order to make adjustments for the effect of significant concomitant variables such as age; sex; cleanliness of the home; location of residence; and occupation, education, and intelligence status of the parents.

Finally, because of the inconsistent results of data anlysis as shown above, we feel that a significant relationship between blood lead and nerve conduction velocity has not been established. It should also be reiterated that there were no clinically anemic children. While Landrigan, et al, might correctly consider a hematocrit of less than 33% to be abnormal, many clinicians feel that a definite threshold value cannot be defined. It is more important clinically to consider changes or depression of hematocrit, since individual differences often show values below 33% in completely healthy children. The subjective and objective impressions of local medical practitioners in the Kellogg area reveal that there does not appear to be wide-spread anemia or hematocrit depression; however, these impressions should be confirmed by further prospective studies.

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* Gamstorp, I. Normal conduction velocity of ulnar, median, and peroneal nerves in infants, children and adolescents. Acta Ped, Suppl., 146; 68, 1963.

TABLE I

COMPARISON OF MATCHED PAIRS AND REGRESSION ANALYSIS SUBJECTS IN SHOSHONE COUNTY

Matched Pairs	Subjects For Regression Analysis	Maximum Age Difference Between Pb Study Group & Control Group	Mean Age Difference Between Pb Study Group & Control Group	Set of Subjects				Maximum Point Difference on Socio-on Socio-Economic Index	Blood Pb/NCV P Value 1 Tailed t Matched Pairs	Blood Pb/NCV P Value Regression Analysis	Blood Pb/IQ P Value 2 Way Analysis of Variance	Blood Pb/IQ 2 tailed t Test Regression Analysis
				Study Group		Control Group						
				N	F	M	F					
				10	12	10	16					
			7 [illegible]						+ - 1.60		F = .307	

146 6 children were found to have nerve conduction velocities within the normal range* (NCV range = 45.1 m/sec - 72.0 m/sec, blood lead range 35 mg% - 62 mg%), thereby indicating reversible nerve conduction velocity depression, or possibly technique differences or recording error in data collection. Further collection of data is planned by the Department of Health and Welfare, State of Idaho.

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34 Landrigan		11+ months	7.50 vs. 7.42	18	16	18	16		t = 1.69 P = ±0.05 Landrigan P = 0.05 Gartside		F = .397 df = 1/66 P = .538 Gregory	
20 Gregory (Recombinant)		11+ months							p = 0.05 Gartside			
21 Landrigan (Recombinant)		11+ months									F = .213 df = 1/106 P = .650 Gregory	
54 Gregory		11+ months	7.32 vs. 7.28	25	29	25	29					
55 Landrigan		11+ months	6.65(±1.32) vs. 6.62(±1.37)	26	29	26	29	16 pt. on a 0-66 pt. continuum	t = 2.58 P < 0.01 Landrigan			
50 Gregory		4 months	7.29 vs. 7.37	24	26	24	26	8 pt. on a 11-77 pt. continuum	52.4 vs. 53.9 p = 0.11 Gartside		Pb Group scored approx. 5 IQ pts lower than controls - P<.001; P=.038; P=.040; P=.037 Gregory	
	202 Landrigan									r = -0.38 t = -2.12 P 0.02 Landrigan		
	168 Gregory											

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TABLE I

COMPARISON OF MATCHED PAIRS AND REGRESSION ANALYSIS SUBJECTS
IN SHOSHONE COUNTY

Matched Pairs	Subjects For Regression Analysis	Maximum Age Difference Between Pb Study Group & Control Group	Mean Age Difference Between Pb Study Group & Control Group	Set of Subjects Study Group M	Study Group F	Control Group M	Control Group F	Maximum Point Difference on Socio-Economic Index	Blood Pb/NCV P Value 1 Tailed t Matched Pairs	Blood Pb/NCV P Value Regression Analysis	Blood Pb/IQ P Value 2 Way Analysis of Variance	Blood Pb/IQ 2 tailed t Test Regression Analysis
34 Landrigan		11+ months	7.50 vs. 7.42	18	16	18	16					
20 Gregory (Recombinant)		11+ months										
21 Landrigan (Recombinant)		11+ months							P = 0.05 Gartside			
54 Gregory		11+ months	7.32 vs. 7.28	25	29	25	29					
55 Landrigan		11+ months	6.65(±1.32) vs. 6.62(±1.37)	26	29	26	29	16 pt. on a 0-66 pt. continuum	t = 2.58 P < 0.01 Landrigan		$\frac{F}{df} = \frac{.213}{1/106}$ $\frac{}{P} = .650$ Gregory	
50 Gregory		4 months	7.29 vs. 7.37	24	26	24	26	8 pt. on a 11-77 pt. continuum	52.4 vs. 53.9 P = 0.11 Gartside			Pb Group scored approx. 5 IQ pts lower than controls - P <.001; p=.038; P=.040; P=.037 Gregory
202 Landrigan										r = -0.38 t = -2.12 P 0.02 Landrigan		
168 Gregory												P <.02 Gregory