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Soil Lead–Blood Lead Relationship Among Boston Children

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Lead contaminated soil has consistently been found to contribute significantly to children's risk of blood lead elevation. For example, the soil lead levels around the homes of 37 poisoned children in Charleston, South Carolina, were significantly greater than the levels at 170 randomly selected sites in the city (Fairey and Gray, 1970). Samples taken from homes of poisoned children were also significantly more likely to have lead concentrations greater than 500 ug/g (the 75th percentile). Eighty-two 2 and 3 year old English children were classified by soil lead category: <1000, 1000-10,000, and > 10,000 ppm. The mean blood lead levels in the three groups were 21, 24, and 29 ug/dl respectively (Barltrop, 1975). Neri et al (1978) grouped children living near a smelter in Trail, British Columbia by residential areas with differing soil lead levels. As mean soil lead rose from 225 to 1800 ppm, mean blood lead levels rose from 17 to 30 ug/dl. A study of 377 generally lower class children in New Haven, Connecticut, found soil lead, paint lead and paint quality, and season to be significant predictors of blood lead level (Stark et al, 1982).

An association between soil lead and blood lead has been reported for children in Holland (Brunekreef et al 1983), Rochester New York (Charney et al , 1980), Omaha, Nebraska (Angle and McIntire, 1982), Christchurch, New Zealand (Shellshear, 1973), and Kellogg, Idaho (Yankel et al, 1977). Table 1 provides a compilation of the published estimated slopes of the relationship between soil and blood lead levels from these studies. Note the units of blood lead are ug/ L.

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Table 1: Compilation of soil - blood lead relationships

Study Location	Range of Soil Lead Values (ug/g)	Sample Size	Estimated Slope (ug/L / ug/kg)
Trail, B.C.	225 - 1,800	87	76 (1-3 yrs)
		103	46 (2-3 yrs)
Omaha, NE	16 - 4,792	1075	68
New Haven,CT	30 - 7,000	153	22 (0-1 yrs)
		334	20 (2-3 yrs)
Charleston, SC	9 - 7,890	194	15
Kellogg, ID	50 - 24,600	860	11
England	420 - 13,969	82	6
Boston, MA	7 - 13,240	195	8.9(st err=2.2)
this study			
less mouthing		70	6
more mouthing		60	16

adapted from US EPA, 1986.

We report here on the connection between soil and blood lead levels among a group of Boston children in order to document this pattern of association in a specific setting which may be analogous to other situations of interest.

MATERIALS AND METHODS

The base population consisted of 11,837 consecutive births at the Boston Hospital for Women between April 1979 and April 1981. Babies were eligible if their umbilical cord blood lead levels were in the highest, middle or lowest decile, if their address was within Route 128, if their consent forms were in English, and if the child had no medical condition which required a hospital stay of more than two weeks. By these criteria, 589 infants were eligible, but some of the families intended to move, some were unreachable, and some refused participation. By April 1981, 249 infants were enrolled.

The participating mothers had a mean age of 29 years and were well educated (15 years mean schooling). Eighty-seven percent were white, 68 % were employed at

the time of conception, and 77% had some childbirth preparation. The annual attrition rate was less than 10% ; a total of 202 children completed the 2 year follow-up. In general these children represent healthy products of uneventful pregnancies and live in areas that placed them at relatively low risk for lead poisoning.

In addition to umbilical cord blood lead measurements (Rabinowitz and Needleman 1982), capillary blood was sampled at 6,12,18 and 24 months of age by trained technicians after extensive skin preparation with alcohol scrubs, and the samples were analyzed in duplicate or triplicate with a model 3010 anodic stripping voltammeter (ESA, Bedford, Massachusetts). The average difference between the duplicates was 1 ug/dl and was 3.5 or greater in only 10% of the pairs. Further details of the blood lead measurements and the lack of stability of a child's blood lead level have appeared elsewhere (Rabinowitz et al 1984).

Environmental samples were collected at 1,6,18, and 24 months of age. Dust samples were collected by wiping a surface (living room floor, furniture top, and windowsill) with a preweighed filter paper using a plastic frame to standardize the area. These were then soaked with perchloric acid (pH 1.8) to extract the leachable lead, and the concentration of lead in this liquid was measured by flameless atomic absorption spectrophotometry. Twelve-hour indoor aerosol samples, paint, and drinking water were also collected and measured for lead (Rabinowitz et al , 1984, 1985). Soil samples were collected at the 18 and 24 month visits from a site further than 3 m from any road or structure. Preference was given to any obvious play area. The top centimeter of soil from 3 points, 1 meter apart was collected in a glass jar. Any object greater than 1 cm was discarded, the sample oven-dried at 80° C, and blended for uniformity. The 10% nitric acid acid-soluble lead from 5 g portions was measured by flame atomic absorption spectrophotometry. Soil values reported here are the means of available 18 and 24 month data. Soil lead values are available for only 195 infants because samples could not be obtained from some apartment houses or when the ground was frozen.

RESULTS AND DISCUSSION

The mean lead level of the 195 soil samples was 702 ug/g with a range of 7 to 13,240. The median was 365 (90 th percentile 1548, 10 th percentile 72) ug/g. The average of 232 available postnatal blood lead levels was 61 ug/L. Figure 1 is a scatter plot showing the soil lead levels and mean of the blood lead values for

a child between the ages 6 and 24 months. The mean level of dust lead on surfaces was 83 ug/m^2, in tap water 5.0 ug/L, and 0.13 ug/m^3 in air.

There are strong correlations between blood lead levels and soil lead and dust lead levels but not between blood lead and air or water lead levels in this population. The correlation of soil lead with blood lead is stronger at ages 18 through 24 months than in the first year. Also, soil and floor dust lead levels are strongly correlated ($r=0.43$, $p < 0.0001$.), as are floor dust lead and table top dust lead ($r=0.58$, $p<0.0001$).

Because of these intercorrelations we calculated the correlations between blood lead and both soil and dust lead after adjusting for the other. These calculations were performed on a restricted data set that included only 187 subjects for whom complete data (soil, dust and blood) were available. A composite dust lead value was calculated for each home by averaging the repeated dust lead measurments; similarly all the post-natal blood lead values for a child were averaged. The correlation coefficient between soil and blood lead levels was 0.30 ($p<.0001$). After adjusting for dust lead, it fell to 0.21 ($p<.005$). The correlation between dust and blood lead was 0.26 ($p<.0005$), and after adjusting for soil lead it fell to 0.15 ($p<.03$). These correlations were still very significant despite the attenuation with adjustment.

Table 2. Spearman Correlation Coefficients Between Blood Lead at Different Ages and Various Environmental Measures.

Environmental Measure	Age of child (months)			
	6	12	18	24
Soil Lead	0.09	0.17	0.37**	0.30**
Floor Dust Lead	0.27**	0.29**	0.49**	0.43**
Surface Dust Lead	0.25*	0.33**	0.39**	0.34**
Tap Water Lead	0.11	0.00	0.12	0.14
Indoor Air Lead	0.00	-0.02	0.17	0.06

* $p < 0.01$ ** $p < 0.0001$

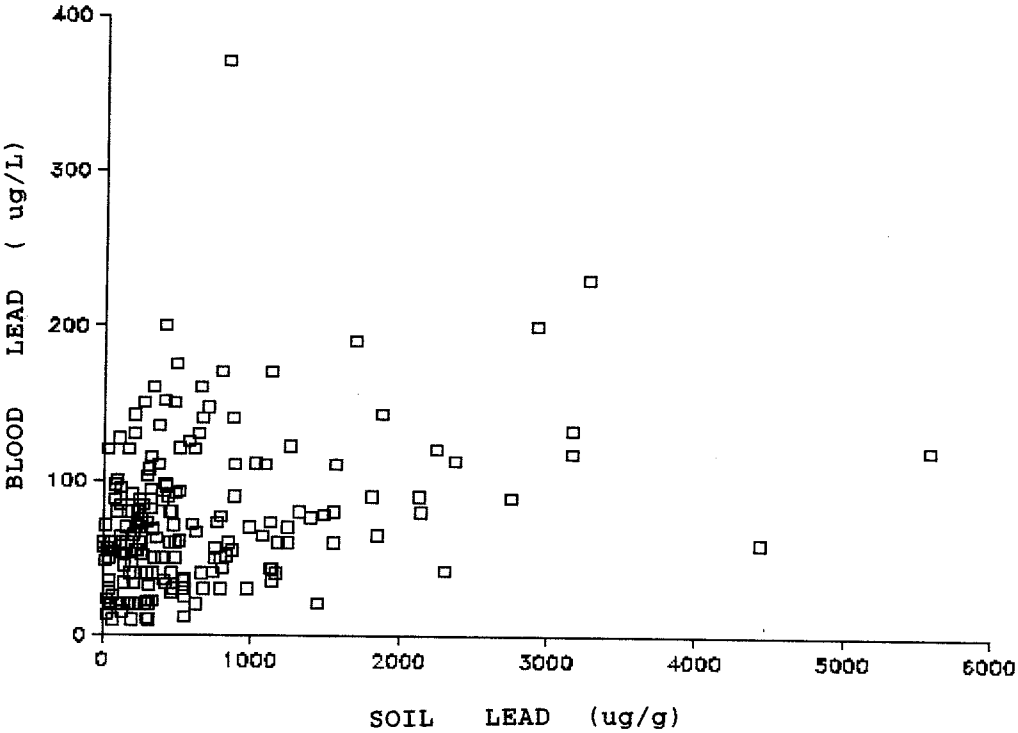

Figure 1: Observed Values of Soil Lead and Blood Lead. Each point is the mean of available 6,12 18, and 24 month blood lead values and the mean soil lead taken from their home. Of the 195 total pairs, one outlying point (soil 13,240; blood 90) is not plotted.

We calculated the slope of the best linear fit between soil lead and blood lead levels, unadjusted for any other variable. Using as the blood lead the mean of samples from ages 6 to 24 months, we obtained a slope of 8.9 (std err 2.2) ug/L / ug/kg and an intercept of 65 (std err 3) ug/L with an r-squared of 0.11 for 195 subjects.

In order to estimate the influence of a child's mouthing activity on the relationship between soil lead and blood lead, children were classified by a composite mouthing score. Briefly, 8 judgments of mouthing activity at 18 and 24 months of age, from the mother and testing psychologist, were reduced by principal component analysis to 2 composite variables. One pertains to the child's mouthing of fingers and hands, and the second to mouthing of toys and other objects. The finger and hand mouthing composite, which correlated best with blood lead levels, is used here to

classify the children into three groups of equal size (Bellinger et al 1986). Among the 60 who displayed the most mouthing activity, the calculated slope of the regression of blood lead on soil lead is 16(std err 5). Among the 70 children who mouthed the least, the slope of 5.7 (std err 2), is significantly lower ($<p<0.001$). Those children judged to mouth the most have 2 to 3 times the response of those who mouth the least to a given soil lead level.

The magnitude of the slope in this population, about 10 ug/L per 1000 ug/g , may not be directly applicable to populations at higher risk for overt lead poisoning. In general, the population we studied did not live in crowded conditions which limit children's play areas very close, within 3 meters, to the homes. These areas are typically more contaminated with lead. Other factors may also contribute to a steeper slope among such high risk children, including the presence of additional high dose sources such as deterioration lead paint and nutritional deficiencies that may increase the efficiency of lead absorption. The estimated slope in the children we studied applies to the more general case of children at lower risk of lead poisoning or blood lead elevations.

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