Lead Exposure and Growth in the Early Preschool Child: A Follow-up Report from the Cincinnati Lead Study

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ABSTRACT. This report is a follow-up of an earlier study of the effects of low to moderate prenatal and postnatal lead exposure on children's growth in stature. Two hundred thirty-five subjects were assessed every 3 months for lead exposure (blood lead level) and stature (recumbent length) up to 33 months of age. Fetal lead exposure was indexed by maternal blood lead level during pregnancy. The adverse effects of lead on growth during the first year of life were reported previously. This analysis covers essentially the second and third years of life. The results indicate that mean blood lead level during this period was negatively associated with attained height at 33 months of age ($P = .002$). This association was, however, evidenced only among those children who had mean blood lead levels greater than the cohort median (≥ 10.77 μg/dL) during the 3- to 15-month interval. The results also suggest that the effect of lead exposure (both in utero as well as during the first year of life) are transient provided that subsequent exposure to lead is not excessive. It appears that maintaining an average blood lead level of 25 μg/dL or more during the second and third year of life was detrimental to the child's attained stature at 33 months of age. Approximately 15% of this cohort experienced these levels of lead exposure. Continued follow-up of this cohort will reveal whether these lead-related deficits persist and whether they continue to be dependent on the level of exposure in an earlier period. *Pediatrics* 1991;88:886–892; *lead toxicity, growth, stature, infant, follow-up.*

In an earlier paper,[1] we reported an apparent interactive effect of prenatal and postnatal lead exposure on infants' growth in stature through the first 15 months of life. It was found that postnatal lead exposure, as indexed by change in average blood lead concentration (PbB) over the interval from 3 to 15 months of life, was negatively associated with infant growth rates during the same period. This effect, however, was exhibited only by those infants born to mothers with above average (>7.7 μg/dL) prenatal PbB levels. This provided support for our initial hypothesis that "high" early postnatal lead exposure would have an adverse effect on an infant's growth in stature provided that a relatively "high" in utero exposure to lead was experienced (see Shukla et al[1] for a review of the pertinent literature that led to this hypothesis). The present paper is a follow-up of the same cohort. This follow-up analysis covers the period of 18 months to 33 months of age. The stature measurements for the infants in the study were obtained in their recumbent position until the age of 3 years whereupon standing height was obtained. To avoid introducing a systematic source of bias in the growth data due to two different stature-measuring techniques, we analyze and report the findings on the data up to 33 months of child's age.

Our objective in this follow-up analysis was to assess the independent association of lead exposure with indices of stature during the period 18 to 33 months of age. We considered lead exposure histories both in utero as well as during early (3 to 15 months) and later postnatal periods (ie, 18 to 33 months). The following questions were examined: (1) Are lead-related deficits in growth still evident during the 18- to 33-month interval? (2) What combination of prenatal and/or postnatal lead exposure is most strongly associated with lead-related growth deficits? (3) Is there any evidence of an exposure threshold for this effect?

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MATERIALS AND METHODS

Subjects

The general design of the Cincinnati Lead Study has been reported elsewhere.[2] Since this study is a prospective follow-up, we continue to examine the same subjects as in the earlier report.[1] For details of inclusion and exclusion criteria for the subjects, the reader is referred to the earlier papers.[1,2] Of all the subjects, 25 had three or fewer length measurements during the 18- to 33-month period and therefore had inadequate growth data (ie, lengths at both 18 months and 33 months were missing). Consequently, they were excluded from further analyses. Thus, of the original 260 subjects, 235 were available for the present study. A comparison was made for several demographic variables between the excluded subjects (n = 25) and the remaining sample (n = 235). The excluded subjects were very similar to those retained in the analysis except for cigarette consumption (packs per day) by mothers during pregnancy (1.12 [excluded subjects] vs 0.71 [included subjects], $P = .02$).

Lead Exposure Indices

Details of the PbB assessments are given elsewhere.[1] Briefly, in utero lead exposure was indexed by maternal PbB measured from samples collected by venipuncture at the first prenatal visit. Postnatal PbB was measured at 10 days of age (corrected for gestational age), at 3 months of age, and every 3 months thereafter. Details of the laboratory's quality assurance and quality control programs have been reported elsewhere.[3] All PbB measurements were adjusted to an average hematocrit for developmental age and transformed to their natural logarithm for statistical analyses.

Longitudinal studies with frequent assessments typically have some "missed" and "mistimed" observations. Approximately 15% of the subjects needed either imputation of missing PbB data or adjustment for mistimed PbB assessments. Missing postnatal PbB levels were imputed from a weighted average of a within-subject regression of PbB on age and the cohort mean PbB at each age. Analyses were conducted with the data set that contained *both* imputed and observed PbB values and also with the data set that contained *only* observed PbB values. This was done to determine whether there were differences in the regression coefficients obtained for PbB in these two overlapping data sets. Very similar results were found in these analyses. Therefore, we report only results obtained from the larger data set, which contained both observed and imputed or adjusted PbB values.

Typical PbB profiles of exposed infants beyond 15 months of age are such that they peak somewhere around 24 months and then gradually decline. Consequently, net change in PbB (the exposure index used in the previous study) may not be an appropriate measure of the degree of lead exposure during this period. Therefore, mean PbB during 18 to 33 months was chosen as a more appropriate integrated index of lead exposure for this later developmental period. For the present follow-up analyses, we used mean PbB during 3 to 15 months as an index of postnatal exposure during that period. This was done for the sake of consistency with the 18- to 33-month measures and for ease of interpretation. The results obtained did not differ with respect to the two alternative ways of characterizing developmental lead exposure during 3 to 15 months. Mean PbB for the cohort in the 18- to 33-month interval was 17 μg/dL (range 5.7 to 53.9 μg/dL). Arithmetic mean total iron-binding capacity was 360 ± 31.2 μg/dL and hemoglobin level averaged 12 ± 0.72 g/dL, a value indicating adequate iron status for most subjects.

Index of Growth

As was done previously,[1] slopes of the least-squares regression, fitted to each subject's length measurements at 18, 21, 24, 27, 30, and 33 months, were used as growth rates. All the subjects had at least four of the six possible length measurements, including both the 18- and 33-month length assessment. Eighty-five percent of the subjects had all six measurements. There were no apparent differences between the subjects when grouped in terms of the available number of length measurements. Coefficient of determination (R^2) exceeded .80 in all the cases, indicating that the slopes of the linear regression fit were an adequate representation of the growth for this period. Table 1 gives some descriptive statistics for the sample. Rate of growth during second and third year was not only reduced by half, vis-à-vis the first year, but also the variability in the growth rates was decreased (from an SD = 0.2 to 0.1 cm/mo).

Statistical Approach

As in the previous analysis,[1] a set of candidate confounders/covariates was chosen based on their a priori probability of being related to growth rate and/or length at 33 months. Two key response variables, namely growth rate (18 to 33 months) and stature at 33 months, plus one key exposure variable, ie, mean blood lead level during 18 to 33 months (mean PbB), were examined for bivariate correlation with a host of candidate confounders/

covariates. Table 2 lists Pearson's product-moment correlation coefficients between the above-mentioned key variables and confounders/covariates.

For exploring the hypothesis that some combination of prenatal and postnatal blood lead levels affect growth during the period 18 to 33 months, we categorized past lead exposure histories (both prenatal and early postnatal) into four groups based on median splits of prenatal PbB (7.78 μg/dL) and mean PbB during 3 to 15 months (10.77 μg/dL). This resulted in four combinations (ie, low-low, low-high, high-low, and high-high) of earlier lead exposure histories. The relationships between mean PbB (18 to 33 months) and growth rate (18 to 33 months) as well as mean PbB (18 to 33 months) and length at 33 months were examined via stepwise multiple regression with particular emphasis on understanding which, if any, of the four lead history combinations were exhibiting lead-growth or lead-stature relationships.

RESULTS

The bivariate correlations between growth rate, length during 33 months, and mean PbB at 18 to 33 months, by lead exposure histories, prenatally and early postnatally (3 to 15 months), are shown in Table 3. The growth rate from 18 to 33 months was essentially uncorrelated either with the growth rate from 3 to 15 months (first column) or with height at 18 months (second column). The attained height at 33 months, however, was very strongly correlated with the attained height at 18 months (third column). The correlations of PbB vs growth (fourth column) and PbB vs length at 33 months (fifth column) were weak but consistent for two

TABLE 1. Descriptive Statistics on Study Sample (N = 235)

Variable	Mean	SD	Minimum	Maximum
Length at 18 mo, cm	81.4	2.9	73.7	91.0
Length at 33 mo, cm	93.2	3.5	85.4	105.0
Growth rate (3–15 mo), cm/mo	1.5	0.2	0.8	2.1
Growth rate (18–33 mo), cm/mo	0.8	0.1	0.4	1.2
Blood lead at 18 mo, μg/dL	17.3	9.1	2.9	62.9
Blood lead at 33 mo, μg/dL	15.9	7.8	2.9	44.4
Mean blood lead (3–15 mo), μg/dL	11.8	5.4	4.1	36.5
Mean blood lead (3–15 mo), μg/dl*	10.7	1.6	4.1	36.5
Mean blood lead (18–33 mo), μg/dL	17.1	8.0	5.7	53.9
Mean blood lead (18–33 mo), μg/dl*	15.5	1.5	5.7	53.9
Mean total iron-binding capacity (18–33 mo), μg/dL	359.4	31.2	283.0	449.3
Mean hemoglobin (18–33 mo), g/dL	12.1	0.7	10.2	14.5
HOME score†	33.0	4.8	17.0	43.0
% Chelated (diagnostic)	5.53			
% Chelated (therapeutic)	0.85			

* Geometric mean and geometric SD.

† Average of Home Observation for Measurement of the Environment scores at 12 months and 24 months of age.

TABLE 2. Correlates of Lead Exposure and Response Variables*

Variables	Mean PbB 18–33 mo	Growth Rate 18–33 mo	Length at 33 mo
Mean PbB during 3–15 mo	.78	−.02	−.03
Prenatal PbB	.32	.02	−.04
Cigarette use	.12	−.05	−.12
Child's race	−.13	.14	.12
HOME†	−.16	.01	.08
Maternal height	.02	.18	.40
Total iron-binding capacity	.02	.18	.20
Socioeconomic status	−.17	−.02	.02
Sex	−.03	.14	.04
Stature at 18 mo	−.09	−.07	.80

* Race and prenatal cigarette use were bona fide confounders. They were forced to remain in the models, irrespective of their significance levels. Correlation coefficient ≥ .11 for $P < .10$. Entries are correlation coefficients. PbB, blood lead concentration.

† Average of total Home Observation for Measurement of the Environment scores at 12 months and 24 months.

TABLE 3. Bivariate Correlations of Response and Exposure Index*

Lead Exposure History† Prenatal/Early Postnatal (3–15 mo)	Growth Rate (18–33 mo) vs Growth Rate (3–15 mo)	Growth Rate (18–33 mo) vs Length at 18 mo	Length at 33 mo vs Length at 18 mo	Growth Rate (18–33 mo) vs Mean PbB (18–33 mo)	Length at 33 mo vs Mean PbB (18–33 mo)
Low-low	.07	−.07	.83	.06	.04
(n = 77)	(.56)	(.56)	(<.0001)	(.63)	(.75)
Low-high	.24	−.09	.70	−.28	−.32
(n = 44)	(.11)	(.55)	(<.0001)	(.07)	(.04)
High-low	.01	−.35	.74	−.01	−.01
(n = 41)	(.94)	(.02)	(<.0001)	(.53)	(.54)
High-high	0	.17	.85	−.22	−.25
(n = 73)	(1.0)	(.17)	(<.0001)	(.07)	(.04)
All	.06	−.07	.79	−.06	−.11
(n = 235)	(.39)	(.30)	(<.0001)	(.36)	(.10)

* Entries in parentheses are *P* values. PbB, blood lead concentration.

† Based on median split (prenatal PbB = 7.78 μg/dL, mean PbB [3–15 mo] = 10.77 μg/dL).

lead exposure history combinations, namely low-high and high-high. This suggested a possible interaction of early postnatal lead exposure history (up to 15 months) with subsequent lead exposure (ie, 18 to 33 months) in determining the effect of PbB on either growth or length at 33 months.

Using our strategy of confounder/covariate selection, followed by a multiple regression analysis (which included two-way interactions), two reduced models (with all significant variables) were obtained, one for growth rate and the other for length at 33 months. The reduced models for both response measures indicated that mean PbB during the 3- to 15-month interval did have a statistically significant interaction with mean PbB during the 18- to 33 month interval (P = .05 for growth rate and P = .02 for length at 33 months). The model R^2 for growth rate was low (R^2 = .13), while that for length at 33 months was high (R^2 = .71).

Results from the multiple regression analysis where length at 33 months was treated as the dependent variable are shown in Table 4. There was a significant interaction (P = .025) between lead exposure during the 3- to 15-month interval and exposure during the 18- to 33-month interval which confirmed our observations based on the bivariate correlations (Table 3). The positive regression coefficient of mean PbB (3 to 15 months) is uninterpretable here because of the presence of a significant interaction in the model involving this PbB variable. The nature of this interaction is graphically displayed in Figs 1 and 2. Covariate-adjusted stature at 33 months was significantly (P = .002) and negatively related to mean PbB during the 18- to 33-month interval for subjects having high (≥10.77 μg/dL) mean PbB during the 3- to 15-month interval of life (Fig 1). On the other hand, no relationship (P = .85) was observed for subjects

TABLE 4. Multiple Regression Results for Recumbent Length at 33 Months*

Independent Variable	Coefficient	SE	*P* Value
Intercept	3.80		
Mean PbB (3–15 mo)	5.60	2.22	.01
Mean PbB (18–33 mo)	−0.13	0.57	.83
Mean PbB (3–15 mo) × mean PbB (18–33 mo)	−1.81	0.80	.025
Sex†	0.55	0.27	.05
Race‡	0.81	0.42	.06
Cigarette use during pregnancy	−0.40	0.18	.03
Mean total iron-binding capacity (18–33 mo)	0.01	0.004	.009
Maternal height (cm)	0.10	0.02	.0001
Length at 18 mo (cm)	0.84	0.05	.0001

* PbB, blood lead concentration.

† 1 = male, 2 = female.

‡ 1 = white, 2 = black.

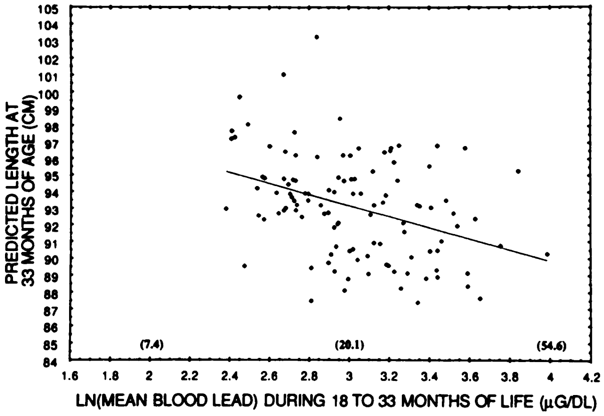

Fig 1. Influence of later postnatal lead exposure on attained height at 33 months for children with moderately elevated blood lead concentration (PbB) from 3 to 15 months of age (mean PbB ≥10.77 μg/dL).

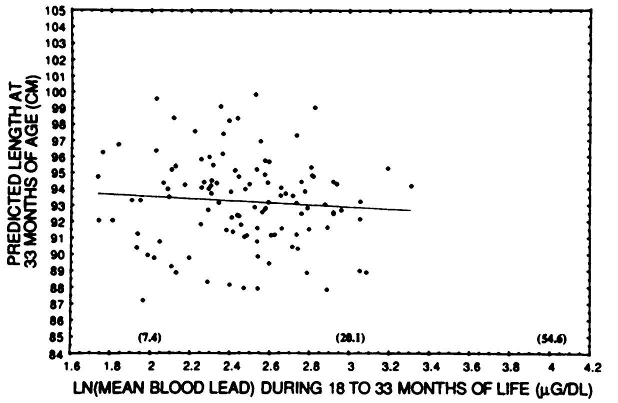

Fig 2. Influence of later postnatal lead exposure on attained height at 33 months for children with low blood lead concentration (PbB) from 3 to 15 months of age (mean PbB <10.77 µg/dL).

with "low" (<10.77 µg/dL) mean PbB for 3 to 15 months of age (Fig 2). It appears that in utero exposure to lead did not directly predict stature at 33 months. On the other hand, lead exposure from 3 to 15 months of age did interact with lead exposure during the second and third year of life and determined whether that child exhibited a negative association between lead exposure and growth.

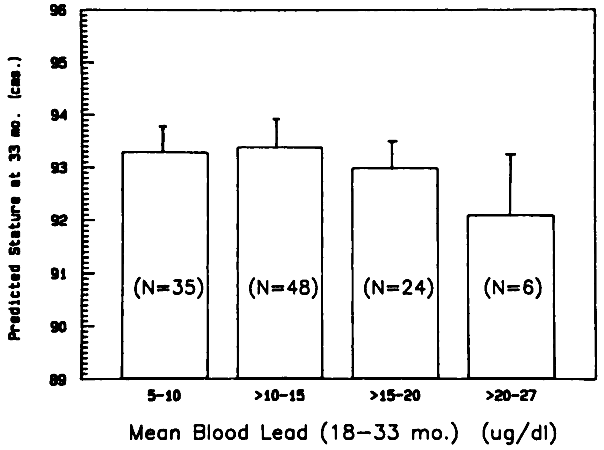

Fig 3. Dose-effect relationship between mean blood lead concentration (PbB) (18 to 33 months) and length at 33 months for children with low PbB from 3 to 15 months (mean PbB <10.77 µg/dL). Note the PbB scale is the *average* size of six quarterly PbB determinations between 18 and 33 months of age.

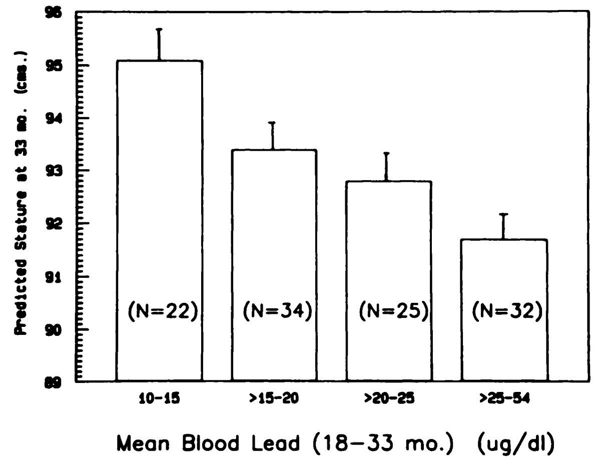

Fig 4. Dose-effect relationship between mean blood lead concentration (PbB) (18 to 33 months) and length at 33 months for children with moderately elevated PbB from 3 to 15 months (mean PbB ≥10.77 µg/dL). Note the PbB scale is the *average* of six quarterly PbB determinations between 18 and 33 months of age.

Figures 3 and 4 show the dose-effect relationship of second- and third-year PbB on the mean height at 33 months, as predicted by the regression analysis. Two features are noteworthy. One is that the "low" first-year PbB group (Fig 3) exhibited almost no change in average length for increasing levels of mean PbB (18 to 33 months), except for a small number of subjects (n = 6) with mean PbB of 20 to 27 µg/dL. The "high" first-year PbB group (Fig 4) exhibited a significant lead-related decrement in mean stature at 33 months. In fact, subjects with a mean PbB (18 to 33 months) of ≥25 µg/dL had a mean predicted length at 33 months of about 1.5 cm below the cohort mean of 93.2 cm. Those children with "low" PbB during the second and third year (mean PbB of 10 to 15 µg/dL) exhibited mean stature well above the cohort mean, suggesting that high levels of lead exposure in the first year suppressed growth and thus did not allow them to attain their full potential for growth in stature in that early period. Lead exposure during the second and third year of life, being relatively "low," may have allowed them to catch up[4] and even overshoot. The subjects with intermediate mean PbB during the second and third year (15 to 20 µg/dL) were perhaps experiencing two opposing effects—the catch-up and the suppression of growth. Perhaps the two impacts canceled each other. These data suggest that an average PbB of 20 to 25 µg/dL over the entire interval from 18 to 33 months of a child's life was perhaps "borderline" while an average PbB of above 25 µg/dL could certainly be considered as having a significant and measurable negative effect on height.

One would expect that complete catch-up resulting from a decrease in lead exposure would result in attainment of stature approximately that of children who had been exposed to low levels of lead throughout the period extending from fetal life through 33 months. Surprisingly, when low exposure in the 18- to 33-month interval is preceded by high exposure earlier, stature at 33 months actually exceeds that of children with persistently low exposure. Figure 5 illustrates this point. Subjects were

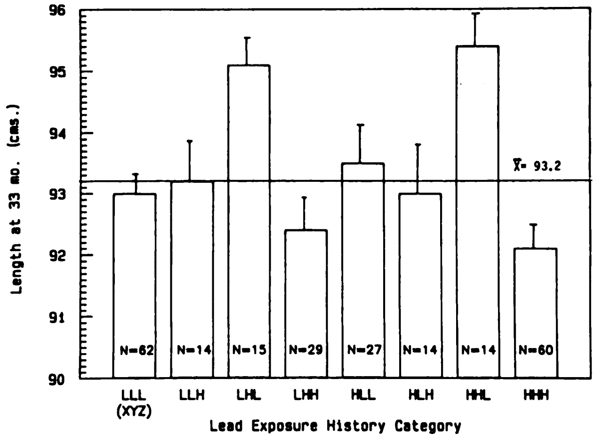

Fig 5. Model-based predicted length at 33 months by lead exposure history (XYZ). X, prenatal; Y, mean blood lead concentration (3 to 15 months); Z, mean blood lead concentration (18 to 33 months).

divided in two halves (based on median PbB of 7.7, 10.77, and 15.37 μg/dL for prenatal, 3 to 15 months, and 18 to 33 months, respectively) for each developmental period. The three periods thus provided eight ($2^3 = 8$) categories of lifetime lead exposure history. These different exposure categories yield different average statures at 33 months of age. For example, a relatively "high" in utero exposure to lead might suppress intrauterine growth and consequently result in lower than expected birth length. If the same infant continues to experience relatively elevated postnatal lead exposure, he or she would continue to exhibit depressed growth and lower than average attained stature. On the other hand, if the same infant avoids elevated postnatal lead exposure, then he or she might exhibit "catch-up"[4] and consequently would exhibit average or above-average attained stature because of overshoot. The subjects in the two lead exposure history categories with the greatest stature (ie, LHL and HHL) also tended to be those with highest rates of growth during 18 to 33 months. These children averaged 0.85 cm/mo compared with 0.76 cm/mo for the entire cohort and 0.75 cm/mo for the children in the consistently low lead category (LLL), in which the lead impact on growth was minimal. We are not aware of any other situation in which previously depressed growth results in a rate of growth that overshoots the norm for the population in question.

The amount of cigarette use by mothers during pregnancy was still independently associated with child's length at 33 months of age ($P = .03$). The other striking finding from the regression analysis is the relationship of a child's iron status (total iron-binding capacity) with stature ($P = .009$). The implications of these two findings are the subject of our future investigation.

DISCUSSION

Our earlier work[1] revealed that lead exposure is associated with deficits in infants' growth during the first year, but only among those who had experienced elevated in utero exposure (maternal prenatal PbB >7.7 μg/dL). The present study is a follow-up of the same cohort during the second and third year of life. We no longer found any indication of a prenatal PbB influence on growth. Rather, the depressive effect of lead during the second and third year was now dependent on lead exposure in the early postnatal period (3 to 15 months). The disappearance of prenatal PbB and appearance of PbB (3 to 15 months) as an interacting variable suggests two things. First, the depression of growth is seen only with a long period of exposure, eg, prenatal plus 3 to 15 months, or 3 to 15 months plus 18 to 33 months. Second, the effect seems to be reversible, at least when elevated prenatal and early postnatal exposure is followed by lower exposure. Another interesting feature is the overshoot phenomenon illustrated in Fig 5. We are at a loss to explain why this occurs and are not aware of any similar situation in which previously depressed growth is followed by attainment of stature that clearly exceeds the expected norms. This may be a transient phenomenon. Continued follow-up should reveal whether this is the case.

Unlike in the first year, when growth rate was an adequate measure of response, this was not found to be the case in the second and third year. Growth velocities during the second and third year of life are not only slow relative to the first year, but the between-subject variability in growth rates decreases.[5] Further, the amount of measurement error inherent in any single length measurement is combined and compounded in obtaining growth rates from several length measurements. This cumulative error effect applies to a total quantity (the amount of growth) that is quite small relative to length measurements. The attained height at a given point in time represents the end result of a cumulative growth process and was found to be a satisfactory measure of response. We were concerned, however, about the robustness of results for other ages. Consequently, similar analyses were conducted using lengths at 27 months, 30 months, 36 months, and 39 months as the response variable. The results obtained were similar in all the cases to those for length at 33 months (data not shown).

The sensitivity of the lead-growth regression coefficient was tested for inclusion/exclusion of other explanatory covariables. The coefficient did not change in magnitude (−1.8 to −2.0) as a function of other covariables. A set of 15 children was chelated one or more times for diagnostic or therapeutic purposes prior to 3 years of age. Thirteen of these subjects had "high" first-year PbB and had mean PbB (18 to 33 months) of more than 25 μg/dL. We found that the regression results were essentially unchanged when those 13 subjects were excluded, indicating that the overall results of this study were not unduly influenced by those children. A number of demographic characteristics and potential confounders were compared between the two groups of subjects created by the median split-on the mean PbB during the 3- to 15-month interval. Except for prenatal maternal smoking (packs per day) (0.84 ± 0.94 for "high" group vs 0.57 ± 0.68 for "low" group), the two groups were very similar.

Another issue worth emphasizing here relates to the epidemiologic nature of the study design and analysis. No amount of data-based evidence from an observational study like this can, by itself, be conclusive "proof" of a cause-effect relationship. Any temptation to draw such a conclusion must be resisted. One such issue that cannot be fully resolved from a study like this relates to PbB as a marker for lead exposure in a growing child. The possibility cannot be ruled out that faster growing children exhibit lower PbB levels because of increased volume of distribution, particularly bone. Other recent studies,[6–9] though not all,[10] also suggest that low to moderate levels of lead exposure are a risk factor for growth in stature during early childhood. Moreover, reduction in linear and ponderal growth due to lead exposure has been clearly shown to occur in weanling rats.[11,12]

In summarizing our findings, we conclude that a sizable portion of young inner-city children experience lead exposures that may have a significant impact (both statistically and otherwise) on growth and attained height. It also appears that prenatal and/or early postnatal effects on growth are "transient" in the sense that a child tends to catch up, provided subsequent lead exposure is reduced. We hope that continued follow-up of the study cohort will reveal whether these Pb-related deficits in growth persist.

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