Annals of Human Biology
2009, 1–11, iFirst article

informa
healthcare

ORIGINAL ARTICLE

Blood lead levels and growth status among African–American and Hispanic children in Dallas, Texas – 1980 and 2002: Dallas Lead Project II

B. B. LITTLE[1,2], S. SPALDING[2], B. WALSH[2], D. C. KEYES[3], J. WAINER[2], S. PICKENS[2], M. ROYSTER[2], J. VILLANACCI[4] & T. GRATTON[5]

[1]*Departments of Mathematics, and of Physics and Engineering, Texas Data Mining Research Institute, and Division of Academic Affairs, Tarleton State University, Stephenville, Texas,* [2]*Parkland Health & Hospital System, Dallas Texas,* [3]*John Peter Smith Health Network, Fort Worth, Texas,* [4]*Environmental and Injury Epidemiology, and* [5]*School of Public Health UNT*

(Received 21 May 2008; revised 30 January 2009; accepted 6 February 2009)

Abstract
Objective: The purpose of this investigation is to analyze childhood blood lead levels and growth status (ages 2–12) in Dallas, Texas lead smelter communities in the 1980s and 2002, where smelters operated from 1936 to 1990.
Methods and materials: A sample of convenience study design was used in two cohorts ($n=360$): 1980–1989 ($n=191$) and 2002 ($n=169$). Multivariate analysis of variance and covariance and tandem multiple regressions were used to evaluate the association between stature and blood lead level in two time periods.
Results: In 2002 average child blood lead level (1.6 μg/dL ± 0.2 SE) was significantly ($p<0.001$) lower compared to the 1980 cohort mean level (23.6 μg/dL ± 1.3 SE). Average height and weight in 2002 were 4.5 cm and 4.0 kg greater, respectively, than in 1980. Lowered blood lead level was associated with 3.9 cm, 3.5 kg and 1.1 units greater height, weight and body mass index (BMI), respectively. Cohort effect was associated with greater height (0.6 cm), weight (0.5 kg) and BMI (0.1).
Conclusion: This investigation reports on child growth in a community before and after the transition from high to low blood lead levels over several decades. Using child growth as a proxy, health status of Dallas's lead smelter communities increased markedly over the past two decades, primarily because of lower blood lead levels, while the poverty rate was only marginally lower.

Keywords: *Child growth, lead exposure, blood lead level, lead smelter, Superfund, 20 year follow-up*

Correspondence: Bert Little, Associate Vice President for Research and Professor, Tarleton State University, Stephenville, TX 76 402-0010, USA

ISSN 0301-4460 print/ISSN 1464-5033 online
DOI: 10.1080/03014460902806615

Introduction

> 'Child growth is a mirror of the material and moral conditions of a community.'
> Jim Tanner, MD, Professor Emeritus, Institute of Child Health, University of London

Growth stunting has been reported in children with elevated blood lead levels. Children are at greater risk than adults for adverse environmental effects (e.g. undernutrition, environmental pollutants) because they are experiencing rapid hyperplastic growth and, to a lesser extent, hypertrophic growth (Bearer 2000). Child growth is a sensitive barometer of community health and nutritional conditions (Tanner 1986, 1992), including environmental pollution (Schell 1986; Denham et al. 2005). Specifically, stunted child growth and delayed sexual maturation are indicators of poor community health and nutritional conditions (Tanner 1992). Delayed age at menarche is associated with elevated blood lead levels (Selevan et al. 2003; Wu et al. 2003; Denham et al. 2005). Stunted stature and weight were associated with elevated lead levels in several investigations (Schwartz et al. 1986; Shukla et al. 1989, 1991; Little et al. 1990; Kafourou et al. 1997; Ballew et al. 1999; Sanín et al. 2001; Ignasiak et al. 2006). As noted, increased poverty is known to confound the investigation of lead level effects on growth but has not been analyzed. Body lead burden and child growth across time (longitudinally) were significantly inversely related in two investigations (Shukla et al. 1989, 1991), but not in several others (Sachs and Moel 1989; Greene and Ernhart 1991). One group of investigators reported persistent lead exposure was associated with increased body mass index (BMI), but no significant effect on physical growth (Kim at al. 1995).

That lead exposure at lower levels may be harmful has only recently been recognized as better information became available (Bellinger 2004). Whether the adverse effects of lead are a threshold effect or a dose response phenomenon remains controversial, but significant effects on human growth have been detected below 10 μg/dL (Schell et al. 2009). Lead exposure during childhood is known to cause irreversible mental retardation (Canfield et al. 2003; Chiodo et al. 2004; Lanphear et al. 2005), growth stunting (Ignasiak et al. 2006) and impaired physical fitness (Ignasiak et al. 2007). Poor socioeconomic conditions (i.e. per cent of households below federal poverty level) among lead-exposed populations are recognized confounders of investigation of human health effects of lead exposure (Agency for Toxic Substances and Disease Registry (ATSDR) 1997; Ballew et al. 1999).

The objective of the present investigation is to analyze the association of changes in blood lead levels and child growth status over two decades (1980, 2002) in a lead smelter community in Dallas, Texas, before and after the closure of smelters and an EPA Superfund Cleanup lead remediation project in 1992–1993 (http://www.epa.gov/superfund/).

Methods and materials

Institutional Review Board oversight and approval

The University of Texas Southwestern Medical Center Institutional Review Board approved the studies that were conducted in 1980–1989 and in 2002 in two separate Human Subjects applications.

Background

Lead smelting in Dallas, Texas began in 1936 when the RSR Metals (formerly Southern Lead) was established in West Dallas. Dixie Metals began operations in South Dallas in 1954, was later owned by Exide Battery, and finally by National Lead (Gratton et al. 2004). The last smelter in West Dallas ceased operations on 24 February 1984. The south Dallas smelter (Cadillac Heights and West Dallas) ceased operation in 1990. It is estimated that 40% of the study children lived within 1 mile of the smelter in both studies, and all lived within a 3 mile radius. These companies processed batteries and slag material on a site in West Dallas, releasing a lead dust cloud over surrounding communities (ATSDR 1997; Khoury and Diamond 2003). The location of the lead smelter communities is west of downtown Dallas, and north of Interstate 30 (Figure 1).

Study design

Two cohorts of children were studied at two points in time, one from the 1980s and one from 2002. Child blood lead levels, height, weight, and BMI were compared for the two time periods in Dallas, Texas. Data for the 1980 cohort consisted of a self-selected group of children who had a medical record with blood lead levels and anthropometrics at a City of Dallas lead clinic or Children's Medical Center, Dallas, Texas. It is estimated that 40% of the children lived within 1 mile of the smelter in both studies, and all lived within a 3 mile radius. The children did not present with symptoms of plumbism, but rather were referred to the clinics for blood level testing because they resided in an area near a lead smelter and were considered at risk for exposure. Symptoms were, however, discovered during clinic visits (Appendix I). In 2002 Parkland Hospital and Health System used funding from a legislatively directed grant to conduct a blood lead screening and administer a health status indicator questionnaire. Subjects were recruited through a community outreach (publicity) program, similar to the solicitation for the 1980s clinic visits (i.e. you or your child may be at risk, testing is free). The EPA conducted a Superfund Cleanup in 1992–1993 in Dallas (US EPA 1993) lead smelter communities in the time period that separated the two cohorts with the 1980s cohort data collected prior to the Superfund Cleanup, and the 2002 cohort data collected after the Superfund Cleanup. The federal poverty level, as determined by the US Census Bureau, was included for the study communities' zip codes and census tracts in 1980, 1990, and 2000 to assess change over time. The use of census data coupled with zip code data are routinely used to assess socio-economic status (SES) at the community level. Microlevel (household, block) data are known to be better indicators of socio-economic status because zip code areas typically contain 30 000 people and are not homogeneous (Geronimus and Bound 1998). Thus, zip code data may often underestimate poverty rates in a given area (Krieger et al. 1997), which argues for the use of census tract data in addition. In the Dallas, Texas lead smelter study communities, socio-economic homogeneity is much greater than is generally the case in the US, being comprised entirely of inner city minority, lower SES households (ATSDR 1997).

Blood lead level and growth status

Data from the 1980s screening program for 404 children included blood lead level, height and weight (Little et al. 1990). Childhood blood lead level and growth status were measured in 390 children in 2002 as part of a phase II health screening by Parkland Health & Hospital System. In both instances, height was measured without shoes, feet together, and head erect

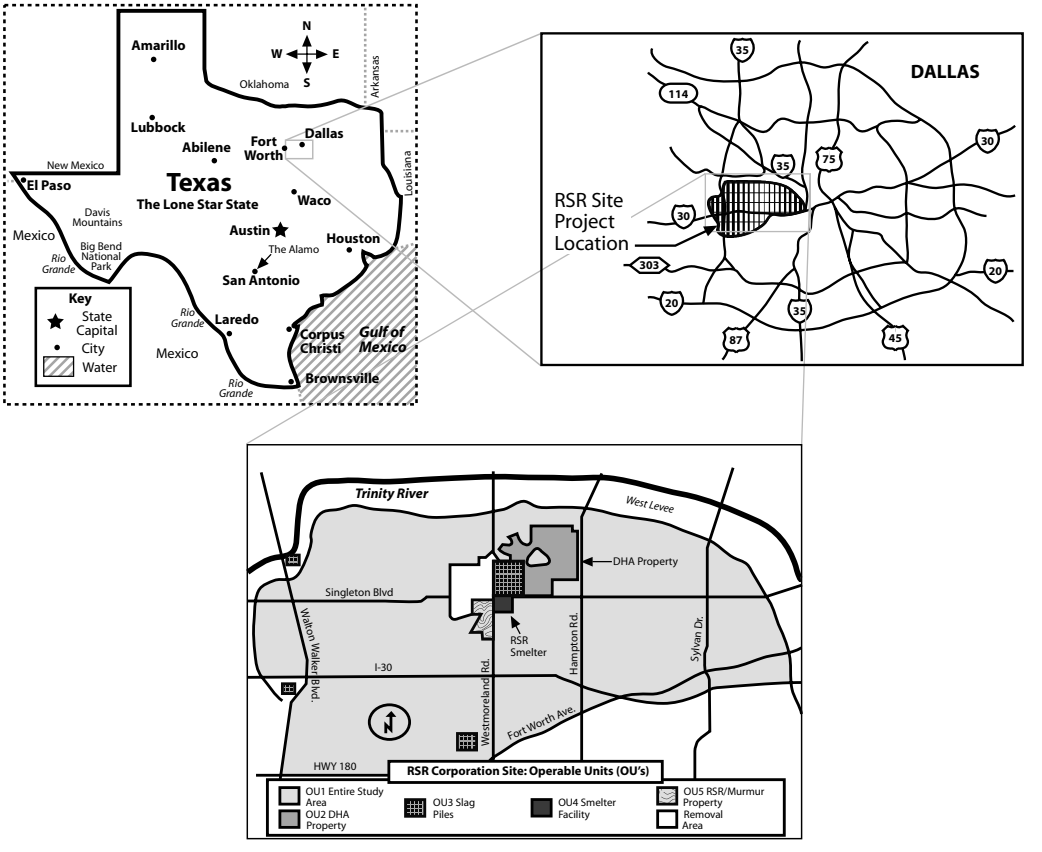

Figure 1. Map of Texas (left, top) city of Dallas lead smelter communities (right, top) showing the location of the study communities (hatched area), smelter locations, and public housing (DHA, Dallas Housing Authority) and street level detail of lead-affected areas (bottom, center).

by clinic nurses. Thus, databases from the 1980 cohort ($n = 404$ unique) and 2002 ($n = 390$ unique) contained a total of 794 children, but some had missing values on anthropometric variables. Listwise deletion resulted in 191 children for the 1980 cohort and 169 children in the 2002 cohort for a total of 360 children (Appendix I). In 1980, mean blood lead level of children with height and weight data was 24.8 μg/dL (SD = 11.0), and mean blood lead level was 22.8 μg/dL (SD = 12.3) in those for whom height and weight information was unavailable. For the 2002 cohort, mean blood lead level was 1.8 μg/dL (SD = 1.8) for children with anthropometrics, while those children without height and weight measurements had a mean blood lead level of 2.4 μg/dL (SD = 1.5). Variance in blood lead levels were homogeneous between children with and without anthropometrics (Levene's test p = NS) in the 1980 cohort. In contrast, blood lead level variance was not homogeneous comparing children with and without height and weight measurements (Levene's test $p < 0.04$). It seems reasonable to assume that those children with anthropometrics are a representative sample of children who had blood lead levels tested, although variances are not homogeneous in the 2002 cohort between the groups with and without measurements.

Age range of children in the study was restricted to 2–12 years because of very small sample sizes at later ages. Age^2 was included to account for possible non-linear components

present in growth data. Height and weight were measured by nurses in 1980 and 2002 under standard protocol, but intra- and inter-observer errors are not available.

Lead determination

In the 1980s, whole blood lead levels were determined by the Texas Department of State Health Services (formerly Texas Department of Health) using a graphite furnace atomic absorption spectrophotometer with a lower limit of detection <1.0 μg/dL (Little, personal communication). In 2002, whole blood lead levels were analyzed by a Varian atomic absorption spectrophotometer with a graphite furnace and Zeeman correction and by a certified external commercial laboratory. The lower limit of detection was <1.0 μg/dL, and the assay calibration curve was linear up to 40.0 μg/dL.

Statistical analysis and confounding effects

Age distributions differ between the two cohorts, with the 1980 cohort having a higher representation of children ≤ 6 years old (76.7%), and the 2002 cohort consisting of more children >6 years of age (72.8%). Age effects were corrected in two ways: (1) data were transformed to age and sex specific z-scores computed from CDC reference data, and (2) age and age^2 were modeled and implemented as covariates in MANOVA/MANCOVAs and regressions. The best practice method for analysis of 'samples of convenience' is to adjust statistically for known possible confounders, as has been done in this investigation. The reason age and age^2 still made a contribution after z-score transformation is because age is a continuous variable. Decimal age still has variation within 1 year increments of the z-score transformation standards data. Therefore, age effects should be well controlled because within and between age group variation is corrected for analytically.

In the 1980s cohort, 64% of children were African–American, and 36% were Hispanic. Ethnicity was evaluated for possible effects on the dependents (height, weight, BMI) and blood lead in the 1980 cohort. Differences between the ethnic groups were not significant (ANOVA, $p =$NS) and variation was homogeneous comparing the two groups (Box and Levene's tests, $p =$NS). It was, therefore, assumed appropriate for the combined sample to be used for analysis. In 2002, African–Americans comprised 97% of survey participants; the category of 'unknown' (3%) was excluded from the present analysis because of missing values on an analytic variable (e.g. ethnicity). Blood lead was not normally distributed (skew and kurtosis >1.0), and was $\log_{10}$ transformed for analysis to obtain skew and kurtosis <1.0. Multivariate analyses of variance (MANOVA) and covariance (MANCOVA) were done to analyze height, weight and BMI and blood lead levels in 1980 and 2002.

BMI was a calculated variable: weight (kg)/height (m)^2. CDC reference child growth status data in EpiInfo software (US CDC, Atlanta, GA, USA 2005) were used to compute age and gender specific z-scores for height, weight, and BMI for use in multiple regression analysis.

Pooled z-scores for growth status were evaluated by tandem multiple regressions for blood lead effects first, and the standardized residuals saved. Cohort effects (1980 vs 2002) on height, weight, and BMI were tested by multiple regression analysis of residuals (i.e. after the effects of blood lead, age and age^2 were removed).

Type III sums of squares were used to assure linear independence of the MANOVA, MANCOVA and regression estimates. Bonferroni correction for multiple comparisons was applied in each analysis. Statistical power was calculated for each dependent variable.

Analyses were performed using SAS V 9.1 (SAS Institute, Inc., Cary, NC, USA, 2006) and SPSS V 14.0 (SPSS Inc., Chicago, IL, USA, 2006).

Results

Socio-economic conditions improved slightly in 22 years in the study community, with a net decrease in poverty between 1980 (44.4%) and 2002 (30.9%) of 13.5% (Table I). The age composition of the study samples is biased toward younger children in 1980 and older children in 2002 (Appendix I). Regression analysis reveals that the relationship of blood lead level with height is very similar (not statistically significantly different) among children ≤ 6 years in 1980 and 2002 cohorts ($B = -0.11$ and -0.09, respectively) and also among those >6 years of age in the two cohorts ($B = -0.21$ and -0.19, respectively) (Table II).

Children living in lead smelter communities in Dallas had exceptionally high blood lead levels in the 1980s compared to current CDC standards. In 2002 average child blood lead level (1.6 μg/dL $\pm$ 0.2 SE) in smelter communities was significantly ($p < 0.001$) reduced compared to the 1980 cohort mean level (23.6 μg/dL $\pm$ 1.3 SE).

MANOVA by gender of blood lead level, height, weight, and BMI with age and age^2 held constant using MANCOVA, indicates that the main effect (cohort) is significant ($p < 0.0001$) for all dependent variables. Male and female children ages one to 12 years old in 2002 were significantly (<0.0001) taller and heavier with higher BMI and lower blood lead than children measured in the 1980s. Power was acceptable (range: 81% to 100%) for effects on height, weight, and BMI. Variance in blood lead level was homogeneous (Levene's $p =$ NS), but not homogeneous for height, weight, and BMI (Levene's $p < 0.0001$).

Blood lead level had a significant ($p < 0.0001$) inverse log–linear relationship with height, weight, and BMI z-scores (Table III). Higher BMI at lower levels of blood lead was unexpected, suggesting a relatively greater weight compared with height at lower blood lead levels observed in the study sample. After the 'lead effect' was removed by regression, analysis of residuals revealed that the cohort effect was negligible (not statistically significant). This implies that the lion's share of the greater body size and BMI was associated with lower blood lead level. This seems reasonable given that blood lead level was 22 μg/dL lower ($p < 0.0001$) among children surveyed in 2002 compared to children in the 1980 cohort while poverty rates at the community level was 13.5% lower over 22 years.

Extrapolation of the regression analyses of pooled z-scores residuals indicated that when blood lead level was 10 μg/dL lower, height was 2.1 cm higher (95% CI: 1.9–2.3) ($p < 0.0001$) (Table IV). Similarly, weight was 1.9 kg greater (95% CI: 1.7–2.1) ($p < 0.0001$) with each 10 μg/dL lowering of blood lead level (Table IV). BMI was 0.5 units greater (95% CI: 0.4–0.7) with a 10 μg/dL lowering of blood lead level (Table IV). The cohort effect was small and not statistically significant when orthogonalized from the effects of lead (Table III).

Table I. Poverty rate in zip code-census tract areas for Dallas study communities 1980, 1990, and 2000.

	Dallas, TX Study area*	Median, USA†	Median household income poverty threshold‡
1980	44.4%	13.0%	$9300
1990	38.3%	13.5%	$12 700
2000	30.9%	11.8%	$17 050

*Zip codes: data from US Census 1980, 1990, and 2000 zip code sub-tables; 1980 data were obtained via CD-ROM; 1990 and 2000 are available online at www.census.gov, with technical assistance from Shirley Witcher.
†From Rushevsky (2002).
‡Family of four.

Table II. Regression of height *z*-score on $\log_{10}$ blood lead level by age (1–6 years, 7–12 years) and cohort (1980 vs 2002).

Age group	*n*	%	Mean age	SD	*B*	SE	R^2
1980 cohort							
<6 years	140	71.5	3.5	1.3	−0.11	0.04	0.72
>6 years	56	28.6	9.7	1.8	−0.21	0.33	0.17
	196						
2002 cohort							
<6 years	46	27.2	4.5	1.7	−0.09	0.13	0.32
>6 years	123	72.8	9.9	1.8	−0.19	0.11	0.43
	169						

Discussion

Markedly lower child blood lead levels (mean decrease = 22 μg/dL) were observed in Dallas lead smelter communities comparing 1980–2002. Statistically significantly greater growth status for height, weight, and BMI was observed among children ages 2–12 years in 2002 compared to children in the 1980s in the same communities. Concurrently, a national trend in reduced environmental lead pollution occurred (unleaded gasoline use, lead paint removal, other lead pollution abatement) (Pirkle et al. 1998; Ballew et al. 1999; US CDC 2000; Bellinger 2004). Larger body size (height, weight) were associated with lower blood lead levels in several prior investigations (Table V), including the 1980 cohort (Little et al. 1990).

Much of the difference in blood lead levels in Dallas smelter communities was present by 1992–1993. Therefore, it is probably not reasonable to attribute the marked reduction in child blood lead levels to the EPA Superfund Cleanup because blood lead levels were already lowered to an average of 5 μg/dL by 1992 when the Superfund Cleanup began (ATSDR 1995, 1997). The local lead abatement efforts implemented before the EPA

Table III. Regression analysis of blood lead level and cohort effects on *Z*-scores for stature, weight, and BMI ($n = 360$).

	Z-score*								
	B†	SE	*p*	β	*A*	SE	*p*	*p* for *F*	Adj. R^2
Height									
Log blood lead	−0.52	0.09	0.0001	−0.30	0.47	0.09	0.0001	0.0001	0.09
Cohort effect‡	0.13	0.05	(0.20)	0.06	−11.66	9.13	(0.20)	(0.20)	0.004
Weight									
Log blood lead	−0.68	0.09	0.0001	−0.36	0.52	0.09	0.0001	0.0001	0.12
Cohort effect‡	0.15	0.099	(0.13)	0.07	−0.06	0.07	(0.33)	(0.13)	0.01
BMI									
Log blood lead	−0.32	0.09	0.0001	−0.19	0.26	0.09	0.005	0.0001	0.03
Cohort effect‡	0.06	0.10	(0.58)	−0.03	−0.03	0.07	(0.71)	(0.58)	0.001

B, unstandardized (scaled) regression coefficient, β, standardized (unscaled) regression coefficient.

*Z-scores for height, weight and BMI were calculated using CDC standards in EpiInfo (2005) for regression analyses.

†*B* may be used to compute the scaled (cm, kg, BMI units) effects associated with each measure and main effect.

‡*B* computed on residuals after effects of log blood lead are removed by regression. Parentheses indicate statistically non-significant result.

Table IV. Scaled effects of changes in blood lead level and cohort on stature, weight, and BMI.

	Z-score converted to scaled measure in study population						Adjusted partial decrement*
	Lead effects† measure		Cohort effect‡ 1980 vs 2002		Total decrement (cohort ± lead effects)		Blood lead effect per 10 μg/dL lead increase
Scaled measure	Estimate	*p*§	Estimate	*p*§	Estimate	*p*§	Mean (95% CI)
Height (cm)	−3.9	0.0001	− 0.6	NS	−4.5	0.0001	−2.1 (−1.9 to −2.3)
Weight (kg)	−3.5	0.0001	− 0.5	NS	−4.0	0.0001	−1.9 (−1.7 to −2.1)
BMI (kg/m^2)	−1.0	0.0001	− 0.1	NS	−1.1	0.0001	−0.5 (−0.4 to −0.7)

*Computed from B in Table III adjusted for age age^2, sex, and cohort effect; generalizable.
†These are effects for the Dallas study and not generalizable. Total decrease between 1980 and 2002 in blood lead level was evaluated at means of 21.3 and 21.5 μg/dL, for males and females, respectively, and z-score was converted to scaled (cm) metric using the pooled population SD. Hence, these results are population specific.
‡See Table I for poverty status of study community.
§p value from Table III for B upon which this estimate is based testing H_o that $B = 0$.
Height, weight, and BMI decrement scaled estimate, $y = [AX^b] \times \text{SD}$, where SD is standard deviation, y is height z-score, x is log blood lead level, A is regression intercept, and b is B (Table III), the scaled (unstandardized) regression slope. Logarithms in linear equations have the property anti-log $y = AX^b$ where $\log y = \log a + b \log X$, scaled estimate $= [AX^b] \times \text{SD}$, where SD is pooled standard deviation scaled for the measure across age and sex groups, and X is log blood lead level.

Superfund cleanup were undoubtedly an important factor because of substantial environmental interventions (i.e. lead contaminated top soil and lead paint removal programs, planting forage to subdue soil dust) in the 1980s, in addition to national trends in lead pollution reduction. Thus, the large difference in blood lead levels from 1980 to 1992 was most likely a result of closure of the smelter which eliminated the air pathway, the most significant pathway of lead exposure.

Child growth status is a reliable surrogate indicator of community health status, including levels of environmental pollution (Tanner 1992; Denham et al. 2005; Schell et al. 2006). Growth status of children in the smelter communities was significantly greater in 2002 compared to 1980, and blood lead levels were significantly lower. Children in the 1980 cohort from West Dallas and Cadillac Heights (Figure 1) had an average blood lead level (23.6 μg/dL) that was close to the CDC level for medical intervention/action (25 μg/dL) in the 1980s. Analysis of the 2002 cohort showed a dramatic difference in blood lead level, accompanied by a marked increase in child height. The 2002 cohort (born 1990–2000) and reared in the smelter neighborhoods had blood lead levels significantly lower than the 1980 cohort. Notably, the magnitude of difference in stature and weight is within the range that would be predicted based upon changes reported by other investigators when expressed as change in height and weight per 10 μg/dL decrease in blood lead level (Table IV).

It is probable that the higher growth status may be related to several non-Superfund cleanup programs such as closure of smelters and the national trend toward lower child blood lead levels. The proportion of households below the federal poverty level in the smelter communities lowered from 44.4% in 1980–33.6% ($\Delta = 13.5\%$) in 2000. Thus, socio-economic growth in the smelter neighborhoods may have positively affected nutrition and healthcare of children in the 2002 cohort. The cohort effect was associated with a slightly greater height and weight (13% of the total) in the 2002 group compared to the 1980 cohort (Table IV), but it is unknown whether or not the association is causal. The

Table V. Decrements in height and weight associated with blood lead*.

Study group	Age range	Decrement	Investigators
Stature decrement			
Cincinnati Lead Study	3–15 months	2.0 cm/10 μg/dL	Shukla et al. (1989)
Cincinnati Lead Study	33 months	1.5 cm/10 μg/dL	Shukla et al. (1991)
Dallas Lead Project I	1–10 years	1.6 cm/10 μg/dL	Little et al. (1990)
Dallas Lead Project II	2–12 years	2.1 cm/10 μg/dL	Little et al. (this study)
Greek Cities ($n=3$)	6–9 years	0.9 cm/10 μg/dL	Kafourou et al. (1997)
NHANES II	1–7 years	1.2 cm/10 μg/dL	Schwartz et al. (1986)
NHANES III	1–7 years	1.6 cm/10 μg/dL	Ballew et al. (1999)
Lower Silesia, Poland Lead Study	7–14 years	3.1 cm/10 μg/dL	Ignasiak et al. (2006)
Weight decrement			
Dallas Lead Project I	1–10 years	1.4 kg/10 μg/dL	Little et al. (1990)
Dallas Lead Project II	2–12 years	1.9 kg/10 μg/dL	Little et al. (this study)
NHANES II	0–7 years	1.1 kg/10 μg/dL	Schwartz et al. (1986)
Mexico City Lead Project	0–1 months	36 g/10 μg/dL	Sanín et al. (2001)
Lower Silesia, Poland Lead Study	7–14 years	3.1 kg/10 μg/dL	Ignasiak et al. (2006)

*Other studies have reported body size decrements associated with elevated blood lead levels, but did not report the estimated decrement, or did not provide information needed to compute it; adapted from Ignasiak et al. (2006), with corrections to Dallas Lead Project II decrements. Analyses in the current report have been revised since the Ignasiak investigation was published.

slightly higher BMI over time suggests a mild-to-moderate betterment of nutritional and health conditions from 1980 to 2002.

In the present investigation we showed that larger body size (growth status) is associated with lower blood lead levels and, to a lesser extent, with a lower community poverty rate. It is likely no fewer than four sources of environmental lead abatement were associated with lower blood lead levels: (1) City of Dallas Court Ordered soil removal in the 1980s, (2) smelter closures, (3) the EPA Superfund Cleanup (soil removal), and (4) the larger national trend toward lower environmental lead levels (e.g. changing to unleaded gasoline). Lower blood lead levels were associated with improved growth status among children in the study sample, and, by inference, community health status. Small effects of reduced poverty (e.g. improved nutrition, socio-economic improvements, housing renovation, new construction) were suggested by the analyses. Based upon dose-dependent negative physiological effects of blood lead levels on chondrocytic proliferation at growth plates (Hicks et al. 1996), it seems likely that the association of lower blood lead level coincident with larger body size is causal among children in the present study, but this remains deductive speculation nonetheless. Recent data suggest that the threshold for lead effects on human growth may be 3 μg/dL (Schell et al. 2009).

In summary, lead abatement programs, including closure of smelters, were associated with lower blood lead levels in Dallas lead smelter communities. Lower blood lead levels were associated with taller children who weighed more and had a slightly higher BMI. These data suggest community health conditions, reflected in child growth, improved substantially from 1980 to 2002.

Acknowledgements

This study was funded by Texas Department of State Health Services with the advocacy of Texas State Senator Royce West and his Legislative Director Roger Jones. An earlier version of this paper was orally presented at the American Public Health Association Annual Meeting in Philadelphia, PA, 2005. Special appreciation is extended to Ann Pawlak for editorial assistance, and John Millet for Figure 1.

References

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Texas Department of Health. Public Health Assessment. RSR Corporation, Dallas, Dallas County, Texas, CERCLIS NO. TXD079348397, 16 August 1995. Available online at: http://www.atsdr.cdc.gov/HAC/pha/rsrcorp/rsr_toc.html [accessed 29 November 2007].

Ballew C, Khan LK, Kaufman R, Mokdad A, Miller DT, Gunter EW. 1999. Blood lead concentration and children's anthropometric dimensions in the Third National Health and Nutritional Examination Survey (NHANES III), 1988–1994. J Pediatr 134:623–630.

Bearer CF. 2000. The special and unique vulnerability of children to environmental hazards. Neurotoxicology 21:925–934.

Bellinger DC. 2004. Lead. Pediatrics. 113:1016–1022.

Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. 2003. Intellectual impairment in children with blood lead concentrations below 10 micrograms per deciliter. New Engl J Med 348:1517–1526.

CDC (Centers for Disease Control and Prevention). 2000. Blood lead levels in young children – United States and selected states, 1996–1999. MMWR 49:1133–1137.

Chiodo LM, Jacobson SW, Jacobson JL. 2004. Neurodevelopmental effects of postnatal lead exposure at very low levels. Neurotoxicol Teratol 26:359–371.

Denham M, Schell LM, Deane G, Gallo MV, Ravenscroft J, DeCrapio AP. 2005. Relationship of lead, mercury, mirex, dichlorodiphenyldichloroethylene, hexachlorobenzene, and polychlorinated biphenyls to timing of menarche among Akwesasne Mohawk girls. Pediatrics 115:e127–e134.

Geronimus AT, Bound J. 1998. Use of census-based aggregate variables to proxy for socio-economic group: Evidence from national samples. Am J Epidemiol 148:475–486.

Gratton TB, Trief NM, Aubesquin S, Juarez D. 2004. A review of health and environmental lead data from a neighborhood exposed to smelter emissions. J Child Health 2:21–27.

Greene T, Ernhart CB. 1991. Prenatal and preschool age lead exposure: Relationship with size. Neurotoxicol Teratol 13:417–427.

Hicks DG, O'Keffe RJ, Reynolds KJ, Cory-Slechta DA, Puzas JE, Judkins A, Rosier RN. 1996. Effects of lead on growth plate chondrocyte phenotype. Toxicol Appl Pharmacol 140:164–172.

Ignasiak Z, Sławińska T, Rożek K, Little BB, Malina RM. 2007. Blood lead level and physical fitness of schoolchildren in the copper basin of south western Poland: Indirect effects through growth stunting. Ann Hum Biol 34:329–343.

Ignasiak Z, Sławińska T, Roażek-Piechura K, Little BB, Malina RM. 2006. Growth status of Polish schoolchildren living near a copper smelter. Ann Hum Biol 33:401–414.

Kafourou A, Touloumi G, Makropoulos V, Loutradi A, Papanagiotou A, Htazakis A. 1997. Effects of lead on somatic growth of children. Arch Env Health 52:377–383.

Khoury GA, Diamond GL. 2003. Risks to children from exposure to lead in air during remedial or removal activities at Superfund sites: a case study of the RSR lead smelter Superfund site. J Expos Anal Environ Epidemiol 13:51–65.

Kim R, Hu H, Rotnitzky A, Bellinger D, Needleman H. 1995. A longitudinal study of chronic lead exposure and physical growth in Boston. Environmental Health Perspectives 103:952–957.

Krieger N, Chen JT, Ebel G. 1997. Can we monitor socio-economic inequalities in health? A survey of US health departments' data collection and reporting practices. Public Health Reports 112:481–491.

Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, et al. 2005. Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. Environ Health Perspect 113:894–899.

Little BB, Snell LM, Johnston WL, Knoll KA, Buschang PH. 1990. Blood lead levels and growth status of children. Am J Hum Biol 2:265–269.

Pirkle JL, Kaufmann RB, Brody DJ, Hickman T, Gunter EW, Paschal DC. 1998. Exposure of the US population to lead, 1991–1994. Environ Health Perspect 106:745–750.

Rushevsky ME. 2002. Public policy in the United States, 3rd edn. Armonk, NY: M.E. Sharpe.

Sachs HK, Moel DI. 1989. Height and weight following lead poisoning in childhood. Am J Dis Child 143:820–822.

Sanín LH, González-Cossío T, Romieu I, Peterson KE, Ruíz S, Palazuelos E, Mauricio Hernández-Avila M, Hu H. 2001. Effect of maternal lead burden on infant weight and weight gain at one month of age among breastfed infants. Pediatrics 107:1016–1023.

Schell LM. 1986. Community health assessment and physical anthropology: Auxological epidemiology. Hum Organiz 45:321–327.

Schell LM, Gallo MV, Denham M, Ravenscroft M. 2006. Effects of pollution on human growth and development: An introduction. J Physiol Anthropol 25:103–112.

Schell LM, Denham MA, Stark AD, Parsons PJ, Schulte EE. 2009. Growth of infants' length, weight, head and arm circumferences in relation to low levels of blood lead measured serially. Am J Hum Biol Schell LM, Denham MA, Stark AD, Parsons PJ, Schulte EE. 2009. Growth of infants' length, weight, head and arm circumferences in relation to low levels of blood lead measured serially. Am J Hum Biol 21:180–189.

Selevan SG, Rice DC, Hogan KA, Euling SY, Pfahles-Hutchens A, Bethel J. 2003. Blood lead concentration and delayed puberty in girls. N Engl J Med 348:1527–1536.

Schwartz J, Angle C, Pitcher H. 1986. Relationship between childhood blood lead levels and stature. Pediatrics 77:281–288.

Shukla R, Bornschein RL, Dietrich KN, Buncher CR, Berger OG, Hammond PB, Succop PA. 1989. Fetal and infant lead exposure: Effects on growth in stature. Pediatrics 84:604–612.

Shukla R, Dietrich KN, Bornschein RL, Berger O, Hammond PB. 1991. Lead exposure and growth in the early preschool child: A follow-up report from the Cincinnati Lead Study. Pediatrics 88:886–892.

Tanner JM. 1986. Growth as a mirror for the conditions of society: Secular trends and class distinctions. In: Demirjian A, ed. Human growth: A multidisciplinary review. London: Taylor & Francis, p. 3.

Tanner JM. 1992. Growth as a measure of the nutritional and hygienic status of a population. Horm Res 38 (Suppl 1):106–115.

US EPA (Environmental Protection Agency). 1993. Site Assessment Report for RSR Smelter (West Dallas Lead) Site, Dallas, Dallas County, Texas. Prepared for Ecology and Environment, Inc., 10 September 1993.

Wu T, Buck GM, Mendola P. 2003. Blood lead levels and sexual maturation in US girls: The Third National Health and Nutrition Examination Survey, 1988–1994. Environ Health Perspect 111:737–741.