

A Comparison of Child Blood Lead Levels in Urban and Rural Children Ages 5–12 Years Living in the Border Region of El Paso, Texas

Juan Alvarez¹ · Michelle Del Rio¹ · Tania Mayorga¹ · Salvador Dominguez¹ · Mayra Gisel Flores-Montoya³ · Christina Sobin^{1,2,4}

Received: 19 April 2018 / Accepted: 9 July 2018 / Published online: 28 July 2018 © Springer Science+Business Media, LLC, part of Springer Nature 2018

Abstract

Lead exposure is an unresolved pediatric health risk and disproportionately affects children in lower-income neighborhoods. Residences with children younger than age 5 years are the focus of mitigation policies; however, studies have shown that older children between the ages of 5 and 12 years also are at risk of central nervous system effects. Whether historically contaminated neighborhoods present ongoing risk to older children also is of concern. This study compared the blood lead levels (BLLs) of older children from an historically contaminated urban neighborhood to those of demographically matched children from a nearby rural locale and predicted significantly higher BLLs in the urban children. The study included 222 children aged 5–12 years, 111 from the urban neighborhood and 111 from local rural townships, matched for age, sex, race/ ethnicity, and family income. Blood lead, cadmium, and mercury were measured using inductively coupled plasma mass spectrometry. General linear models tested whether geographic location (urban vs. rural) predicted child heavy metal levels, controlling for sex and age. Only location predicted only child BLL (R^2 =0.36); children living in the urban setting had significantly higher BLLs as compared with matched rural township children (F = 125, $df_{220,2}$, p < 0.001). Neighborhoods with a history of lead contamination can present current risk of lead exposure for older children between the ages of 5 and 12 years, as well as for infants and toddlers. More studies are needed to better characterize the risk of lead exposure to older children, particularly in lower-income neighborhoods with a history of lead contamination.

Early exposure to environmental lead is a major ongoing child public health problem in the United States, as suggested for example by events in Flint, Michigan (Hanna-Attisha et al. 2016). Currently available NHANES data has suggested that 2.5% of U.S. children have blood lead levels (BLLs) \geq 5 µg/dL, justifying the designation of this value for determining "elevated." (The value will be updated as new data becomes available.) Two years after changes to the Flint water supply, 6% of children were identified as having elevated BLLs, more than double the rate suggested

Christina Sobin casobin@utep.edu

- ¹ Department of Public Health Sciences, University of Texas, El Paso, 500 West University, El Paso, TX 79968, USA
- ² Border Biomedical Research Center, Toxicology Core, University of Texas, El Paso, El Paso, TX, USA
- ³ Department of Psychology, University of Texas, El Paso, El Paso, TX, USA
- ⁴ Laboratory of Neuroendocrinology, The Rockefeller University, 1230 York Avenue, New York, NY 10065, USA

by the NHANES data. Subsequent investigative reporting by Reuters identified hundreds of cities nationwide with estimated child lead exposure rates far worse than those found in Flint (Pell and Schneyer 2016). One such city was El Paso, Texas. Using aggregated census tract and zip code level data, it was estimated that 10–15% of children living in downtown El Paso had "elevated" blood lead (Pell and Schneyer 2016). Complicating the problem, statistical modeling has suggested that blood lead is either never tested or never reported for hundreds of thousands of children nationwide (Roberts et al. 2017).

It is widely accepted that no level of lead exposure is "safe" for children. Once a child is exposed to lead, there are no interventions available that reverse its damaging physiological effects. Environmental lead source mitigation is the only known method for prevention and intervention; however, national child lead exposure estimates suggest that many high-risk neighborhoods remain un-remediated. Current mitigation policies can vary from state to state, nonetheless focus almost exclusively on homes with children younger than age 5 years. The most common source of child lead exposure continues to be un-remediated or poorly remediated residential lead paint. Old plumbing and historically contaminated soil contribute to a somewhat lesser extent, and all of these tend to occur more frequently in lower-income neighborhoods. Perhaps for this reason, risk of child lead exposure has been associated with socioeconomic and demographic characteristics, such as family income and race/ethnicity (Adler 2005; Kennedy et al. 2016; Morales et al. 2005; Morrison et al. 2013; Sobin et al. 2015), as well as child age (Bose-O'Reilly et al. 2010; Flora et al. 2012; Goyer 1997; Kostial et al. 1978; Lanphear et al. 2002; Morrison et al. 2013) and sex (Chłopicka et al. 1998; Hanna-Attisha et al. 2016; Morrison et al. 2013; Sobin et al. 2015).

Risk of lead exposure is greatest among children younger than age 5 due largely to hand-to-mouth behavior. This assumption has influenced state and local policy guidelines for residential lead mitigation. Many studies have suggested that older children between the ages of 5 and 12 years also are vulnerable to lead exposure and its effects on neurocognitive function. For example, results from studies conducted by our laboratory showed that in more than 600 children ages 5-12 years, living in downtown El Paso neighborhoods, 14% had levels \geq 5 µg/dL; 60% of children had BLLs between 2.5 and 7 µg/dL (Sobin et al. 2009, 2011, 2015). These exposure levels were associated with deficits in motor dexterity and working memory. Another issue concerns the extent to which historically contaminated urban settings continue to pose risk of exposure for children of all ages. Studies are needed to characterize lead exposure in older children living in neighborhoods, particularly those previously identified as "high-risk" for child lead exposure.

This study compared the BLLs of children aged 5–12 years living in an urban neighborhood to the BLLs of demographically matched children living in two nearby rural communities (Fig. 1). The urban neighborhood was one of seven in the downtown region designated by federal and state agencies approximately 30 years earlier as "highrisk" for child lead exposure, largely because of its proximity to a smelter site (Fig. 1). The urban and rural children were matched for age, sex, race, ethnicity, and family income level. We hypothesized that geographic location would predict increased lead exposure in older children and that the BLLs of children living in the previously designated "highrisk" urban neighborhood would have significantly higher BLLs compared with o rural children.

Methods

The studies followed all current standards for human subjects' research and were approved by the University of Texas Institutional Review Board (IRB Protocol #564493-1 and #79085-14), by the El Paso Independent School District Research Board, and by the Canutillo Independent School District Board and Superintendent.

These studies used extant data collected by our laboratory for research on child heavy metal exposure in the southwest United States. Identical methods were used with all study participants, yielding the same demographic, health, and biological data for all children and families. Child participants were recruited from elementary schools in two geographically distinct locations, including one urban area and two adjacent rural townships located within approximately 20 miles of the urban setting. Inclusion criteria were current enrollment at one of the participating elementary schools and being between 5 and 12 years of age. The exclusion criterion was previous diagnosis of lead poisoning (no children had been previously tested positive for lead exposure and none were excluded from participation). Fewer children were available from the rural setting, and to maximize the available samples, data from all of the participating rural children were included (n = 111). Urban child matches were randomly selected according to age (within 6 months), sex, race, ethnicity, and family income level. Children from the rural settings were from two neighboring townships (Rural 1, n=39 and Rural 2, n=72); children from the urban setting were from one elementary school. Figure 1 shows the geographic boundaries of children's urban and rural neighborhoods.

All forms and materials were available in Spanish and English versions. Researchers participating in this study were bilingual and interacted with parents and children in their preferred language throughout the course of the study. Parents were recruited during parent-teacher meetings at the elementary schools; informed consent was obtained at the time of recruitment following full explanation and discussion of the study. Child assent was obtained immediately before the start of child testing. All testing was conducted during physical education periods and included anthropometric measurements and finger stick blood sample collection. Children first washed their hands. Fingers were then wiped with metal-removing towelettes (D-WipeTM, Esca-Tech, Inc. Milwaukee, WI). Saf-T-ProTM 1.8-mm lancets were used to prick the fourth finger of the left hand, and 50 µl of blood was collected into EDTA microvials and refrigerated until analysis at 4 °C. All blood samples were analyzed via inductively coupled plasma mass spectrometry (ICP-MS) and were tested for lead, cadmium, and mercury. Specific methods for the ICP-MS analyses conducted have been reported in detail elsewhere (Sobin et al. 2009, 2011, 2015).



Fig. 1 The map shows the locations of urban and rural neighborhoods from which children in the study were sampled. The urban neighborhood is located in the downtown city center

Statistical Analyses

All variables were examined for outliers and distribution properties. Descriptive statistics were calculated for demographic and clinical characteristics. To determine whether data from the two rural settings could be combined, the demographic, anthropometric, and BLL values ($\mu g/dL$) of children from the two rural schools were

compared using ANOVA. The characteristics of rural children did not differ by site (Table 1), and for the urban/rural comparisons, data from the two rural sites were combined. ANOVA and general linear model regression were used to examine whether geographic location predicted child BLL, controlling for gender and age. For comparison purposes, the same regression models were calculated for cadmium and mercury.

Table 1	Clinical and demographic characteristics of	children in urban and rural settings and	parent's demographic characteristics ($n = 222$)
---------	---	--	--

Variable	Rural 1		Rural 2		Urban	
	Females	Males	Females	Males	Females	Males
	n = 12 (30.8%)	n = 27 (69.2%)	n = 42 (58.3%)	n = 30 (41.7%)	n = 54 (48.6%)	n = 57 (51.4%)
Age (SD)	8.20 (±1.78)	7.86 (±1.79)	8.07 (±1.88)	7.56 (±1.88)	8.18 (±1.79)	7.85 (±1.75)
Weight lbs. (SD)	70.17 (±21.42)	65.76 (±27.09)	65.36 (±27.13)	$64.50(\pm 26.23)$	68.21 (±22.32)	70.59 (±25.64)
Height in. (SD)	51.24 (±4.85)	50.03 (±5.07)	50.05 (±5.15)	50.12 (±4.81)	$51.49 (\pm 6.06)$	$60.39(\pm 4.18)$
BMI (SD)	18.43 (±3.77)	17.80 (±4.11)	17.62 (±3.78)	17.43 (±4.32)	$17.90(\pm 2.87)$	19.35 (±6.37)
Pb µg/dL (SD) (222/222)	$1.00(\pm 0.94)$	1.25 (±1.47)	$1.00 (\pm 0.80)$	$1.17 (\pm 0.90)$	$2.81 (\pm 1.20)$	2.91 (±1.38)
Cd µg/dL (SD) (222/222)	$0.038 (\pm 0.023)$	$0.042 (\pm 0.073)$	$0.029(\pm 0.052)$	$0.033 (\pm 0.056)$	$0.041 (\pm 0.060)$	$0.047 (\pm 0.086)$
Hg µg/dL (SD) (141/222)	$0.025 (\pm 0.0005)$	$0.037 (\pm 0.037)$	$0.037 (\pm 0.017)$	$0.042 (\pm 0.022)$	$0.049(\pm 0.11)$	$0.075(\pm 0.17)$
Mother ethnicity/race (200/222))					
Hispanic (%)	7 (58.3%)	14 (51.9%)	21 (61.8%)	21 (70.0%)	28 (57.1%)	23 (47.9%)
Mexican (%)	3 (25.0%)	7 (25.9%)	10 (29.4%)	4 (13.3%)	18 (36.7%)	18 (37.5%)
Other (%)	2 (16.7%)	8 (29.6%)	3 (8.8%)	5 (16.7%)	3 (6.1%)	7 (14.6%)
White (%)	12 (100%)	27 (100%)	42 (100%)	30 (100%)	54 (100%)	57 (100%)
Mother education level complet	ed (199/222)					
Graduated high school (%)	5 (41.7%)	12 (44.4%)	3 (8.8%)	9 (30.0%)	11 (22.9%)	16 (33.3%)
Completed some college (%)	4 (33.3%)	7 (25.9%)	13 (38.2%)	9 (30.0%)	6 (12.5%)	4 (8.3%)
Other (%)	3 (24.9%)	8 (29.6%)	18 (53.0%)	12 (40.0%)	31 (64.6%)	28 (58.4%)
Father ethnicity/race (175/222)						
Hispanic (%)	4 (33.3%)	12 (46.2%)	20 (60.6%)	20 (69.0%)	19 (48.7%)	18 (50.0%)
Mexican (%)	6 (50.0%)	8 (30.8%)	8 (24.2%)	6 (20.7%)	17 (43.6%)	14 (38.9%)
Other (%)	2 (16.7%)	6 (23.1%)	5 (15.2%)	3 (10.3%)	3 (7.7%)	2 (11.1%)
White (%)	12 (100%)	27 (100%)	42 (100%)	30 (100%)	52 (96%)	54 (95%)
Father education level complete	ed (175/222)					
Graduated high school (%)	4 (33.3%)	13 (50.0%)	9 (27.2%)	5 (17.9%)	9 (26.5%)	7 (19.5%)
Completed some college (%)	4 (33.3%)	3 (11.5%)	2 (6.1%)	5 (17.9%)	5 (14.7%)	5 (13.9%)
Other (%)	4 (33.3%)	10 (38.5%)	22 (66.7%)	18 (64.3%)	20 (58.8%)	29 (80.6%)
Income (189/222)						
=10 K (%)	1 (10.0%)	1 (4.0%)	8 (24.3%)	5 (17.9%)	20 (43.5%)	28 (59.6%)
> 10 and = 20 K (%)	6 (60.0%)	13 (52.0%)	15 (45.5%)	11 (39.3%)	18 (39.1%)	12 (25.5%)
> 20 and $= 30$ K (%)	1 (10.0%)	5 (20.0%)	5 (15.2%)	3 (10.7%)	3 (6.5%)	6 (12.8%)
Other (%)	2 (20.0%)	6 (24.0%)	5 (15.2%)	9 (32.1%)	5 (10.9%)	1 (2.1%)
	To	tal Rural 1	r	Total Rural 2		Total Urban
	n=	=39 (17.6%)	I	n=72 (32.4%)		n = 111 (50.0%)
Age (SD)	7	2.97 (±1.77)		7.86 (±1.88)		8.01 (±1.77)
Weight lbs. (SD)	67	7.12 (±25.28)		$65.00(\pm 26.58)$		$69.43 (\pm 24.00)$
Height in. (SD)	50	0.41 (±4.97)	:	50.08 (±4.97)		50.93 (±5.19)
BMI (SD)	17	7.99 (±3.97)		17.54 (±3.98)		18.64 (±5.02)
Pb µg/dL (SD) (222/222)	1	.18 (±1.32)		$1.07 (\pm 0.84)$		2.86 (±1.29)
Cd µg/dL (SD) (222/222)	0.	041 (±0.062)		$0.031 (\pm 0.053)$		$0.044~(\pm 0.074)$
Hg µg/dL (SD) (141/222)	0.	033 (±0.032)		0.039 (±0.019)		$0.064 (\pm 0.15)$
Household Size	5	5.13 (±1.26)		$5.34(\pm 1.56)$		$5.00(\pm 1.65)$

Results

Table 1 shows the demographic and clinical characteristics of the analyzed sample of 222 children by site. Mean ages for rural Site 1 and Site 2 and Urban were 7.97 ± 1.77 , 7.86 ± 1.88 , and 8.01 ± 1.77 , respectively. Children living in the urban setting had a slightly higher mean body weight compared with children living in the rural sites; mean height was very similar across all sites. Higher mean BMIs were found in children living in the urban setting (18.64 ± 5.02) as compared to rural sites (17.99 ± 3.97 in Rural 1 and 17.54 ± 3.98 in Rural 2). These differences were not statistically significant.

As shown in Table 1, the majority of the children's mothers and fathers in all three sites identified themselves as Hispanic or of Mexican descent, and white. Across all sites, the highest education level for a majority of parents was high school. For all sites, the majority of households had an income of ≤ 20 K per year. The mean number of persons living in households also was similar across all sites (5.13 ± 1.26 Rural 1, 5.34 ± 1.56 Rural 2, and Urban, 5.00 ± 1.65) (the U.S. Federal Poverty Level for 2017 for a

family of 4 was \$24,600). As shown in Table 1, the mean BLL for urban children was higher $(2.86 \pm 1.29 \ \mu g/dL)$ compared with rural children $(1.18 \pm 1.32 \ \mu g/dL$, Rural 1 and $1.07 \pm 0.84 \ \mu g/dL$ Rural 2). The same trends were observed for cadmium and mercury.

To determine whether findings from the two rural settings could be combined for models comparing urban versus rural geographic location, one-way ANOVAs were conducted comparing the two rural sites with regard to child blood lead, cadmium, and mercury levels, controlling for age and gender. As shown in Table 2, the differences in heavy metal levels for the two rural sites were not statistically significant (lead, $F_{1,109} = 0.26$, p = 0.61; cadmium, $F_{1,109} = 0.84$, p = 0.36; mercury, $F_{1,109} = 1.17$, p = 0.28). Data from the two rural sites were combined for the remaining analyses.

Table 3 summarizes the Type III fixed effects and parameter estimates for associations between child BLL and geographic location, controlling for gender and age. Location predicted BLL ($F_{1,221}$ =126.02, p < 0.001). The effects of age ($F_{1,221}$ =1.80, p=0.18) and gender ($F_{1,221}$ =0.740, p=0.39) were not significant predictors of BLL; the interaction of location and gender also was not significant ($F_{1,221}$ =0.120, p=0.73). The nonsignificant effects were dropped from

Metal	Source	df	SS	MS	F	р
Pb	Between groups	1	0.28	0.282	0.26	0.61
	Within groups	109	117.0	1.07		
	Total	110	117.3			
Cd	Between groups	1	0.003	0.003	0.84	0.36
	Within groups	109	0.4	0.003		
	Total	110	0.35			
Hg	Between groups	1	0.001	0.001	1.17	0.28
	Within groups	109	0.07	0.001		
	Total	110	0.07			

Table 3 T	ype III fixed effects
and paran	neter estimates for
associatio	ns between child
blood lead	d levels, controlling for
gender an	d age, living in rural
and urban	settings $(n=222)$

 Table 2
 One-way analysis of variance comparing blood lead, cadmium and mercury levels in children from two rural settings

Type III fixed effect			Solutions for fixed effects							
	F	р		Est	SE	DF	t value	р		
Full model			Intercept	3.29	0.39	1	8.38	0.00		
Age	1.80	0.18	Age	-0.059	0.044	1	-1.34	0.18		
Location	126.01	0.00	Location urban	1.75	0.25	1	-6.93	0.00		
Gender	0.74	0.39	Location rural	0.00	-	-	_	-		
Location × gender	0.12	0.73	Gender male	0.081	0.22	1	0.37	0.72		
			Gender female	0.00	_	_	_	-		
			Location urban × gender male	0.11	0.341	1	0.35	0.73		
			Location urban × gender female	0.00	-	_	-	_		
			Location rural × gender male	0.00	-	-	_	-		
			Location rural × gender female	0.00	-	_	-	_		
Reduced model			Intercept	2.86	0.12	1	23.83	0.00		
Location	125.12	0.00	Location	1.72	0.17	1	-10.12	0.00		

the model and the reduced model was recalculated predicting child BLL from geographic location. In the reduced model, location was a significant predictor of child BLL $(F_{1,221} = 125.12, p < 0.001)$.

Tables 4 and 5 summarize the Type III fixed effects and parameter estimates for associations between child blood cadmium levels and blood mercury levels respectively, controlling for gender, age, and location. The effect of location was not a significant predictor of child blood mercury levels ($F_{1,140}=2.61, p=0.11$). Interestingly, mercury level was predicted by age alone ($F_{1,140}=4.67, p=0.03$); however, the amount of variance explained was negligible.

Multiple linear regression analyses also were conducted to predict blood lead, cadmium, or mercury levels based on location, age, and gender (Table 6). Consistent with the ANOVA results, the regression equation predicting BLL from location was significant ($F_{3,218}$ = 125.13, p < 0.001, R^2 = 0.363). Only location was a significant predictor of BLL in children, p < 0.001. Children living in the urban setting had significantly higher BLLs than children living in the rural area. Also consistent with the ANOVA models, the regression analyses predicting cadmium and mercury from location, controlling for age and gender, were not statistically significant (cadmium, $F_{3,217} = 1.23$, p = 0.27, $R^2 = 0.015$; mercury, $F_{3,137} = 3.36$, p = 0.069, $R^2 = 0.024$).

Discussion

There is heightened national awareness that early chronic lead exposure continues to be a major unresolved pediatric health threat. Remediation and risk abatement policies focus on children below the age of 5 years. It also is critical to understand the risk to children older than 5 years of age. This study compared 5-12-year-old children living in an urban neighborhood designated 30 years earlier as "highrisk" for lead exposure, to demographically matched rural children living 20 miles to the north. The older urban children had significantly higher BLLs. Cadmium and mercury levels were within current limits in all children, and geographic location did not predict cadmium or mercury levels. It is important to note that the 111 urban children were randomly selected for matching from a database of more than 600 tested children and thus did not represent urban children with the highest lead exposures. The BLLs of a majority of children in this study did not exceed 5 μ g/dL.

Type III fixed effect			Solutions for fixed effects							
	III fixed effectSolutions for fixed effectsFpEstSEDmodelIntercept 0.065 0.022 1e 1.42 0.24 Age -0.003 0.002 1cation 1.32 0.25 Location urban -0.1 0.013 1nder 0.38 0.54 Location rural 0.00 $ -$ cation×gender 0.00 0.99 Gender male 0.00 $ -$ Location urban×gender female 0.00 $ -$ Location urban×gender male -0.00007 0.018 1Location urban×gender female 0.00 $ -$ Location rural×gender male 0.00 $ -$	DF	<i>F</i> t value							
Full model			Intercept	0.065	0.022	1	2.93	0.00		
Age	1.42	0.24	Age	-0.003	0.002	1	-1.19	0.24		
Location	1.32	0.25	Location urban	-0.1	0.013	1	-0.8	0.43		
Gender	0.38	0.54	Location rural	0.00	-	-	-	-		
Location × gender	0.00	0.99	Gender male	0.01	0.013	1	0.439	0.66		
			Gender female	0.00	-	_	_	_		
			Location urban × gender male	-0.00007	0.018	1	-0.004	0.99		
			Location urban × gender female	0.00	-	-	-	_		
			Location rural × gender male	0.00	-	-	-	_		
			Location rural × gender female	0.00	-	-	-	-		

Table 5 Type III fixed effects
and parameter estimates
for associations between
child blood mercury levels,
controlling for gender and
age, living in rural and urban
settings $(n=222)$

 Table 4
 Type III fixed effects

 and parameter estimates
 for associations between

 child blood cadmium levels,
 controlling for gender and

 age, living in rural and urban
 for and

settings (n=222)

Type III fixed effect			Solutions for fixed effects							
	F	р		Est	SE	DF	t value	р		
Full model			Intercept	-0.008	0.033	1	-0.23	0.82		
Age	4.67	0.03	Age	0.007	0.003	1	2.16	0.03		
Location	2.61	0.11	Location urban	-0.015	0.022	1	-0.69	0.49		
Gender	1.40	0.24	Location rural	0.00	-	-	_	-		
Location × gender	0.35	0.55	Gender male	0.03	0.026	1	0.99	0.32		
			Gender female	0.00	-	-	_	-		
			Location urban × gender male	-0.017	0.029	1	-0.59	0.55		
			Location urban × gender female	0.00	-	_	_	-		
			Location rural × gender male	0.00	-	_	_	-		
			Location rural \times gender female	0.00	-	_	_	-		

Table 6Blood lead, cadmium,and mercury levels regressedhierarchically on gender, age,and living in rural and urbansettings

Metal	Variable	Model	1		Model 2	Model 2			Model 3			
		В	SE B	β	В	SE B	β	В	SE B	β		
Pb	Location	0.88	0.079	0.6	0.88	0.078	0.61	0.88	0.078	0.60		
	Age				-0.063	0.044	-0.077	-0.059	0.044	-0.073		
	Gender							-0.014	0.16	-0.047		
	R^2		0.36			0.40			0.37			
	F for change in R^2		125.13			2.08			0.74			
	p value		0.00			0.15			0.39			
Cd	Location	0.005	0.004	0.075	0.005	0.004	0.078	0.005	0.004	0.08		
	Age				-0.003	0.002	-0.085	-0.003	0.002	-0.081		
	Gender							-0.006	0.009	-0.042		
	R^2		0.006			0.01			0.015			
	F for change in R^2		1.23			1.60			0.38			
	p value		0.27			0.21			0.54			
Hg	Age	0.007	0.003	0.183	0.007	0.003	0.176	0.007	0.003	0.183		
	Location				0.025	0.14	0.145	0.024	0.014	0.141		
	Gender							-0.012	0.12	-0.086		
	R^2		0.033			0.054			0.062			
	F for change in R^2		4.81			3.05			1.06			
	p value		0.03			0.083			0.305			

509

Similar to hundreds of cities nationwide, the urban locale studied had a well-documented history of lead contamination followed by environmental remediation. A smelter built in 1887 within 1 mile of what eventually became the downtown center, and active until 1999, was one major contamination source. Data collected for a lawsuit in this region initiated in 1970 and claiming violations of the Texas Clean Air Act determined that in one 3-year period, between 1969 and 1971, the smelter had emitted more than 1000 metric tons of lead, 560 tons of zinc, 12 tons of cadmium, and 1 ton of arsenic (Landrigan and Baker 1981). The company declared bankruptcy in August 2005, and when the EPA denied permission to restart the facility in March 2009, the property was placed in an environmental custodial trust. In a December 2009 settlement, the company agreed to pay \$1.79 billion to settle pollution claims at 80 sites in 20 states. Cleanup of the El Paso site began in 2010, the stack was demolished in 2011, and site remediation was completed in late 2016. The BLLs of children in this study suggested that risk of lead exposure continues among children 5-12 years of age.

Implications of Lead Exposure in Older Children

While historical contamination from industrial emissions are relevant in the urban area studied, nationwide lead-based paint remains the most common source of exposure for children (Centers for Disease and Control Prevention 2013). Using data from the Texas Childhood Blood Lead Surveillance Program for El Paso County, the Texas Department of State Health Services showed that the median age of houses (by census tract) predicted child BLLs (Agency for Toxic Substances and Disease Registry 2018). In the urban neighborhood studied, > 80% of residences were built before bans on lead paint were enacted (1978) (Census: El Paso, TX 2014).

Risk of lead exposure to younger children often is attributed to ingestion of paint chips, contaminated household dirt and/or dust, lead-contaminated water, and/or contaminated soil through frequent hand-to-mouth behavior. Remediation efforts attempt to identify these ingestible sources. Lead exposure in children ages 5-12 may instead suggest that exposure sources are from inhaled contaminated dust, ingested contaminated food or water, or exposure from jewelry. Pernicious sources can include leached lead when acid-based foods, such as tomatoes, are prepared with and/or stored in leaded cookware, lead-glazed pottery, and leaded utensils (Landrigan et al. 1975; Morse 1979; Romero 1997). Children's jewelry can have high lead content absorbed through abraded skin or piercings. Particularly for exposure among older children, region-specific factors, such as seasonal wind patterns, bioavailability, and lead source isotype, could help to identify lead sources responsible for child blood levels.

It is important to note that there is growing awareness of the discrepancies between the demonstrated ill effects in children of lower-range BLLs and contamination mandates. For example, on December 29, 2017, the U.S. Court of Appeals for the Ninth Circuit delivered a rarely issued *writ of mandamus* to the EPA. In this action, the EPA was ordered immediately to address the grievances cited in a consumer-generated 2009 petition and, within 90 days of the *writ*, finally establish lower "acceptable" limits of interior lead-paint residue.

Limitations

This study focused only on child BLLs and did not attempt to identify possible sources of exposure in either the urban or rural locales. While previous studies showed that the estimated child BLLs of children in the urban locale were associated with neurocognitive deficits, the child BLLs represented exposure during only one 6-month period. Longitudinal studies are needed to further characterize the effects of lead exposure over time in older children.

Conclusions

Older urban children living in historically contaminated neighborhoods have significant ongoing risk of lead exposure. In hundreds of cities nationwide, exposure to environmental lead is a violation of environmental justice and requires the development of practical systematic approaches for its characterization and remediation (CDC 2013).

Acknowledgements The authors thank Jesus Placencia, M.S., for creation of the map. Funding was provided by National Institute of Child Health and Human Development (Grant No. R21HD060120, CS PI), National Center for Research Resources (Grant No. 5G12RR008124) and J. Edward and Helen M.C. Stern Professorship in Neuroscience (CS).

Compliance with Ethical Standards

Conflict of interest The authors have no potential conflicts of interest to disclose.

Human and Animal Rights The studies followed all current standards for human subjects' research and was approved by the University of Texas Institutional Review Board (IRB Protocol #564493-1 and #79085-14), by the El Paso Independent School District Research Board, and by the Canutillo Independent School District Board and Superintendent.

Informed Consent Informed consent was obtained from all parents before children's participation; child assent was obtained from each child immediately before study participation. The study methods and procedures underwent annual review by University Institutional Review Board.

References

Adler T (2005) Questioning lead standards: even low levels shave points off IQ. Environ Health Perspect 113(7):A473–A474

- Agency for Toxic Substances and Disease Registry (2018) Health consultation: analysis of risk factors for childhood blood lead levels El Paso, Texas, 1997–2002. Accessed 10 Jan 2018. https://www.dshs.texas.gov/epitox/consults/elppasblpbgisfinal4_23.pdf
- Bose-O'Reilly S, McCarty KM, Steckling N, Lettmeier B (2010) Mercury exposure and children's health. Curr Probl Pediatric Adolesc Health Care 40(8):186–215. https://doi.org/10.1016/j.cpped s.2010.07.002
- Census: El Paso, Texas (2014) Accessed 10 Jan 2018. https://www. unitedstateszipcodes.org
- Centers for Disease and Control Prevention (2013) Blood lead levels in children aged 1–5 years—United States, 1999–2010. MMWR. Morbidity and mortality weekly report 62, no. 13 (April 5, 2013): 245–248
- Chinaro K, Yard E, Dignam T, Buchanan S, Condon S, Brown MJ, Raymond J et al (2016) Blood lead levels among children aged < 6 years—Flint, Michigan, 2013–2016. MMWR Morbid Mortal Weekly Rep 65:650–654. https://doi.org/10.15585/mmwr. mm6525e1
- Chłopicka J, Zachwieja Z, Zagrodzki P, Frydrych J, Słota P, Krośniak M (1998) Lead and cadmium in the hair and blood of children from a highly industrial area in Poland. Biol Trace Elem Res 62:229–234. https://doi.org/10.1007/BF02783973
- Gagan F, Gupta D, Tiwari A (2012) Toxicity of lead: a review with recent updates. Interdiscip Toxicol. https://doi.org/10.2478/v1010 2-012-0009-2
- Goyer RA (1997) Toxic and essential metal interactions. Ann Rev Nutr 17:37–50. https://doi.org/10.1146/annurev.nutr.17.1.37
- Hanna-Attisha M, LaChance J, Sadler RC, Schnepp AC (2016) Elevated blood lead levels in children associated with the flint drinking water crisis: a spatial analysis of risk and public health response. Am J Public Health 106:283–290. https://doi. org/10.2105/AJPH.2015.303003
- Kostial K, Kello D, Jugo S, Rabar I, Maljković T (1978) Influence of age on metal metabolism and toxicity. Environ Health Perspect 25:81–86
- Landrigan PJ, Baker EL (1981) Exposure of children to heavy metals from smelters: epidemiology and toxic consequences. Environ Res 25:204–224. https://doi.org/10.1016/0013-9351(81)90090-6
- Landrigan PJ, Gehlbach SH, Rosenblum BF, Shoults JM, Robert PE, Candelaria M, Barthel WM et al (1975) Epidemic lead absorption near an Ore Smelter: the role of particulate lead. N Engl J Med 292:123–129. https://doi.org/10.1056/NEJM197501162920302
- Lanphear BP, Hornung R, Ho M, Howard CR, Eberly S, Knauf K (2002) Environmental lead exposure during early childhood. J Pediatrics 140:40–47. https://doi.org/10.1067/mpd.2002.120513
- Morales LS, Gutierrez P, Escarce JJ (2005) Demographic and socioeconomic factors associated with blood lead levels among Mexican-American children and adolescents in the United States. Public Health Rep 120:448–454. https://doi.org/10.1177/0033354905 12000412
- Morrison D, Lin Q, Wiehe S, Liu G, Rosenman M, Fuller T, Wang J, Filippelli G (2013) Spatial relationships between lead sources and children's blood lead levels in the urban center of Indianapolis (USA). Environ Geochem Health 35:171–183. https://doi.org/10.1007/s10653-012-9474-y
- Morse DL (1979) El Paso revisited: epidemiologic follow-up of an environmental lead problem. JAMA 242:739. https://doi. org/10.1001/jama.1979.03300080037022
- Pell MB, Schneyer J (2016) The thousands of U.S. locales where lead poisoning is worse than in Flint. Reuters website, December 19, 2016. https://www.reuters.com/investigates/special-report/usalead-testing/
- Roberts EM, Madrigal D, Valle J, King G, Kite L (2017) Assessing child lead poisoning case ascertainment in the US,

1999–2010. Pediatrics 139:e20164266. https://doi.org/10.1542/ peds.2016-4266

- Romero M (1997) The death of Smeltertown: a case study of lead poisoning in a Chicano community. Chicano Stud Surv Anal 115:26–37
- Sobin C, Gutierrez M, Alterio H (2009) Polymorphisms of deltaaminolevulinic acid dehydratase (ALAD) and peptide transporter 2 (PEPT2) genes in children with low-level lead exposure. NeuroToxicology 30:881–887. https://doi.org/10.1016/j.neuro .2009.08.006
- Sobin C, Parisi N, Schaub T, Gutierrez M, Ortega AX (2011) δ-Aminolevulinic acid dehydratase single nucleotide

polymorphism 2 and peptide transporter 2*2 haplotype may differentially mediate lead exposure in male children. Arch Environ Contamin Toxicol 61:521–529. https://doi.org/10.1007/s0024 4-011-9645-3

Sobin C, Flores-Montoya MG, Gutierrez M, Parisi N, Schaub T (2015) δ-Aminolevulinic acid dehydratase single nucleotide polymorphism 2 (ALAD2) and peptide transporter 2*2 haplotype (HPEPT2*2) differently influence neurobehavior in low-level lead exposed children. Neurotoxicol Teratol 47:137–145. https://doi. org/10.1016/j.ntt.2014.12.001