Blood Lead Levels of Refugee Children Resettled in Massachusetts, 2000 to 2007

Katherine W. Eisenberg, PhD, Edwin van Wijngaarden, PhD, Susan G. Fisher, PhD, Katrina S. Korfmacher, PhD, James R. Campbell, MD, MPH, I. Diana Fernandez, MD, PhD, MPH, Jennifer Cochran, MPH, and Paul L. Geltman, MD, MPH

Objectives. We described elevated blood lead level (BLL; ≥10 μg/dL) prevalence among newly arrived refugee children in Massachusetts. We also investigated the incidence of BLL increases and BLLs newly elevated to 20 μg/dL or higher in the year following initial testing, along with associated factors.

Methods. We merged data from the Massachusetts Department of Public Health's Refugee and Immigrant Health Program and the Childhood Lead Poisoning Prevention Program on 1148 refugee children younger than 7 years who arrived in Massachusetts from 2000 to 2007.

Results. Elevated BLL prevalence was 16% among newly arrived refugee children. The rate ratio for BLL elevation to 20 μg/dL or higher after arrival was 12.3 (95% confidence interval [CI]=6.2, 24.5) compared with children in communities the state defines as high-risk for childhood lead exposure. Residence in a census tract with older housing (median year built before 1950) was associated with a higher rate of BLL increases after resettlement (hazard ratio=1.7; 95% CI=1.2, 2.3).

Conclusions. Refugee children are at high risk of lead exposure before and after resettlement in Massachusetts. A national surveillance system of refugee children's BLLs following resettlement would allow more in-depth analysis. (*Am J Public Health.* 2011;101:48–54. doi:10.2105/AJPH.2009.184408)

Refugee children who resettle in the United States may arrive with substantially higher blood lead levels (BLLs) than those among children in the general US population.[1,2] For recently arrived refugees, the risk of additional lead exposure after immigration may also be substantial. In 2000, a 2-year-old Sudanese refugee child died as a result of exposure to lead in her family's New Hampshire home.[3] A subsequent case series among children resettled in New Hampshire raised concerns that African refugee children may be at particularly high risk of lead exposure. In that study, 22 of 71 children (31%) who arrived without an elevated BLL (≥10 μg/dL) had an elevated BLL at follow-up testing, indicating new exposure to lead after immigration.[2]

Preventing postimmigration lead exposure among refugee children is an important public health priority because of the irreversible behavioral and cognitive deficits caused by even low levels of lead exposure in early childhood.[4] Families may be placed in housing with inherently high health risks—generally older, nonrehabilitated housing with lead exposure hazards from deteriorating paint or contaminated soil. Such families may also have difficulty accessing health care over time, so cases of lead poisoning may not be identified. To prevent lead exposure and its adverse consequences in refugee children, information regarding the sources of lead, its prevalence, and the severity of exposure is necessary.

Our primary goal was to describe the risk of elevated BLLs among refugee children upon arrival in the United States and in the period following resettlement. Secondary goals were to determine whether African origin was associated with an increased risk of elevated BLL at initial and follow-up testing and whether residence in housing built prior to 1950 (after which use of lead in residential paint declined) was associated with postimmigration increases in BLL.

METHODS

The term *refugee* is defined by US law (the Immigration and Nationality Act, as amended by the Refugee Act of 1980) as a person who is unable or unwilling to return to his or her country because of a well-founded fear of persecution. Refugees who arrive in the United States are entitled to a free medical screening within 90 days of US arrival (45 CFR §400.107). Young children generally receive lead testing as a part of this screening. The Refugee and Immigrant Health Program (RIHP) of the Massachusetts Department of Public Health maintains a database of information collected during screenings. The Centers for Disease Control and Prevention (CDC) also recommends that refugee children aged 6 months to 6 years receive follow-up lead testing 3 to 6 months after placement in permanent housing.[5] Our study included all children younger than 7 years who (1) arrived in Massachusetts from January 1, 2000, through December 31, 2007, (2) were eligible for refugee medical screening, and (3) had a blood lead test within 90 days of arrival.

Blood Lead Level Measurements

Massachusetts requires reporting of blood lead testing results (105 CMR §460.070). The RIHP records were matched with all available BLLs since immigration as documented in the database of the state's Childhood Lead Poisoning Prevention Program. Specimens were tested at laboratories operating under Clinical Laboratories Improvement Act standards for blood lead analyses. The lower limit of detection for the study period was 2 μg/dL. For BLLs below the detection limit, we replaced values with the detection limit divided by the square root of 2.[6]

To minimize false positives, BLLs obtained via capillary sampling were replaced in the analysis with samples obtained via venipuncture if the venous blood draw was done within 45 days. After replacement, 59 (5%) rather

than 66 of 1148 initial BLLs were obtained from capillary sampling, and 32% of follow-up BLLs were from capillary sampling, compared to 34% of BLLs prior to replacement. The disparity in the proportion of capillary blood draws between initial and follow-up testing is likely because of additional blood work performed during the initial health screening that required a venous blood draw.[7]

Clinical and Demographic Information

Data abstracted from RIHP records included the variables in Table 1. For some children, precise birth dates were not available at the time their overseas documents were prepared for processing. The US Department of Homeland Security typically assigns a birth date of January 1 to meet documentation requirements for US immigration, and it is this date that is in all subsequent records. Consistent with US Department of State practice in 2007, world regions were defined for birth purposes as Africa, East Asia and the Pacific, Europe and Central Asia, Latin America and the Caribbean, and Near East and South Asia.

Children were considered anemic if their hemoglobin values were below age- and ethnicity-specific cutoffs. For children younger than 6 months, the cutoff used was 10.5 g/dL, consistent with the physiologically lower hemoglobin levels that are typical in children younger than 6 months. Cutoffs were 11.0 g/dL for children aged 6 months to 60 months and 11.5 g/dL for children older than 60 months who were not of African nationality, and 1.0 g/dL lower for all children from Africa, regardless of age.[8] People of African descent living in the United States have lower hemoglobin values than do other ethnic groups, and the World Health Organization indicates that the hemoglobin cutoff for anemia should be adjusted downward for people of African descent of all ages.[9] Because of limitations in the database, children who tested negative for pathologic intestinal parasites were grouped in the analysis with children who did not undergo parasite testing.

Growth parameter z scores (body mass index [BMI]-for-age and height-for-age; BMI defined as weight in kilograms divided by height in meters squared) were calculated on the basis of World Health Organization Child Growth Standards by using the Stata igrowup macro version 2 (StataCorp LP, College Station, TX).[10]

TABLE 1—Characteristics of Refugee Children Younger Than 7 Years Who Arrived in Massachusetts From 2000 to 2007 and Received a Blood Lead Test Within 90 Days of Arrival

Characteristic	Initial BLL ≥ 10 µg/dL	Initial BLL < 10 µg/dL	*P*
Total	184 (16)	964 (84)	
Age at arrival			.025
Months, mean (SD)	48.0 (21.3)	43.1 (22.8)	
< 3 y, no. (%)	59 (13)	394 (87)	
≥ 3 y, no. (%)	125 (18)	570 (82)	
Gender, no. (%)			.537
Male	96 (17)	479 (83)	
Female	88 (15)	485 (85)	
Year of arrival,[a] no. (%)			< .001
2007	7 (6)	104 (94)	
2006	13 (13)	91 (88)	
2005	24 (13)	166 (87)	
2004	46 (18)	203 (82)	
2003	28 (23)	92 (77)	
2002	8 (10)	72 (90)	
2001	15 (11)	120 (89)	
2000	43 (27)	116 (73)	
World region of birth,[b] no. (%)			< .001
Europe and Central Asia	24 (6)	362 (94)	
Africa	134 (23)	443 (77)	
East Asia and the Pacific	5 (6)	84 (94)	
Near East and South Asia	9 (17)	43 (83)	
Latin America and the Caribbean	12 (27)	32 (73)	
Median year home built in census tract of residence[c] at first lead test, no. (%)			.767
≥ 1950	45 (16)	233 (84)	
< 1950	136 (17)	666 (83)	
Missing	3 (4)	65 (96)	
Time from arrival in United States to first lead test			.889
Days, mean (SD)	26.1 (16.5)	28.1 (16.4)	
< 4 w, no. (%)	118 (16)	613 (84)	
≥ 4 w, no. (%)	66 (16)	351 (84)	
Initial blood draw, no. (%)			.843
Venous	174 (16)	915 (84)	
Capillary	10 (17)	49 (83)	
Season of initial testing, no. (%)			< .001
November–April (winter)	48 (11)	401 (89)	
May–October (summer)	136 (19)	563 (81)	
Anemia, no. (%)			.024
Yes	45 (21)	172 (79)	
No	120 (14)	710 (86)	
Missing	19 (19)	82 (81)	
Pathologic intestinal parasite, no. (%)			< .001
≥ 1	54 (24)	171 (76)	
0/missing[d]	130 (14)	793 (86)	

Continued

TABLE 1—*Continued*

Height-for-age z score, mean (SD)	–0.8 (1.9)	–0.4 (1.9)	.043
≥ –2	123 (14)	728 (86)	
< –2	38 (20)	154 (80)	
Missing	23 (22)	82 (78)	
BMI-for-age z score, mean (SD)	0.2 (1.5)	0.3 (1.4)	.127
≥ –2	152 (15)	849 (85)	
< –2	9 (21)	33 (79)	
Missing	23 (22)	82 (78)	

Notes. BLL = blood lead level; BMI = body mass index. Sample size was n = 1148. BMI was defined as weight in kilograms divided by height in meters squared.
[a]Percentages may not add to 100 because of rounding.
[b]Based on US Department of State designations of world regions, 2007.
[c]Based on census tract data from 2000 US Census, Summary File 3.[11]
[d]Unable to distinguish between children who were tested and found not to have pathologic intestinal parasites and children who were not tested.

We assumed that height values were measured as recommended by the CDC, with children younger than 2 years measured supine and children aged 2 years and older measured upright. The BMI-for-age z score assessed wasting, and the height-for-age *z* score assessed stunting.

Because of confidentiality concerns, housing age was available only at the census tract level. Median housing age data for census tracts were from the 2000 US Census, Summary File 3.[11] The median year of housing construction in the census tract of residence was analyzed as before 1950 versus 1950 or later.

Data Analysis

Initial lead levels. We calculated the prevalence of elevated initial BLLs in the study population by world region of birth and other characteristics. We used Poisson regression with robust standard errors to estimate prevalence ratios and 95% confidence intervals (CIs) for elevated BLLs comparing African children with children from other world regions, as well as other covariates.[12] Europe and Central Asia was the reference region because it had the lowest prevalence of BLL elevation. Independent variables from Table 1 were included if the variables were associated with elevated initial BLLs at $P \leq .2$ in bivariate analysis. Multivariable models were based on study participants with complete data, as few variables had missing data.

Changes in lead levels after resettlement. We log-transformed the BLLs to approximate a normal distribution. We used a paired *t* test to compare transformed BLLs from initial testing with the first follow-up test, stratified by initial BLL category (<10 μg/dL vs ≥10 μg/dL). To ensure a consistent time frame, we included only follow-up tests performed within a year of initial testing.

We calculated rate ratios to compare the rate of incident cases of BLL 20 μg/dL or higher among the refugee population to the rate among Massachusetts children in the general population, and in traditionally high-risk communities (defined as communities with at least 15 newly confirmed cases of BLL ≥ 20 μg/dL and with a case rate adjusted for income and housing age no less than the state rate of 1.0 for a given 5-year period) from July 2001 to June 2006.[13] Because of comparison data availability, this analysis was limited to children with venous confirmation of the relevant BLLs, and to children aged 6 months to 72 months.

We performed multivariable analysis with a conditional risk set model, an extension of the Cox model that can incorporate multiple events,[14] to test whether housing age and other variables were associated with increases in BLL after resettlement. We defined events as an increase in BLL of 2 μg/dL or more at any time after initial testing. Time to event was the time since US arrival. We used the Efron approximation for failure times and robust variance estimation in all models.[12]

We included age (≥3 years vs <3 years), season of testing, and housing age in the model as time-varying covariates because they could change by lead test. Initial BLLs were entered as categorical (<5 μg/dL, 5–9 μg/dL, or ≥10 μg/dL). We assessed the proportional hazards assumption graphically by plotting log–log plots against analysis time. This assumption was appropriate for all variables associated with initial BLLs in bivariate analysis except for year and birth region, which were consequently not included in the final model. We used Stata version 10.1 (StataCorp LP, College Station, TX) to perform statistical analyses.

RESULTS

A total of 1582 refugee children arrived in Massachusetts during the study period. No lead test was available within 90 days of arrival for 484 children (27%), leaving 1148 children (73%) in the analysis. Of those children, 413 had follow-up BLLs performed within 1 year of the first test. Children who did not have a lead test result available were older (71% aged 3 years and older vs 61% among those who had a BLL reported; $P < .001$) and were more likely to be from Africa ($P < .001$).

Initial Lead Levels and Children's Characteristics

Among children included in the analysis, an average of 144 children arrived each year. Yearly arrivals ranged from 80 in 2002 to a high of 249 in 2004. Most children were born in Africa (50%) or Europe and Central Asia (34%), and most (70%) lived in census tracts with older homes at the time of initial testing.

Sixteen percent of initial BLLs were elevated (Table 1). More children aged 3 years and older had an elevated initial BLL compared with younger children ($P = .025$). Elevated initial BLL was associated with arrival year ($P < .001$), ranging from 27% in 2000 to 6% in 2007, with lower BLLs in later arrival years. Summer testing ($P < .001$), anemia ($P = .024$), and pathologic intestinal parasites ($P < .001$) were associated with initial BLL elevation.

The prevalence ratio of initial BLL elevation for children born in Africa (most often, Somalis and Liberians) was 3.8 (95% CI = 2.3, 6.1) compared with the ratio for children born in Europe and Central Asia (most commonly from the former Soviet Union and the former Yugoslavia; Table 2). Compared with children from Europe and Central Asia, West African

children had a high prevalence of initially elevated BLLs, at 36%, and an adjusted prevalence ratio of 5.6 (95% CI=3.3, 9.3) for initial elevation. Children born in the Near East and South Asia region also had a significantly elevated prevalence ratio at arrival of 3.6 (95% CI=1.9, 7.8). Children who had their first lead test in summer were nearly twice as likely to have an elevated BLL compared with children tested in winter.

Postresettlement Changes in Blood Lead Levels

The 413 children who received follow-up testing within a year arrived with significantly higher BLLs than children who did not receive follow-up testing, with 31.5% versus 7.4% of elevated BLLs at arrival, respectively. The geometric mean was 6.1 μg/dL (95% CI=5.7, 6.5) for those who received follow-up testing compared with 4.5 μg/dL (95% CI=4.3, 4.7) for those without follow-up testing. Children aged 3 years and older were more likely to receive follow-up testing (68% vs 60%; P=.022) compared with children younger than 3 years; children from Africa (73%) and East Asia and the Pacific (77%) were more likely to receive follow-up testing compared with children from Latin America and the Caribbean (63%), the Near East and South Asia (56%), and Europe and Central Asia (46%).

Of those 413 children who received follow-up testing, 24 (6%) had a BLL increase of 5 μg/dL or higher within that year. Among the 283 children who did not have an elevated BLL at initial testing and who received follow-up testing, 21 (7%) had a newly elevated BLL at follow-up. Children who arrived with a BLL 10 μg/dL or higher experienced a statistically significant decrease in geometric mean BLL, with a decline from 14.3 μg/dL (95% CI=13.4, 15.3) at initial testing to 11.0 μg/dL (95% CI=10.1, 11.9) at the first follow-up test (P<.001). Children who arrived with a BLL less than 10 μg/dL also experienced a significant decline (P=.02), from 4.4 μg/dL (95% CI=4.2, 4.7) to 4.1 μg/dL (95% CI=3.9, 4.3).

TABLE 2—Relative Risk Regression Model Prevalence Ratios for Blood Lead Level Elevation at Arrival: Refugee Children Younger Than 7 Years Who Arrived in Massachusetts From 2000 to 2007

Variable	Prevalence Ratio (95% CI)
Age <3 y	0.79 (0.58, 1.08)
Year of arrival	
2007 (Ref)	1.00
2006	2.96 (1.18, 7.42)
2005	2.32 (0.99, 5.45)
2004	2.71 (1.19, 6.15)
2003	3.90 (1.69, 9.00)
2002	2.04 (0.65, 6.40)
2001	2.56 (1.00, 6.59)
2000	6.83 (2.97, 15.70)
World region of birth[a]	
Europe and Central Asia (Ref)	1.00
Africa	3.77 (2.34, 6.09)
East Asia and the Pacific	1.11 (0.38, 3.24)
Latin America and the Caribbean	2.42 (0.83, 7.07)
Near East and South Asia	3.64 (1.85, 7.84)
Median census tract[b] year home built before 1950 at initial testing	0.88 (0.62, 1.23)
Venous blood draw	0.89 (0.40, 2.00)
Testing done May–October	1.96 (1.40, 2.75)
Anemia at arrival	1.10 (0.80, 1.53)
≥1 pathologic intestinal parasite at arrival	1.21 (0.90, 1.65)
Height-for-age z score <−2	1.03 (0.72, 1.47)
BMI-for-age z score <−2	1.25 (0.72, 2.14)

Notes. BMI = body mass index; CI = confidence interval. Sample size was n = 948. BMI was defined as weight in kilograms divided by height in meters squared.
[a]Based on US Department of State designations of world regions, 2007.
[b]Based on census tract data from 2000 US Census, Summary File 3.[11]

Comparisons With Massachusetts Children

The rate of BLLs newly 20 μg/dL or higher was significantly higher among refugee children compared with the population of children living in high-risk communities in Massachusetts

TABLE 3—Rate Ratios for Incident BLLs ≥20 μg/dL With Venous Confirmation Among Refugee Children Aged 6 Months to 72 Months Who Arrived in Massachusetts From 2000 to 2007 Compared With Local Massachusetts Children in General and Those Living in High-Risk Communities

Population	No. Screened per Year	No. With BLL Newly ≥20 μg/dL	Rate per 1000 Children Screened	Rate Ratio (95% CI)
General population	1 161 813	1189	1.0	. . .
High-risk communities (Ref)	415 457	773	1.9	1.0
Refugees	349	8	22.1	12.3 (6.2, 24.5)

Notes. BLL = blood lead level, CI = confidence interval. High-risk communities are defined as those with at least 15 newly confirmed cases of BLLs ≥20 μg/dL and with a case rate adjusted for income and housing age no less than the state rate of 1.0 for a given 5-year period.

TABLE 4—Hazard Ratios Modeling Time to 2 μg/dL Increases in Blood Lead Levels Over Time in the United States: Refugee Children Resettled in Massachusetts, 2000–2007

Variable	Hazard Ratio (95% CI)
Age < 3 y	1.09 (0.82, 1.47)
Median year home built before 1950 in census tract of residence[a] at time of lead testing	1.69 (1.23, 2.33)
Venous blood draw (follow-up tests)	0.79 (0.61, 1.04)
Testing done May–October	2.27 (1.71, 3.03)
Anemia at arrival	1.86 (1.37, 2.53)
≥1 pathologic intestinal parasite at arrival	1.33 (0.97, 1.82)
Height-for-age *z* score < -2	1.14 (0.82, 1.57)
BMI-for-age *z* score < -2	1.51 (0.89, 2.58)
Initial blood lead level	
< 5 μg/dL (Ref)	1.00
5–9 μg/dL	0.86 (0.64, 1.16)
≥10 μg/dL	1.28 (0.89, 1.86)

Notes. BMI = body mass index; CI = confidence interval. Sample size was n = 559; total of 226 events observed. BMI was defined as weight in kilograms divided by height in meters squared.
[a]Based on census tract data from 2000 US Census, Summary File 3.[11]

(Table 3; $P<.001$). Compared with children in high-risk communities in Massachusetts the risk ratio for having a BLL newly 20 μg/dL or higher among refugees with follow-up was 12.3 (95% CI=6.2, 24.5).

Conditional Risk Model

Follow-up ranged from 11 to 2437 days (6.7 years), with a median of 443 days (1.2 years; Table 4). To further explore the effect of birth region, we ran the same model as in Table 4, but using only children born in Africa or Europe and Central Asia. The hazard ratio associated with African birth in that model was 1.5 (95% CI=1.0, 2.1; data not shown).

Older median housing in a child's census tract of residence at the time of blood lead testing was associated with a hazard ratio of 1.7 (95% CI=1.2, 2.3) for a 2-μg/dL increase in BLL between any 2 lead tests. When housing age was entered into the model as initial housing age, rather than as a time-varying covariate, the hazard ratio was 1.2 (95% CI=0.8, 1.8).

As with the model of initial BLLs, season of testing was significantly associated with time to increase in BLL, with a hazard ratio of 2.3 (95% CI=1.7, 3.0) associated with lead tests performed during the summer months. Anemia at arrival was also associated with a significantly increased hazard ratio of 1.9 (95% CI=1.4, 2.5), whereas the remaining covariates (age, pathologic intestinal parasites, initial BLL, venous blood draw at follow-up testing, and nutritional parameters) were not.

DISCUSSION

We update and expand previous work from Massachusetts[1] to reflect the geographic and cultural distribution of refugee arrivals since 2000. Our analysis includes follow-up levels for substantially more children over a longer period than are reflected in the existing literature.

Initial Lead Levels and Correlates

In the United States, children aged 1 to 5 years tested from 1999 to 2004 had a 1.4% prevalence of BLLs 10 μg/dL or higher.[15] In contrast, refugee children who arrived in Massachusetts from 2000 to 2007 had a 16% prevalence of elevated BLLs at arrival, up from 11% in the previous study of Massachusetts refugee arrivals.[1] Initial BLLs primarily reflect overseas exposure to lead. However, the increase in our study more likely suggests a shift toward refugee source countries with more lead hazards rather than any increasing rates of elevated BLLs overseas.

Children born in Africa or the Near East and South Asia had prevalence ratios for initial BLL elevation higher than 3.5 compared with children born in Europe and Central Asia, with a particularly high prevalence (36%) among children from West Africa. However, the minimum prevalence for a world region was 6%, even among children born in lower-risk areas. These results suggest pervasive preimmigration environmental exposures, and indicate a substantially higher burden of lead exposure among refugee children from all world regions compared with the general population of US children.

In sub-Saharan Africa, lead continued to be used in gasoline throughout most of the study period, which could account for much of the lead burden in this population. Lead was removed from gasoline in 2006 from all of sub-Saharan Africa,[16] so we would expect to see lower levels in later years. The lower BLLs among children who arrived in 2007 are consistent with this hypothesis. Other sources of lead in refugee camps likely reflect those in the general population in developing countries, such as industrial activities in residential areas, lead-based paint, and lead-contaminated dust and soil.[17] Exposure can be exacerbated by social and health conditions, including overcrowding, a dusty environment, and iron deficiency, which can enhance lead absorption.

Changes in Blood Lead Levels and Housing Age

Overall, BLLs declined significantly after resettlement for refugee children who arrived in Massachusetts from 2000–2007. However, a subgroup of children experienced BLL increases over time, dramatically so in some cases. Six percent of children receiving a follow-up lead test within a year of their initial test had a BLL increase of 5 μg/dL or higher, indicating a new source of lead exposure after immigration. Even more strikingly, the risk for a recently arrived refugee having a BLL increase to 20 μg/dL or higher in the first 12 to 15 months after arrival was 12 times that of local children of the same age living in high-risk communities in Massachusetts. The fact that 20 of 24 children with a large BLL increase were younger than 3 years is consistent with an increased risk of lead exposure in younger children that peaks at about 2 years of age.[18]

When lead tests were matched with the median age of housing in a child's census tract at the time of testing, residence in a census tract with median housing construction pre-1950

was associated with a 69% increase in the hazard of having an increase in BLL at any time after immigration. This finding has important implications for refugee service providers because it suggests that even if families are placed in lead-safe homes initially, they may move soon after resettlement, in some cases to housing with greater lead hazards. Our ability to detect this increase, even with the inexact tool of census tract-level information, indicates a strong association, despite relatively strict Massachusetts housing laws and regulations (105 CMR §460.000 and MGL 111 §198A-199B) to prevent lead poisoning.

Disparate rates of newly elevated BLL reported in this study and others suggest the importance of differences in population and local environment in determining lead exposure risk. Our data showed a much smaller proportion of newly BLL over time (7%), compared with data from African refugee children who arrived in New Hampshire in 2003 and 2004 (31%).[2] Researchers in Minnesota found an even lower risk of postimmigration lead exposure, with only 1 child who had a newly elevated BLL (0.7%) out of 140 who received follow-up testing, although that study included children aged 16 years or younger.[19]

Limitations

The major limitation of this study is that children who arrived with a higher BLL were more likely to receive follow-up testing than were children who arrived with a lower BLL. This differential follow-up meant that less information was available for children who arrived with a lower BLL, so the postresettlement exposure risk is less well-defined for these children. This lack of precise information could represent a group at lower risk of postresettlement lead exposure if the initial BLL represents an indicator of general risk of lead exposure before or after resettlement. However, the conditional risk model did not demonstrate that either higher or lower initial BLLs were associated with subsequent BLL increases. In addition, we designed our analyses of postresettlement BLLs to focus on changes in BLL over time, rather than on the prevalence of elevated BLLs, to minimize the impact of loss to follow-up on our results.

An additional limitation is the inexact nature of BLL as a reflection of a child's exposure to lead. In particular, uncertainty regarding the half-life of lead in blood in children with unknown exposure chronicity can make it difficult to interpret changes in BLL over time. We considered only increases in BLL in our longitudinal analysis because increases should not occur in the absence of new lead exposure. The downside of this strategy is that we necessarily underestimated the degree of low-level postimmigration exposure reflected in BLLs that remained stable or were slow to decline. Our results should therefore be interpreted as conservative estimates of postimmigration exposure to lead (see Appendix, available as a supplement to the online version of this article at http://www.ajph.org, for further discussion of this issue).

The use of BLLs obtained by capillary sampling is another limitation because capillary sampling is more prone to contamination from skin than is venipuncture, and it can therefore result in artificially high results.[7] However, removing all capillary BLLs from consideration was not desirable because the population that received venous-only BLLs may have been at a higher risk than the overall population. We minimized the effects of potential false-positive results in a number of ways, including replacing capillary BLLs with venous ones when possible and controlling for the type of blood draw in the conditional risk model. The fact that we were able to detect significant associations between some variables and small increases in BLL in the conditional risk model suggests that the use of capillary BLLs introduced a relatively small amount of error into our analysis.

Conclusions

Children from Africa and certain other world regions, including children older than those considered at highest risk in the United States, experience much higher BLLs than do children in the general US population because of more exposure to lead overseas. Refugee children who have recently arrived in the United States are a high-risk group for lead exposure, with high rates of BLL increases compared with rates among US children. Lead paint and dust hazards in housing, anemia, pathologic intestinal parasites, and summer season of arrival all contribute to this risk. To prevent postimmigration lead exposure in refugee children, refugee service providers must ensure prompt initial screening, provide follow-up testing regardless of initial BLL or age, provide safe housing placement initially, and provide families with information on safe housing choices. A surveillance system of refugee children's initial and follow-up BLLs nationally would provide an important source of data that would allow for more in-depth analysis than is available in the existing literature. ■

About the Authors

Katherine W. Eisenberg, Edwin van Wijngaarden, Susan G. Fisher, and I. Diana Fernandez are with the Department of Community and Preventive Medicine, University of Rochester School of Medicine and Dentistry, Rochester, NY. Katrina S. Korfmacher is with the Department of Environmental Medicine, University of Rochester School of Medicine and Dentistry. James R. Campbell is with the Department of Pediatrics, University of Rochester School of Medicine and Dentistry. Jennifer Cochran and Paul L. Geltman are with the Refugee and Immigrant Health Program, Massachusetts Department of Public Health, Jamaica Plain, MA. Paul L. Geltman is also with the Department of Pediatrics, Boston University School of Medicine, Boston, MA.

Correspondence should be sent to Katherine W. Eisenberg, University of Rochester School of Medicine and Dentistry, 601 Elmwood Ave, Box 402, Rochester, NY, 14642 (e-mail: katherine_eisenberg@urmc.rochester.edu). Reprints can be ordered at http://www.ajph.org by clicking the "Reprints/Eprints" link.

This article was accepted April 3, 2010.

Contributors

K.W. Eisenberg originated the study, completed the analysis, and led the writing and revisions. E. van Wijngaarden, S.G. Fisher, J.R. Campbell, K.S. Korfmacher, I.D. Fernandez, J. Cochran, and P.L. Geltman supervised the study and contributed to the study design and analysis. All authors edited and approved the article.

Acknowledgments

The project was supported by National Center for Research Resources (NCRR; grant UL1 RR024160), a component of the National Institutes of Health (NIH) and the NIH Roadmap for Medical Research.

Thanks to the Massachusetts Department of Public Health's Refugee and Immigrant Health Program and Childhood Lead Poisoning Prevention Program for the use of their data and for their invaluable assistance. Thanks in particular to Thinh Nguyen for assistance with data set management.

Note. The article's contents are solely the responsibility of the authors and do not necessarily represent the official view of NCRR or NIH. Information on NCRR is available at http://www.ncrr.nih.gov.

Human Participant Protection

This study was approved by the University of Rochester's Research Subjects Review Board and received approval for public release by the Massachusetts Department of Public Health.

References

1. Geltman PL, Brown MJ, Cochran J. Lead poisoning among refugee children resettled in Massachusetts, 1995 to 1999. *Pediatrics.* 2001;108(1):158–162.

2. Plotinsky RN, Straetemans M, Wong LY, et al. Risk factors for elevated blood lead levels among African refugee children in New Hampshire, 2004. *Environ Res.* 2008;108(3):404–412.

3. Fatal pediatric lead poisoning–New Hampshire, 2000. *MMWR Morb Mortal Wkly Rep.* 2001;50(22): 457–459.

4. Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10μg per deciliter. *N Engl J Med.* 2003;348(16):1517–1526.

5. Refugee Health Guidelines [Centers for Disease Control and Prevention Web site]. Updated February 9, 2009. Available at: http://www.cdc.gov/ncidod/dq/refugee/rh_guide/index.htm. Accessed October 26, 2009.

6. Hornung RW, Reed LD. Estimation of average concentration in the presence of non-detectable values. *Appl Occup Environ Hyg.* 1990;5(1):46–51.

7. Sargent JD, Dalton MA. Rethinking the threshold for an abnormal capillary blood lead screening test. *Arch Pediatr Adolesc Med.* 1996;150(10):1084–1088.

8. Nestel P. Adjusting hemoglobin values in program surveys. Washington, DC: International Nutritional Anemia Consultative Group; 2002:2–3.

9. *Iron Deficiency Anaemia, Assessment, Prevention and Control: a Guide for Programme Managers.* Geneva, Switzerland: World Health Organization; 2001:34.

10. World Health Organization. The WHO Child Growth Standards. 2007. Available at: http://www.who.int/childgrowth/standards/en/index.html. Accessed October 26, 2009.

11. Census 2000 Summary File 3–Massachusetts. Washington, DC: US Census Bureau; 2002.

12. Barros AJ, Hirakata VN. Alternatives for logistic regression in cross-sectional studies: an empirical comparison of models that directly estimate the prevalence ratio. *BMC Med Res Methodol.* 2003;3:21.

13. Childhood Lead Poisoning Screening and Incidence Statistics by Community [Massachusetts Department of Public Health Childhood Lead Poisoning Prevention Program Web site]. Available at: http://www.mass.gov/?pageID=eohhs2terminal&L=5&L0=Home&L1=Researcher&L2=Community+Health+and+Safety&L3=Environmental+Health&L4=Lead+Research+and+Statistics&sid=Eeohhs2&b=terminalcontent&f=dph_environmental_lead_r_screen_stats&csid=Eeohhs2. Accessed August 1, 2008.

14. Prentice RL, Williams BJ, Peterson AV. On the regression analysis of multivariate failure time data. *Biometrika.* 1981;68(2):373–379.

15. Jones RL, Homa DM, Meyer PA, et al. Trends in blood lead levels and blood lead testing among US children aged 1 to 5 years, 1988-2004. *Pediatrics.* 2009;123(3):e376–e385.

16. Timberg C. Era of leaded gas comes to an end in most of Africa. *The Washington Post.* January 1, 2006:A14.

17. Nriagu JO, Blankson ML, Ocran K. Childhood lead poisoning in Africa: a growing public health problem. *Sci Total Environ.* 1996;181(2):93–100.

18. Meyer PA, Pivetz T, Dignam TA, Homa DM, Schoonover J, Brody D. Surveillance for elevated blood lead levels among children–United States, 1997-2001. *MMWR Surveill Summ.* 2003;52(10):1–21.

19. Zabel EW, Smith ME, O'Fallon A. Implementation of CDC refugee blood lead testing guidelines in Minnesota. *Public Health Rep.* 2008;123(2):111–116.