The Association Between State Housing Policy and Lead Poisoning in Children

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ABSTRACT

Objectives. This study examined the effect of an active program of household lead paint hazard abatement, applied over 22 years, on childhood lead poisoning in Massachusetts.

Methods. A small areas analysis was used to compare screening blood lead levels of children in Worcester County, Mass (n = 27 590), with those in Providence County, RI (n = 19 071). Data were collapsed according to census tract.

Results. The percentage of children with lead poisoning (blood lead level ≥ 20 μg/dL [Pc20]) was, on average, 3 times higher in Providence County census tracts (3.2% vs 0.9% in Worcester County census tracts, $P < .0001$), despite similar percentages of pre-1950s housing in both counties. The ratio of Pc20 in Providence vs Worcester County census tracts was 2.2 (95% confidence interval = 1.8, 2.7), after adjustment for differences in housing, sociodemographic, and screening characteristics. This estimate was robust to alternative regression methods and sensitivity analyses.

Conclusions. Massachusetts policy, which requires lead paint abatement of children's homes and places liability for lead paint poisoning on property owners, may have substantially reduced childhood lead poisoning in that state. (*Am J Public Health.* 1999;89:1690–1695)

Blood lead levels in children have decreased dramatically in the past 20 years; data from national samples of children show that the prevalence of blood lead levels of 10 μg/dL or greater in children aged 1 to 5 years fell from 88.2 % in 1976 to 4.4% in 1991–1994.[1] Despite these gains, hazards to children from exposure to lead paint in older, deteriorating housing continue to be a problem, especially in the Northeast and Midwest.[2] This repository of household lead presents a policy problem because of the lack of consensus regarding who should be responsible for the costs of removal. Not surprisingly, lead paint housing policy varies markedly from state to state.

This study was designed to compare childhood lead poisoning in Providence County, RI, and Worcester County, Mass. Both counties have central cities, only 40 miles apart, with between 160 000 and 170 000 persons and similar demographics, housing age, climate, industrial base, and air lead concentrations. Both states have laws mandating universal annual lead screening of children aged 6 to 60 months, but Massachusetts has actively enforced legal requirements for abatement of housing lead paint hazards since 1973. Moreover, Massachusetts enforces a policy of "strict liability," in which property owners may be held accountable for damages to children who acquire lead poisoning in their rental properties. Several highly visible lawsuits have awarded large sums to plaintiffs who were lead poisoned in that state, substantially raising the stakes for property owners who choose not to address lead hazards.

In contrast, Rhode Island had no official policy for the management of lead hazards before 1992; before 1994, lead paint hazards were seldom abated in that state. In addition, Rhode Island law includes the concept of the "innocent owner," which makes it very difficult to find property owners accountable for damages to children. Thus, Rhode Island affords a control for possible effect of Massachusetts housing policy.

In a small areas analysis, we compared the percentage of children with lead poisoning (blood lead level ≥ 20 μg/dL [Pc20]) living in Worcester County census tracts with that of children living in Providence County census tracts. We hypothesized that even after controlling for possible differences in housing and sociodemographics, we would find lead poisoning in Providence County to be more prevalent because of state differences in housing policy.

Methods

Study Sample

Screening was done in a variety of settings, including doctors' offices; community health centers; Women, Infants, and Children programs; and public clinics. In Rhode Island, door-to-door screening also was conducted during the summer in high-risk neighborhoods. We examined lead screening data from both states for calendar year 1994. Fifty-two percent (n = 27 590) of the children in Worcester County and 47% (n = 19 071) of the children in Providence County were screened.

Records for children with multiple tests and multiple identification numbers were merged. Overall, 12.1% of the children in

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Worcester County and 13.9% of the children in Providence County had more than 1 lead test in 1994. The arithmetic mean was used for children who had multiple samples. For children who had both capillary and venous samples, venous blood lead results were used preferentially, with a single venous result preferred over the mean of multiple capillary results.

Laboratory Analysis

The respective state laboratories analyzed more than 80% of the specimens from both counties by graphite furnace atomic absorption spectroscopy. During 1994, each laboratory analyzed 36 specimens with identical blood lead levels through the Centers for Disease Control and Prevention/Wisconsin Proficiency Program; in no case did either laboratory fail. During 1994, we also evaluated the proficiency of both laboratories by sending 9 blood specimens of known lead concentration disguised as clinical specimens.[3]

We found a high correlation ($R^2 = 0.97$) between Massachusetts and Rhode Island results for these 45 specimens. However, the slope coefficient (0.95) indicated that the Rhode Island results were, on average, 5% higher than the Massachusetts results. The upper bounds for the positive Rhode Island (or negative Massachusetts) laboratory bias was 10%.

Geographic Unit of Observation

Addresses for children were geocoded to census tract with Matchmaker/2000 for Windows software (Geographic Data Technology Co, Lebanon, NH). Laboratory results were aggregated by census tract, resulting in population lead exposure values for each of 153 census tracts in Worcester County and 136 census tracts in Providence County. We excluded 15 census tracts in which fewer than 30 children were screened.

Measurement of Average Lead Exposure

An intermediate- to high-dose lead source is usually present in the environment of children with lead poisoning.[4] Multiple epidemiologic studies in the United States and other countries support the association between elevated risk of childhood lead poisoning and lead-based paint sources.[5–14] Moreover, lead can be ingested through household dust[15–17] and soil adjacent to the house[18] that has been contaminated by deteriorating paint. Because household lead paint is the most likely source in children in the United States with blood lead levels of 20 µg/dL or greater, we used the percentage of children with Pe20 to represent the average paint-related lead exposure of children within a census tract.

A secondary outcome variable reported herein is the percentage of children with blood lead levels of 10 µg/dL or greater (Pe10). The Centers for Disease Control and Prevention has suggested that Pe10 be used as a community marker for lead exposure. However, many factors in addition to lead paint might be responsible for mild lead exposure (blood lead level in the 10–19 µg/dL range). Moreover, it is not clear at this time that a paint or dust source within houses can be reliably identified for children who have blood lead levels in this range. Thus, this measure may not be as closely linked to lead paint as Pe20.

Covariates

Despite the many similarities between these counties, several factors could have confounded a possible housing policy effect in this study. We categorized these factors into housing, sociodemographic, and screening program characteristics to be used as independent variables in regression analyses.[19] Census tract demographic and housing data were derived from the 1990 US Bureau of the Census STF 3A tape.[20]

We chose the percentage of houses built before 1950 as a proxy for the amount of lead paint present in a census tract. This date was chosen as a marker for when latex paint rapidly replaced lead-based interior house paint in the US market.[21,22] Another housing characteristic of interest is the percentage of rental housing. It reflects the greater likelihood that paint in old rental housing will be allowed to deteriorate and become accessible to children. A third factor that has been linked to childhood lead exposure is the percentage of houses vacant,[19] also thought to be a marker for housing deterioration.

Because poor people frequently have no choice but to live in the oldest and most dilapidated housing, poverty has long been associated with childhood lead poisoning.[23,24] Census tract measures of poverty include the percentage of households receiving public assistance, median per capita income, percentage of the population that is Black, and percentage of the population that are recent immigrants (immigrants who came to the continental United States between 1985 and 1990). Another variable, percentage of the population that is Hispanic, was excluded because it was highly correlated with percentage of recent immigrants ($r = 0.86$) and because it was not significant in preliminary regression analyses that included recent immigrants as one of the independent variables. We included within-county mobility (percentage of households that moved within the county between 1985 and 1990) because it should affect the chances that a family would live in a house with high lead exposure. Finally, we included the number of persons and the number of houses in the census tract as measures of population size.

Screening penetration, age of the children screened, date of screening, and type of blood sample are all characteristics of the screening programs that could affect aggregate blood lead values. We used the percentage of resident children screened per census tract [(number screened in 1994/1990 census estimates of resident children aged 0 to 5 years) × 100] as a measure of screening penetration. Blood lead levels were highest in children aged 24 to 35 months, so we used the percentage of children screened who were in this age range as a covariate in the analysis.[1] Blood lead levels were highest in the summer, so we used the proportion of samples taken between July 1 and September 30 as a covariate.[7,25] Finally, previous studies have shown that lead level in blood drawn by capillary techniques is biased upward by 0.5 to 1 µg/dL compared with that in venous blood, so we used the proportion of children whose blood lead level was determined by capillary sampling.[21,22,26]

Statistical Analysis

We began with a bivariate comparison of measures of lead exposure in Worcester County and Providence County census tracts with an unpaired t test. We used least squares regression analysis to examine lead exposure, controlling for confounding influences. Logarithmic transformation of all variables was used to normalize distributions and minimize heteroscedasticity in regression analyses. A state dummy variable (0 for Worcester County and 1 for Providence County) was used to model the ratio of Pe20 among children screened in Providence County census tracts to Pe20 among children screened in Worcester County census tracts. The bivariate model can be written as $\ln(\text{Pe20}) = a + b \times (\text{state dummy})$. A model with controls for old housing can be written as $\ln(\text{Pe20}) = a + b \times (\text{state dummy}) + c \times (\ln[\text{percentage of houses built before 1950}])$. In the bivariate model, e^b = Pe20 (Providence County census tracts)/Pe20 (Worcester County census tracts) is an estimate of the ratio of Pe20 in Providence County to that in Worcester County census tracts. In the multivariate model, e^b is a measure of the increased lead exposure in Providence County that remains unexplained after controlling for confounding. We allowed for the addition of covariates in the stepwise model until no fur-

TABLE 1—Mean Values for Aggregate Measures of Lead Exposure in Children Aged 0 to 5 Years Screened in Providence County and Worcester County Census Tracts (CTs)

Aggregate Measure	Mean ± SD	
	Providence (n = 136 CTs)	Worcester (n = 153 CTs)
Blood lead level ≥ 10 μg/dL, %	20.2 ± 12.7	10.3 ± 7.0
Blood lead level ≥ 20 μg/dL, %	3.2 ± 3.6	0.9 ± 1.10
Blood lead level ≥ 30 μg/dL, %	0.8 ± 1.1	0.2 ± 0.51
Arithmetic mean blood level, μg/dL	6.7 ± 2.06	5.4 ± 1.10
Geo mean blood level, μg/dL	6.4	5.3

ther gains were made in the predictive validity of the model and multicolinearity emerged.

Sensitivity Analysis

We tested the robustness of the state dummy variable estimate in the least squares ecological model by reestimating with other dependent variables, including Pe20 and ln(Pe20+1); this model would include communities with no children with Pe20. To test for ecological bias with respect to some variables that were measured at the individual level, we used logistic regression to reestimate the across-county difference in likelihood of lead poisoning; individual children were categorized as 0 (blood lead level < 20 μg/dL) or 1 (Pe20). In this model, individual characteristics (season, age, sex, and sample type) were entered with community characteristics of the census tract of resident children (e.g., age of housing, proportion of rental units, screening penetration). It is not possible to evaluate the effect of clustering on the confidence interval for these community controls, because variance within census tracts is not known. However, our goal was not to estimate the association between community variables and lead poisoning or to evaluate its statistical significance, but only to control for confounding. Measurement error theory indicates that estimates derived from these clustered variables should overadjust for confounding, resulting in a conservative estimate of the across-state adjusted odds ratio.[27]

We also used a series of logistic regression analyses to test whether the higher likelihood of lead poisoning in Providence County children could be sensitive to the following factors: use of an alternative dependent variable (blood lead level < 10 vs ≥ 10 μg/dL), positive bias in the Rhode Island laboratory, greater emphasis on targeted summer screening in Rhode Island, or choice of variable to model old housing risk. To evaluate the effect of a systematic upward bias of the Rhode Island laboratory, we reestimated after multiplying all Rhode Island laboratory results by reduction factors (0.95, 0.90, and 0.85), modeling the effect of possible systematic laboratory biases of 5%, 10%, and 15%, respectively. We examined the effect of targeted summer screening in Providence County by excluding from the analysis all children who were screened from July 1 to September 30 (n = 11 934). Finally, we reanalyzed the results with alternative measures of old housing—percentage of houses built before 1940 and percentage of houses built before 1960.

Results

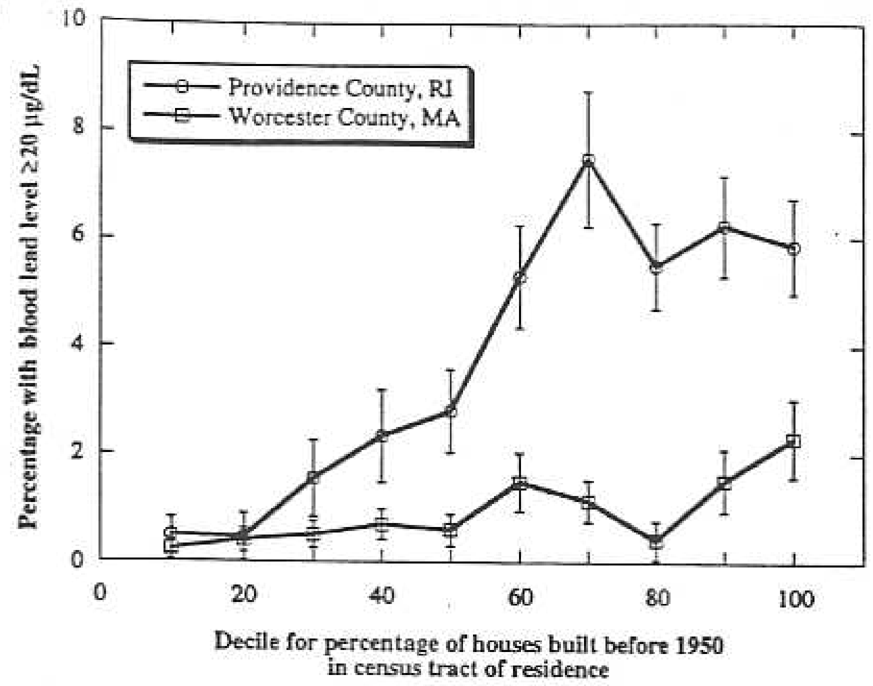
Table 1 shows mean values for aggregate measures of lead exposure for children aged 0 to 5 years screened in Worcester County and Providence County census tracts. However calculated, lead exposure was significantly higher in Providence County. The percentage of children with Pe10 was twice as high in Providence as in Worcester County and 4 times higher in Providence County for cutoff values of 20 and 30 μg/dL. Aggregate lead concentrations were highest among census tracts in the central cities, where differences between the 2 counties are accentuated.

Census tract–level data for housing, screening, and sociodemographic characteristics of each county are shown in Table 2. Age of housing and vacancy rates were similar, but

TABLE 2—Percentage (± Standard Deviation) of Children Aged 0 to 5 Years With Blood Lead Levels of 20 μg/dL or Greater and Sociodemographic and Housing Variables for Providence County and Worcester County Census Tracts (CTs)

Childhood Lead Exposure	Providence (n = 136 CTs)	Worcester (n = 153 CTs)	Comparison P
Children with blood lead level ≥20 μg/dL, %	3.2 ± 3.6	0.9 ± 1.10	<.0001
Housing characteristics			
No. of houses, ×1000	1.8 ± 0.7	1.8 ± 0.7	.7
Pre-1950 housing, %	51.3 ± 22.6	47.6 ± 20.5	.15
Rental housing, %	48.3 ± 24.5	38.7 ± 23.1	.0008
Vacant housing, %	7.2 ± 4.6	6.9 ± 3.8	.51
Screening characteristics			
Resident children screened, %	46.6 ± 16.3	53.1 ± 15.2	.0005
Children aged 24–35 months screened, %	22.5 ± 4.5	23.1 ± 3.5	.19
Children screened during summer months, %	28.0 ± 5.5	27.4 ± 4.3	.33
Blood level determined by capillary sample only, %	49.3 ± 17.1	77.3 ± 10.2	<.0001
Sociodemographic characteristics			
No. of persons, ×1000	4.4 ± 1.7	4.5 ± 1.9	.4
Blacks, % of population	6.0 ± 11.6	1.9 ± 2.9	.0001
Recent immigrants,[a] % of population	3.8 ± 5.2	2.0 ± 2.9	.0006
Family mobility[b]	27.8 ± 6.7	26.6 ± 7.2	.16
Median family income, ×$1000	29.6 ± 10.4	36.2 ± 11.6	<.0001
Households receiving public assistance income, %	10.2 ± 8.3	8.0 ± 7.3	.02

[a]Percentage of the population that immigrated to the United States within the last 5 years (includes people who moved from Puerto Rico).
[b]Percentage of households that moved from one residence to another inside the county within 5 years before 1989.



Note. For the percentage of houses built before 1950, cutoff levels for each decile among census tracts are 10th percentile = 21%, 20th percentile = 27%, 30th percentile = 32%, 40th percentile = 41%, 50th percentile = 50%, 60th percentile = 57%, 70th percentile = 63%, 80th percentile = 70%, 90th percentile = 78%, and 100th percentile = 98%.

FIGURE 1—Percentage of children with blood lead levels of 20 µg/dL or greater within each decile for the percentage of houses built before 1950 in Providence and Worcester Counties.

Figure 1 shows the percentage of children with Pe20 by decile of the percentage of houses built before 1950 for each county. Children in the 10th percentile lived in census tracts where fewer than 21% of the houses were built before 1950, whereas children in the 90th percentile lived in census tracts where more than 78% of the houses were built before 1950. As indicated in Figure 1, the percentage of children with Pe20 was similar across counties for children living in census tracts in the 10th and 20th percentiles. Beyond that, children in Providence County had substantially and significantly higher blood lead levels, as Pe20 climbs to between 6% and 8% for Providence County children living in census tracts beyond the 50th percentile but remains between 0.5% and 2% for children in Worcester County census tracts.

All of the independent variables listed in Table 2 were entered into a stepwise multivariate regression with ln(Pe20) as the dependent variable. Results for the first 5 steps of this procedure are shown in Table 3. Regression 1 included only a dummy variable modeling the Providence vs Worcester County comparison of lead exposure. Without adjustment, Pe20 was, on average, 2.9 times higher in Providence County census tracts. With controls for housing and screening characteristics (4-variable model), Pe20 was 2.2 times higher in Providence County census tracts, and the model predicted 62% of the variance in Pe20 among census tracts. We tested up to 12 independent variables in a full stepwise procedure. Adding additional variables to the 4-variable model did not improve predictive validity and resulted in multicolinearity after more than 6 variables were added. Use of alternative

Providence County census tracts had significantly higher amounts of rental housing. Residents of Providence County census tracts were significantly poorer, with lower median family income and higher rates of public assistance, minorities, and recent immigration than Worcester County census tracts. Worcester County had somewhat higher screening rates and more capillary screening than Providence County.

TABLE 3—Results From Least Squares Multiple Regression Analyses: Children Aged 0 to 5 Years With Blood Lead Levels of 20 µg/dL or Greater (Pe20) Against Sociodemographic and Housing Variables in Providence County and Worcester County Census Tracts

No. of Variables	1	2	3	4	5
Pe20: Providence County/ Worcester County (95% CI)	2.9[a] (2.3, 3.7)	1.8 (1.4, 2.4)	2.4 (1.9, 3.0)	2.2 (1.8, 2.8)	2.2 (1.8, 2.7)
Controls					
Housing characteristics					
ln % pre-1950 housing				0.50	0.51
ln % vacant housing			0.82	0.66	0.64
Screening characteristics					
ln % blood lead level determined by capillary sample		−0.87	−0.29 (.05)	−0.31 (.02)	−0.34 (.01)
ln % screened during summer months					0.31 (.20)
Adjusted R^2	0.31	0.41	0.57	0.62	0.62

Note. CI = confidence interval. Regression 1 represents a crude estimate of Pe20 (Providence County) census tracts/Pe20 (Worcester County) census tracts; equations 2 through 5 represent adjusted estimates after control for housing, sociodemographic, and screening characteristics. Only β coefficients for covariates are shown; all $P < .0001$ unless given in parentheses.

TABLE 4—Sensitivity Analysis: Adjusted Odds Ratios (ORs)[a] for Blood Lead Levels of 20 and 10 µg/dL or Greater (Pe20 and Pe10) Given Residence in Providence County, RI[b]

	Increased Odds of Having Pe20			Increased Odds of Having Pe10		
	OR	95% CI	*P*	OR	95% CI	*P*
Full model[c]	2.8	2.4, 3.4	<.0001	1.8	1.7, 1.9	<.0001
Blood lead level [RI] × 0.95[c]	2.1	1.7, 2.5	<.0001	1.4	1.3, 1.4	<.0001
Blood lead level [RI] × 0.90[c]	1.8	1.5, 2.2	<.0001	1.1	1.0, 1.1	.19
Blood lead level [RI] × 0.85[c]	1.5	1.3, 1.9	<.0001	1.0	1.0, 1.1	.59
Excluding summer screening[d]	2.3	1.9, 2.9	<.0001	1.6	1.5, 1.7	<.0001

Note. CI = confidence interval.

[a]All models include controls for age (5 dummy variables for age in years), sex, season (4 dummy variables for winter [January–March], spring [April–June], summer [July–September], fall [October–December]), sampling method (capillary vs venous), % pre-1950s housing, % rental housing, % vacant housing, % children screened, % Black, % recent immigrants, family mobility, and % households receiving public assistance income.

[b]The full model is followed by 3 models in which blood lead level in Providence County children has been reduced by 5%, 10%, and 15% to model the effects of systematic laboratory error. The final model excludes children who were screened between July and September, the period during which targeted door-to-door screening occurred in Providence County.

[c]n = 43 155.

[d]n = 31 221; summer includes the months July, August, and September.

dependent variables resulted in different combinations of independent variables in stepwise regressions but did not alter the above result.

When the data were reanalyzed at the individual level with a full set of individual and community controls (Table 4), the adjusted odds ratio for Pe20 for Providence County vs Worcester County children was 2.8 (*P* < .0001, n = 43 155). This suggests that ecological bias is, if anything, conservative with respect to variables measured at the individual level (age, sex, season of screening, and type of sample). The odds ratio of a Providence County child having Pe10 was 1.8 (95% confidence interval = 1.7, 1.9). The adjusted odds ratio for Pe20 was robust to a systematic error of 15%. The adjusted odds ratio for Pe10 lost statistical significance for systematic errors of 10% or more. The findings were not substantially affected by the exclusion of children screened during the summer (n = 31 221). Finally, use of other cutoff levels for old housing (1940 or 1960) did not alter the results.

Discussion

This study determined that children screened in Providence County, RI, were significantly more likely to have blood levels of 20 µg/dL or higher than were children in Worcester County, Mass, despite similar age and type of housing and even after adjustment for differences in various individual and community characteristics. The sensitivity analysis indicated that the higher likelihood of lead poisoning in Providence County children is not explained by choice of dependent variable in the community model, choice of regression method, systematic upward bias in the Rhode Island state laboratory results, targeted summer screening in Providence County, or choice of cutoff year for old housing covariates. Moreover, these findings are conservative because the increased use of capillary screening in Massachusetts would be expected to elevate average blood lead levels there as a result of specimen contamination.[21,22,26]

Higher likelihood of lead poisoning in Providence County children is probably not the result of poorer education of parents about lead poisoning in that county. A 1994 survey conducted by the Department of Housing and Urban Development used random-digit-dial methods to sample knowledge about lead poisoning among US households. Twenty-seven percent of the Massachusetts respondents answered all 7 questions about lead hazards correctly, compared with 32.9% of the Rhode Island respondents (B. A. Haley, US Department of Housing and Urban Development, personal communication, January 2, 1997).

We suggest that the risk of lead poisoning may be lower in Massachusetts children because of active enforcement of the state's long-standing housing policy that requires abatement of any lead hazards in homes built before 1978 in which children younger than 6 years live. Abatement is accomplished by permanently covering or removing lead hazards (1) on all moveable or impactable parts of windows, (2) up to 5 feet from the floor and 4 inches from an outside corner on accessible mouthable surfaces, and (3) in loose paint on other surfaces. Abatement using dust-generating methods must be done by individuals certified to perform deleading work.

In Massachusetts, property owners are legally responsible for damage sustained to lead-exposed children.[24] As a corollary to the Massachusetts law, a requirement for obtaining housing subsidies is a certificate that the housing unit to be occupied is free of lead hazards; by 1994, more than 25% of the families receiving public assistance income were living in housing units officially certified as having had their lead paint hazards abated (E. Fitzgerald and S. Snyder, Worcester Housing Authority, unpublished data, July 1997). In each case, abatement of lead paint hazards as a result of legal requirements, as a condition for obtaining liability insurance or to qualify for housing assistance, permanently removes the hazard.

Although few question that the removal of lead from the environment of a growing child is a laudable goal, many are skeptical that abatement of housing lead hazards has a beneficial effect on blood lead levels, especially for children with mild blood lead elevation (10–19 µg/dL). Indeed, studies of the short-term effects of abatement through scraping lead-painted surfaces[13,28] suggested that this activity may result in higher short-term exposure caused by the release of dust.[29] Despite these short-term effects, subsequent tenants may experience lower lead exposure once the dust disperses. Only 1 small study has examined the long-term (>6 months) effects of abatement; 13 homes that had undergone a modified abatement procedure were found to have long-term declines in dust lead levels.[28] More research is needed to identify and refine safe, efficient lead abatement strategies.

We caution against overinterpreting the results of this study. We have identified a difference in blood lead levels between 2 counties that are otherwise similar, but because we did not directly measure lead in housing, we cannot directly confirm that our findings are due to abatement of lead hazards in Massachusetts. Our conclusions related to housing policy are based on the reasonable assumptions that use of lead paint was not greater in Rhode Island and that level of industrial pollution is similar in both cities because of the close proximity and similar industrial history of these counties. Other factors in Massachusetts, such as energy-saving inducements to replace old windows, could have resulted in greater removal of lead paint in Worcester County, independent of lead paint housing policy.

In places where there are large tracts of old lead-painted rental housing units, the prevention of lead poisoning through improvement in housing should be the primary goal of state health policy. Areas at high risk for lead poisoning can be reliably identified with ecological models by using only census data.[19,30–32] We suggest the following areas of focus: (1) creating and enforcing housing regulations that emphasize lead paint abatement; (2) crafting elements to housing code, such as strict liability, so that property owners perceive that it is in their best economic interest to remove lead paint hazards before children become poisoned; (3) improving screening tests for bioavailable lead in houses; (4) initiating routine surveillance of bioavailable lead in high-risk housing; and (5) improving lead abatement and containment strategies.

Contributors

J. D. Sargent, M. Dalton, and R. Z. Klein designed the study and wrote the paper. E. Demidenko analyzed the data. P. Simon contributed to the design of the study and the interpretation of the results.

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References

1. Centers for Disease Control and Prevention. Update: blood lead levels—United States, 1991–1994. *MMWR Morb Mortal Wkly Rep.* 1997;46(7):141–146.
2. Brody DJ, Pirkle JL, Kramer RA, et al. Blood lead levels in the US population: phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1991. *JAMA.* 1994;272:277–283.
3. Sargent JD, Johnson L, Roda S. Large disparities in clinical laboratory performance on the blood lead analysis. *Arch Pediatr Adolesc Med.* 1996;150:609–614.
4. Committee on Environmental Hazards. Statement on childhood lead poisoning. *Pediatrics.* 1987;79:457–465.
5. Lanphear BP, Weitzman M, Eberly S. Racial differences in urban children's environmental exposures to lead. *Am J Public Health.* 1996; 86:1460–1463.
6. Sutton PM, Athanasoulis M, Flessel P, et al. Lead levels in the household environment of children in three high-risk communities in California. *Environ Res.* 1995;68:45–57.
7. Schwartz J, Levin R. The risk of lead toxicity in homes with lead paint hazard. *Environ Res.* 1991;54:1–7.
8. Centers for Disease Control and Prevention. Targeted screening for childhood lead exposure in a low prevalence area—Salt Lake City, Utah, 1995–1996. *MMWR Morb Mortal Wkly Rep.* 1997;46(10):213–215.
9. Bates M, Malcolm M, Wyatt R, et al. Lead in children from older housing areas in the Wellington region. *N Z Med J.* 1995;108:400–404.
10. Rabinowitz M, Leviton A, Bellinger D. Home refinishing, lead paint, and infant blood lead levels. *Am J Public Health.* 1985;75:403–404.
11. Gilbert C, Tuthill RW, Calabrese EJ, Peters HA. A comparison of lead hazards in the housing environment of lead poisoned children versus nonpoisoned controls. *J Environ Sci Health.* 1979;A14(3):145–168.
12. Bornschein RL. Influence of social factors on lead exposure and development. *Environ Health Perspect.* 1985;62:343–351.
13. Chisolm JJ, Mellits ED, Quaskey SA. The relationship between the level of lead absorption in children and the age, type, and condition of housing. *Environ Res.* 1985;38:31–45.
14. Clark CS, Bornschein RL, Succop P, Que Hee SS, Hammond PR, Peace B. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. *Environ Res.* 1985;38:46–53.
15. Charney E. Lead poisoning in children: the case against household lead dust. In: Chisolm JJ, O'Hara DM, eds. *Lead Absorption in Children.* Baltimore, Md: Urban & Schwarzenberg; 1979:35–42.
16. Lanphear BP, Weitzman M, Winter NL, et al. Lead-contaminated house dust and urban children's blood lead levels. *Am J Public Health.* 1996;86:1416–1421.
17. Sayre JW, Charney E, Vostal J, Pless IB. House and hand dust as a potential source of childhood lead exposure. *Am J Dis Child.* 1974;127: 167–170.
18. Weitzman M, Aschengrau A, Bellinger D, Jones R, Hamlin JS, Beiser A. Lead-contaminated soil abatement and urban children's blood lead levels [see comments]. *JAMA.* 1993;269:1647–1654.
19. Sargent JD, Bailey A, Simon P, Blake M, Dalton M. Census tract analysis of lead exposure in Rhode Island children. *Environ Res.* 1997;74: 159–168.
20. Available at: http://venus.census.gov/cdrom/lookup/CMD = LIST/DB = C90STF3ALEV = STATE. Accessed October 2, 1997.
21. Sargent JD, Dalton M, Stukel TA, Roda S, Klein RZ. Evaluation of capillary collection methods for blood lead screening in children. *Amb Child Health.* 1995;1:112–122.
22. Schonfeld DJ, Cullen MR, Rainey PM, et al. Screening for lead poisoning in an urban pediatric clinic using samples obtained by fingerstick. *Pediatrics.* 1994;94(2, pt 1):174–179.
23. Lin-fu JS. Vulnerability of children to lead exposure and toxicity. *N Engl J Med.* 1973;289: 1229–1233, 1289–1293.
24. Klein RZ. Lead poisoning. *Adv Pediatr.* 1977; 24:103–130.
25. Hunter JM. The summer disease: an integrative model of the seasonality aspects of childhood lead poisoning. *Soc Sci Med.* 1977;11(14–16): 691–703.
26. Schlenker TL, Fritz CJ, Mark D, et al. Screening for pediatric lead poisoning: comparability of simultaneously drawn capillary and venous blood samples. *JAMA.* 1994;271:1346–1348.
27. Carroll RJ, Ruppert D. *Measurement Error in Nonlinear Models.* London, England: Chapman & Hall; 1995.
28. Farfel MR, Chisolm JJJ. Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. *Am J Public Health.* 1990;80:1240–1245.
29. Amitai Y, Graef JW, Brown MJ, Gerstle RS, Kahn N, Cochrane PE. Hazards of "deleading" homes of children with lead poisoning. *Am J Dis Child.* 1987;141:758–760.
30. Bailey AJ, Sargent JD, Goodman DC, Freeman J, Brown MJ. Poisoned landscapes: the epidemiology of environmental lead exposure in Massachusetts children 1990–1991. *Soc Sci Med.* 1994;39:757–766.
31. Sargent JD, Brown MJ, Freeman JL, Bailey A, Goodman D, Freeman D. Childhood lead poisoning in Massachusetts communities: its association with sociodemographic and housing characteristics. *Am J Public Health.* 1995;85:528–534.
32. Lanphear BP, Byrd RS, Auinger P, Schaffer SJ. Community characteristics associated with elevated blood lead levels in children. *Pediatrics.* 1998;101:264–271.