

The Effectiveness of Housing Policies in Reducing Children's Lead Exposure

ABSTRACT

Mary Jean Brown, RN, ScD, Jane Gardner, RN, ScD, James D. Sargent, MD, Katherine Swartz, PhD, Howard Hu, MD, ScD, MPH, and Ralph Timperi, MPH

Objectives. This study evaluated the relation of housing policies to risk of subsequent lead exposure in addresses where lead-poisoned children had lived.

Methods. Addresses where children with lead poisoning lived between May 1992 and April 1993 were selected from lead screening registries in 2 northeastern states differing in their enforcement of lead poisoning prevention statutes. Blood lead levels of subsequently resident children, exterior condition, tax value, age, and census tract characteristics were collected. The odds of elevated blood lead levels in subsequently resident children were calculated with logistic regression.

Results. The risk of identifying 1 or more children with blood lead levels of 10 $\mu\text{g}/\text{dL}$ or greater was 4 times higher in addresses with limited enforcement. Controlling for major confounders had little effect on the estimate.

Conclusions. Enforcement of housing policies interrupts the cycle of repeated lead exposure. (*Am J Public Health*. 2001;91:621-624)

The influence of lead exposure on children's development and later success is well documented.¹⁻⁴ The risk for lead poisoning is greatest in poor, urban, and minority communities and between 18 and 36 months of age.⁵⁻⁷ Paint, dust, and soil are the most common sources of lead for US children.^{8,9} Studies of efforts to reduce elevated blood lead levels in children by reducing residential contamination indicate that the benefit of intervening when children are already poisoned is small.¹⁰⁻¹² Efforts to prevent lead exposure in high-risk children through dust control measures also have been disappointing.^{13,14} However, the rate of case identification varies greatly between geographic areas with similar housing stock and sociodemographic factors but different capacity to intervene when children are poisoned, which suggests that lead poisoning prevention laws may indeed have a preventive effect.^{15,16}

In this retrospective cohort study, we assessed the risk that in addresses where children had been lead poisoned (blood lead level ≥ 25 $\mu\text{g}/\text{dL}$) in the past, at least 1 subsequently resident child would have a blood lead level of 10 $\mu\text{g}/\text{dL}$ or greater. The study was conducted in adjacent areas in 2 states in the northeastern United States. Enforcement of lead poisoning prevention statutes differed between the states during the 5-year study period (1993-1998). In one state, where such statutes were strictly enforced, addresses where lead-poisoned children lived were subject to enforcement, including criminal and civil penalties against property owners who failed to abate lead hazards. Inspectors reported dangerous levels of lead to the owner, to all tenants at the address, and to the state lead poisoning prevention program. Tenants living in units other than the one inspected were informed of the process for obtaining an inspection of their units. In the other state, with limited enforcement of lead poisoning prevention statutes, inspection was limited to the unit where the poisoned child lived and seldom resulted in lead hazard abatement. Criminal and civil sanctions were not initiated against property owners, and tenants were not notified of the presence of lead hazards.

Methods

Sample Selection

A listing of all addresses with lead-poisoned children (blood lead level ≥ 25 $\mu\text{g}/$

dL) identified between May 1, 1992, and April 30, 1993, was generated from the lead screening registries (N=183). We enrolled addresses where subsequently resident children were tested for lead poisoning between May 1, 1993, and April 30, 1998 (n=143).

For children whose blood lead levels during the study period were below 10 $\mu\text{g}/\text{dL}$, 1 sample per child per year was selected. For children with elevated blood lead levels, all tests following the elevated test were excluded, and records were searched to exclude children whose blood lead levels were elevated before the children moved into the index residence (n=5). Under these criteria, a final sample of 138 addresses (33 strict and 105 limited enforcement) was available for analysis.

Housing Data

Addresses where a child with lead poisoning lived in 1992 were evaluated between August 1998 and March 1999 with an instrument developed by the National Center for Lead Safe Housing. Data were collected regarding the condition of the exterior, grounds, and potential point sources of lead (e.g., metal stamping plants) within 1500 feet of the property. Addresses were scored from 0 to 10 based on the number of exterior elements in disrepair or the presence of a point source. The number of units in the address, the year the structure was built, and the tax valuation were extracted from the tax assessors' records.

Census Data

The census tract of each address was identified, and data associated with community level risk for lead poisoning, including the number of children younger than 7 years, the percentage of Black residents, mobility, the number of households receiving public assistance,

Mary Jean Brown and Ralph Timperi are with the Massachusetts Department of Public Health, State Laboratory Institute, Jamaica Plain, Mass. Mary Jean Brown, Jane Gardner, Katherine Swartz, and Howard Hu are with Harvard School of Public Health, Boston, Mass. James D. Sargent is with Dartmouth Medical School, Lebanon, NH.

Requests for reprints should be sent to Mary Jean Brown, RN, ScD, Department of Maternal and Child Health, Harvard School of Public Health, 677 Huntington Ave, Boston, MA 02115 (e-mail: mbrown@hsph.harvard.edu).

This brief was accepted June 24, 2000.

the age of housing, and tenancy, were extracted from the 1990 census STF 3A file.^{15,16}

Statistical Analyses

Census tract characteristics were compared for addresses where children were tested during the study period. Because the number of addresses in the census tracts varied from 1 to 21, a weight was constructed from the inverse of the variance of the number of addresses to reduce the influence on the group mean of census tracts with only 1 observation.

For the 4 addresses that were vacant lots in 1998, the mean housing condition value, 1 element in disrepair, was imputed. It was assumed that the interior windows were not replaced before demolition. For vacant lots and 8 other addresses, tax values were unknown and the median value of owner-occupied housing for the census tract was used. Models with imputed values did not differ significantly from models in

which missing values were permitted to "float."

A bivariate logistic regression model was fitted to determine the odds that an address would house at least 1 subsequent child with blood lead levels of 10 µg/dL or greater in comparisons of strict and limited enforcement addresses. The model was adjusted for differences between enforcement groups. Three variable models that showed a 10% or greater change in the exposure covariate were classified as potential confounders of the relation. Including an interaction term, exterior condition and wooden exterior, did not improve the fit of the final model (log likelihood ratio test, $P > .10$).

Results

In all, 679 test results from children 6 years or younger were recorded during the study period. In both groups, the median number of children tested was 3 per address.

The mean venous blood lead levels for the children were 7.6 µg/dL (± 4.8 ; $n = 111$) and 8.9 µg/dL (± 7.2 ; $n = 547$) for the strict and limited enforcement addresses, respectively ($P = .02$).

Census Tract Characteristics

For several important census tract characteristics, the mean value of the limited enforcement addresses was significantly different from that of the strict enforcement addresses (Table 1). However, when the values were adjusted with weights to account for the number of addresses in a given census tract (range = 1–21 addresses per census tract), none of the differences in characteristics approached statistical significance.

Address Characteristics

In both groups, most addresses were 3-unit buildings (84% in strict and 87% in limited) built during the 1920s and early 1930s (Table 2). Six addresses (4 strict and 2 limited enforcement, $P = .01$) were built after 1950. Most addresses were in good condition, with an average of 1.3 structural elements in disrepair in strict enforcement addresses and 0.9 structural elements in disrepair in limited enforcement addresses. The tax valuation differed significantly—strict enforcement addresses were valued approximately \$40,000 more on average than were limited enforcement addresses. Limited enforcement addresses were more likely to have wooden exteriors (45 vs 8, $P = .055$) and less likely to have replacement windows ($P = .001$).

New Cases

Limited enforcement addresses were 4.6 (95% confidence interval [CI] = 2.0, 11.0) times more likely to house at least 1 subsequent child with blood lead levels of 10 µg/dL or greater (Table 3). In models that controlled for the major covariates, the risk of identifying at least 1 child with blood lead levels of 10 µg/dL or greater was 4.4 times (95% CI = 1.3, 15.3) higher in limited compared with strict enforcement addresses ($P = .02$). Limited enforcement addresses also were 6.6 (95% CI = 0.85, 51.5) times more likely to house at least 1 child with blood lead levels of 25 µg/dL or greater (data not shown).

Discussion

Despite differences in the assessed value, the exterior conditions of the buildings were very similar. The groups differed by the number of addresses with painted, wooden exteriors and the number of addresses where inte-

TABLE 1—Comparison of Average Census Characteristics^a in Tracts With Addresses With Lead-Poisoned Children in 1992 and Children Tested From 1993 to 1998

Census Variable, 1990	Strict Enforcement (n=27 Census Tracts)	Limited Enforcement (n=34 Census Tracts)
No. of persons, mean (SD)	4279 (± 1672)	4530 (± 1978)
Percentage of persons in urban areas, mean (SD)**	94% ($\pm 14\%$)	99.7% ($\pm 2\%$)
Tracts with proportion of Black residents, no. (%)		
<0.01	8 (30%)	8 (24%)
0.01–0.03	8 (30%)	7 (21%)
>0.03–0.08	8 (30%)	7 (21%)
>0.08	3 (11%)	12 (35%)
No. of children younger than 6 y, mean (SD)	439 (± 225)	478 (± 218)
Percentage of persons older than 6 y living in same house in 1985, mean (SD)	53% ($\pm 10\%$)	50% ($\pm 11\%$)
Median income (SD)**	\$26 933 (± 8340)	\$21 654 (± 7399)
Proportion of households receiving public assistance, no. (%)*		
<0.08	6 (22%)	7 (21%)
0.08–0.15	11 (41%)	5 (15%)
>0.15–0.22	5 (19%)	11 (32%)
>0.22	5 (19%)	11 (32%)
Proportion of units in buildings with >4 housing units, no. (%)		
<0.14	7 (26%)	10 (29%)
0.14–0.25	5 (19%)	8 (24%)
>0.25–0.39	7 (26%)	8 (24%)
>0.39	8 (30%)	8 (24%)
Median year built (SD)	1943 (± 8)	1946 (± 11)
No. of units built before 1950, mean (SD)	1147 (± 499)	1113 (± 477)
Percentage of owner-occupied units (SD)	40% ($\pm 21\%$)	34% ($\pm 17\%$)

^aThe number of addresses in census tracts ranged from 1 to 21. Weights based on the inverse of the variance would reduce the influence of census tracts with few addresses. When weights were used to compare the enforcement areas, none of the characteristics were statistically different.

**t* test for means or χ^2 for categoric variables, comparison $.05 > P \leq .15$.

***t* test for means or χ^2 for categoric variables, comparison $P \leq .05$.

TABLE 2—Housing Characteristics in 1998 for 133 Units^a With Children Tested in 1993 to 1998, by Enforcement Status

	Strict Enforcement (n=33), No.(%)	Limited Enforcement (n=105), No.(%)
Vacant lot in 1998	2 (6%)	3 (3%)
Type of exterior*		
Wood	8 (26%)	45 (44%)
Other	23 (74%)	57 (56%)
Interior windows**		
>Half old	9 (29%)	63 (62%)
Replaced/new	22 (71%)	39 (38%)
Exterior structural element in disrepair or missing, mean (SD)	1.3 (±2)	0.9 (±1.6)
Mean tax valuation, \$**		
<63 500	4 (13%)	29 (28%)
63 500–85 600	4 (13%)	29 (28%)
>85 600–116 600	6 (19%)	27 (27%)
>116 600	17 (55%)	17 (17%)
Addresses with 3 dwelling units		
Yes	26 (84%)	89 (87%)
No	5 (16%)	13 (13%)
Built before 1950**		
Yes	27 (87%)	100 (98%)
No	4 (13%)	2 (2%)
Median no. of blood tests per address (n=137)	3.0	3.0
Tests for children aged 18–36 mo (n=137)*		
0	19 (59%)	33 (31%)
1–24	2 (6%)	13 (12%)
25–49	4 (13%)	30 (29%)
50–74	6 (19%)	20 (19%)
>75	1 (3%)	9 (9%)

^aVacant lots (n=5) not included in calculations.

*t test for means or χ^2 for categoric variables, .05 > P ≤ .15.

**t test for means or χ^2 for categoric variables, P ≤ .05.

rior windows had been replaced, both factors related to enforcement. Although these findings suggest that residential lead hazards were more likely in the limited enforcement addresses, the difference in risk of subsequent cases of blood lead elevation was not explained solely by these factors. Nor was it adequately explained by underlying poverty and related sociodemographic differences between the census tracts where the addresses were located.

Public policy is the result of a complex interplay of laws, regulations, and custom. Although policies are implemented across com-

munities, they are designed to influence the lives of individuals. Thus, residents are “exposed” to the public policies in force in their communities. For lead poisoning, these policies include abatement of lead hazards in individual units, property owner liability, notification and referral for services of affected parties, screening, and public education.

The contribution of each factor is not well understood. However, both states in this study had established lead poisoning prevention programs with nearly universal screening and widespread public education. The difference

between the rates of subsequent cases of blood lead elevation in addresses with lead-poisoned children in the past was likely the result of differences in enforcement of state housing statutes. In addition, although a direct association cannot be inferred between the overall prevalence of blood lead levels of 10 $\mu\text{g}/\text{dL}$ or greater and the risk of recurrence in housing with poisoned children, the prevalence of blood lead elevation in the limited enforcement county was approximately twice that in the strict county.^{15,17} This also may reflect the long-term effect of limited enforcement capacity.

This study had several limitations. First, blood lead testing was not controlled by the investigators. Differences in screening procedures may account for some of the differences in case identification, although the number of children tested in an address did not vary by enforcement status. Second, because the concentration of lead in paint was not measured at the addresses, addresses with limited enforcement may have been painted with more highly leaded paint. However, no evidence showed that paint sold in either area varied by lead concentration or that housing in either area was more or less likely to be painted with lead paint.

Our inability to measure some factors, such as race/ethnicity, known to increase risk for lead poisoning at the individual level, was clearly a limitation. However, our goal in this study was not to quantify the contribution of these factors to risk for lead poisoning but to control for confounding. Measurement theory implies that using aggregate values rather than individual values for these factors provides an attenuated estimate of enforcement status.^{18,19} Finally, it is unlikely that unidentified factors both varied significantly between the adjacent areas and were more influential than the covariates evaluated in this and other studies.

Conclusions

This study, to our knowledge the first to evaluate the effectiveness of housing policies

TABLE 3—Adjusted and Unadjusted Odds Ratios (ORs) for Identifying At Least 1 Child With Blood Lead Levels of 10 $\mu\text{g}/\text{dL}$ or Greater, by Enforcement Status

	Strict Enforcement	Limited Enforcement	Unadjusted ^a OR (95% CI)	Adjusted ^b OR (95% CI)
Addresses with ≥ 1 child identified	12 (38%)	77 (73%)	4.6 (2.0, 11.0)	4.4 (1.3, 15.3)
Total no. of addresses	32 ^c	105		

Note. CI=confidence interval.

^aLimited vs strict enforcement addresses.

^bAdjusted for proportion of households in the census tract receiving public assistance, percentage of toddlers living at the address, median census tract income, proportion of Black residents in census tract, and exterior condition of address.

^cOne property where child was tested with a capillary blood sample was excluded.

in reducing lead exposure, suggested that strict enforcement of lead poisoning prevention statutes is an effective primary prevention strategy. It also confirmed health practitioners' experience that lead-poisoned children are identified repeatedly in the same housing. Because relocation of lead-poisoned children is frequently the goal of lead poisoning prevention programs that lack the capacity to enforce abatement, our research also suggests the need to develop address-specific surveillance systems to track the blood lead levels of children living in the housing units. Such surveillance systems would allow programs to evaluate their effectiveness and would serve as lead-safe housing registries. Research regarding factors that influence owners' maintenance practices, including owner occupancy, availability of funding, local enforcement capacity, liability, and the effect of lead hazard reduction on property values, also is needed. □

Contributors

M. J. Brown planned the study, analyzed the data, and wrote the paper. J. Gardner contributed to the study design and implementation, the data analysis, and the writing of the paper. J. D. Sargent contributed to the study design, the interpretation of the data, and the writing of the paper. K. Swartz and H. Hu participated in the study design and the writing of the paper. R. Timperi contributed to the study design, the data analysis, and the writing of the paper.

Acknowledgments

The work was supported in part by the generosity of John and Virginia Taplin.

The authors would like to acknowledge the research assistance of Peter Simon, MD (Family Health Services, Rhode Island Department of Health); Robert Klein, MD (Dartmouth Medical School); and Bela Matyas, MD (Massachusetts Department of Public Health). Joseph Schirmer's (Wisconsin Bureau of Public Health) thoughtful review was greatly appreciated. Sidney Atwood (Harvard School of Public Health)

provided invaluable assistance with computer programming. Sean Carter, MS (Harvard School of Public Health), provided biostatistical consultation. Kevin McCarthy's assistance with environmental assessments and transportation was essential to the project.

The study was approved by the Human Subjects Committees at the Harvard School of Public Health, the Rhode Island Department of Health, and Dartmouth Medical School.

References

1. Needleman HL, Gatsonis CA. Low level lead exposure and the IQ of children: a meta-analysis of modern studies. *JAMA*. 1990;263:673-678.
2. Needleman HL, Schell A, Bellinger D, Leviton A, Allred E. The long term effects of exposure to low doses of lead in childhood: an 11 year follow-up report. *N Engl J Med*. 1990;311:83-88.
3. Bellinger DC, Leviton A, Wateraux C, Needleman H, Rabinowitz M. Longitudinal analysis of prenatal and postnatal lead exposure and early cognitive development. *N Engl J Med*. 1987;316:81-87.
4. White R, Diamond R, Proctor S, et al. Residual cognitive deficits 50 years after lead poisoning during childhood. *Br J Ind Med*. 1993;50:613-622.
5. Pirkle J, Brody D, Gunter E, et al. The decline in blood lead levels in the United States: the National Health and Nutrition Examination Surveys (NHANES). *JAMA*. 1994;272:284-291.
6. Brody DJ, Pirkle JL, Kramer R, et al. Blood lead levels in the US population, phase one of the Third National Health and Nutrition Examination Survey (NHANES III 1988-1991). *JAMA*. 1994;272:277-283.
7. Sargent J, Brown MJ, Freeman JL, et al. Childhood lead poisoning in Massachusetts communities: its association with sociodemographic and housing characteristics. *Am J Public Health*. 1995;85:528-534.
8. *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials*. Atlanta, Ga: Centers for Disease Control and Prevention; 1997.
9. *Comprehensive and Workable Plan for the Abatement of Lead-Based Paint in Privately Owned Housing*. Washington, DC: Dept of Housing and Urban Development; 1990.
10. Farfel MR, Chisolm JJ. Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. *Am J Public Health*. 1990;80:1240-1245.
11. Swindell S, Charney E, Brown MJ, Delaney J. Home abatement and blood lead changes in children with class III lead poisoning. *Clin Pediatr*. 1994;33:536-541.
12. Weitzman M, Aschengrau A, Bellinger D, Jones R, Hamlin J, Beiser A. Lead-contaminated soil abatement and urban children's blood lead levels. *JAMA*. 1993;269:1647-1654.
13. Charney E, Kessler B, Farfel M, Jackson D. Childhood lead poisoning: a controlled trial of the effects of dust control measures on blood lead levels. *N Engl J Med*. 1983;309:1089-1093.
14. Lanphear B, Howard C, Eberly S, et al. Primary prevention of childhood lead exposure: a randomized trial of dust control. *Pediatrics*. 1999;103:772-777.
15. Sargent J, Dalton M, Demidenko E, Simon P, Klein R. The association between state housing policies and lead poisoning in children. *Am J Public Health*. 1999;89:1690-1695.
16. 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.
17. *Massachusetts' Fight Against Childhood Lead Poisoning: Updated Trends, 1993-1998*. Boston: Massachusetts Department of Public Health; 1999.
18. Stefanski LA, Carroll RJ. Covariate measurement error in logistic regression. *Ann Stat*. 1985;13:1335-1351.
19. Carroll RJ, Ruppert D, Stefanski LA. *Measurement Error in Nonlinear Models (Berkson Measurement Error Model)*. London, England: Chapman & Hall; 1995.