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Environmental Research 92 (2003) 182–190

Environmental
Research

<http://www.elsevier.com/locate/envres>

The effect of lead-based paint hazard remediation on blood lead levels of lead poisoned children in New York City[☆]

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Received 5 June 2002; received in revised form 26 December 2002; accepted 17 January 2003

Abstract

Despite the widespread use of lead paint hazard control for children with lead poisoning, few controlled studies that estimate the effect of such control on children's blood lead levels have been published. This retrospective follow-up study examined the effects of lead hazard remediation and its timing on the blood lead levels of lead-poisoned children. From the New York City child blood lead registry, 221 children were selected who had an initial blood lead level of 20–44 µg/dL between 1 July 1994 and 31 December 1996; were 6 months to 6 years of age; had a report of a follow-up blood lead test between 10 and 14 months after the initial test; had a lead-based paint hazard identified in the primary dwelling unit prior to the 10- to 14-month follow-up blood lead test; had resided or spent time at only one address with an identified lead-based paint hazard; and were not chelated. The decline in geometric mean blood lead levels from baseline to 10–14 months later was compared for children whose homes were remediated and whose homes were not remediated during the follow-up period. Regardless of remediation, geometric mean blood lead levels declined significantly from 24.3 µg/dL at the initial diagnosis to 12.3 µg/dL at the 10- to 14-month follow-up blood lead test ($P < 0.01$). Among the 146 children whose homes were remediated the geometric mean blood lead levels declined 53% compared to 41% among the 75 children whose homes were not remediated by the follow-up blood lead test, a remediation effect of approximately 20% ($P < 0.01$). After adjusting for potential confounders, the remediation effect was 11%, although it was no longer significant. Race was the only factor that appeared to confound the relationship: Black children had higher follow-up blood lead levels even after controlling for other factors, including the natural logarithm of the initial blood lead level. The effect of remediation appeared to be stronger for younger (10 to <36 months old) than for older (36 to 72 months old) children ($P = 0.06$). While children in homes with earlier remediation (within less than 3 months) appeared to have greater declines in blood lead levels at the follow-up test than children in homes with later remediation (after 3 or more months), this trend was not significant when controlling for confounding factors. The findings of this study suggest that early identification of lead-poisoned children and timely investigation and abatement of hazards contribute to reducing blood lead levels. However, the apparent effect is modest and further research is needed to systematically test and improve the effectiveness of lead hazard controls.

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Keywords: Abatement; Blood lead levels; Childhood lead poisoning; Lead paint; Lead hazard remediation

[☆]The work for this project was conducted as part of the regular program activities of the Childhood Lead Poisoning Prevention Program (LPPP) at the New York City Department of Health and Mental Hygiene, 253 Broadway, New York, NY 10007. The activities were funded through the regular program budget of LPPP and were conducted in accordance with national and New York City Department of Health guidelines for the protection of human subjects.

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1. Introduction

Interior lead-based paint has been identified as the most common source of childhood lead poisoning in the United States (Lanphear and Roghmann, 1997; Reece et al., 1972; Shannon and Graef, 1992). When such paint

is peeling, cracking, or chipping, or when it is abraded to dust by wear or remodeling, it becomes hazardous to young children, who have frequent hand-to-mouth activity (Chisolm et al., 1985; Clark et al., 1985). The abatement or remediation of lead-based paint hazards is recommended and widely used as part of the management of children with lead poisoning. In a number of studies, declines in blood lead levels after lead hazard reduction were seen for children with initial blood lead levels of 25 µg/dL and above (Amitai et al., 1991; Rosen et al., 1991; Staes et al., 1994; Swindell et al., 1994). However, other studies suggest that lead hazard remediation may not be effective in reducing blood lead levels of children with lower levels (<20 µg/dL) (Aschengrau et al., 1997; Swindell et al., 1994).

In New York City (NYC) from 1994 to 1996, between approximately 1400 and 2000 children were identified each year with blood lead levels ≥ 20 µg/dL. Under the authority of the New York City Health Code, the Department of Health and Mental Hygiene (DOHMH) must inspect and order lead hazard remediation in the homes of these children and require that this remediation be performed safely (City of New York 1992, 1993). The effect of these lead hazard remediation efforts on blood lead levels among NYC children has not yet been investigated. The purpose of this study was to determine (1) the effect of lead hazard remediation as ordered by the NYC DOHMH Lead Poisoning Prevention Program (LPPP) on the blood lead levels of lead-poisoned children, (2) whether the effect varied with the timing of the remediation and (3) other potential modifiers of the remediation effect.

2. Methods

2.1. Study population

Data for this study were obtained from the LPPP's computerized tracking system of blood lead levels and case and environmental management events associated with lead-poisoned children. Selection criteria for this investigation included age between 6 months and 6 years; initial diagnosis of a blood lead level of 20–44 µg/dL between 1 July 1994 and 31 December 1996; report of a follow-up blood lead test between 10 and 14 months after the initial test; and the identification of lead-based paint hazards in the primary dwelling unit prior to the 10- to 14-month follow-up blood lead test. The specified time period ensured that we included only children who were identified as lead poisoned after mandatory safety regulations became effective in 1994 (New York City Health Code, City of New York, 1993). To allow for the comparison of baseline blood lead levels with follow-up blood lead levels obtained during the same season, children were identified who had at least one blood lead

test collected between 10 and 14 months after the baseline test. There were 941 children who met these criteria. However, 720 of them were excluded for one or more of the following reasons: they had been chelated; they had more than one address on the computer system; or they had moved from their primary residence before the 10- to 14-month follow-up blood lead test.

There were no statistically significant differences in follow-up blood lead levels, racial/ethnic distribution, or gender between the 221 study group children and the 720 excluded children. Study group children had significantly lower initial blood lead levels (geometric mean: 24.8 vs 26.2 µg/dL; $P < 0.01$), were significantly older (mean age: 35.5 vs 30.5 months; $P < 0.01$), and were more likely to be diagnosed in the warmer months (73% vs 64%; $P < 0.05$) than the 720 excluded children. Study group children also had a greater mean number of violations identified at their primary residence (26.6 vs 23.6; $P < 0.01$) than the excluded children.

2.2. LPPP investigation and remediation procedures

During the period under study, standard LPPP procedures included an inspection of homes of children <18 years old identified with a blood lead level ≥ 20 µg/dL. All interior components with peeling paint were tested with an X-ray fluorescence (XRF) analyzer (Princeton Gamma-Tech). Each component tested with an XRF reading of 0.7 mg/cm² or higher was classified as positive and considered in violation of the NYC Health Code (City of New York, 1992). Property owners were ordered to perform lead hazard remediation for each of these violations (City of New York, 1993). The allowable methods of lead hazard remediation were specified in these orders and included enclosure of walls with wallboard, sheet rock, plasterboard, or paneling; enclosure or wet scraping and painting of ceilings; replacement or removal of baseboards and moldings; replacement, removal, or enclosure of doors, cabinets, radiators, and non-friction surfaces of windows; and replacement or removal of window friction surfaces. Wet scraping and painting were only allowed in limited circumstances. The following methods of paint removal were prohibited: dry scraping, grinding, or sanding without HEPA exhaust; heat guns above 1100°F; open-flame gas-fired torching; and chemical strippers containing suspected carcinogens.

It was not required that children be relocated during the lead hazard remediation. However, since March 1994, and thus during the period under study, the NYC Health Code has required that the lead hazard remediation be performed safely according to specified methods. These methods included covering or removing furniture, covering floors, sealing the work area, correcting underlying conditions that may cause the

paint to deteriorate, cleaning the work area, performing dust wipe testing to ensure that the area has been properly cleaned, and keeping occupants out of the work area until completion.

A lead-based paint hazard was determined to be remediated when an LPPP inspector visually inspected the dwelling unit and certified that the work had been completed and an independent third party submitted lead dust levels from the floors and windows in the dwelling unit which were below 100 micrograms of lead per square foot ($\mu\text{g}/\text{ft}^2$) on floors; 500 $\mu\text{g}/\text{ft}^2$ on window sills, and 800 $\mu\text{g}/\text{ft}^2$ on window wells (City of New York, 1993).

2.3. Data analysis

To estimate the effect of remediation on blood lead levels, children were categorized into two groups: a remediated group, comprising children whose homes were determined to have been remediated within the period before the follow-up blood test (10–14 months after the diagnosis), and an unremediated group, including those whose remediation was incomplete or whose remediation status was unknown before the follow-up blood lead test. Children in the group with unknown remediation status were those for whom access to their dwelling unit could not be obtained to determine whether the work had been performed.

To investigate the effect of the timing of the remediation on the follow-up blood lead level, children in the remediation group were classified into three categories according to the time between their initial diagnosis of lead poisoning and the determination that their lead hazard violations had been remediated: Less than 3 months; ≥ 3 to ≤ 6 months; and > 6 months.

To obtain crude estimates of the remediation effect and the effect of other potential predictors on the changes in the blood lead levels, two-sided *t* test analyses were used. The initial blood lead level, age, race, and gender of the child, as well as the season of the lead poisoning diagnosis and the number of lead-based paint violations at the primary residence (as indicated by the number of positive XRF readings), were selected as covariates based on their theoretical or previously observed association with elevated blood lead levels in children.

The natural logarithm of blood lead levels was used in all analyses. Analysis of covariance (ANCOVA) was performed to assess the effect of remediation and its timing on follow-up blood lead levels, controlling for initial blood lead levels and potential confounders or modifiers of the remediation effect.

3. Results

The characteristics of the 221 study children are shown in Table 1. Overall, the geometric mean blood

lead level declined from 24.3 to 12.3 $\mu\text{g}/\text{dL}$, a 50% decline between the initial and follow-up testing periods (Table 1). As shown in Table 2, there was a 53% decline in geometric mean blood lead levels among the 146 children in the three groups whose homes were remediated (from 24.6 to 11.6 $\mu\text{g}/\text{dL}$). By contrast, there was only a 41% decline in geometric mean blood lead levels among the 75 children in homes that were unremediated (from 23.8 to 13.9 $\mu\text{g}/\text{dL}$). These differences were significant at $P < 0.01$.

Of the potential confounding factors available, only blood lead level at diagnosis and a child's race were associated with the change in blood lead levels in the crude analyses (Table 2). Children with higher (25–44 $\mu\text{g}/\text{dL}$) initial blood lead levels had a decline of 55% compared to a 46% decline in children with lower (20–24 $\mu\text{g}/\text{dL}$) initial blood lead levels ($P < 0.06$). The percentage declines in geometric mean blood lead level were smaller for Black children (43%) than for Hispanic children (50%) or children in the other ethnic groups (Asian, White, and Unknown: 63%) in crude analyses. The differences in the percentage decline between the race/ethnicity categories were significant ($P < 0.01$). The season of diagnosis, a child's age, a child's gender, and the number of violations were not associated with the percentage change in blood lead levels.

As shown in Table 3, Model 1, in an analysis of covariance controlling for potential confounders

Table 1
Characteristics of the study children ($n = 221$)

Characteristic	
Initial blood lead level ($\mu\text{g}/\text{dL}$)	
Mean $\pm$ SD	24.8 $\pm$ 4.9
Geometric mean $\pm$ geom.SD	24.3 $\pm$ 1.2
Range	20–43
Follow-up blood lead level ($\mu\text{g}/\text{dL}$)	
Mean $\pm$ SD	14.4 $\pm$ 6.8
Geometric mean $\pm$ geom.SD	12.3 $\pm$ 1.9
Range	0–40
Age (in months)	
Mean $\pm$ SD	35.5 $\pm$ 16.3
Range	10–72
Race	
% Black	50.7
% Hispanic	29.9
Gender	
% male	56.1
Season of diagnosis	
% in April–September	73.3
Number of positive XRF measures	
Mean $\pm$ SD	26.6 $\pm$ 18.9
Range	1–99

Table 2

Percentage change in geometric mean blood lead level 10–14 months after diagnosis by selected potential predictors of change

Predictor	Number of children	Baseline geometric mean blood lead level	Follow-up geometric mean blood lead level	Change in geometric mean (95% CI)	<i>P</i> value ^a
Remediation status					
Remediation within <3 months	57	24.9	10.2	–59% (–66%, –50%)	0.006
Remediation within 3–6 months	41	24.5	12.2	–50% (–61%, –36%)	
Remediation after more than 6 months	48	24.4	12.8	–48% (–56%, –38%)	
Remediated (all)	146	24.6	11.6	–53% (–58%, –37%)	
Not remediated	75	23.8	13.9	–41% (–47%, –35%)	
Estimated effect of remediation ^b				–20.3%	
Child's blood lead level at diagnosis					
<25 µg/dL	140	21.7	11.7	–46% (–51%, –40%)	0.057
≥25 µg/dL	81	29.6	13.4	–55% (–61%, –48%)	
Child's age					
<36 months	117	24.7	12.4	–50% (–56%, –43%)	0.813
≥36 months	104	23.9	12.3	–49% (–55%, –42%)	
Child's race					
Black	114	23.7	13.5	–43% (–49%, –37%)	0.002
Hispanic	69	25.4	12.7	–50% (–57%, –43%)	
Other ^c	38	24.5	9.0	–63% (–72%, –52%)	
Child's sex					
Female	97	24.8	12.0	–52% (–57%, –46%)	0.325
Male	124	24.0	12.6	–47% (–53%, –41%)	
Season diagnosis was made					
April–September	162	24.2	12.4	–49% (–53%, –44%)	0.608
October–March	59	24.8	12.1	–51% (–60%, –41%)	
Number of violations documented at the primary address					
<25	115	24.3	11.6	–52% (–58%, –46%)	0.176
≥25	106	24.4	13.1	–46% (–52%, –39%)	

^a Based on *t* test or ANOVA. Dependent variable: $\ln(\text{follow-up blood lead level}) - \ln(\text{initial blood lead level})$.^b Estimated effect of remediation is computed as $[(1 - \% \text{ change remediated}) / (1 - \% \text{ change unremediated}) - 1] \times 100 = [(1 - 0.53) / (1 - 0.41) - 1] \times 100 = 20.3\%$.^c Includes: Asian (*n* = 23), White (*n* = 11), Unknown (*n* = 4).

(natural logarithm of the initial blood lead level, age, race, gender, the number of lead-based paint violations), the adjusted geometric mean of the follow-up blood lead levels of children in remediated homes was 11% lower than that of children in unremediated homes, but the effect was no longer significant. The only variable that remained strongly associated with follow-up blood lead levels was race: White and Asian children had an adjusted geometric mean follow-up blood lead level that was 30% lower than that among Black children ($P < 0.01$).

In the crude analyses, there was a trend in blood lead level decline associated with the timing of remediation, ranging from a 59% decline in children with the earliest remediation (within <3 months) to a 48% decline in children whose homes were remediated after 6 months (Table 2). A similar but nonsignificant effect of early remediation was also seen after controlling for potential confounding factors: Children whose dwellings were documented to be remediated in less than 3 months had adjusted follow-up mean blood lead levels that were 18% lower, on average, than children in unremediated

Table 3

Results of the analysis of covariance including all potential confounders of the remediation effect (Model 1) and age as a modifier of the remediation effect (Model 2)

Model	Regression coefficient (<i>b</i>) [difference in adjusted mean ln follow-up blood lead level]	<i>P</i> value	Difference in adjusted geometric mean follow up blood lead level ^a (95% CI)
Model 1			
$(R^2 = 0.075; F = 2.471; P < 0.05)$			
Intercept	1.524		
ln initial blood lead level	0.386	0.113	
Remediation status ^b	−0.117	0.215	−11% (−26%, 7%)
Number of Violations ^b	−0.082	0.355	−8% (−23%, 10%)
Race1 ^b	−0.080	0.413	−8% (−24%, 12%)
Race2 ^b	−0.362	0.003	−30% (−45%, −11%)
Gender ^b	−0.077	0.383	−7% (−22%, 10%)
Age ^b	−0.010	0.903	−1% (−16%, 17%)
Model 2			
$(R^2 = 0.090; F = 2.626; P < 0.01)$			
Intercept	1.680		
ln initial blood lead level	0.373	0.124	
Remediation status ^b	−0.271	0.030	−24% (−40%, −23%)
Number of violations ^b	−0.099	0.266	−9% (−24%, 8%)
Race1 ^b	−0.065	0.506	−6% (−23%, 14%)
Race2 ^b	−0.360	0.003	−30% (−45%, −11%)
Gender ^b	−0.097	0.271	−9% (−24%, 8%)
Age ^b	−0.232	0.113	−21% (−41%, 6%)
Age by remediation	0.337	0.062	40% (−2%, 100%)

^a The percentage difference in adjusted geometric mean blood lead level is computed as: $(e^{\text{regression coefficient}} - 1) \times 100$.

^b Codes: Remediation status: remediated = 1, not remediated = 0; Number of violations: less than 25 = 1, 25 or more = 0; Race1: Hispanic = 1, Black = 0; Race2: Other = 1, Black = 0; Gender: female = 1, male = 0; Age: 36 months or older = 1, less than 36 months old = 0.

homes. However, children whose homes were remediated 3 months or after but before the follow-up blood lead test had declines of only 8–9% relative to children in unremediated homes (Fig. 1).

The interaction between remediation status and age was also investigated to determine whether a remediation effect was dependent on the age of a child. As shown in Table 3, Model 2, there was a borderline significant interaction between remediation and the age of a child ($P = 0.06$). For younger children, the adjusted geometric mean follow-up blood lead level was 24% lower among those in remediated homes than among those in unremediated homes ($p < 0.05$). However, older children in remediated homes had a nonsignificant increase (7%) in adjusted geometric mean blood lead level compared to those in unremediated homes (Fig. 2). Similar analyses were performed with other potential modifiers of remediation but none approached significance.

4. Discussion

This study suggests that remediation of lead-based paint hazards in homes of children with elevated blood lead levels can have a modest protective effect on blood

lead levels and points to the importance of early identification of children with lead poisoning and prompt remediation of any lead-based paint hazards. Although not significant after adjustment for other factors, children in homes in which remediation was confirmed to have been completed had 1-year follow-up blood lead levels which were 11% lower, on average, than children in homes where remediation was either incomplete or the status was unknown at follow-up (“unremediated” homes). This adjusted remediation effect was similar to the 13% ($p < 0.07$) found by others who investigated children with blood lead levels $\geq 25 \mu\text{g/dL}$ (Staes et al., 1994).

As has been shown in other studies, the blood lead levels of the lead-poisoned children in this study declined on average 50% after a year regardless of remediation status. While children in homes with earlier remediation (within < 3 months) had the greatest declines in blood lead level (59%), even children in unremediated homes had considerable declines (41%). Factors that might contribute to this decline in blood lead levels even in the absence of remediation include aging of the child (and a concomitant reduction in mouthing behaviors), secular trends, regression to the mean, and lead poisoning prevention education to parents and medical providers.

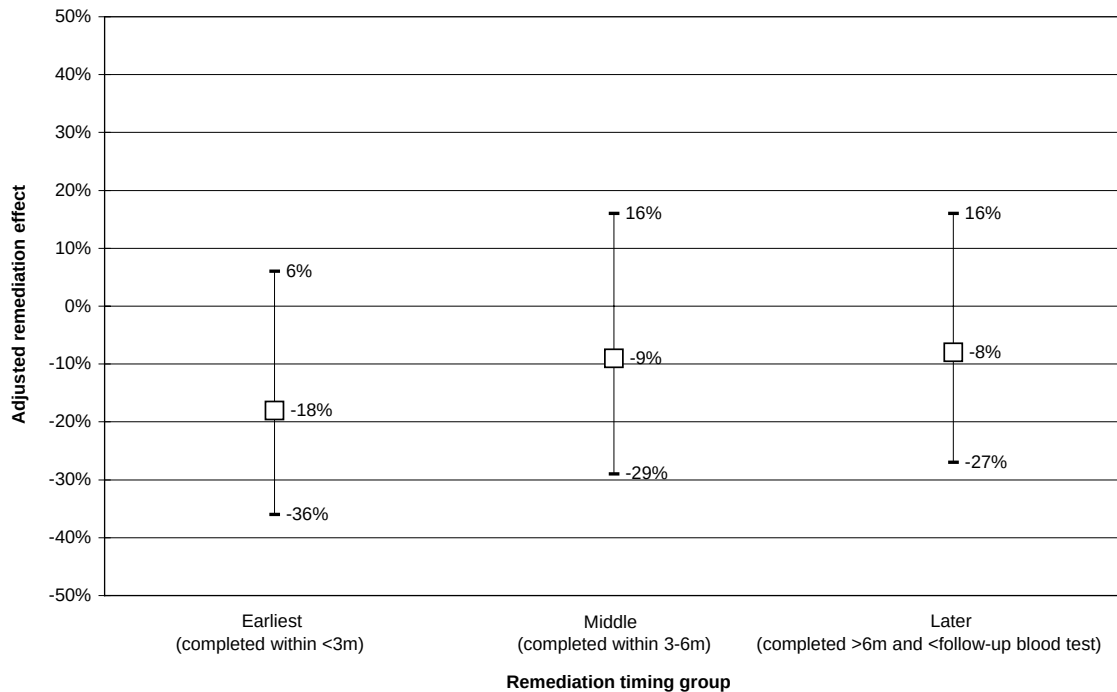


Fig. 1. Effect of remediation on blood lead levels for children receiving earliest, middle and later remediation. The remediation effect in each remediation timing group is the comparison of the adjusted follow-up blood lead levels of children in remediated residences to those of children in unremediated residences. Follow-up blood lead levels are adjusted for initial blood lead levels, number of violations, race, gender, and age. Whiskers represent the 95% confidence interval.

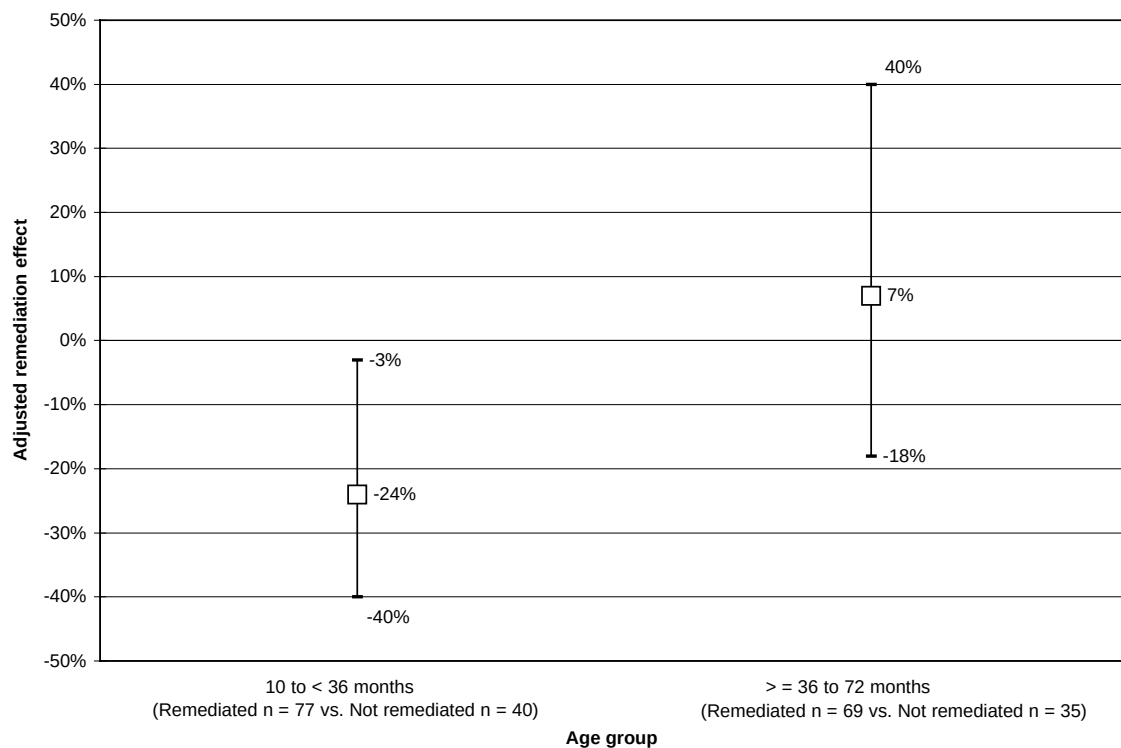


Fig. 2. Remediation effect by age group. The remediation effect in each age group is the comparison of the adjusted follow-up blood lead levels of children in remediated residences to those of children in unremediated residences. Follow-up blood lead levels are adjusted for initial blood lead levels, number of violations, race, and gender. Whiskers represent the 95% confidence interval.

The overall declines observed in this study are substantially greater than declines found by others who investigated follow-up blood lead levels at periods from 6 to 14 months after diagnosis (Amitai et al., 1991; Aschengrau et al., 1997; Charney et al., 1983; Staes et al., 1994; Swindell et al., 1994). Blood lead level changes in these other studies ranged from an increase of 2.6% in children with mean initial blood lead levels of approximately 11 $\mu\text{g}/\text{dL}$ (Aschengrau et al., 1997) to a decline of 29% in children with initial blood lead levels of approximately 36 $\mu\text{g}/\text{dL}$ (Amitai et al., 1991). Except for the studies by Aschengrau et al. and Swindell et al., initial blood lead levels in these children were generally 25 $\mu\text{g}/\text{dL}$ or above (Aschengrau et al., 1997; Swindell et al., 1994).

A number of factors might explain the greater overall blood lead level declines observed in our study compared with those of others. First is the possibility that the effect of regression to the mean was greater in our study than in previous studies. This could occur if the population mean blood lead level in NYC during the time period under study was lower than in other studies. It is likely that due to declining population mean blood lead levels throughout the country over the last 20 years, this did occur. Second, it is also possible that blood lead levels in our study declined more because regulations in NYC since 1994 have required that lead abatement work be performed according to safety requirements and that clearance dust wipe tests must be performed. Third, it is possible that families in NYC were receiving more intensive lead poisoning prevention education than those in other communities, either from their providers or from LPPP staff. Differences in educational interventions among communities would have to be assessed to determine whether this was a possible cause for the greater declines in blood lead levels observed in our study.

Investigators in previous studies have found that the declines in blood lead levels have been greatest for children with the highest initial blood lead levels (Staes et al., 1994; Swindell et al., 1994). In our study, similar results were found in crude analyses, but in the adjusted models the initial blood lead level was not associated with the follow-up blood lead level. Also, after adjusting for potential confounders, the effect of remediation did not appear to depend on the initial blood lead levels. We found a trend toward early (within <3 months after diagnosis) lead hazard remediation being associated with modest reductions in blood lead levels 10–14 months after diagnosis. The greater decline observed for children whose homes were remediated earliest probably reflects the benefit of the longer period of time that the children were removed from the source of the exposure before the follow-up blood lead test. Others (Staes et al., 1994) also found this modest effect of earlier remediation.

Our data also suggest that the benefit of remediation is greater for children <3 years of age than in older children, for whom remediation was actually associated with a small, nonsignificant increase in blood lead levels. This finding of an interaction between abatement and the age of a child has, as far as we know, not been previously identified. It is possible that the blood lead levels of older children would have declined more regardless of remediation, since older children have less of a tendency for hand-to-mouth behavior and therefore have less continued exposure to lead hazards. On the other hand, unless the lead exposure is removed (remediated), younger children are more likely to be continually at risk due to their hand-to-mouth behavior. It is also possible that, compared to preschool-aged children, toddlers have less lead stored in bones and thus can have greater declines once the source is removed.

Race had an important influence on follow-up blood lead levels in this study. Black and Hispanic children had higher follow-up blood lead levels than children of other race/ethnicity groups. This association has not previously been reported. This effect of race may reflect an influence of other socioeconomic factors that were not measured. For example, although the lead poisoning prevention records do not contain information on family income, Black families in New York City are more likely than White or Asian families to live in poverty, and thus it is likely that other factors such as access to preventive services, education, and nutrition rather than race itself contributed to the lesser declines in follow-up blood lead levels. It is also important to note that “other” race was mostly accounted for by Asians. For the Asian children included in this study the average time of remediation was earlier than that for Black or Latino children. Although even when both race and timing of abatement were included in the model, Black children still had higher follow-up blood lead levels than children of other races/ethnicities, it is likely that other, unmeasured factors could eliminate this relationship.

Certain limitations of our study deserve mention. First, some misclassification of remediation status and timing may have occurred. As noted earlier, the unremediated group included some children for whom access could not be obtained to determine whether the remediation had been completed at the time of the 10- to 14-month follow-up blood lead test. It is, therefore, possible that abatement may have actually been completed in some of these dwellings. Misclassification of such children into the unremediated group would have had the effect of minimizing the differences from the other three abatement groups. However, excluding all 21 children for whom remediation status was unknown did not affect the results.

A second concern is the generalizability of these results to the larger population of lead-poisoned

children. The study sample represents only a small fraction (5%) of all lead-poisoned children identified during the time period under study. By further restricting the study sample to those children for whom only one address was identified and inspected and who had a follow-up blood lead test 10–14 months later, the majority of lead-poisoned children was excluded. Whether the remediation effects were consistent for children with multiple addresses is unknown. While it is very possible that similar results would be found among children with multiple addresses which had been remediated, this cannot be confirmed in this study.

A third concern is that even 1 year after the initial diagnosis of a blood lead level $\geq 20 \mu\text{g/dL}$, the geometric mean blood lead levels (BLL) of the 221 children in this study were $\geq 10 \mu\text{g/dL}$. Even with remediation, it may be difficult to reduce BLLs below $10 \mu\text{g/dL}$ within 1 year. Possible explanations for this finding are the body burden of lead and continued exposure to interior lead-contaminated dust or other potential environmental sources of lead. This continuation of elevated blood lead levels even after a year suggests that the most important intervention is the primary prevention of lead poisoning.

The importance of dust control during remediation is paramount. Interior lead-contaminated dust has been consistently associated with measurable elevations in young children's blood lead levels (Lanphear et al., 1996, 1998a,b). The potential for interior lead dust levels to increase, if safe abatement and clean-up procedures are not followed, has been well documented (Amitai et al., 1987; Farfel and Chisolm, 1990; Swindell et al., 1994). At the time this study was conducted (1994–1996), dust lead clearance levels were 100, 500, and $800 \mu\text{g/ft}^2$ on floors, window sills, and window wells, respectively (City of New York, 1993)—levels which have since been associated with measurable increases in children's blood lead levels (Lanphear et al., 1998a,b). Despite requirements for daily and final clean-up, and although independent third-party dust clearance testing was required as part of the remediation process, the actual levels of lead dust before, during, and after remediation were not available for analysis. Thus, objective evaluations of the safety of the work performed by landlords and their contractors were not available for evaluation.

Another limitation of this study is that it did not assess the impact of educational and case management efforts on behavioral change and subsequent blood lead levels. It is possible that the differences between the groups may have been partially explained by differences in education or management.

Lastly, as population blood lead levels decline, proportionally more children are found to have mildly elevated levels. Future studies should assess the effect of remediation for mildly lead-poisoned children.

The findings of this study demonstrate that lead paint hazard remediation has a modest impact on blood lead levels in lead poisoned children. Available evidence suggests that developmental toxicity from lead is greatest when levels are elevated in the early years of life and when high blood lead levels are sustained for longer periods (Shukla et al., 1991; Tong et al., 1996). In addition, it appears that harm from elevated blood lead levels is only partially reversible when blood lead levels later decline (Soong et al., 1999; Tong et al., 1998). To achieve the maximum benefit of remediation, lead-poisoned children must thus be identified at as young an age as possible and remediation must be timely. To better assess and improve the effectiveness of lead hazard controls, additional field-based, longitudinal studies are needed. In addition, to prevent irreversible lead toxicity, strategies and programs for primary prevention of lead poisoning associated with housing should be developed.

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