Long-Term Effect of Dust Control on Blood Lead Concentrations

Bruce P. Lanphear, MD, MPH*; Shirley Eberly, MS§; and Cynthia R. Howard, MD, MPH‡

ABSTRACT. Background. Dust control is recommended to prevent children’s exposure to residential lead hazards, but the long-term effect of dust control on children’s exposure to environmental lead is unknown.

Objective. To determine the effect of dust control on children’s exposure to lead, as measured by blood lead concentration at 48 months of age.

Design. A randomized, controlled trial.


Participants. A total of 275 urban children were randomized at 6 months of age; 189 (69%) were available for the 48-month follow-up blood test.

Intervention. Children and their families were randomly assigned to an intervention group that received cleaning equipment and up to 8 visits by a trained lead hazard control advisor or to a control group. The intervention was terminated when the children were 24 months of age.

Outcome Measures. Geometric mean blood lead concentration and prevalence of elevated blood lead concentration (ie, ≥10 μg/dL, ≥15 μg/dL, and ≥20 μg/dL), by group assignment.

Results. For children with 48-month blood tests, baseline geometric mean blood lead concentrations were 2.8 μg/dL (95% confidence interval [CI]: 2.6,3.0); there were no significant differences in baseline characteristics or lead exposure by group assignment. At 48 months of age, the geometric mean blood lead was 5.9 μg/dL (95% CI: 5.3,6.7) for the intervention group and 6.1 μg/dL (95% CI: 5.5,6.9) for the control group. The percentage of children with a 48-month blood lead ≥10 μg/dL, ≥15 μg/dL, and ≥20 μg/dL was 19% versus 19%, 2% versus 9%, and 1% versus 2% in the intervention and control groups, respectively.

Conclusions. We conclude that dust control, as performed by families and in the absence of lead hazard controls to reduce ongoing contamination from lead-based paint, was not effective in preventing children’s exposure to residential lead hazards. Pediatrics 2000;106(4). URL: http://www.pediatrics.org/cgi/content/full/106/4/e48; blood lead, lead-contaminated house dust, randomized trial, children, environmental exposure, lead poisoning, prevention.

ABBREVIATION. CI, confidence interval.

From the *Children’s Hospital Medical Center, Cincinnati Ohio, and the Departments of ‡Pediatrics and §Biostatistics, University of Rochester School of Medicine and Dentistry, Rochester, New York.

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Reprint requests to (B.P.L.) Children’s Hospital Medical Center, 3333 Burnet Ave, Cincinnati, OH 45229-3039. E-mail: bruce.lanphear@chmcc.org

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Despite the dramatic decline in the prevalence of children having blood lead of 10 μg/dL or higher,1 undue lead exposure remains endemic among children living in some cities, especially those in the Northeastern United States.2,3 Numerous recommendations exist to reduce a child’s risk of exposure to residential lead hazards, such as dust control, close supervision, and hand-washing. But it is uncertain that any of these efforts, when performed by families, are effective at reducing children’s lead exposure, because they fail to repair or remove lead-based paint hazards from the child’s environment.4

The American Academy of Pediatrics and the Centers for Disease Control and Prevention recommend educating families to conduct dust control to reduce children’s exposure to lead-contaminated house dust—a major source of lead intake.5–10 Unfortunately, trials of dust control involving children who had blood lead levels <25 μg/dL have not consistently demonstrated a reduction in blood lead levels.11–14 Rhoads et al11 reported a 17% decline in blood lead concentration among children who received professional cleaning compared with a control group. In contrast, in an earlier analysis of the cohort described in this present study, we found no significant difference in children’s blood lead concentration at 24 months of age in those assigned to a dust control intervention performed by a family member.12 There was, however, a trend toward lower blood lead concentrations in the experimental group. It was unknown, however, if the beneficial effect of dust control would become more evident with extended follow-up.

The purpose of this study was to assess the long-term effect of dust control in preventing children’s exposure to lead, as measured by blood lead concentration at 48 months of age.

METHODS

Children and their families were eligible for the study if: they lived in the city of Rochester, New York; they denied having plans to relocate in the next 3 months; and their child was 6 months of age (± 1 month) at the time of the baseline visit.12 Subjects were identified and recruited by using sequential lists of live births from 5 urban hospitals. After the combined list was checked for errors, the entries were ordered chronologically and current addresses and phone numbers were obtained by using information from 5 hospitals, 4 inner-city clinics, and the Monroe County Department of Social Services and Health Department. To determine eligibility, interviewers dialed each telephone number until the family was contacted or until at least 6 calls were made. Once a family was deemed eligible and agreed to participate, a study team visited their home, obtained a blood sample, conducted an interview, and collected environmental samples.

After baseline sampling, families and their children were ran-
tation compare the proportions of children having blood lead concentra-
using Student's tests, as appropriate. Geometric mean blood lead concentrations of
children in the intervention and control groups were compared
(Table 1). The geometric mean blood lead levels for
children followed until 48 months of age are shown
were compared by
x
2 tests, Fisher's exact tests, and Student’s t
tests, as appropriate. Geometric mean blood lead concentrations of children in the intervention and control groups were compared using Student’s t tests. x2 tests or Fisher’s exact tests were used to
cmare the proportions of children having blood lead concentra-
tion ≥ 5 μg/dL, ≥ 10 μg/dL, ≥ 15 μg/dL, and ≥ 20 μg/dL. All
significance tests were 2-tailed.

RESULTS

Of the 1878 potential subjects in the sampling
frame, we contacted 751 families. Of these, 429 fam-
ilies (57%) were eligible and 275 of the eligible fam-
ilies (64%) agreed to participate in the trial (Fig 1).
One hundred eighty-nine (69%) were available at
48-month follow-up visit. There was no difference in
attrition by study group; 44 of 140 children in the
intervention group (31%) were lost to follow-up
compared with 42 of 135 in the control group (31%).
There were no differences in the 189 children who were
retained in the study by household income,
rental housing, condition of housing, or any of the
environmental lead measures, compared with the 86
children lost to follow-up. There was, however, a
difference in attrition by race. The attrition rate
among black children was 27% versus 38% for chil-
dren of other racial or ethnic backgrounds (P = .044).
Comparisons of baseline characteristics of the 189
children followed until 48 months of age are shown
(Table 1). The geometric mean blood lead levels for
children in the intervention and control groups were
2.7 μg/dL (95% confidence interval [CI]: 2.4,3.1) and
2.9 μg/dL (95% CI: 2.6,3.2), respectively (P = .51).
There were no statistically significant differences in
baseline characteristics by group assignment, but poor
housing condition was marginally more prev-

tent in the control group compared with the inter-
vention group (24% vs 13%, respectively; P = .06).
There was no significant difference in blood lead
concentration by intervention status at 48 months of
age. The geometric mean blood lead concentration for children at 48 months of age in the intervention and control groups were 5.9 μg/dL (95% CI: 5.3,6.7)
and 6.1 μg/dL (95% CI: 5.5,6.9), respectively (P = .73). Adjusting for black race (P = .86) and for bad
housing condition (P = .94) did not alter the effect of
the intervention (Table 2). There was no significant
interaction of black race by dust intervention status
(P = .36) or of housing condition by intervention
status (P = .63).
The prevalence of children with elevated blood
lead concentration (ie, ≥ 5 μg/dL, ≥ 10 μg/dL, ≥ 15
μg/dL, and ≥ 20 μg/dL) at 48 months of age was
generally lower in the intervention group. But these
differences were not significant. There was, however,
a marginal but nonsignificant difference in the per-
centage of children who had a blood lead concentra-
tion exceeding 15 μg/dL at 36 and 48 months of age
(Table 3).

DISCUSSION

The results of this study indicate that despite
efforts to inform families about lead poisoning pre-
vention, recommend cleaning techniques to reduce
lead-contaminated house dust and to provide high-
quality cleaning equipment and supplies, there was
no significant effect of the intervention on children’s
blood lead concentration at 48 months of age. There also were no differences in the percentage of children who had elevated blood lead concentration, with the exception of a marginal reduction in the proportion of children who had a blood lead concentration exceeding 15 mg/dL.

Other controlled trials have not demonstrated a reduction in blood lead levels in children assigned to a dust control intervention. Hilts et al13 conducted a randomized trial of dust control—as performed by a professional cleaner with a HEPA vacuum every 6 weeks over a 10-month period—among 111 children, 6 to 70 months of age. The setting was a community with an active smelter. There was no significant reduction in the blood lead concentrations of children assigned to the intervention group compared with those assigned to the control group.13 In a randomized trial of 94 children who were 12 to 31 months of age, investigators reported no significant difference in blood lead concentrations 7 months after enrollment.14 That intervention, however, consisted of minimal education and provision of paper towels, detergent, and instructions about mopping. No cleaning equipment was provided.14 In our earlier analysis of the cohort presented in this study, we found no significant effect of dust control on children’s blood lead concentrations at 24 months of age.12

In contrast, some controlled trials found that dust control was associated with a significant decline in children’s blood lead concentration. In the earliest controlled trial of dust control, involving 39 children who had blood lead concentration $\geq 30$ mg/dL, Charney et al15 reported a 6.9-mg/dL decline (18%) in blood lead concentration in children assigned to the dust control group.15 This study, however, included abatement in both treatment groups and professional cleaners performed dust control for the 14 children assigned to the experimental group. In a randomized trial of 99 children, investigators reported a 2.1-mg/dL reduction (17%) in blood lead concentrations among children in the intervention group after a median of 17 cleaning visits by a professional cleaning team performed over the 12-month trial.11 For children whose homes were cleaned $\geq 20$ times, there was a 3.9-mg/dL decrease (34%) in blood lead concentration.11

Lead-contaminated house dust is clearly a major source of lead intake for children.9–11 But is dust control effective in reducing childhood lead exposure? Taken together, existing data indicate that there is some benefit if professional cleaners perform dust control. Moreover, because these studies examined the effect of dust control only after children were exposed,11,13–15 they probably underestimated the contribution of lead-contaminated house dust to

### TABLE 1. Baseline Comparisons of 189 Children Enrolled in Dust Control Intervention Study by Group Assignment Who Were Followed Until 48 Months of Age

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Intervention Group (n = 96)</th>
<th>Control Group (n = 93)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood lead levels ($\mu$/dL)</td>
<td>Geometric Mean (95% CI)</td>
<td>Geometric Mean (95% CI)</td>
<td></td>
</tr>
<tr>
<td>Serum ferritin (ng/dL)</td>
<td>2.7 (2.4,3.1)</td>
<td>2.9 (2.6,3.2)</td>
<td>.56</td>
</tr>
<tr>
<td>Age (mo; mean)</td>
<td>37.3 (32.2,43.1)</td>
<td>36.8 (32.2,42.1)</td>
<td>.91</td>
</tr>
<tr>
<td>Floor lead loading ($\mu$/ft$^2$)</td>
<td>6.7 (6.6,6.7)</td>
<td>6.6 (6.6,6.7)</td>
<td>.43</td>
</tr>
<tr>
<td>Interior window sill lead ($\mu$/ft$^2$)</td>
<td>7.4 (5.9,9.5)</td>
<td>7.5 (5.9,9.5)</td>
<td>.97</td>
</tr>
<tr>
<td>Window-trough lead ($\mu$/ft$^2$)</td>
<td>418 (295,593)</td>
<td>453 (299,627)</td>
<td>.90</td>
</tr>
<tr>
<td>Interior paint lead hazard index</td>
<td>17 (11,27)</td>
<td>17 (11,27)</td>
<td>.90</td>
</tr>
<tr>
<td>Exterior paint lead hazard index</td>
<td>1.9 (1,4,2,6)</td>
<td>1.8 (1,3,2,4)</td>
<td>.71</td>
</tr>
<tr>
<td>Soil lead ($\mu$/g)</td>
<td>926 (731,1173)</td>
<td>1151 (924,1433)</td>
<td>.19</td>
</tr>
<tr>
<td>Water lead levels (&gt;0.0025 mg/L)</td>
<td>20 (21)</td>
<td>12 (13)</td>
<td>.15</td>
</tr>
<tr>
<td>Soil present</td>
<td>95 (99)</td>
<td>90 (97)</td>
<td>.36</td>
</tr>
<tr>
<td>Poor housing condition</td>
<td>12 (13)</td>
<td>22 (24)</td>
<td>.06</td>
</tr>
<tr>
<td>Black race</td>
<td>60 (63)</td>
<td>61 (66)</td>
<td>.66</td>
</tr>
<tr>
<td>Household income $\leq$15 500</td>
<td>66 (69)</td>
<td>69 (75)</td>
<td>.40</td>
</tr>
<tr>
<td>Rental housing</td>
<td>81 (84)</td>
<td>77 (84)</td>
<td>.90</td>
</tr>
</tbody>
</table>

### TABLE 2. Children’s Geometric Mean Blood Lead Concentrations by Group Assignment

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Intervention Group</th>
<th>Control Group</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Lead ($\mu$/dL)</td>
<td>No. μg/dL (95% CI)</td>
<td>No. μg/dL (95% CI)</td>
<td></td>
</tr>
<tr>
<td>Age (mo)</td>
<td>No. μg/dL (95% CI)</td>
<td>No. μg/dL (95% CI)</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>140 2.8 (2.5,3.1)</td>
<td>135 2.9 (2.7,3.2)</td>
<td>.51</td>
</tr>
<tr>
<td>12</td>
<td>127 5.5 (4.9,6.2)</td>
<td>126 5.9 (5.3,6.6)</td>
<td>.40</td>
</tr>
<tr>
<td>18</td>
<td>124 5.9 (5.3,6.7)</td>
<td>122 6.2 (5.5,7.0)</td>
<td>.58</td>
</tr>
<tr>
<td>24</td>
<td>126 7.3 (6.6,8.2)</td>
<td>119 7.8 (6.9,8.7)</td>
<td>.47</td>
</tr>
<tr>
<td>36</td>
<td>106 6.0 (5.3,6.8)</td>
<td>97 6.9 (6.1,7.8)</td>
<td>.13</td>
</tr>
<tr>
<td>48</td>
<td>96 5.9 (5.3,6.7)</td>
<td>93 6.1 (5.5,6.9)</td>
<td>.73</td>
</tr>
</tbody>
</table>
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Table 3. Percentage of Children With Elevated Blood Lead Level by Group Assignment, 6 to 48 Months of Age

<table>
<thead>
<tr>
<th>Months of Age</th>
<th>Intervention Group</th>
<th>Control Group</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>≥5 µg/dL</td>
<td>21 (15)</td>
<td>20 (15)</td>
</tr>
<tr>
<td></td>
<td>≥10 µg/dL</td>
<td>3 (2)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>12</td>
<td>≥5 µg/dL</td>
<td>78 (61)</td>
<td>79 (63)</td>
</tr>
<tr>
<td></td>
<td>≥10 µg/dL</td>
<td>21 (17)</td>
<td>22 (17)</td>
</tr>
<tr>
<td></td>
<td>≥15 µg/dL</td>
<td>5 (4)</td>
<td>9 (7)</td>
</tr>
<tr>
<td></td>
<td>≥20 µg/dL</td>
<td>2 (2)</td>
<td>4 (3)</td>
</tr>
<tr>
<td>18</td>
<td>≥5 µg/dL</td>
<td>81 (65)</td>
<td>78 (64)</td>
</tr>
<tr>
<td></td>
<td>≥10 µg/dL</td>
<td>25 (20)</td>
<td>31 (25)</td>
</tr>
<tr>
<td></td>
<td>≥15 µg/dL</td>
<td>11 (9)</td>
<td>14 (11)</td>
</tr>
<tr>
<td></td>
<td>≥20 µg/dL</td>
<td>2 (2)</td>
<td>6 (5)</td>
</tr>
<tr>
<td>24</td>
<td>≥5 µg/dL</td>
<td>92 (73)</td>
<td>88 (74)</td>
</tr>
<tr>
<td></td>
<td>≥10 µg/dL</td>
<td>39 (31)</td>
<td>43 (36)</td>
</tr>
<tr>
<td></td>
<td>≥15 µg/dL</td>
<td>15 (12)</td>
<td>17 (14)</td>
</tr>
<tr>
<td></td>
<td>≥20 µg/dL</td>
<td>6 (5)</td>
<td>8 (7)</td>
</tr>
<tr>
<td>36</td>
<td>≥5 µg/dL</td>
<td>65 (61)</td>
<td>68 (70)</td>
</tr>
<tr>
<td></td>
<td>≥10 µg/dL</td>
<td>26 (25)</td>
<td>30 (31)</td>
</tr>
<tr>
<td></td>
<td>≥15 µg/dL</td>
<td>5 (5)</td>
<td>11 (11)</td>
</tr>
<tr>
<td></td>
<td>≥20 µg/dL</td>
<td>2 (2)</td>
<td>6 (6)</td>
</tr>
<tr>
<td>48</td>
<td>≥5 µg/dL</td>
<td>59 (61)</td>
<td>58 (62)</td>
</tr>
<tr>
<td></td>
<td>≥10 µg/dL</td>
<td>18 (19)</td>
<td>18 (19)</td>
</tr>
<tr>
<td></td>
<td>≥15 µg/dL</td>
<td>2 (2)</td>
<td>8 (9)</td>
</tr>
<tr>
<td></td>
<td>≥20 µg/dL</td>
<td>1 (1)</td>
<td>2 (2)</td>
</tr>
</tbody>
</table>

* Sample size for each age group is shown in Table 2.

children’s lead intake. Still, further research is necessary to examine the effect of lead hazard controls combined with more aggressive professional cleaning and to compare the cost-effectiveness of professional cleaning with other lead hazard controls. But the key to reduce children’s blood lead levels is to make leaded paint inaccessible and to clean to achieve dust lead levels (ie, clearance tests) that are safe.4,9,10

Other educational efforts to reduce lead exposure in children have not been rigorously tested or shown to be ineffective. Sargent et al16 reported that calcium-supplemented formula did not result in sustained reduction in children’s blood lead concentrations. There are no published randomized, controlled trials of multifactorial interventions, such as calcium supplementation combined with dust control. Thus, there are no data showing that educational efforts—the cornerstone of lead poisoning prevention for the majority of children with undue lead exposure—are effective in preventing lead exposure from residential lead hazards.

CONCLUSION

The results of this study suggest that dust control, as performed by families and in the absence of lead hazard controls to reduce ongoing contamination from leaded paint, was not effective in the primary prevention of childhood lead exposure. These results underscore the fact that dust control, one of the primary strategies to control lead exposure for children with low to moderate elevations in blood lead concentration, does not seem to be effective unless it is performed by professional dust control teams. Taken together, these and other data indicate that we can no longer rely on dust control, as performed by families, as a panacea to prevent subclinical lead toxicity in children.4

ACKNOWLEDGMENTS

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