

Archives of Environmental Health: An International Journal

Publication details, including instructions for authors and subscription information:

<http://www.tandfonline.com/loi/vzeh20>

Effect of a Follow-Up Professional Home Cleaning on Serial Dust and Blood Lead Levels of Urban Children

Carla Campbell ^a, Donald F. Schwarz ^a, David Rich ^b & Douglas W. Dockery ^b

^a The Children's Hospital of Philadelphia and University of Pennsylvania School of Medicine Department of Pediatrics Philadelphia, Pennsylvania

^b Harvard School of Public Health Environmental Epidemiology Program Boston, Massachusetts

Published online: 07 Aug 2010.

To cite this article: Carla Campbell , Donald F. Schwarz , David Rich & Douglas W. Dockery (2003) Effect of a Follow-Up Professional Home Cleaning on Serial Dust and Blood Lead Levels of Urban Children, Archives of Environmental Health: An International Journal, 58:12, 771-780, DOI: [10.3200/AEOH.58.12.771-780](https://doi.org/10.3200/AEOH.58.12.771-780)

To link to this article: <http://dx.doi.org/10.3200/AEOH.58.12.771-780>

PLEASE SCROLL DOWN FOR ARTICLE

Taylor & Francis makes every effort to ensure the accuracy of all the information (the "Content") contained in the publications on our platform. However, Taylor & Francis, our agents, and our licensors make no representations or warranties whatsoever as to the accuracy, completeness, or suitability for any purpose of the Content. Any opinions and views expressed in this publication are the opinions and views of the authors, and are not the views of or endorsed by Taylor & Francis. The accuracy of the Content should not be relied upon and should be independently verified with primary sources of information. Taylor and Francis shall not be liable for any losses, actions, claims, proceedings, demands, costs, expenses, damages, and other liabilities whatsoever or howsoever caused arising directly or indirectly in connection with, in relation to or arising out of the use of the Content.

This article may be used for research, teaching, and private study purposes. Any substantial or systematic reproduction, redistribution, reselling, loan, sub-licensing, systematic supply, or distribution in any form to anyone is expressly forbidden. Terms & Conditions of access and use can be found at <http://www.tandfonline.com/page/terms-and-conditions>

Effect of a Follow-Up Professional Home Cleaning on Serial Dust and Blood Lead Levels of Urban Children

CARLA CAMPBELL

DONALD F. SCHWARZ

Department of Pediatrics

The Children's Hospital of Philadelphia

and

University of Pennsylvania School of Medicine

Philadelphia, Pennsylvania

DAVID RICH

DOUGLAS W. DOCKERY

Environmental Epidemiology Program

Harvard School of Public Health

Boston, Massachusetts

ABSTRACT. Children residing in Philadelphia, Pennsylvania, who were enrolled in a clinical trial of oral chelation therapy ($n = 73$) were studied to determine the effects of a follow-up professional lead dust cleaning of their homes 18 mo after an initial cleaning and commencement of therapy. Home dust lead levels were determined from dust-wipe specimens collected from the kitchen and playroom floors, and from a playroom windowsill, prior to, immediately following, and 3 and 6 mo after the second cleaning. Children's blood lead levels were assessed at 3-mo intervals before and after the follow-up cleaning. Professional cleaning produced immediate decreases in dust lead levels; however, dust lead re-accumulated to precleaning levels within 3–6 mo. Frequent, repeated cleanings may be required if blood lead or dust lead levels are to be reduced and sustained at low levels in urban homes.

<Key words: blood lead, cleaning, dust, environment, lead, lead poisoning>

FOLLOWING THE DRAMATIC REDUCTION in exposures to airborne lead from leaded gasoline, the contribution of dust in the home as a source of lead exposure for young children has come under closer scrutiny and study. For example, in several studies^{1–6} investigators identified lead-contaminated house dust as a major source of lead exposure in children. In a meta-analysis of exposure pathways, Lanphear et al.⁷ concluded that "lead-contaminated house dust is the major source of lead exposure for children." The Treatment of Lead-Exposed Children (TLC) Trial was a multicenter, double-blind, randomized clinical trial of lead chelation using the oral agent succimer (meso 2,3-dimercaptosuccinic acid) vs. a placebo, among children in 4 U.S. cities. As part of that protocol, the children's homes were professionally cleaned with the goal of minimizing continued dust lead exposure during treatment (i.e., for up to 6 mo). We measured serial dust and blood lead levels to

monitor the effectiveness of the intervention.^{8,9} An analysis of the primary cleaning intervention revealed a moderate reduction in dust lead levels following cleaning activities.¹⁰

After completion of the treatment phase of the TLC trial, families of TLC subjects at the Philadelphia Center ($n = 165$) were offered a second home cleaning. This allowed us the opportunity to assess the effect of a second professional home cleaning on both dust and blood lead levels. We report here on the serial blood lead and home lead dust levels of the Philadelphia subjects who chose to participate ($n = 73$), before and after their homes received a second professional lead dust cleaning.

Materials and Method

General. The protocol for the TLC trial has been described elsewhere.⁸ Briefly, the trial enrolled 780 tod-

dlers with blood lead levels between 20 µg/dl and 44 µg/dl to examine the effectiveness of chelation therapy with oral succimer vs. placebo. The study was conducted at the following clinical sites in the United States: Newark, New Jersey; Baltimore, Maryland; Cincinnati, Ohio; and Philadelphia, Pennsylvania. The U.S. Centers for Disease Control and Prevention in Atlanta, Georgia (CDC) provided a central laboratory for blood lead measurements, and the Harvard School of Public Health in Boston, Massachusetts, served as the data coordination center. Children were followed for 36 mo after randomization. The children were assessed for blood lead levels and growth approximately every 3 mo, and for neuropsychological development at baseline and 3 times during the study. Eighteen months after the TLC trial began, families of children ($n = 165$; 41% female, 88% African American; mean age at study entry: 25.8 mo) who were enrolled at the Philadelphia site were offered a second professional home cleaning. Of these, 73 elected to participate.

The TLC trial was approved by the Institutional Review Board of the Children's Hospital of Philadelphia.

Environmental protocol. The preliminary home assessment and cleaning protocols for the TLC trial have been described previously.⁸ In summary, each child's home was assessed by a trained lead inspector, using a specific inspection protocol, at the time of recruitment—and upon notification of any change in housing—for potential sources of lead, damage and deterioration, and cleanability. The study protocol required that each child's home receive a baseline cleaning prior to initiation of treatment. In Philadelphia, homes were cleaned by professional contractors trained in the TLC cleaning protocol and in techniques for the safe handling of materials that contain lead. Floors were vacuumed with a high-efficiency particulate air (HEPA) filter vacuum cleaner at a floor-specific rate, uncarpeted floors were wet-washed with a high-phosphate detergent, and all floors were again HEPA-vacuumed. All horizontal surfaces (e.g., heating, ventilation, and air-conditioning equipment; light fixtures; cabinets) were cleaned and vacuumed. Areas of loose or peeling paint were removed. Dust-wipe samples were collected for a sample of homes (37 in Philadelphia) before and after the initial cleaning; results are reported elsewhere.¹⁰ Throughout the 3-yr study, families periodically received instructions on how to wet mop and wet dust their homes, were encouraged to complete both cleaning processes, and were provided cleaning supplies and equipment.

At the 18-mo follow-up visit after initiation of treatment (postrandomization), we offered each of the 165 study families in Philadelphia a second professional lead dust cleaning of their home. Seventy-three families accepted the offer, and each of their family homes was cleaned ($n = 75$). Dust-wipe lead samples were collect-

ed prior to the cleaning (precleaning), within 1 week following the cleaning (postcleaning), and at 3 mo and 6 mo postcleaning. Samples were taken from the kitchen floor (under a window, if possible), from a playroom windowsill, and from the floor area below the playroom sill. Floor specimens were categorized as being from either hard or carpeted surfaces. Prior to sample collection, surfaces were assessed as "intact," "minor problems," or "deteriorated." Inasmuch as 95–96% of the samples collected were taken from surfaces judged to be intact, the samples were not stratified by the condition of the surface.

Dust-wipe samples were collected in accordance with U.S. Department of Housing and Urban Development protocol.¹¹ A measured area of the floor (0.09 m^2 [1 ft^2]) or windowsill (variable area) was sampled with a standard baby wipe prewetted towelette (Little Ones Baby Wipes® were used initially, followed by Wash-A-Bye Baby Wipes® when manufacturing of the first brand was discontinued). A comparison of side-by-side dust-wipe specimens using both brands of baby wipes revealed no appreciable difference in collected dust lead values. Specimens were analyzed (Azimuth, Inc. [Charleston, South Carolina]) with flame atomic absorption spectrometry (modified U.S. National Institute of Occupational Safety and Health [NIOSH] method 7082^{12,13}) with a laboratory limit of detection of 25 µg/sample. The instrument (PerkinElmer, Inc. [Wellesley, Massachusetts]) was calibrated with every sample set analyzed, and a sample with a known quantity of lead was included with every 10th sample run. An analysis of spiked dust wipes with known concentrations of lead dust was sent periodically, in a blinded fashion, to the laboratory. Recovery rates were within 80–120% for 19 of 20 specimens (1 specimen was measured at 79%). Blank specimens were sent regularly with study specimens; none of the blank-specimen results ($n = 249$) were greater than 25 µg (mean = –2.7 µg).

Homes were reassessed whenever a subject moved, or if remodeling or repair activities took place in the home. The homes of subjects whose parent(s) had agreed to a second professional cleaning were re-inspected prior to that cleaning. We used the home characteristics recorded at the assessment just prior to the second cleaning to characterize these children's homes. For those children whose primary residences did not receive a second cleaning, we estimated home characteristics on the basis of the residence that was evaluated during the time period closest to their 18-mo follow-up visit.

Blood lead level measurements. Venous blood samples were collected during clinical visits, which were scheduled every 1–3 wk during treatment and every 3–4 mo posttreatment (i.e., for the 36-mo study period). For this analysis, we used the blood lead levels for 159 children at the 15-mo, 18-mo (just prior to the second

cleaning), 21-mo, and 24-mo clinical follow-up visits (i.e., $n = 73$ children with homes cleaned and $n = 86$ children with homes not cleaned). Blood lead levels were measured by atomic absorption spectrometry¹⁴ at the CDC central laboratory.

Statistical analysis. All analyses were run using SAS statistical software, version 8.02 (SAS Institute [Cary, North Carolina]). We determined the frequency of home characteristics in homes that were cleaned vs. those that were not cleaned. Fisher's Exact Test was used to determine if those differences were statistically significant ($p < 0.05$), excluding those for which values were missing.

Actual measurements of lead concentrations were obtained from the laboratory for all dust samples. Truncation of lead measurements at the laboratory limit of detection has the unintended result of producing biased sample means and population statistics. Although various statistical corrections have been proposed for this bias, the use of actual laboratory measurements for all samples has been shown to provide a direct, unbiased result estimate.¹⁵⁻¹⁷ For this reason, we analyzed the actual laboratory measurements for all dust-wipe samples.

We calculated a visit-specific mean and standard deviation of the log-transformed dust lead levels for each surface (i.e., kitchen floor, playroom floor, and playroom windowsill). A lead value of 1 μg was substituted for all detected lead levels less than 1 μg (21% of sample). Geometric mean (GM) dust lead levels were calculated as the exponential of the mean of the log-transformed values, and geometric standard deviations ($GSDs$) were calculated as the exponential of the standard deviations of the log-transformed dust lead levels.

Kitchen and playroom floor samples were combined and categorized by type of surface (i.e., hard or carpeted). To adjust for multiple samples per home at each visit, we estimated visit-specific least-square means, and standard deviations by using a mixed model (PROC MIXED (SAS Institute [Cary, North Carolina]) that included a random intercept for each home. Similarly, we used the mixed model with a random intercept for each home to estimate the percentages of precleaning lead levels at postcleaning, and at 3- and 6-mo follow-ups.

To determine if the cleaning was more or less effective in homes that would not have been identified for intervention on the basis of U.S. Environmental Protection Agency (EPA) clearance criteria,¹⁸ we stratified the sample into "low-exposure" and "high-exposure" homes. Low-exposure homes were those in which all 3 surface samples were below EPA clearance standards (i.e., $> 40 \mu\text{g}/\text{ft}^2$ for hard-floor or carpet samples; $> 250 \mu\text{g}/\text{ft}^2$ for windowsill samples). High-exposure homes had at least 1 surface sample that exceeded an EPA clearance standard. We then compared the GM lead loadings for each sample and surface type on the basis of low- or high-exposure status.

The relationship between cleaning status and blood lead level was evaluated using mixed models, with random intercepts for each child—regressing the log of the blood lead level against clinic visit, child, and month. Given the limited age range among the children (12–34 mo at enrollment), age was not a significant factor in the analyses.

Results

Seventy-five homes of 73 children in the Philadelphia TLC trial were cleaned at 18 mo following commencement of the trial. Two children lived in more than 1 home—both of which were cleaned—and no cleaned home was occupied by more than 1 study child. The homes that were cleaned were more likely (75% rowhouses) to be "connected houses" than were homes that were not cleaned (55% rowhouses). That is, homes not cleaned tended to be single-family homes (10% vs. 1% of cleaned homes). The cleaned homes were less likely than those that were not cleaned to have structural problems (14% vs. 22%, respectively; $p = 0.11$), rat infestations (4% vs. 22%, respectively; $p = 0.0006$), or paint problems (31% vs. 70%, respectively; $p < 0.0001$) (Table 1). The cleaned homes also had better overall maintenance than the uncleaned homes (76% vs. 33%, respectively, rated "good"; $p < 0.0001$). They also had lower overall lead exposure (76% vs. 37%, respectively, rated "low-exposure"; $p < 0.0001$).

Effect of cleaning on dust lead levels. Precleaning dust lead levels in the cleaned homes were, on average,

Table 1.—Percentage of Baseline Home Characteristics Determined at the Time of Precleaning (Homes Cleaned) or from Information Elicited from the 18-mo Clinical Follow-Up (Homes Not Cleaned)

Baseline home characteristic	Homes cleaned (%) ($n = 74$)*	Homes not cleaned (%) ($n = 91$)*	p †
Inherent problems*			
Heating system	4	7	0.51
Plumbing leaks	23	15	0.32
Roof	18	14	0.67
Structure	14	22	0.11
Rats	4	22	0.0006
Paint	31	70	< 0.0001
Overall maintenance			
Good	76	33	< 0.0001
Fair or Poor	24	64	
Missing	0	3	
Lead exposure			
Low	76	37	< 0.0001
Moderate or High	24	60	
Missing	0	3	

*Missing values were included in calculating percentages.

†Fisher's Exact Test. A p value < 0.05 indicated a statistically significant difference.

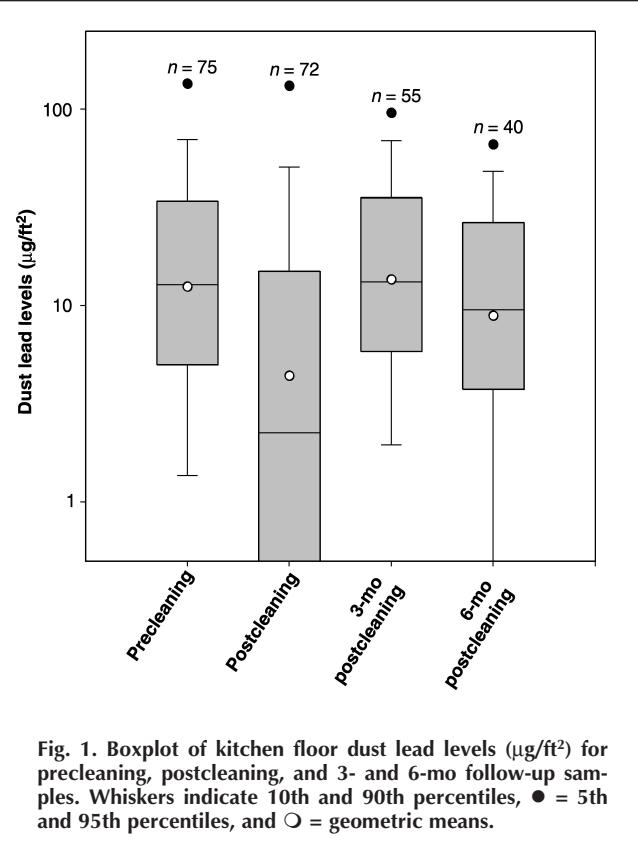


Fig. 1. Boxplot of kitchen floor dust lead levels ($\mu\text{g}/\text{ft}^2$) for precleaning, postcleaning, and 3- and 6-mo follow-up samples. Whiskers indicate 10th and 90th percentiles, ● = 5th and 95th percentiles, and ○ = geometric means.

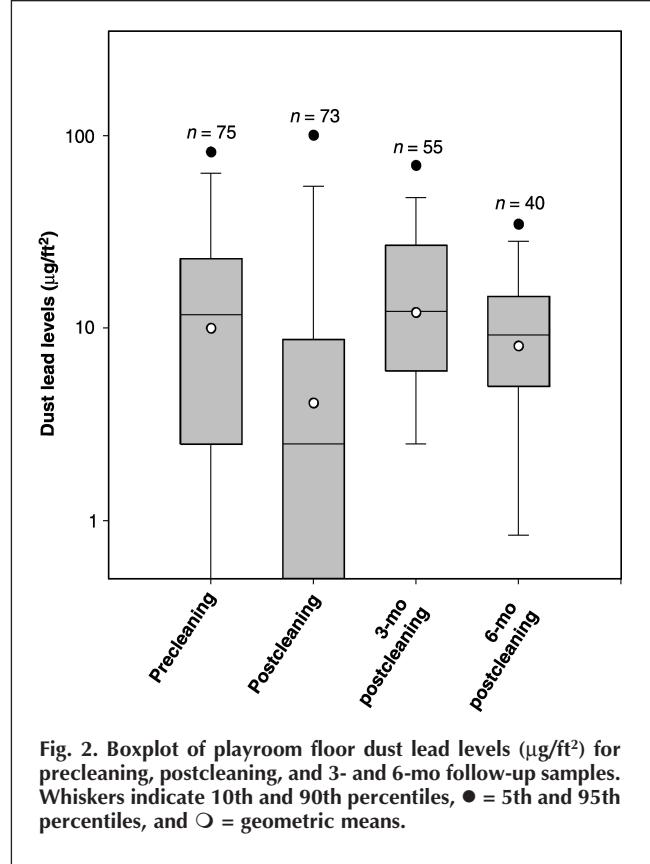


Fig. 2. Boxplot of playroom floor dust lead levels ($\mu\text{g}/\text{ft}^2$) for precleaning, postcleaning, and 3- and 6-mo follow-up samples. Whiskers indicate 10th and 90th percentiles, ● = 5th and 95th percentiles, and ○ = geometric means.

low (Figs. 1–3), with *GM* dust levels of 12.5 $\mu\text{g}/\text{ft}^2$ and 10.0 $\mu\text{g}/\text{ft}^2$ for the kitchen and playroom floor samples, respectively, and 82.8 $\mu\text{g}/\text{ft}^2$ for the playroom windowsills. Twenty percent of the kitchen and 15% of the playroom floor samples contained dust lead levels that exceeded the EPA standard of 40 $\mu\text{g}/\text{ft}^2$. Thirty-four percent of the windowsill samples exceeded the EPA standard of 250 $\mu\text{g}/\text{ft}^2$. Across all 3 sampling areas, 34 (45%) of the 75 homes had at least 1 sample that exceeded an EPA dust lead clearance standard.

Professional cleaning produced immediate and substantial reductions in dust lead levels on the kitchen and playroom floors and playroom windowsill areas sampled (Table 2). The kitchen floor *GMs* dropped from 12.5 $\mu\text{g}/\text{ft}^2$ (precleaning) to 4.5 $\mu\text{g}/\text{ft}^2$ (postcleaning) (36% of precleaning values). However, at the 3-mo and 6-mo follow-up visits, dust levels had returned to precleaning values. Follow-up *GM* dust levels returned to 13.6 $\mu\text{g}/\text{ft}^2$ at 3 mo postcleaning and to 9.0 $\mu\text{g}/\text{ft}^2$ at 6 mo postcleaning. The playroom floor and playroom windowsill samples displayed similar patterns.

Precleaning dust lead levels, adjusted for multiple measures within a home (Table 2), were higher ($p = 0.003$) on hard-surface floors (101 samples from 72 homes; *GM* = 12.9 $\mu\text{g}/\text{ft}^2$) than on carpeted floors (49 samples from 46 homes; *GM* = 6.5 $\mu\text{g}/\text{ft}^2$). Postcleaning dust lead *GMs*, also adjusted for multiple measures

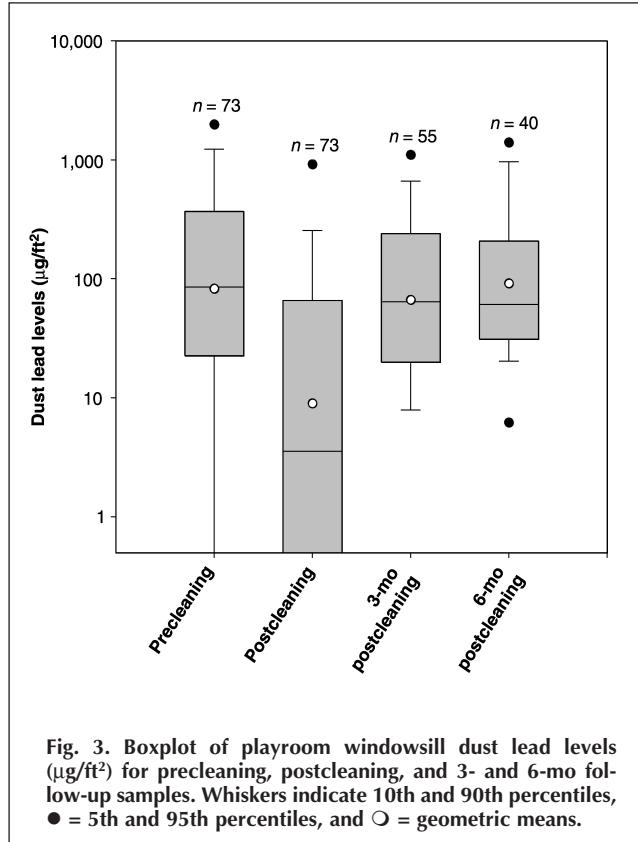


Fig. 3. Boxplot of playroom windowsill dust lead levels ($\mu\text{g}/\text{ft}^2$) for precleaning, postcleaning, and 3- and 6-mo follow-up samples. Whiskers indicate 10th and 90th percentiles, ● = 5th and 95th percentiles, and ○ = geometric means.

Table 2.—Dust Lead Loadings for Cleaned Homes, by Surface Type

Surface type	Precleaning	Postcleaning	3-mo postcleaning visit	6-mo postcleaning visit
Hard floor*				
n_1 = number of samples	101	97	74	57
GM ($\mu\text{g}/\text{ft}^2$)	12.9	5.1†	15.2	8.5†
GSD	1.2	1.2	1.1	1.2
Precleaning loading (%)	100	39†	118	66†
Percentage > 40 $\mu\text{g}/\text{ft}^2$	20	16	26	12
Carpet				
n_1 = number of samples	49	48	36	23
GM ($\mu\text{g}/\text{ft}^2$)	6.5	3.4‡	8.0	6.7
Within-home GSD	1.2	1.2	1.3	1.4
Precleaning loading (%)	100	52‡	124	100
Percentage > 40 $\mu\text{g}/\text{ft}^2$	10	6	8	4
Windowsill				
n = number of samples	73	73	55	40
GM ($\mu\text{g}/\text{ft}^2$)	82.8	9.0†	66.7	91.8
GSD	1.3	1.3	1.4	1.4
Precleaning loading (%)	100	11†	81	111
Percentage > 250 $\mu\text{g}/\text{ft}^2$	34	12	25	25

Note: GM = geometric mean, and GSD = geometric standard deviation.

*Hard-floor analysis was adjusted for multiple measures per home.

† $p < 0.01$, compared with precleaning values, by Fisher's Exact Test; $p < 0.05$ indicated statistical significance.

‡ $p < 0.05$, compared with precleaning values, by Fisher's Exact Test; $p < 0.05$ indicated statistical significance.

within a home, were reduced to 39% of precleaning values on hard-surface floors and to 52% on carpeted floors ($p < 0.01$ and < 0.05 , respectively) (Table 2). For hard-surface floors, follow-up dust lead levels at 3-mo postcleaning did not differ from precleaning values, although the adjusted 6-mo follow-up values were 66% of precleaning values ($p < 0.016$). For carpeted surfaces, follow-up dust lead levels at 3 mo and 6 mo were not different from precleaning values. Likewise, windowsill dust lead GMs were reduced to 11% of precleaning values following cleaning, but values at 3 mo and 6 mo postcleaning were not statistically significantly different from precleaning values.

Although cleaning had little effect on the GM at the 6-mo follow-up, there was a suggestion of an effect in the higher-exposure homes. Among all the precleaning dust samples, 20% of the hard-surface, 10% of the carpeted, and 34% of the windowsill samples exceeded EPA dust lead clearance standards. At the 6-mo follow-up postcleaning, those frequencies had decreased to 12%, 4%, and 25%, respectively. We questioned whether these results might vary depending on the level of exposure, and stratified the homes by level of lead exposure. Table 3 shows the results for the homes in which none of the dust samples yielded levels that exceeded EPA dust lead clearance standards (low-exposure homes). Following cleaning, 10% and 4% of hard-surface and carpeted floors, respectively, and 5% of windowsill samples contained lead levels that exceed-

ed the EPA standards. Postcleaning dust-wipe lead levels were 51%, 66%, and 19% of precleaning values for the hard-surface floors, carpeted floors, and windowsills, respectively. However, at the 3-mo and 6-mo follow-up assessments, GMs were higher than the pre-cleaning means in these homes. Therefore, in the homes that initially had low levels of lead, lead dust might have been resuspended during cleaning and might have settled on common household surfaces, or there might have been new contamination from environmental lead sources.

In the high-exposure homes (Table 4), 44%, 25%, and 74%, of the hard-surface floors, carpeted floors, and windowsill samples, respectively, exceeded the EPA's clearance standards. Cleaning produced a substantial improvement: only 9% and 0% of the hard-surface floor and carpeted-floor samples, respectively, and 40% of the windowsill samples, exceeded EPA standards 6 mo postcleaning. Postcleaning dust-wipe lead levels were 21% and 38% of precleaning values for hard-surface and carpeted floor samples, respectively, and 6% for windowsill samples. Mean dust lead levels at the 6-mo follow-up visits were approximately 30–40% of the precleaning values. Thus, among the high-exposure homes, GM dust loadings were lower and there were substantially fewer homes in this category that contained dust lead levels that remained above EPA standards at 6 mo postcleaning.

There was considerable attrition of participants be-

Table 3.—Dust Lead Loadings, by Surface Type, for Cleaned Homes in Which Dust Lead Levels for All 3 Surfaces Sampled Were Below the U.S. Environmental Protection Agency (EPA) Clearance Standard

Surface type	Precleaning	Postcleaning	3-mo postcleaning visit	6-mo postcleaning visit
Hard floor				
n_1 = number of samples	53	49	42	35
GM ($\mu\text{g}/\text{ft}^2$)	7.6	3.9*	12.3	8.0
GSD	1.2	1.2	1.2	1.2
Precleaning loading (%)	100	51*	161	105
Percentage > 40 $\mu\text{g}/\text{ft}^2$	0	10	12	14
Carpet				
n_1 = number of samples	29	28	22	15
GM ($\mu\text{g}/\text{ft}^2$)	3.4	2.2	8.5†	7.6†
Within-home GSD	1.3	1.3	1.3	1.4
Precleaning loading (%)	100	66	251†	255†
Percentage > 40 $\mu\text{g}/\text{ft}^2$	0	4	9	7
Windowsill				
n	39	39	32	25
GM ($\mu\text{g}/\text{ft}^2$)	21.4	4.1*	36.8	44.3
GSD	1.3	1.3	1.4	1.4
Precleaning loading (%)	100	19*	171	207
Percentage > 250 $\mu\text{g}/\text{ft}^2$	0	5	13	16

Note: GM = geometric mean, and GSD = geometric standard deviation.

* $p < 0.01$, compared with precleaning values, by Fisher's Exact Test; $p < 0.05$ indicated statistical significance.

† $p < 0.05$, compared with precleaning values, by Fisher's Exact Test; $p < 0.05$ indicated statistical significance.

Table 4.—Dust Lead Loadings, by Surface Type, for Cleaned Homes in Which the Dust Lead Level on at Least 1 Surface Sampled Exceeded the U.S. Environmental Protection Agency (EPA) Clearance Standard

Surface type	Precleaning	Postcleaning	3-mo postcleaning visit	6-mo postcleaning visit
Hard floor				
n_1 = number of samples	48	48	32	22
GM ($\mu\text{g}/\text{ft}^2$)	29.8	6.2*	23.2	12.3
GSD	1.3	1.3	1.3	1.4
Precleaning loading (%)	100	21*	78	41†
Percentage > 40 $\mu\text{g}/\text{ft}^2$	44	21	44	9
Carpet				
n_1 = number of samples	20	20	14	8
GM ($\mu\text{g}/\text{ft}^2$)	16.6	6.3	7.3	5.3
Within-home GSD	1.4	1.4	1.5	1.7
Precleaning loading (%)	100	38†	44	32
Percentage > 40 $\mu\text{g}/\text{ft}^2$	25	10	7	0
Windowsill				
n	34	34	23	15
GM ($\mu\text{g}/\text{ft}^2$)	390.3	22.3*	153.6	309.1
GSD	1.4	1.4	1.6	1.7
Precleaning loading (%)	100	6*	39	79
Percentage > 250 $\mu\text{g}/\text{ft}^2$	74	21	43	40

Note: GM = geometric mean, and GSD = geometric standard deviation.

* $p < 0.01$, compared with precleaning values, by Fisher's Exact Test; $p < 0.05$ indicated statistical significance.

† $p < 0.05$, compared with precleaning values, by Fisher's Exact Test; $p < 0.05$ indicated statistical significance.

tween the professional cleaning and the 6-mo visit. Of the 75 homes enrolled in the study, dust-wipe samples were collected from 73 (97%) homes postcleaning, from 55 (73%) homes at the 3-mo follow up, and from 40 (53%) homes at the 6-mo follow up. To directly assess whether the rebound of dust lead levels resulted from attrition, we calculated *GMs* for 31 homes at which dust-wipe measurements were obtained at all 4 sampling times. The *GMs* for these homes were similar to those noted for the complete sample. Specifically, kitchen floor *GM* lead levels were 13.0 $\mu\text{g}/\text{ft}^2$, 5.8 $\mu\text{g}/\text{ft}^2$, 11.3 $\mu\text{g}/\text{ft}^2$, and 9.6 $\mu\text{g}/\text{ft}^2$, respectively, at precleaning, postcleaning, 3-mo, and 6-mo follow ups. Therefore, a rebound in dust lead levels was seen in homes for which we had complete observations.

Effect of cleaning on blood lead levels. *GM* blood lead levels, adjusted for month and child, declined monotonically among the 73 children whose homes were cleaned a second time (Fig. 4). In comparison, blood lead levels of the 86 children in the uncleaned homes also declined over time, although there was an unexplained increase at the 3-mo postcleaning follow-up visit. There was no significant difference in *GM* blood lead levels at any clinical visit between children whose homes were cleaned and those whose homes were not cleaned (Fig. 4).

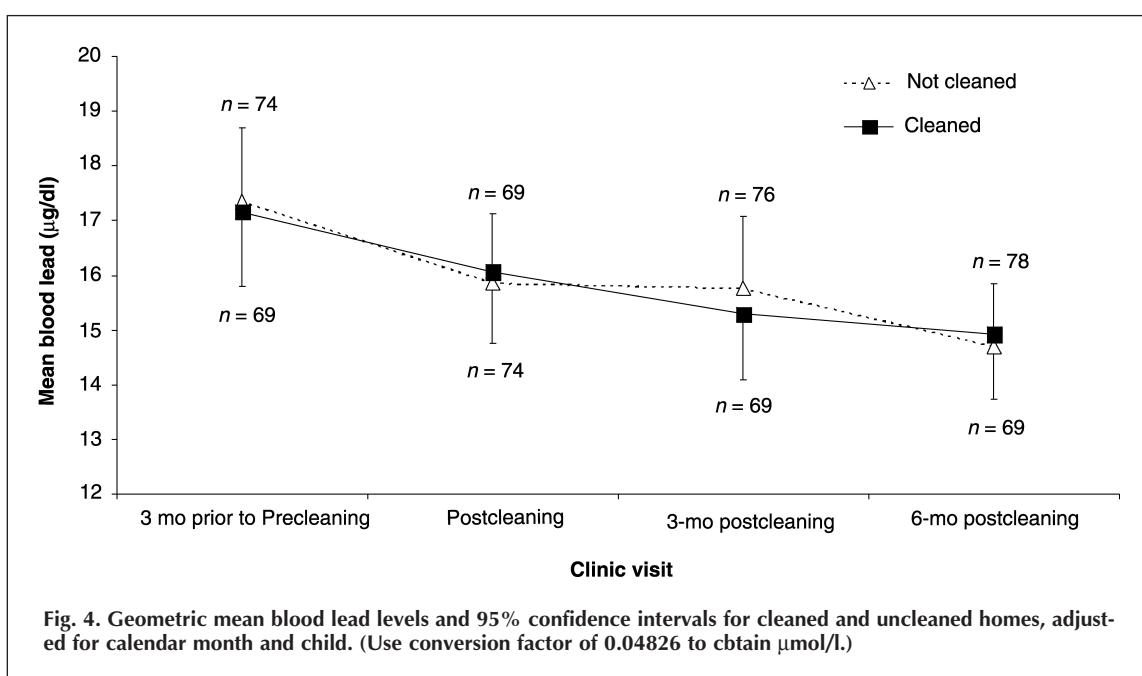
GM blood lead levels prior to the cleaning were higher among children who lived in high-exposure homes (*GM* = 18.1 $\mu\text{g}/\text{dl}$), compared with those who lived in low-exposure homes (*GM* = 14.5 $\mu\text{g}/\text{dl}$). This difference persisted throughout the cleaning and during the 6 mo of follow-up (Fig. 5). There were only small differences in *GM* blood lead levels when these children were fur-

ther stratified by treatment: 18.3 $\mu\text{g}/\text{dl}$ and 17.1 $\mu\text{g}/\text{dl}$ for children given active vs. placebo drugs, respectively, in high-exposure homes; and 14.5 $\mu\text{g}/\text{dl}$ and 13.5 $\mu\text{g}/\text{dl}$, respectively, in low-exposure homes.

Discussion

Families of children enrolled in the TLC trial in Philadelphia were given the opportunity to again have their homes professionally cleaned for lead dust 18 mo after enrollment in the study. Thirty-seven homes in Philadelphia had initial dust-wipe sampling, including 10 of the 75 homes cleaned in this study. Prerandomization *GM* dust lead levels for the Philadelphia cohort were 55 and 38 $\mu\text{g}/\text{ft}^2$ for the kitchen and playroom floor samples, respectively, and 627 $\mu\text{g}/\text{ft}^2$ for the windowsill samples,¹⁰ whereas *GM* dust lead levels prior to the 18-mo cleaning were 12.5 $\mu\text{g}/\text{ft}^2$, 10.0 $\mu\text{g}/\text{ft}^2$, and 82.8 $\mu\text{g}/\text{ft}^2$, respectively.

In several prior studies, investigators characterized lead dust levels in the homes of inner-city children. In a study of 205 toddlers in Rochester, New York,¹⁹ the following *GM* dust lead loading levels were found: 16 $\mu\text{g}/\text{ft}^2$ for noncarpeted floors, 11 $\mu\text{g}/\text{ft}^2$ for carpeted floors, 166 $\mu\text{g}/\text{ft}^2$ for interior windowsills, and 2,759 $\mu\text{g}/\text{ft}^2$ for window wells (with a wide variability in measurements). Dust lead loading levels were highest for window well areas, intermediate in windowsill areas, and lowest for floor areas—results which are consistent with other environmental sampling results.^{20–22} A meta-analysis⁷ examined 12 cross-sectional studies of lead-exposure pathways—using data on dust and soil lead and measures of paint lead content and paint condition



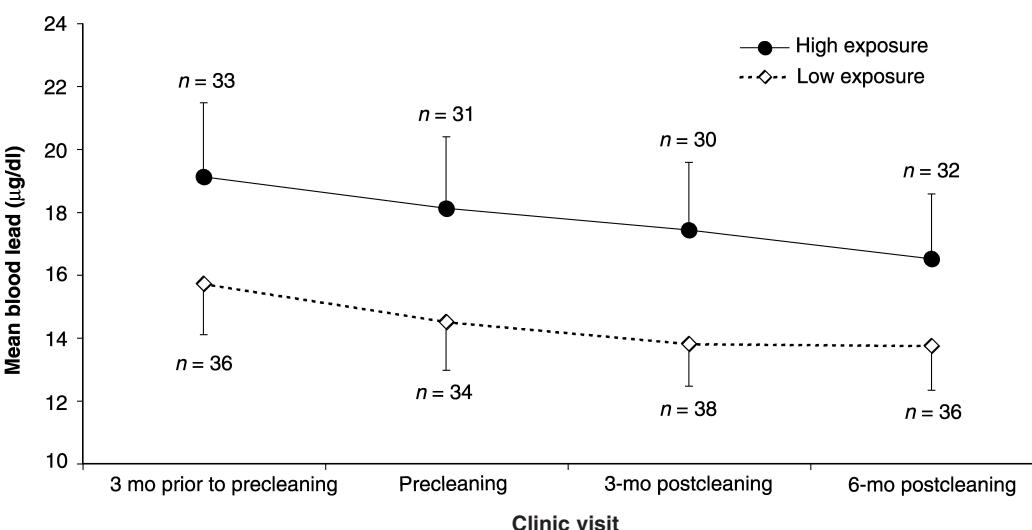


Fig. 5. Geometric mean blood lead levels and 95% confidence intervals for cleaned homes, stratified by pre-cleaning status (high- vs. low-exposure), compared with the U.S. Environmental Protection Agency's dust lead clearance standards. (Use conversion factor of 0.04826 to obtain $\mu\text{mol/l}$.)

from children's homes—found GM interior floor dust lead loading levels that ranged from $1.9 \mu\text{g}/\text{ft}^2$ to $20.4 \mu\text{g}/\text{ft}^2$ (with the exception of one study of inner-city children in Cincinnati, in which the GM was $293.4 \mu\text{g}/\text{ft}^2$). The GM blood lead level was $5.1 \mu\text{g}/\text{dl}$, and 19% of the children studied had blood lead levels of $10 \mu\text{g}/\text{dl}$ or higher. In multivariate regression analyses, floor dust lead loading was the most significant environmental predictor of children's blood lead levels.

All of the TLC study children had blood lead levels in the 20 – $44 \mu\text{g}/\text{dl}$ range at study initiation, with a GM blood lead level of $26 \mu\text{g}/\text{dl}$ ²³ and mean dust lead loadings of $43 \mu\text{g}/\text{ft}^2$ and $39 \mu\text{g}/\text{ft}^2$ for kitchen and playroom floors, respectively, and $308 \mu\text{g}/\text{ft}^2$ for windowsills.¹⁰ At the second precleaning visit, the GM blood lead level was $15.7 \mu\text{g}/\text{dl}$ and GM dust lead loadings were $12.5 \mu\text{g}/\text{ft}^2$ for kitchen floors and $10.0 \mu\text{g}/\text{ft}^2$ for playroom floors—relatively higher than those reported in other studies. Despite the fact that the homes had lower pre-cleaning levels than those demonstrated at the time of the first precleaning sampling, a second professional cleaning reduced dust lead levels significantly (approximately $1/3$ to $1/2$ of precleaning values for floors; $1/10$ for windowsills). Immediate reductions in dust lead levels were achieved in homes that were both above and below the EPA lead clearance guidelines; however, reductions were sustained at 6 mo postcleaning only in the high-exposure homes. This suggests that a single professional cleaning intervention has some effect on long-term control of dust lead levels for high-exposure homes, but that the effect is more transient for low-exposure homes. It may be that participation in a lead-poisoning treatment program, as well as in-

creased awareness through education and improved hygiene practices are all important determinants of home dust lead levels. We found a much greater reduction in dust lead levels immediately after cleaning in the high-exposure homes relative to baseline values, and this might have influenced the longevity of the cleaning effect.

The children who lived in the low-exposure homes had lower blood lead levels at the second precleaning clinical visit (18 mo after randomization), compared with children living in high-exposure homes. The blood lead levels declined in both groups over time, but the blood lead levels of the high-exposure children remained about $4 \mu\text{g}/\text{dl}$ higher throughout the second half of the study. Children who lived in homes with higher dust lead levels (homes in which at least 1 surface exceeded an EPA clearance standard) had higher blood lead levels 18 mo into the TLC study, and they continued to have higher blood lead levels than children who resided in low-exposure homes. This difference persisted even with a second professional lead dust cleaning. Overall hygiene of the home may be more important for a child's blood lead level than the infrequent professional cleanings performed in the TLC study. Regular and frequent cleaning by household members—or living in a home with lower dust lead levels (perhaps resulting from better maintenance and/or less deterioration)—may be effective in keeping down children's blood lead levels.

Some studies in which repeated professional cleanings were offered have demonstrated the effectiveness of this intervention. A randomized control trial of multiple cleanings of lead-exposed urban children's

homes²⁴ revealed significant decreases in house dust levels and blood lead levels in the intervention group approximately 1 yr after baseline testing, compared with a control group; cleanings occurred every 2 wk and included HEPA vacuuming and wet cleaning. A nonrandomized, controlled study of wet mopping twice monthly by a dust-control team for 1 yr in homes of children with significantly elevated blood lead levels at study enrollment demonstrated a significant reduction in blood lead levels of intervention vs. control children.²⁵ In contrast, a randomized control trial of only HEPA vacuuming of floors every 6 wk for 10 mo with no wet cleaning resulted in no significant effects on either blood lead levels or GM dust lead loadings on carpeted surfaces.²⁶ In several studies in which both education and cleaning supplies were provided to families of children, no significant reductions in (or prevention of increases in) blood lead levels, or significant reductions in dust levels, were found.^{27–29}

The data from these studies suggest that frequent cleaning, including wet wiping and mopping, is important in decreasing both blood lead and home lead dust levels. The 2 studies in which significant decreases in children's blood lead levels occurred after cleaning interventions used professional cleaners who performed many cleanings, presumably with great investment of time and money. Our study families received only 2 professional cleanings, coupled with frequent encouragement of parents to perform repeated home cleanings and provision of equipment and supplies. However, because we did not ask parents to keep cleaning diaries, we could not demonstrate objectively the frequency of cleaning performed by study families.

The homes in which we did a second cleaning were judged to have better overall maintenance and a lower lead exposure potential, although they did have an equal or higher percentage of reported problems with plumbing and/or roof leaks—problems that can result in chipping and peeling of paint. Selection bias might have played a role as well, inasmuch as those caregivers who consented to the cleaning intervention might have been different than parents who did not agree to participate (i.e., more likely to maintain and clean their home regularly, to restrict mouthing behaviors, or to perform other activities to improve the health of their child). The homes of families who consented to the cleaning are not likely to represent the entire range of household dust lead levels in the TLC Philadelphia housing stock. It is, therefore, not appropriate for us to generalize our results to the entire TLC population or to all urban housing. Other limitations of this study included the relatively small number of homes that received the second cleaning, the lack of follow-up samples as a result of attrition, the large percentage of negative dust-wipe

values, and the absence of a cleaning diary to document cleaning activity.

Conclusions

A professional cleaning of the homes of children who had elevated blood lead levels produced immediate reductions in dust lead levels; however, lead dust levels rebounded to precleaning values within 3–6 mo. We documented lower blood lead levels among children in homes with low precleaning dust lead levels, compared with the blood lead levels of children who lived in high-exposure homes. The blood lead levels of children in the low-exposure homes were not responsive to the single professional cleaning intervention, irrespective of the treatment arm of the study. More frequent, repeated cleanings may be required in order to produce sustained reductions in blood lead levels or dust lead levels in urban homes. Additional studies that track the effects of lead dust suppression over time are needed to better characterize the overall effect of cleaning interventions.

* * * * *

The authors acknowledge the assistance and cooperation of the entire staff of the TLC Trial, especially Suzette Harper and Judy Quinn in Philadelphia, the TLC staff at the Harvard School of Public Health, and Adrienne Ettinger for her contributions to the data analysis.

This study was funded by the National Institute of Environmental Health Sciences, in cooperation with the Office of Research on Minority Health, under contract #NO1-ES-35361.

Submitted for publication August 13, 2002; revised; accepted for publication August 4, 2003.

Requests for reprints should be sent to Carla Campbell, M.D., M.S., The Children's Hospital of Philadelphia, Primary Care Center, Suite 120, 39th and Chestnut Streets, Philadelphia, PA 19104.

E-mail: campbellc@email.chop.edu

* * * * *

References

1. Sayre JW, Charney E, Vostl J. House and hand dust as a potential source of childhood lead exposure. *Am J Dis Child* 1974; 127:167–70.
2. Charney E, Sayre JW, Coulter M. Increased lead absorption in inner-city children: where does the lead come from? *Pediatrics* 1980; 65:226–31.
3. Bornschein RL, Succop P, Dietrich KN, et al. The influence of social and environmental factors on dust lead, hand lead, and blood lead levels in young children. *Environ Res* 1985; 38:108–18.
4. Bellinger D, Leviton A, Rabinowitz M, et al. Correlates of low-level lead exposure in urban children at 2 years of age. *Pediatrics* 1986; 77:826–33.
5. Lanphear BP, Emond M, Jacobs DE, et al. A side-by-side comparison of dust collection methods for sampling lead-contaminated house dust. *Environ Res* 1995; 68:114–23.
6. Lanphear BP, Roghmann KJ. Pathways of lead exposure in urban children. *Environ Res* 1997; 74:67–73.

7. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. A pooled analysis of 12 epidemiologic studies. *Environ Res* 1998; 79:51–68.
8. Treatment of Lead-Exposed Children Trial Group. The Treatment of Lead-Exposed Children (TLC) Trial: design and recruitment for a study of the effect of oral chelation on growth and development in toddlers. *Paediatr Perinat Epidemiol* 1998;12:313–33.
9. Treatment of Lead-Exposed Children Trial Group. Safety and efficacy of succimer in toddlers with blood lead levels of 20–44 µg/dl. *Pediatr Res* 2000; 48:593–99.
10. Ettinger AS, Bornschein RL, Farfel M, et al. Assessment of cleaning to control lead dust in homes of children with moderate lead poisoning: Treatment of Lead-Exposed Children Trial. *Environ Health Perspect* 2002; 110(12): A773–79.
11. U.S. Department of Housing and Urban Development (HUD). Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing. Washington, DC: HUD, 1995.
12. National Institute for Occupational Safety and Health (NIOSH). NIOSH Manual of Analytical Methods (NMAM). 4th ed. Method 7082: Lead by Flame AAS. Washington, DC: Government Printing Office, 15 August 1994; Issue 2:1–7.
13. Department of Health and Human Services, National Institute for Occupational Safety and Health. January 1997. Protecting Workers Exposed to Lead-based Paint Hazards: A Report to Congress. Chapter 5, Lead-based Paint Hazards. Publication No. 98-112; 86 pp. <<http://www.cdc.gov/niosh/pdfs/98-112.pdf>> Accessed 2/11/05.
14. Miller DT, Paschal DC, Gunter EW, et al. Determination of lead in blood using electrothermal atomisation atomic absorption spectrometry with a L'vou platform and matrix modifier. *Analyst* 1987; 112:1701–04.
15. Porter PS, Ward RC, Bell HF. The detection limit. *Environ Sci Technol* 1988; 22:856–61.
16. Lambert D, Peterson B, Terpenning I. Nondetects, detection limits, and the probability of detection. *J Am Stat Assoc* 1991; 86:266–72.
17. Wendelberger JR. Methods for handling values below detection limits. In: Proceedings of the Section on Statistics and the Environment. Alexandria, VA: American Statistical Association, 1995; pp 38–43.
18. U.S. Environmental Protection Agency (EPA). Lead; Identification of Dangerous Levels of Lead. Final Rule 40 CFR Part 745. Fed Reg 2001; 66(4):1205–40.
19. Lanphear BP, Weitzman M, Winter NL, et al. Lead-contaminated house dust and urban children's blood lead levels. *Am J Public Health* 1996; 86:1416–21.
20. Staes C, Rinehart R. Does Residential Lead-Based Paint Hazard Control Work? A Review of the Scientific Evidence. Columbia, MD: The National Center for Lead-Safe Housing, 1995.
21. U.S. Environmental Protection Agency (EPA). Review of Studies Addressing Lead Abatement Effectiveness: Updated Edition. Washington, DC: U.S. EPA, 1998.
22. The National Center for Lead-Safe Housing. Evaluation of the HUD lead-based paint hazard control grant program. Fifth Interim Report. Columbia, MD: The National Center for Lead-Safe Housing, 1998.
23. Rogan WJ, Dietrich KN, Ware JH, et al. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. *N Engl J Med* 2001; 344:1421–26.
24. Rhoads GG, Ettinger AS, Weisel CP, et al. The effect of dust lead control on blood lead in toddlers: a randomized trial. *Pediatrics* 1999; 103:551–55.
25. Charney E, Kessler B, Farfel M, et al. A controlled trial of the effect of dust-control measures on blood lead levels. *N Engl J Med* 1983; 309:1089–93.
26. Hilts SR, Hertzman C, Marion SA. A controlled trial of the effect of HEPA vacuuming on childhood lead exposure. *Can J Public Health* 1995; 86:345–50.
27. Lanphear BP, Winter NL, Apetz L, et al. A randomized trial of the effect of dust control on children's blood lead levels. *Pediatrics* 1996; 98:35–40.
28. Lanphear BP, Howard C, Eberly S, et al. Primary prevention of childhood lead exposure: a randomized trial of dust control. *Pediatrics* 1999; 103:772–77.
29. Lanphear BP, Eberly S, Howard CR. Long-term effects of dust control on blood lead concentration. *Pediatrics* 2000; 106(4):E48.