The Effect of Interior Lead Hazard Controls on Children's Blood Lead Concentrations: A Systematic Evaluation

Erin Haynes,[1,2] Bruce P. Lanphear,[2] Ellen Tohn,[3] Nick Farr,[4] and George G. Rhoads[5]

[1]University of Michigan School of Public Health, Ann Arbor, Michigan, USA; [2]Children's Hospital Medical Center, Cincinnati, Ohio, USA; [3]ERT Associates, Wayland, Massachusetts, USA; [4]The National Center for Lead-Safe Housing, Columbia, Maryland, USA; [5]The Environmental and Occupational Health Sciences Institute, University of Medicine and Dentistry of New Jersey School of Public Health Piscataway, New Jersey, USA

Dust control is often recommended to prevent children's exposure to residential lead hazards, but the effect of these controls on children's blood lead concentrations is uncertain. We conducted a systematic review of randomized, controlled trials of low-cost, lead hazard control interventions to determine the effect of lead hazard control on children's blood lead concentration. Four trials met the inclusion criteria. We examined mean blood lead concentration and elevated blood lead concentrations (≥ 10 µg/dL, ≥ 15 µg/dL, and ≥ 20 µg/dL) and found no significant differences in mean change in blood lead concentration for children by random group assignment (children assigned to the intervention group compared with those assigned to the control group). We found no significant difference between the intervention and control groups in the percentage of children with blood lead ≥ 10 µg/dL, 29% versus 32% [odds ratio (OR), 0.85; 95% confidence interval (CI), 0.56–1.3], but there was a significant difference in the percentage of children with blood lead ≥ 15 µg/dL between the intervention and control groups, 6% versus 14% (OR, 0.40; 95% CI, 0.21–0.80) and in the percentage of children with blood lead ≥ 20 µg/dL between the intervention and control groups, 2% versus 6% (OR, 0.29; 95% CI, 0.10–0.85). We conclude that although low-cost, interior lead hazard control was associated with 50% or greater reduction in the proportion of children who had blood lead concentrations exceeding 15 µg/dL and ≥ 20 µg/dL, there was no substantial effect on mean blood lead concentration. *Key words:* **blood lead, children, environmental exposure, lead-contaminated house dust, lead poisoning, prevention, randomized trial.** *Environ Health Perspect* **110:103–107 (2002). [Online 19 December 2001]** *http://ehpnet1.niehs.nih.gov/docs/2002/110p103-107haynes/abstract.html*

Despite dramatic reductions in children's blood lead concentrations over the last two decades, subclinical lead toxicity remains a significant risk for urban infants and children (*1–3*). Low-level elevation in blood lead concentration has been associated with cognitive deficits, aggressive behavior, and hearing dysfunction (*4–7*). The Centers for Disease Control and Prevention (CDC) have estimated that 890,000, or 4.4%, of U.S. children 5 years and younger have blood lead concentrations of 10 µg/dL or higher (*1*). Moreover, there is increasing evidence that no detectable threshold exists for the adverse effects of lead exposure on neurodevelopment (*6–8*).

Efforts to prevent exposure of children to residential lead hazards include education and lead hazard controls. For the vast majority of children, educational efforts—such as dust control, hand washing, and reducing children's mouthing behaviors—represent the major strategy to reduce lead exposure, ingestion, and absorption (*9,10*). One professional dust intervention trial led to significant reductions in highly exposed children (*11*), but it is clear that education alone is not adequate to prevent children's exposure to lead, as measured by blood lead concentration (*12–16*). Moreover, despite considerable evidence that higher dietary calcium intake is associated with lower blood lead concentration, the beneficial effects of calcium supplementation on children's blood lead concentration remains uncertain (*17*).

Lead hazard controls typically are implemented only after a child is identified with a blood lead concentration consistently above 15 µg/dL or 20 µg/dL. For these children, there is a spectrum of lead hazard controls, including full abatement (complete removal of lead-contaminated paint), encapsulation (making lead-based paint inaccessible with construction material or polymers that are applied like paint), replacement of window and door frames, stabilizing deteriorated paint, and professional dust control (*18*). The advantage of lead hazard controls is that they do not rely on modifying a family's behavior to reduce environmental exposures to lead. On the other hand, they are more expensive, ranging in cost from $500 to $15,000 or more (*18*).

There is some evidence that abatement or paint stabilization can reduce blood lead concentrations in children with concentrations above 30 µg/dL (*11,19*), but the evidence is inconsistent (*20,21*). Moreover, there are no randomized trials that demonstrate the efficacy or safety of lead hazard controls for children who have blood lead concentrations below 30 µg/dL (*12*). Indeed, one controlled trial found that paint abatement was associated with a 6.5 µg/dL increase in blood lead concentration among children in the abatement group, despite using the U.S. Department of Housing and Urban Development's postabatement clearance testing (*21*). In contrast, there is some evidence that the long-term benefit of paint abatement is considerable (*22*).

The purpose of this study was to use meta-analysis to determine whether low-cost strategies (defined as < $2,500 per housing unit or family) aimed at controlling lead-contaminated dust effectively prevent childhood lead exposure, as measured by children's blood lead concentrations.

Methods

Search strategy. We searched the PubMed (National Library of Medicine, Bethesda, MD) and Cochrane Library (Oxford, England) databases. We combined "lead*" and "dust*" as a title word or text word with "control*," "trial*," "controlled study," "blood lead levels," and "hazard" in all fields. In addition, we reviewed summary reports of lead hazard controls conducted by the U.S. Environmental Protection Agency and the National Center for Lead-Safe Housing (*23,24*). Primary authors were contacted to obtain additional information, if necessary.

Articles eligible for inclusion in the meta-analysis met the following criteria: *a*) randomized allocation of children to either a control group or intervention group; *b*) low-cost interventions, defined as < $2,500; *c*) blood lead concentration used as a measured outcome; and *d*) trial was not conducted in a community with a continual lead emission source. We evaluated the quality of each included trial using a modified version of

Address correspondence to B.P. Lanphear, Division of General and Community Pediatrics, Children's Hospital Medical Center, 3333 Burnet Avenue, Cincinnati, Ohio 45229-3039 USA. Telephone: (513) 636-3778. Fax: (513) 636-4402. E-mail: bruce.lanphear@chmcc.org

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Prendiville's criteria (*25,26*). We examined each trial according to three methodologic aspects: adequacy of allocation concealment at enrollment, control of selection bias (extent to which analyses are based on all randomized participants), and control of information bias (blinding observers).

We illustrated the utility of dust control by using measures of clinical efficacy: the absolute risk reduction (ARR), relative risk reduction (RRR), and the number needed to be treated (NNT). The absolute risk reduction, the difference in event rates between the control and intervention groups, expresses the consequences of not providing the intervention. The relative risk reduction, the difference in the event rates or ARR divided by the event rate in the control group, is the reduction of adverse events achieved by the intervention. The number needed to be treated (NNT), the inverse of the ARR, is the number of children who must be treated to prevent one adverse event. In this study, an adverse event is a blood lead concentration ≥ 10 µg/dL, ≥ 15 µg/dL, or ≥ 20 µg/dL.

Statistical Methods

We divided the studies into two types of intervention trials: education combined with cleaning equipment or supplies, and dust control performed by cleaning professionals. Changes in mean blood lead concentration from baseline to follow-up are reported by random group assignment. The mean differences were calculated for each study, and the variances for each difference were calculated as

$$\frac{(n_1-1)sd_1^2+(n_2-1)sd_2^2}{(N-2)}.$$

Because blood lead concentration was an outcome measure in all studies, we calculated the pooled mean differences between intervention and control groups. The mean differences were weighted according to the reciprocal of their variance, and a pooled mean difference was calculated using these weights:

$$\Sigma \text{ weight} \times \text{mean difference} / \Sigma \text{ weight}.$$

We used two-tailed p-values of < 0.05 as the level of statistical significance, and also calculated 95% confidence intervals (CIs).

We performed all statistical analyses using Review Manager (RevMan) software using a fixed-effect model (*27,28*). The fixed-effect model assumes that the true effects of treatment are the same in all studies. We tested for heterogeneity using the Mantel-Haenszel Q-statistic for pooled effect sizes (*29*).

We also examined the proportion of children with elevated blood lead concentration by random group assignment after the intervention. Reported comparisons of blood lead concentrations in excess of 10 µg/dL, ≥ 15 µg/dL, and ≥ 20 µg/dL were inclusive.

Because all the studies included in this meta-analysis were randomized controlled trials, we used the intention-to-treat principle: Study subjects that were randomly assigned to either an intervention or control group remained in that group for the analysis, regardless of compliance to the proposed intervention. We did not adjust for baseline differences in study population, such as age, season, race, or initial blood lead concentration.

Results

Trials included in the analysis. The literature search returned 405 articles. Of these, nine lead hazard control trials were identified (*11,13–16,19,30–32*), and five fulfilled the inclusion criteria (*13–15,30,31*). One trial was published twice: once following the 24-month follow-up of children and again following the 48-month follow-up (*14,31*). For this analysis, we excluded the shorter follow-up (*14*). Four other studies were excluded from the meta-analysis: One was conducted in a community with an active lead smelter (*16*) and three were not randomized, controlled trials (*11,19,32*) (Table 1).

All studies used venous blood samples to measure blood lead concentration (Table 1). Three studies included parental education (*13,15,31*), two studies provided the families with cleaning supplies or equipment (*15,31*), two provided professional cleaning (*13,30*), and one made minor housing repairs (*30*). The length of follow-up ranged from 6 to 48 months.

Results of meta-analysis. The weighted mean change in blood lead concentration from baseline to follow-up in all studies was –0.62 µg/dL (95% CI, –1.55 to 0.32) (Figure 1, Table 2). There was no significant difference in the change in blood lead concentrations between the intervention and control groups for either the educational dust control trials (–0.33 µg/dL; 95%CI, –1.4 to 0.74) or the professional dust control trials (–1.52 µg/dL; 95% CI, –3.41 to 0.37).

There was no significant difference in the frequency of children who had blood lead concentrations ≥ 10 µg/dL (p = 0.46) (Table 3). There was, however, a significant difference between children in the intervention and control groups who had blood lead concentrations ≥ 15 µg/dL (p= 0.008) and ≥ 20 µg/dL (p = 0.024) (Table 3). The risk of having a blood lead concentration ≥ 15 µg/dL or ≥ 20 µg/dL was 2–3 times lower for children who received low-cost, lead hazard control. The odds ratio for children assigned to the experimental group having blood lead concentrations ≥ 15 µg/dL and ≥ 20 µg/dL after the intervention was 0.40 (95% CI, 0.21, 0.79) and 0.29 (95% CI, 0.01, 0.85), respectively (Table 3).

To test whether the professional dust control trial was driving our results, we examined the effect of the intervention after removing the Rhoads trial. Consistent with the primary analysis, there was no significant difference in

Table 1. Characteristics of included and excluded studies in the meta-analysis of interventions to reduce blood lead concentrations in children.

Study/year (reference)	RCT	Type of Intervention	Mean baseline blood lead (µg/dL)	Mean age or age range at baseline (months)	Length of follow-up (months)	No. in study at baseline	Percent attrition	Absolute change	p-Value
Included studies									
Lanphear 1996 (*15*)	Y	Education and supplies	6.7*	12–31	7	104	8.7	–0.55	0.50*
Aschengrau 1998 (*30*)	Y	Dust control, repair, and painting	16.9	24.5	6	41[a]	41.5	1.1	0.58
Rhoads 1999 (*13*)	Y	Professional dust control	12	20 ± 3	12	113	12.4	–1.9	< 0.05[b]
Lanphear 2000 (*31*)	Y	Education and equipment	2.8	6	48	275	31.3	–0.2	0.73
Excluded studies[c]									
Charney 1983 (*11*)	N	Dust control and abatement	38.5	15–70	6	78	37.2	–6.1	< 0.001
Staes 1994 (*19*)	N	Paint stabilization	35	< 6	10–14	185	70.8	–4.0	0.07
Hilts 1995 (*16*)	Y	Dust control–active smelter	11.56	32.4	9	122	9	0.3	0.85
Schultz 1999 (*32*)	N	Multifactorial education	20–24	40	6–7	413	0	–3.1	< 0.001

Abbreviations: N, no; RCT, randomized controlled trial; Y, yes. [a]Includes children randomized to control and intervention groups. [b]One-sided test. [c]Studies were excluded if they were not RCTs, were conducted in an interior lead hazard control intervention exceeding $2,500, did not use blood lead as a measured outcome, and were conducted in a community with an active lead emissions source.
*Used median (interquartile range).

the frequency of children who had blood lead concentrations ≥ 10 µg/dL (p = 0.65). There was still a significant difference between intervention and control group children who had blood lead concentrations ≥ 15 µg/dL (p = 0.035) and ≥ 20 µg/dL (p = 0.042).

Inclusion of the randomized, controlled trial by Hilts et al. (*16*)—the community with an active lead smelter—altered the findings slightly. Consistent with our primary analysis, there was no significant difference in the blood lead concentration (–0.30 µg/dL; 95% CI, –1.07 to 0.47) by random group assignment. There was, however, only a marginally significant reduction in the proportion of children who had a blood lead concentration of 15 µg/dL or higher (p = 0.075). The reduction in the proportion of children in the dust control groups who had a blood lead concentration ≥ 20 µg/dL remained statistically significant (p = 0.034).

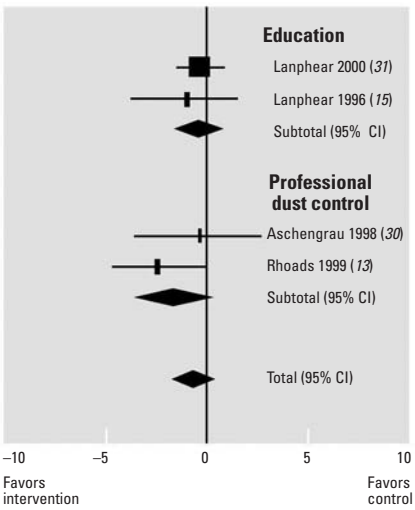

Figure 1. Weighted mean change in blood lead concentrations in children by random allocation to an intervention or control group, from baseline to follow-up.

Additional clinical significance of the data is obtained by pooling the included studies (*12,14,27,30*), and determining the RRR, ARR, and the NNT (*33*). The interventions decreased the risk of developing a blood lead concentration of ≥ 15 µg/dL or ≥ 20 µg/dL by 57% and 67%, respectively. The ARR was 8% and 4% for blood lead concentrations ≥ 15 µg/dL and ≥ 20 µg/dL, respectively (Table 3). Thirteen children need to be treated to prevent one child from developing a blood lead concentration of ≥ 15 µg/dL, and 25 children need to be treated to prevent one child from developing a blood lead concentration of ≥ 20 µg/dL (Table 3).

The quality of each study included was high. All authors were blinded to the random allocation of children to the intervention and control groups, randomly assigned subjects by telephone or opaque sealed envelopes, and used the intention-to-treat principle.

Discussion

We did not find a significant decline in mean blood lead concentrations among children who received low-cost, lead hazard interventions compared with the control groups, but there was a significant reduction in the proportion of children who had blood lead concentrations > 15 µg/dL and > 20 µg/dL. The interventions produced a ≥ 50% reduction in the number of children developing blood lead concentrations > 15 µg/dL and > 20 µg/dL. This finding is consistent with previous research indicating that lead hazard controls produce a greater reduction in blood lead concentration for children who have higher blood lead concentration (*11,19*). Collectively, these studies confirm that lead-contaminated house dust is an important source of lead exposure among children, especially for urban children who have higher blood lead concentrations. Still, the overall effect of dust control was modest.

One reason for the modest effect of dust control was that the interventions did not eliminate ongoing lead contamination of house dust from exterior sources or from interior lead-based paint. For instance, there was less effective dust control in the community with an active smelter (*16*). Presumably, this was caused by ongoing contamination of house dust and inhalation from lead emissions. Consistent with other trials, it is unlikely that dust control will dramatically reduce lead exposure unless the ultimate source of lead—industrial or residential—is controlled (*13–15, 31*).

Lioy and others measured changes in dust lead loading three times during a 12-month intervention (*34*). They found a 35% decline in dust lead loading by the third visit in the intervention homes (p = 0.011) from biweekly professional dust control, with each session consisting of about five person-hours of effort (*13*). In contrast, there was no significant difference in dust lead loading by group assignment in the educational dust control trials (*15,31*). Thus, the cleaning regimen—or adherence to the cleaning regimen—in the educational trials may not have been adequate to reduce dust lead levels. Families in one trial were instructed to clean interior windowsills and floors near windows once every month and the whole house once every 3 months (*31*).

Interventions involving parental cleaning as the sole dust-control intervention rely heavily on the effect of the educational intervention that is intended to motivate parents to engage in complex cleaning tasks. In one trial (*15*), the educational session lasted only

Table 2. Weighted mean change in blood lead concentrations in children by random allocation to an intervention or control group, from baseline to follow-up.

Study	Blood lead concentrations: Intervention No. children	Intervention Mean (SD)	Control No. children	Control Mean (SD)	Weight (%)	Weighted mean difference (95% CI)
Education						
Lanphear 2000 (*31*)	96	3.73 (3.79)	93	3.90 (4.65)	59.2	−0.17 (−1.38–1.04)
Lanphear 1996 (*15*)	52	−0.47 (2.75)	43	0.42 (7.26)	16.5	−0.89 (−3.19–1.41)
Subtotal (95% CI)	148		136		75.7	−0.33 (−1.40–0.74)
χ^2 = 0.30, *df* = 1, *z* = 0.60						
Professional dust control						
Aschengrau 1998 (*30*)	11	−6.20 (3.70)	13	−5.90 (4.20)	8.7	−0.30 (−3.46–2.86)
Rhoads 1999 (*13*)	46	−2.10 (5.70)	53	0.10 (6.30)	15.5	−2.20 (−4.56–0.16)
Subtotal (95% CI)	57		66		24.1	−1.52 (−3.41–0.37)
χ^2 = 0.89, *df* = 1, *z* = 1.57						
Combined total (95% CI)	205		202		100.0	−0.62 (−1.55–0.32)
χ^2 = 2.34, *df* = 3, *z* = 1.29						

Abbreviations: *df*, degrees of freedom; *z*, z-score. [a]The weight of the study is indicated by the thickness of the mean marker.

Table 3. Summary of postintervention blood lead concentrations[a] and measures of clinical efficacy by random allocation to an intervention or control group.

Blood lead (µg/dL)	No. of children (%): Intervention	Control	Odds ratio (95% CI)	*p*-Value	RRR (%)	ARR (%)	NTT
< 10	146 (71)	137 (68)	1.17 (0.77–1.79)	0.46			
≥ 10	59 (29)	65 (32)	0.85 (0.56–1.30)	0.46			
≥ 15	13 (6)	29 (14)	0.40 (0.21–0.79)	0.008	57	8	13
≥ 20	4 (2)	13 (6)	0.29 (0.10–0.85)	0.024	67	4	25

[a]Blood lead concentrations exceeding 10 µg/dL include blood lead concentrations ≥ 15 and ≥ 20; blood lead concentration ≥ 15 µg/dL include blood lead concentrations ≥ 20 µg/dL.

5 min whereas in the other urban trial (*14,31*) an interactive and extensive educational model called facilitation was used to conduct the training session. Although families received, on average, over 6 intensive visits and were provided with cleaning supplies and equipment (*14,31*), they may not have had adequate motivation to clean their homes. The extent of home cleaning by families was estimated by monitoring the amount of cleaning supplies that needed replenishing (*14,31*). Still, adjusting for this behavior did significantly affect blood lead concentration (*14,31*).

Published, randomized, controlled trials indicate that regular visits by professional dust control teams led to greater reductions in dust lead loading and blood lead concentrations (*13,30*). One advantage of such efforts is that dust control can be initiated immediately after a child is identified with lead poisoning. Still, there are several reasons not to rely on dust controls as the primary strategy to prevent childhood lead exposure. First, the magnitude of the reduction in blood lead concentration was modest. Second, the effects may not benefit children who have lower baseline blood lead concentrations < 15 μg/dL, and there is growing evidence of substantial adverse health effects for blood lead concentration < 10 μg/dL (*6–8*). Third, professional cleaning—which led to the largest reductions in blood lead concentration—is not available for most high-risk families in substandard housing. Finally, although there was evidence that dust control was efficacious in reducing children's blood lead concentrations, the adverse effects of undue lead exposure persist even after blood lead levels decline (*35,36*). Thus, from a societal perspective, it is unethical to rely on interventions that occur only after children are unduly exposed (*12*).

Trials excluded from the analysis. The only randomized, controlled trial excluded from the analysis was reported by Hilts et al. (*16*). We excluded the trial because it involved children living near an active smelter. The other three studies excluded from the analysis were not randomized, controlled trials (*11,19,32*) (Table 1). Although they reported a significant decline in blood lead concentration after the intervention, the study children had baseline blood lead concentrations of ≥ 20 μg/dL (*11,19,32*).

The retrospective analysis of paint stabilization intervention by Staes and others (*19*) appeared to benefit children who had blood lead concentration of ≥ 35 μg/dL, but not children who had blood lead concentrations < 35 μg/dL. Charney and others (*11*) removed peeling or deteriorated interior and exterior lead-based paint before conducting dust control. Blood lead concentrations were significantly reduced ($p = 0.001$) in children with blood lead concentrations 30–49 μg/dL, but the study was not a randomized controlled trial (*11*). Although Schultz and others (*32*) observed a significant decline in blood lead concentration for children in their education group compared to a reference group ($p < 0.001$), the reference group was comprised of historical controls and families whom the investigators were unable to contact for the intervention. The secular downward trend in children's blood lead concentration makes any comparison with historical controls suspect, whereas families who could not be contacted may differ from those families that were enrolled in their study (*1,32*).

To our knowledge, there are no randomized, controlled trials examining the efficacy of lead hazard controls to reduce children's blood lead concentrations other than low-cost interventions reported in our analysis. There also were no published randomized, controlled trials that examined the effect of a multifactorial intervention (i.e., a combination of dust control, calcium supplementation, and behavioral modification) on children's blood lead concentration. Thus, until randomized controlled trials are conducted to test whether existing lead hazards controls are safe and efficacious, we will continue to rely on unproven lead hazard controls to reduce childhood lead exposure.

The major limitation was that three of the four trials in this meta-analysis were secondary prevention trials. Theoretically, reducing children's exposure to lead in early infancy or before birth would produce more dramatic effects from lead hazard controls. A second limitation is that it was often difficult to mask interviewers or technicians in this type of community-based research, although some researchers did make an attempt (*14,15,31*). Failure to mask researchers can cause an overestimation of the effectiveness of various interventions (*37*). Indeed, although blood lead concentrations were greater in nonrandomized controlled trials, we also found that there was a greater estimated effect in the nonrandomized trials (*11,19,30*).

Low-cost, lead hazard controls produce a modest, but significant decline in the proportion of children with blood lead concentrations ≥ 15 μg/dL. It is unknown whether a multifactorial intervention will produce a greater reduction in blood lead concentration than dust control alone. We also do not know if other low-cost (< $2,500) environmental lead hazard controls (e.g., window treatments, paint stabilization, and/or creating smooth and cleanable surfaces) will produce a significant decline in children's blood lead concentrations, especially for children who have blood lead concentrations < 30 μg/dL. It is time to test the effectiveness of lead hazard controls using randomized, controlled trials to ensure that children are adequately protected against subclinical lead toxicity.

References and Notes

1. Pirkle JL, Kaufmann RB, Brody DJ, Hickman T, Gunter EW, Paschal DC. Exposure of the U.S. population to lead, 1991–1994. Environ Health Perspect 106:745–750 (1998).
2. Lanphear BP, Byrd RS, Auinger P, Schaffer SJ. Community characteristics associated with children's blood lead levels. Pediatrics 101:264–271 (1998).
3. Sargent JD, Brown MJ, Freeman JL, Bailey A, Goodman D, Freeman DH. Childhood lead poisoning in Massachusetts communities: its association with sociodemographic and housing characteristics. Am J Public Health 85:528–534 (1995).
4. Baghurst PA, McMichael AJ, Wigg NR. Environmental exposure to lead and children's intelligence at the age of seven years. The Port Pirie Cohort Study. N Engl J Med 327:1279–1284 (1992).
5. Friscancho AR, Ryan AS. Decreased stature associated with moderate blood lead concentrations in Mexican-American children. Am J Clin Nutr 54:516–519 (1991).
6. Schwartz J, Otto D. Lead and minor hearing impairment. Arch Environ Health 46:300–305 (1991).
7. Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. Environ Res 65:42–55 (1994).
8. Lanphear BP, Dietrich KN, Auinger P, Cox C. Cognitive deficits associated with blood lead levels < 10 μg/dL in U.S. children and adolescents. Public Health Rep 115:521–529 (2000).
9. CDC. Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials. Atlanta, GA:Centers for Disease Control and Prevention, 1997.
10. AAP. Screening for elevated blood lead levels. American Academy of Pediatrics, Committee on Environmental Health. Pediatrics 101:1072–1078 (1998).
11. Charney E, Kessler B, Farfel M, Jackson D. Childhood lead poisoning: a controlled trial of the effect of dust-control measures on blood lead levels. N Engl J Med 309:1089–1093 (1983).
12. Lanphear BP. The paradox of lead poisoning prevention. Science 281:1617–1618 (1998).
13. Rhoads GG, Ettinger AS, Weisel CP, Buckley TJ, Goldman KD, Adgate J, Lioy PJ. The effect of dust control on blood lead in toddlers: a randomized trial. Pediatrics 103:551–555 (1999).
14. Lanphear BP, Howard CR, Eberly S, Auinger P, Kilassa J, Weitzman M, Schaffer SJ, Alexander K. Primary prevention of childhood lead exposure: a randomized trial of dust control. Pediatrics 103:772–777 (1999).
15. Lanphear BP, Winter NL, Apetz L, Eberly S, Weitzman M. A randomized trial of the effect of dust control on children's blood lead levels. Pediatrics 98:35–40 (1996).
16. Hilts SR, Hertzman C, Marion SA. A controlled trial of the effect of HEPA vacuuming on childhood lead exposure. Can J Public Health 86:345–350 (1995).
17. Sargent JD, Dalton MA, O'Connor GT, Olmstead EM, Klein RZ. Randomized trial of calcium glycerophosphate-supplemented infant formula to prevent lead absorption. Am J Clin Nutr 69:1224–1230 (1999).
18. U.S. HUD. Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing. Washington, DC:Department of Housing and Urban Development, Office of Lead-Based Paint Abatement and Poisoning Prevention, 1995.
19. Staes C, Matte T, Copley CG, Flanders D, Binder S. Retrospective study of the impact of lead-based paint hazard remediation on children's blood lead levels in St. Louis, Missouri. Am J Epid 139(10):1016–1026 (1994).
20. Rich DQ, Rhoads GG, Wartenberg D, Sweatlock J. A retrospective follow-up of the effects of home lead abatements on childhood blood lead levels. J Environ Health (in press).
21. Aschengrau A, Beiser A, Bellinger D, Copenhafer D,

Weitzmann M. Residential lead-based-paint hazard remediation and soil lead abatement: their impact among children with mildly elevated blood lead levels. Am J Public Health 87:1698–1702 (1997).
22. Sargent JD, Dalton M, Demidenko E, Simon P, Klein RZ. The association between state housing policy and lead poisoning in children. Am J Public Health 89:1690–1695 (1999).
23. U.S. EPA. A review of studies addressing lead abatement effectiveness: updated edition. EPA 747-B-98-001. Washington, DC:U.S. Environmental Protection Agency, 1998.
24. Staes C, Rinehart R. Does Residential Lead-based Paint Hazard Control Work? A Review of the Scientific Evidence. Columbia, MD:National Center for Lead-Safe Housing, 1995.
25. Prendiville W, Elbourne D, Chalmers I. The effects of routine oxytocic administration in the management of the third stage of labor: an overview of the evidence from controlled trials. Br J Obstet Gynaecol 95:3–16 (1988).
26. Roberts I, Kramer MS, Suissa S. Does home visiting prevent childhood injury? A systematic review of randomised controlled trials. Br Med J 312:29–33 (1996).
27. Review Manager: Version 3.1 for Windows. Oxford: Cochrane Collection, 1998.
28. Petitti D. Meta-Analysis, Decision Analysis, and Cost-Effectiveness Analysis. New York:Oxford University Press, 1994.
29. Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. J Natl Cancer Inst 22: 719–748 (1959).
30. Aschengrau A, Hardy S, Mackey P, Pultinas D. The impact of low technology lead hazard reduction activities among children with mildly elevated blood lead levels. Environ Res 79:41–50 (1998).
31. Lanphear BP, Eberly S, Howard CR. Long-term effect of dust control on blood lead concentrations. Pediatrics 106(4):48–52 (2000).
32. Schultz B, Pawel D, Murphy A. A retrospective examination of in-home educational visits to reduce childhood lead levels. Environ Res 80:364–368 (1999).
33. Laupacis A, Sacket D, Roberts R. An assessment of clinically useful measures of the consequences of treatment. N Engl J Med 318(26):1728–1733 (1988).
34. Lioy P, Yiin L, Adgate J, Weisel C, Rhoads G. The effectiveness of a home cleaning intervention strategy reducing potential dust and lead exposures. J Expo Anal Environ Epidemiol 8(1):17–35 (1998).
35. Burns JM, Baghurst PA, Sawyer MG, McMichael AJ, Tong SL. Lifetime low-level exposure to environmental lead and children's emotional and behavioral development at ages 11-13 years. The Port Pirie Cohort Study. Am J Epidemiol 149(8):740–749 (1999).
36. Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood: an 11 year follow-up report. N Engl J Med 322:83–88 (1990).
37. Kunz R, Oxman AD. The unpredictability paradox: a review of empirical comparisons of randomised and non-randomised clinical trails. Br Med J 317(7167):1185–1190 (1998).