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SPECIAL ARTICLE

Screening for Elevated Lead Levels in Childhood and Pregnancy: An Updated Summary of Evidence for the US Preventive Services Task Force

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ABSTRACT

BACKGROUND. In 1996, the US Preventive Services Task Force provided recommendations for routine screening of asymptomatic children and pregnant women for elevated blood lead levels. This review updates the evidence for the benefits and harms of screening and intervention for elevated blood lead in asymptomatic children and pregnant women.

METHODS. We searched Medline, reference lists of review articles, and tables of contents of leading pediatric journals for studies published in 1995 or later that contained new information about the prevalence, diagnosis, natural course, or treatment of elevated lead levels in asymptomatic children aged 1 to 5 years and pregnant women.

RESULTS. The prevalence of elevated blood lead levels among children and women in the United States, like that in the general population, continues to decline sharply, primarily because of marked reductions in environmental exposure, but still varies substantially among different communities and populations. Similar to the findings in 1996, our searches did not identify direct evidence from controlled studies that screening children for elevated blood lead levels results in improved health outcomes, and there was no direct evidence identified from controlled studies that screening improves pregnancy or perinatal outcomes. No new relevant information regarding the accuracy of screening for lead toxicity was identified during the update, and we did not identify evidence that demonstrates that universal screening for blood lead results in better clinical outcomes than targeted screening. Substantial new relevant information regarding the adverse effects of screening and interventions was not identified.

CONCLUSIONS. There is no persuasive evidence that screening for elevated lead levels in asymptomatic children will improve clinical outcomes. For those children who are screened and found to have elevated levels, there is conflicting evidence demonstrating the clinical effectiveness of early detection and intervention.

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Key Words
lead levels, children, pregnancy, screening, intervention

Abbreviations
USPSTF—US Preventive Services Task Force
BLL—blood lead level
AHRQ—Agency for Healthcare Research and Quality
EPC—Oregon Evidence-Based Practice Center
KQ—key question
CDC—Centers for Disease Control and Prevention
CI—confidence limit
TLC—Treatment of Lead-Exposed Children
RCT—randomized, controlled trial
DMSA—meso-2,3-dimercaptosuccinic acid
SES—socioeconomic status
GM—geometric mean
FTII—Fagan Test of Infant Intelligence
RBC—red blood cell
HUD—US Department of Housing and Urban Development

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IN 1996, THE US Preventive Services Task Force (USPSTF) recommended screening for elevated blood lead levels (BLLs) at 12 months of age in all children with identifiable risk factors and in all children living in communities in which the prevalence of elevated BLLs was high or unknown. There was insufficient evidence, however, to recommend a specific community prevalence below which targeted screening could be substituted for universal screening. The USPSTF found insufficient evidence to recommend for or against routine screening for lead exposure in asymptomatic pregnant women. The USPSTF also found insufficient evidence to recommend for or against trying to prevent lead exposure by counseling families to control lead dust by repeated household cleaning or to optimize caloric, iron, and calcium intake specifically to reduce lead absorption.[1]

METHODS

Problem Formulation

USPSTF members defined the scope of this update in cooperation with the Agency for Healthcare Research and Quality (AHRQ) and the Oregon Evidence-Based Practice Center (EPC) personnel. The USPSTF's goal for this update was to review the literature published since its 1996 recommendation to identify new evidence addressing the previously identified gaps in the literature, including the accuracy of risk-assessment questionnaires in children with varying BLLs, the population prevalence at which to change from targeted screening to universal screening, the effectiveness of interventions to lower lead levels, and cost-effectiveness analyses of lead screening programs. (See Appendix 1 and Fig 1 for key questions and analytic framework.)

Literature Review and Synthesis

We developed literature-search strategies and terms for each key question (KQ) and then searched Medline, CINAHL (Cumulative Index to Nursing and Allied Health Literature), and the Cochrane Library, assisted by an EPC reference librarian, to comprehensively update the literature from 1995 to August 2005 that contained new information about the prevalence, diagnosis, natural course, or treatment of elevated lead levels in asymptomatic children aged 1 to 5 years and pregnant women. The search was supplemented with reference lists of review articles, references from experts in the field, and reports, guidelines, and recommendations from government, nongovernment, and medical professional organizations. Inclusion criteria included the following:

1. The study must have been an original meta-analysis, prospective cohort study, controlled trial, quasi-experimental study with concurrent controls, or case-control study.
2. The study must not have been included in the 1996 review.
3. The study must have been rated at least "fair quality" using USPSTF criteria (Appendix 2).

Consistent with the scope of USPSTF recommendations, interventions needed to be relevant to primary care and feasible for delivery in primary care or by referral. Interventions were classified as pharmaceutical (chelation), environmental (residential lead paint, dust, or soil abatement), or nutritional. A primary reviewer abstracted relevant information from included studies for each of the intervention categories in KQ5.

RESULTS

KQ1: Screening in Asymptomatic Children and Pregnant Women

Similar to the 1996 findings, our searches did not identify direct evidence that screening children for elevated BLLs improved health outcomes. There was also no direct evidence that screening improves pregnancy or perinatal outcomes.

KQ2: Prevalence and Risk

The prevalence of elevated BLLs among children and women in the United States, like that in the general population, continues to decline sharply, primarily because of marked reductions in environmental exposure to lead (eg, gasoline, air, dietary sources, and residential paint). These reductions are largely the result of regulatory interventions at the federal, state, and local levels of government. The prevalence of elevated BLLs, however, varies substantially among different communities and populations, and children and pregnant women share many of the same risk factors for lead exposure. Correlates of higher BLLs at all ages include minority race/ethnicity, urban residence, low income, low educational attainment, older (pre-1950) housing, home renovation or remodeling, pica, use of ethnic remedies, cosmetics, lead-glazed pottery, occupational exposures, and recent immigration. Alcohol use and smoking are known risk factors among pregnant women (see Appendix 3 for a complete discussion).

Recent observational studies have demonstrated an inverse relationship between historical BLLs in children and subsequent measures of behavioral and cognitive performance at BLLs of <10 μg/dL. Observational studies of infants provide preliminary data that prenatal BLLs <10 μg/dL may be associated with neurodevelopmental delay or impairment. Study design and measurement issues, however, limit interpretation of these studies. Studies also suggest that levels of maternal exposure in this range may be associated with increased risk for spontaneous abortion, hypertension in pregnancy, and adverse effects on fetal growth[2] (Appendix 3).

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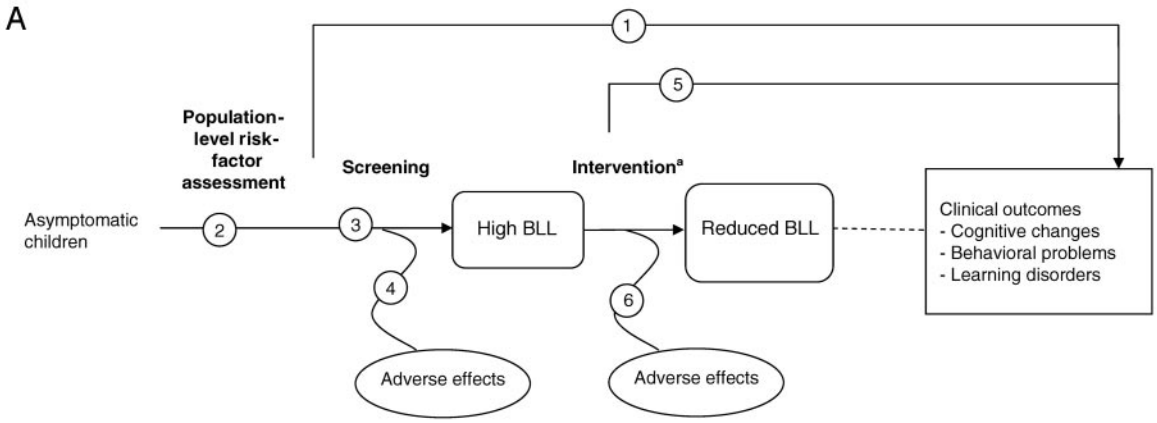


KQ1: Is there direct evidence that screening in asymptomatic children for lead results in improved health outcomes (ie, cognitive changes, behavioral problems, learning disorders)?

KQ2: What is the prevalence of elevated lead in children? Are there population-level risk factors that identify children at higher risk for elevated lead levels (ie, geography, racial/ethnicity, SES, age)?

KQ3: Can screening tests accurately detect elevated BLL?

A. What is the accuracy of using questionnaires (or other tools) for risk-factor assessment at various BLLs?

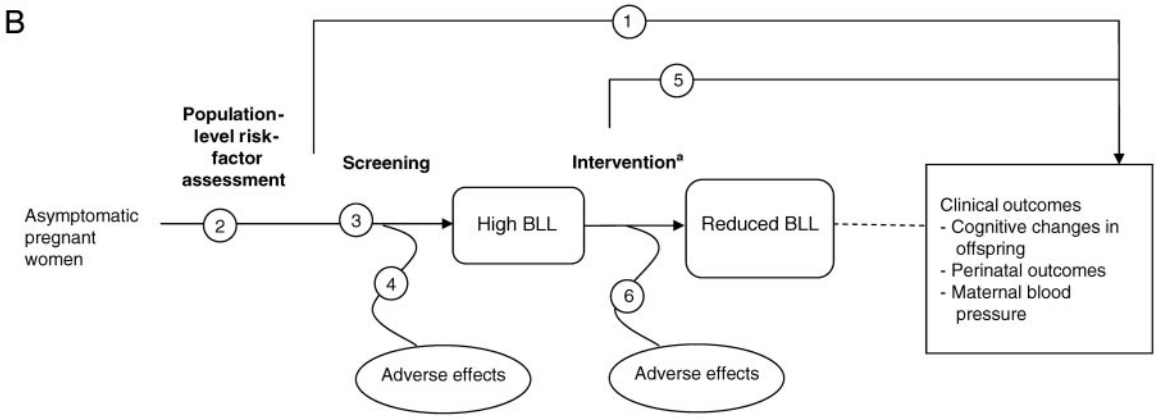
B. What is the optimal frequency for screening? What is the optimal frequency for repeat testing?

KQ4: What are the adverse effects of screening?

KQ5: Do interventions (ie, counseling families to reduce lead exposure, nutritional interventions, residential lead hazard-control techniques, chelation therapy) for elevated lead levels result in improved health outcomes?

KQ6: What are the adverse effects of the interventions?

KQ7: What are the cost-effectiveness issues?



KQ 1: Is there direct evidence that screening in asymptomatic pregnant women for lead results in improved health outcomes (ie, cognitive changes in offspring, perinatal outcomes including birth weight/preterm delivery etc, maternal blood pressure)?

KQ 2: What is the prevalence of elevated lead in asymptomatic pregnant women? Are there population-level risk factors that identify pregnant women at higher risk for elevated lead levels (ie, geography, racial/ethnicity, SES, age)?

KQ 3: Can screening tests accurately detect elevated BLLs? What is the accuracy of using questionnaires (or other tools) for risk-factor assessment at various BLLs?

KQ 4: What are the adverse effects of screening?

KQ 5: Do interventions (ie, counseling families to reduce lead exposure, nutritional interventions, residential lead hazard-control techniques, chelation therapy) for elevated lead levels result in improved health outcomes?

KQ 6: What are the adverse effects of the interventions?

KQ 7: What are cost-effectiveness issues?

FIGURE 1

Analytic frameworks and KQs. The analytic frameworks represent an outline of the evidence review and includes patient populations, interventions, outcomes, and adverse effects. The KQs examine a chain of evidence about the effectiveness, accuracy, and feasibility of screening asymptomatic children for elevated BLLs in primary care settings, prevalence rates and risk factors, adverse effects of screening, effectiveness of interventions for children identified with elevated BLLs, adverse effects of interventions, and cost-effectiveness issues. A, Children; B, pregnant women. [a] Interventions include counseling families to reduct lead exposure, nutritional interventions, residential hazard-control techniques, and chelation therapy.

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KQ3: Accuracy of Screening Tests

Can Screening Tests Accurately Detect Elevated BLLs?

We identified no new relevant information regarding the accuracy of screening for lead toxicity (refer to the 1996 USPSTF statement[1]). Blood lead testing has largely supplanted protoporphyrin levels as a screening tool because of poor performance of the latter at BLLs <25 μg/dL.[3]

What Is the Accuracy of Using Questionnaires (or Other Tools) for Risk-Factor Assessment at Various BLLs?

In communities where there is a low prevalence of elevated BLLs, screening will identify few cases and yield a significant proportion of false-positive test results. Older cross-sectional studies in urban and suburban populations showed that 1 or more positive responses to 5 questions (about exposures to deteriorated paint from older or renovated housing, to other lead-poisoned children, or to lead-related hobbies or industry) detected 64% to 87% of children with BLLs >10 μg/dL.[1] Higher sensitivities (81%–100%) for BLLs >15 to 20 μg/dL were reported,[1] but none of these studies evaluated the ability of questionnaires to detect levels >20 μg/dL, in part because so few patients had levels so high. Specificity among the studies ranged from 32% to 75%. False-negative results were predictably low (0.2%–3.5%) in low-prevalence (2%–7%) samples but increased to 19% when the population prevalence of elevated lead levels was higher (17%–28%). Questionnaires, therefore, may have greater utility in identifying children at low risk of elevated blood lead (negative predictive value) where the population prevalence is low and local risk factors are known. Negative predictive values of 96% to 100% have been reported in these settings.[1,4]

More recent studies of questionnaires in urban and rural settings, however, demonstrated a low prevalence of elevated BLLs and poor sensitivity and specificity.[5–8] Studies of questionnaires modified for local use provide some evidence of improved clinical utility for identifying children with elevated BLLs,[8–10] when compared with the panel of screening questions recommended by the Centers for Disease Control and Prevention (CDC) in 1991.[11]

Other studies have reported high false-positive rates for questionnaires[6,8] and that resource considerations[5] are important when formulating a screening program. A population-based follow-up study (n = 31 904) showed that raising the action level for screening to 15 μg/dL in this sample would have eliminated the unnecessary follow-up of 5162 children, 3360 of whom were falsely identified as having elevated lead levels.[12]

A recent study identified housing risk factors associated with elevated BLLs (>10 μg/dL) among 481 children residing in Rochester, New York. Housing characteristics including rental status, lead-contaminated floor dust, and poor housing conditions were all associated with elevated BLLs (sensitivity: 47%–92%; specificity: 28%–76%; positive predictive value: 25%–34%; negative predictive value: 85%–93%), suggesting that housing characteristics and floor dust lead levels can be used to identify homes in which a lead hazard may exist before or during occupancy.[13]

Prenatal Screening With Questionnaires

A maternal survey using 4 questions recommended by the CDC was evaluated in a study of 314 new prenatal patients. The prevalence of elevated maternal lead levels (≥10 μg/dL or 0.483 μmol/L) was 13%. Subjects with a positive response to at least 1 question were more likely to have elevated blood lead than those who answered negatively to all 4 questions (relative risk: 2.39; 95% confidence interval [CI]: 1.17 to 4.89; P = .01). The CDC questionnaire had a sensitivity of 75.7%. Among women who answered "no" to all 4 questions, the probability of having an elevated lead level was reduced from 13% to 6.9% (negative predictive value: 93.1%). The most predictive single item was "home built before 1960." The study also identified a high prevalence of elevated blood lead among children living with women with elevated BLLs.[14]

KQ5: Effectiveness of Early Detection

Detecting elevated BLLs before the development of clinical manifestations allows a clinician to recommend interventions to limit additional exposure and, when necessary, begin medical treatment with chelating agents. Early detection may also result in interventions that prevent lead exposure in other children (the child with an elevated BLL acting as a sentinel for a hazardous environment). There is relatively little convincing evidence, however, that these interventions effectively improve health outcomes. First, most available studies of asymptomatic children evaluated the effects of various interventions on BLLs rather than on clinical outcomes. Second, BLLs in childhood, after peaking at ~2 years of age, decrease without intervention,[1,15] a result attributable in part to regression to the mean, random variation, laboratory error, and redistribution of lead from blood to other tissue compartments. Studies must account for these changes over time, preferably by using controls who do not receive the intervention, to adequately evaluate the interventions' effects on BLLs or health outcomes.

Effect of Screening on Clinical Outcomes

The EPC staff did not identify evidence demonstrating that universal screening for blood lead results in better clinical outcomes. The 1996 USPSTF recommendation cited several older studies that reported intensive screening programs targeting children in high-risk neighborhoods reduced case fatality rates, mortality rates, and

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proportions of children detected with very high BLLs or who developed symptomatic lead poisoning.[1] Lacking concurrent controls, however, it was possible that the reported reductions in mortality and case fatality rates were caused by other factors such as advances in medical care rather than the effect of screening. The reduction in mean BLLs in the US population is primarily the result of diminishing exposure in the environment through regulatory interventions. The available evidence regarding the efficacy of screening programs, therefore, is weak.

Do Interventions for Elevated Lead Levels Result in Improved Health Outcomes?

Although chelating agents benefit children with symptomatic lead poisoning, no studies have demonstrated clinical benefits of chelation therapy in asymptomatic children. The Treatment of Lead-Exposed Children (TLC) trial, a large multicenter randomized, controlled trial (RCT) sponsored by the US National Institute for Environmental Health Science, enrolled children from 1994 to 1997 to assess the effect of oral chelation therapy with succimer on IQ in young children with venous BLLs of 20 to 45 μg/dL.[16] Follow-up testing at 36 months demonstrated a mean IQ 1 point lower, and poorer parental ratings of behavior, among those in the succimer group compared with those in the placebo group. Although succimer-treated children did slightly better on a test of learning ability, none of the differences between groups were statistically significant.[17] Reanalysis of the same data using the change in BLL as the independent variable demonstrated a 4.0-point improvement in cognitive scores for every 10 μg/dL reduction in BLL, but only in the placebo group, suggesting that factors other than declining blood lead contributed to cognitive improvement or that treatment had an adverse effect on cognitive performance.[18] Assessment of neurobehavioral outcomes at 7 years of age revealed no statistically significant differences on a battery of neurobehavioral tests except that those in the succimer group had worse attention-executive function scores.[19] Treatment also seemed to have an adverse effect on mean height.[20] The TLC group concluded that chelation therapy was not indicated for children with BLLs <45 μg/dL.[17,19]

Despite evidence of efficacy in lowering blood lead on a short-term basis, there is little evidence confirming a clinical benefit from chelation therapy for children with lead levels <45 μg/dL.

We found no studies that evaluated clinical outcomes after environmental or nutritional interventions.

Effects of Chelation Therapy on BLLs

In the previously cited US National Institute for Environmental Health Science–sponsored RCT of oral chelation in young children with venous BLLs of 20 to 45 μg/dL (TLC study), which reported no effects of chelation on IQ[16–19,21] (Tables 1 and 2), BLLs fell steeply in the treatment-group subjects in the first week (mean: 11 μg/dL lower) but rebounded afterward. BLLs also dropped in the placebo-group subjects but more slowly. BLLs were 77% of baseline in the succimer-treated subjects (88% of baseline among placebo) at 7 weeks after initiation of therapy. Mean BLLs among those in the treatment group were 4.5 and 2.7 μg/dL at 6 and 12 months, respectively, but the difference between those in the treatment and placebo groups at 24 months was not significant.[21]

Chelating agents have demonstrated short-term reductions in BLLs in children whose pretreatment values ranged from 20 to 70 μg/dL in studies in which chelation therapy was often combined with environmental interventions, but these reductions were not sustained over longer periods in the absence of repeated or continuing chelation therapy or environmental interventions.[1,22–24]

These data provide good evidence that chelating agents may result in short-term reductions in BLLs in children but suggest that these reductions may not be sustained over longer periods in the absence of repeated or continuing chelation therapy or environmental interventions. In addition, there is no evidence that these reductions result in improved neurobehavioral or health outcomes.

Effect of Residential Lead Hazard Control on BLLs

Recent studies of household dust and paint hazard control through cleaning, abatement, and education have shown mixed results. Of the 8 controlled studies published since 1995, 1 has shown a modest, but significant, decline; 5 have shown nonsignificant declines; and 2 have shown nonsignificant elevations in BLLs among children. Reduced BLLs were seen among children with higher baseline BLLs ($\geq$15 or $\geq$20 μg/dL) in 2 studies (1 meta-analysis and 1 retrospective chart review with no comparison group) but not in children with lower baseline levels. Recent studies have differed from older studies in that newer paint hazard-control techniques result in lower lead-dust levels. Population venous lead levels have decreased over time, and lead-poisoned children in older studies had higher mean BLLs than those in recent studies (see Tables 3 and 4 and Appendix 4 for a detailed assessment).

Effect of Counseling and Education Interventions on BLLs

Overall, the evidence to determine if education and counseling improve outcomes among children with moderately elevated BLLs is weak and conflicting (see Appendix 5 for a detailed assessment).

Effect of Soil Abatement on BLLs

Recent studies of soil remediation in residential areas have shown only modest or nonsignificant effects.[25–27] Soil remediation in communities near lead-mining,

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TABLE 1 **Chelation Interventions**

Author (Year)	Type of Intervention	*N*	Population/Risk Factors	BLL, μg/dL	Duration of Follow-up	Results	Adverse Effects
Besunder et al[22] (1995)	Chelation with DMSA and abatement of domestic lead hazards	46 treated, 18 excluded: $n = 28$	Referral population: 35% black, 10% Hispanic	25–49	80 d	BLL: posttreatment (day 18), −43% (±20.8%) and day 80 −31% (±20.2%); ZPP posttreatment (day 18), −12% (±21.7%) and day 80, −32% (±21.9%)	Neutropenia ($n = 1$)
Chisolm[24] (2000)	Chelation with DMSA, relocation to lead-safe housing	59	Children aged 12–65 mo	25–70	21 d	Mean BLL decreased to below 35% of pretreatment value after 4 wk of DMSA treatment; rebounded to 58% of pretreatment level 2–3 wk after termination of treatment	Elevated alkaline phosphate levels ($n = 2$); eosinophilia ($n = 1$)
Dietrich et al[19] (2004) (TLC trial)	Chelation with DMSA after domestic cleaning with HEPA vacuum and damp-cloth wiping	1854 evaluated, 780 randomized	Children aged 12–33 mo; 77% black; most had poor, single mothers and lived in older, poorly maintained residences	20–44	6 y (until 7 y of age)	No statistically significant difference in neurobehavioral outcomes except DMSA-treated children did worse on attention/executive functions	No statistically significant difference compared to placebo; excess noted: trauma, scalp rashes, neutropenia/thrombocytopenia, elevated ALT
Liu et al[18] (2002) (TLC trial)	Chelation with DMSA after domestic cleaning with HEPA vacuum and damp-cloth wiping	1854 evaluated, 780 randomized; results from 741 reanalyzed for this study	Children aged 12–33 mo; 77% black; most had poor, single mothers and lived in older, poorly maintained residences	20–44	36 mo	6 mo after treatment, BLL had fallen a similar amount in both DMSA and placebo groups; there was no association between change in BLL and change in cognitive test score; BLL continued to fall, but 36 mo after treatment, cognitive test scores improved 4.0 points for every 10 μg/dL drop in BLL in the placebo group only	No statistically significant difference compared to placebo; excess noted: trauma, scalp rashes, neutropenia/thrombocytopenia, elevated ALT
O'Connor and Rich[23] (1999)	Chelation with DMSA, domestic cleaning and repair	39	Low-income black inner-city children, 2.5–5 y old	30–45	6 mo	DMSA: baseline, 34.9 ± 4.7 μg/dL,1 mo, 27.4 ± 7.5 μg/dL, 6 mo, 28.8 ± 6.4 μg/dL; placebo: baseline, 33.0 ± 6.2 μg/dL,1 mo, 33.2 ± 10.3 μg/dL, 6 mo, 25.1 ± 6.8 μg/dL ($P = .06$); differences in BLL between groups were not statistically significant ($P = .16$ at 1 month; $P = .06$ at 6 mo)	ND
Peterson[20] (2004) (TLC trial)	Chelation with DMSA after domestic cleaning with HEPA vacuum and damp-cloth wiping	1854 evaluated, 780 randomized	Children aged 12–33 mo; 77% black; most had poor, single mothers and lived in older, poorly maintained residences	20–44	34 mo	Difference in mean change in height, DMSA vs placebo: 0–9 mo, −0.27 cm (CIs: −0.42 to −0.11) and 0–34 mo, −0.43 cm (CIs: −0.77 to −0.01)	Small, marginally significant decrease in height among treatment group compared to placebo; excess noted: trauma, scalp rashes, neutropenia/thrombocytopenia, elevated ALT
Rogan[16] (1998) (TLC trial)	Chelation with DMSA after domestic cleaning with HEPA vacuum and damp-cloth wiping	1854 evaluated, 780 randomized	Children aged 12–33 mo; 77% black; most had poor, single mothers and lived in older, poorly maintained residences	20–44	36 mo	Description of baseline measurements, group characteristics, study methodology	ND
Rogan[21] (2000) (TLC trial)	Chelation with DMSA after domestic cleaning with HEPA vacuum and damp-cloth wiping	1854 evaluated, 780 randomized	Children aged 12–33 mo; 77% black; most had poor, single mothers and lived in older, poorly maintained residences	20–44	12 mo	DMSA group: BLL 11 μg/dL lower at 1 wk; rebound began at 1 wk, and at 7 wk DMSA group mean BLL was 72% of baseline (placebo group mean BLL: 88% of baseline); during the 6 mo after initiation of treatment, the DMSA group had a mean BLL 4.5 μg/dL lower than the control group; at 12 mo, mean DMSA group BLL was 2.7 μg/dL lower than the control group, but CIs overlap; at 12 mo, groups were similar	No statistically significant difference compared to placebo; excess noted: trauma, scalp rashes, neutropenia/thrombocytopenia, elevated ALT
Rogan et al[17] (2001) (TLC trial)	Chelation with DMSA after domestic cleaning with HEPA vacuum and damp-cloth wiping	1854 evaluated, 780 randomized	Children aged 12–33 mo; 77% black; most had poor, single mothers and lived in older, poorly maintained residences	20–44	36 mo	First 6 mo: DMSA mean BLL 4.5 μg/dL lower than placebo; at 36 mo, DMSA group scored on average 1 IQ point lower than the control group and had slightly worse behavior by parental rating compared to the placebo group; the placebo group fared slightly better on a developmental neuropsychological battery of tests; Overall, there was no statistically significant difference	No statistically significant difference compared to placebo; excess noted: trauma, scalp rashes, neutropenia/thrombocytopenia, elevated ALT

ALT indicates alanine transferase; HEPA, high-efficiency particulate air; ND, not described; ZPP, zinc protoporphyrin.

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TABLE 2 **Summary of Chelation Interventions**

Author (Year) (Quality)	Study Design	Type of Intervention	Years Conducted	*N*	Child Age	Duration of Follow-up	Baseline BLL, μg/dL	BLL Results	Summary of Effect
Rogan[16] (1998), Rogan[21] (2000), Rogan et al[17] (2001), Liu et al[18] (2002), Dietrich et al[19] (2004), and Peterson et al[20] (2004); (TLC trial) (Good)	Randomized multicenter, placebo-controlled, double-blind trial	DMSA lasting 26 d, dose based on body surface area; treatment repeated up to 3 times for persistently elevated BLL; domestic cleaning with HEPA vacuum and damp-cloth wiping	NR	1854 evaluated; 780 randomized; 741 of randomized reanalyzed for Liu 2002 (cognitive function)	12–33 mo	At 12 mo after initiation of treatment, test BLL; at 36 mo, planned behavioral, cognitive, and biochemical tests; retest at 72 mo (to age 7); also, at 9 and 34 mo, test height-weight	20–44	DMSA vs placebo: 1 wk, BLL 11 μg/dL lower in DMSA group; 7 wk, 72% vs 88% of baseline; first 6 mo: BLL 4.5 μg/dL lower in DMSA group; 12 mo: groups are similar	DMSA produced short-term reduction in BLL; rebound began at 1 and 7 wk; follow-up outcomes do not support the hypothesis that lead-induced cognitive defects are reversible by chelation therapy; those in the DMSA group scored worse on some measures
Besunder et al[22] (1995) (NA)	Retrospective case series	DMSA 10 mg/kg every 8 h for 5 d, followed by 10 mg/kg every 12 hours for 14 d; abatement of domestic lead hazards	June 1991 to May 1993	46 treated, 18 excluded ($n = 28$)	NR	80 d	25–49	BLL posttreatment: 18 d, −43% (±20.8%), and 80 d, −31% (±20.2%); ZPP posttreatment: 18 d, −12% (±21.7%) and 80 d: −32% (±21.9%)	No control group; cannot exclude other intervention effects (abatement of domestic lead hazards)
Chisolm[24] (2000) (NA)	Open-label case series	DMSA 1050 mg/m^2 per d, divided in 3 doses for 5 d, followed by 700 mg/m^2 per d, divided in 2 doses for 21–23 d; relocation to lead-safe housing	NR	59	12–65 mo	21 d	25–70	BLL posttreatment: 1 d, <35% of pretreatment level; 2–3 wk, rebounded to 58% of pretreatment level	No control group; cannot exclude other intervention effects (abatement of domestic lead hazards)
O'Connor and Rich[23] (1999) (Fair)	Randomized, placebo-controlled, double-blind trial	DMSA (child weight <15 kg) 1000 mg tid for 5 d, followed by 100 mg bid for 14 d; DMSA (child weight >15 kg) 200 mg tid for 5 d, followed by 200 mg bid for 14 d	NR	39	2.5–5 y	6 mo	30–45; DMSA group: 34.9 ± 4.7; placebo group, 33.0 ± 6.2	DMSA vs placebo: 1 mo, 27.4 ± 7.5 vs 33.2 ± 10.3 μg/dL; 6 mo, 28.8 ± 6.4 vs 25.1 ± 6.8 μg/dL	Both treatment and control groups demonstrated significant BLL reductions; differences between groups were not significant

bid indicates 2 times per day; HEPA, high-efficiency particulate air; NR, not reported; tid, 3 times per day; ZPP, zinc protoporphyrin.

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TABLE 3 **Environmental Interventions**

Author (Year)	Type of Intervention	*N*	Population/Risk Factors	BLLs	Duration of Follow-up	Results	Adverse Effects
Aschengrau et al[26] (1994); Aschengrau et al[27] (1997)	Soil and interior dust abatement and loose-paint stabilization	152	Children <4 y	7–24 μg/dL	2 y	BLL and change (μg/dL) (95% CI): phase I, mixed interventions: pre 13.10, post 10.65, change −2.44 (−3.32 to −1.57); phase II, soil abatement for homes not already deleaded: group A, pre 12.94, post 7.69, change −5.25 (−6.51 to −3.99); group B: pre 10.54, post 9.15, change −1.30 (−4.03 to +1.26); all groups, all phases combined: pre 12.66, post 9.77, change −2.89 (−3.64 to −2.13); soil lead reduction of 2060 ppm is associated with a 2.25–2.70 μg/dL decline in BLLs; low levels of soil recontamination 1–2 y after abatement indicate the intervention is persistent; paint hazard remediation alone was associated with a BLL increase of 6.5 μg/dL ($P = .05$); paint hazard remediation combined with soil abatement suggested an insignificant increase of 0.9 μg/dL ($P = .36$)	ND
Aschengrau et al[76] (1998)	Dust, domestic cleaning with HEPA vacuum, wash window surfaces, seal flaking paint, and repair holes in wall	63	Children ≤4 y	16.9 μg/dL	6 mo	BLL and change (μg/dL): automatic intervention group (high risk): pre 17.5, post 9.1, change −8.4; randomized intervention group: pre 17.6, post 11.5, change −6.2; randomized control group: pre 16.3, post 10.4, change −5.9; relative change, treatment vs control: −0.3 (95% CI −3.8 to +3.3); automatic intervention vs control: −2.5 (CI −7.0 to +2.1)	ND
Campbell et al[79] (2003)	Dust, second cleaning follows 18–21 mo after TLC study cleaning	73 treatment, 86 control	Toddlers aged 12–34 mo	20–44 μg/dL	6 mo	BLL declined in both treatment and control groups; GM BLL, adjusted for month and child, declined monotonically among 73 children whose homes were cleaned a second time; BLLs of the 86 children whose homes did not receive a second cleaning also declined over time, although there was an unexplained increase at the 3-mo postcleaning follow-up visit; BLLs before the cleaning were higher among children in high-exposure homes (GM BLL: 18.1 μg/dL), compared with those in low-exposure homes (GM BLL: 14.5 μg/dL); stratified by randomized treatment, there were only small differences in BLL for children in chelation vs placebo (18.3 and 17.1 μg/dL) in high-exposure homes and for chelation vs placebo (14.5 vs 13.5 μg/dL) in low-exposure homes	ND
Clark et al[73] (2004)	Lead-based paint and dust hazard-control program and survey	869 children	HUD hazard-control program participants in 14 states	ND	6 wk	Postintervention, 81 (9%) participants had BLL increases ≥5 μg/dL (range: 5–25 μg/dL; average: 8.4 μg/dL); logistic regression analysis indicated 4 factors were significantly associated with increases: (1) child's age at preintervention ($P = .006$); (2) female caregiver's education ($P = .002$); (3) general exterior building condition ($P = .0071$); and (4) second season of blood-sample collection ($P < .001$); odds ratio of BLL increase decreased sharply as child's age increased; when the female parent had not completed high school, likelihood of BLL increase was 2.5 times higher than families in which the female parent had completed high school	ND

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TABLE 3 **Continued**

Author (Year)	Type of Intervention	*N*	Population/Risk Factors	BLLs	Duration of Follow-up	Results	Adverse Effects
Farrell et al[25] (1998)	Soil	Enrolled 408 children in 263 houses; 187 completed study	Children aged 6 mo to 6 y	Baseline: 11 μg/dL; 54% of properties had soil samples >1000 ppm	1 y	1 y postabatement: BLL in both groups fell below baseline; differences between treatment and control groups were not significant in any of the cross-sectional or longitudinal models; 2 y postabatement: soil sampling showed significant lead reaccumulation	ND
Galke et al[82] (2001)	Dust	240 children in 1212 dwellings	Children aged 6 mo to 6 y	Median: 10 μg/dL (range: 2–48)	12 mo	12 mo postintervention: BLL declined from 11.0 to 8.2 μg/dL (−2.8 μg/dL), a 26% reduction	ND
Haynes et al[78] (2002)	Dust, meta-analysis	4 studies, total subjects = 533	NR	6.7–16.9 μg/dL	6–48 mo	Weighted mean change in BLL: −0.62 μg/dL (95% CI −1.55 to 0.32); no significant difference between intervention and control groups, combined from educational dust-control and professional-dust-control trials	ND
Jordan et al[85] (2003)	Education	594 mothers and 378 of their children	Inner-city, poor, ethnically diverse (78% nonwhite)	Before intervention, all levels were <10 μg/dL	2 y	Intervention vs control: maintained BLL <10 μg/dL: 81% vs 73% ($P = .08$); >90% completed 19–20 sessions; half completed first year of follow-up sessions; <5% completed second year	ND
Lanphear et al[86] (1999); Lanphear et al[56] (2000); Lanphear et al[94] (2002)	Education	275	Children aged 6 mo	2.9 μg/dL (95% CI: 2.7 to 3.1) at age 6 mo	48 mo	No significant difference in BLL by intervention status at 24 or 48 mo; intervention vs control BLL: age 24 mo, 7.3 vs 7.8 μg/dL, age 48 mo, 5.9 vs 6.1 μg/dL; dust lead levels declined sharply in both the treatment and control groups; there was no significant difference in dust lead levels at 24 mo by group nor a difference in change in dust lead levels from 6 to 24 mo by group; other results (Lanphear et al[94]): dietary iron intake, but not calcium intake, was inversely associated with BLL ($P < .05$); also, BLL was >50% higher in black than in white children ($P = .0001$)	ND
Lanphear et al[88] (2003)	Soil	198 in first survey; 215 in second survey	Children	Mean: 5.6 μg/dL with soil >500 ppm (11% ≥10 μg/dL); mean: 3.0 μg/dL with soil <500 ppm (3% ≥10 μg/dL)	NA	BLL change (μg/dL) before and after soil abatement: intervention group: pre 5.6, post 3.0, change −3.6 ($P = .0001$); nonintervention group: pre 3.0, post 2.6, change −1.4 ($P = .06$); stratified by age and adjusted for mouthing behavior score and SES: age 36–72 mo: change −2.3 μg/dL (NS), age 6–36 mo: change −2.5 μg/dL ($P = .03$)	ND
Leighton et al[77] (2003)	Lead-paint hazard remediation	221	Lead-poisoned children	20–44 μg/dL	10–14 mo	BLL declined significantly for all groups: 24.3 μg/dL at baseline to 12.3 μg/dL at 10- to 14-mo follow-up, a 50% decline ($P < .01$); intervention ($n = 146$) vs nonintervention ($n = 75$): BLL reduction 53% vs 41%, relative reduction ±20% ($P < .01$); after adjusting for confounders, remediation effect was 11% (NS); race was the only factor that confounded the relationship; black children had higher BLLs in follow-up after remediation; mean BLL for white and Asian children was 30% lower than that for black children ($P < .01$); effect of remediation seemed to be stronger in younger children (10–36 mo) than in older children (36–72 mo) ($P = .06$); timing of remediation produced no significant effect on BLL	ND

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TABLE 3 **Continued**

Author (Year)	Type of Intervention	*N*	Population/Risk Factors	BLLs	Duration of Follow-up	Results	Adverse Effects
Rhoads et al[74] (1999)	Dust	113 enrolled; final blood levels obtained from 99	Children, mean age: 1.7 y	Intervention mean: 12.4 μg/dL (SD: 5.7 μg/dL); control mean: 11.6 μg/dL (SD: 6.2 μg/dL)	1 y	Significant effect on BLL (μg/dL) change: intervention: pre 12.4, post 10.3, change −2.1 (17%); control: pre 11.6, post 11.6, change +0.1 (+1%); estimated intervention effect = −1.9 μg/dL ($P < .05$); mother's final knowledge score was not a highly significant predictor of BLL change; the contribution of the educational intervention could not be clearly distinguished from the effects of cleaning	ND
Schultz et al[87] (1999)	Education	187	Black, white, Native American, Asian, other	20–24 μg/dL	6 mo	Intervention vs reference group BLL decline (μg/dL) significant: 4.2 (±21%) vs 1.2 (±6%); net reduction 3.1 μg/dL ($P < .001$)	ND
Strauss et al[83] (2005)	Paint	1179	Children aged ≤36 mo	Preintervention means: untreated, 4.5 μg/dL; treated, 7.0 μg/dL	From 1 y preintervention to 3 y postintervention	Comparison of case vs control change in BLL (μg/dL) showed significant differences, adjusted for time, seasonality, age, and gender: controls matched on housing criteria only: HUD-treated, 7.04 (42.7%) vs 3.54 (13.2%), untreated control: 4.57 (19.7%) vs 3.45 (10.0%) ($P < .001$); controls matched on combination of pre-BLL and housing information: HUD-treated, 7.07 (42.8%) vs 3.57 (12.5%), untreated control, 5.76 (29.1%) vs 3.96 (15.9%) ($P = .116$); controls matched on pre-intervention BLL information: HUD-treated, 7.07 (42.9%) vs 3.59 (12.6%), untreated control, 6.62 (36.9%) vs 4.28 (16.0%) ($P = .015$)	ND
Swindell et al[80] (1994)	Paint; dust	132	Children with high BLLs; mean age: 35 mo (range: 12–91 mo); 52% boys	Preabatement level = 26.0 μg/dL (± 6.5 μg/dL)	2 wk to 6 mo after abatement	BLL declined significantly: 26.0 to 21.2 μg/dL ($P < .001$); BLL reduction varied by baseline BLL: 97% with BLL ≥30 μg/dL had reductions within 1 y, 81% with BLL 20–29 μg/dL had reductions, and 35% with BLL <20 μg/dL had reductions; in this group, BLL increased after abatement, 16.7 to 19.2 μg/dL ($P = .053$); there was no meaningful change in preabatement to postabatement levels by calendar year of abatement	ND
Taha et al[75] (1999)	Paint; dust	42 eligible, data analyzed for 37	Children aged 1–3 y	28.8 μg/dL	±69 d after abatement	Posttreatment mean BLL 24.6 μg/dL represented a 6.2 μg/dL reduction (22%); adjusted for season and age of child, the BLL reduction was 6.0 μg/dL (18%); adjusted BLL (μg/dL) initial/follow-up/change/percentage change: intervention ($n = 37$), pre 28.8, post 22.8, change −6.0 (−18%) ($P = .05$); control ($n = 65$), pre 31.1, post 29.5, change −1.6 (−1.8%) (NS)	ND

NS indicates not significant.

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TABLE 4 **Summary of Environmental Interventions**

Author (Year) (Quality)	Study Design	Type of Intervention	Years Conducted	*N*	Age	Duration of Follow-up	Baseline BLL, μg/dL	BLL Results, μg/dL (Initial/Final/Change)	Summary of Effect
Aschengrau et al[26] (1994); Aschengrau et al[27] (1997) (NA)	Randomized environmental intervention; no untreated comparison group	Soil	1989–1990	152	≤4 y	2 y	7–24	TG1: 13.10/10.65/−2.44 (95% CI −3.32 to −1.57); TG2: 12.94/7.69/−5.25 (95% CI −6.51 to −3.99); TG3: 10.54/9.15/−1.30 (95% CI −4.03 to +1.26); all TGs: 12.66/9.77/−2.89 (95% CI −3.64 to −2.13); CG: none	NA
Aschengrau et al[76] (1998) (Fair)	RCT	Dust, paint	1993–1995	63	≤4 y	6 mo	16.9	TG1 (high BLL, not randomized): 17.5/9.1/−8.4; TG2 (random): 17.6/11.5/−6.2; CG (random): 16.3/10.4/−5.9; TG1 vs CG: −0.3 (95% CI −3.8 to +3.3); TG2 vs CG: −2.5 (95% CI −7.0 to +2.1)	No effect
Campbell et al[79] (2003) (Fair)	Non-RCT; follow-up at the Philadelphia site of TLC trial, a chelation RCT	Dust	NR	73	12–34 mo	3–6 mo posttreatment	20–44	No significant difference in mean BLL at any clinic visit between children whose homes were cleaned vs those whose homes were not cleaned; BLL declined among both groups	No effect
Clark et al[73] (2004) (NA)	Observational, no untreated comparison group	Dust, paint	NR	869 children	6 mo to 6 y	6 wk	ND	Mean change after intervention: +8.4 μg/dL; predictors of BLL increase of >5 μg/dL: child's age at baseline ($P = .006$), mother's education ($P = .002$), exterior building condition ($P = .007$), and season of sample collection ($P < .001$)	NA
Farrell et al[25] (1998) (Fair)	RCT	Soil	1990	408 enrolled in 263 houses; 187 completed the study	6 mo to 6 y	1 y	11	TG: 12.1 (1988)/9.7 (1991); CG: 10.9 (1988)/8.4 (1991): treatment effect, adjusted for effects of time, seasonality, SES, age, and mouthing behavior: TG (pre-post): 0.030 (SE: 0.034); CG (pre-post): 0.075 (SE: 0.036); TG vs CG: −0.045 (SE: 0.037)	No effect
Galke et al[82] (2001) (NA)	Descriptive study, no comparison group	Dust	1994–1997	240 children in 1212 dwellings	6 mo to 6 y	12 mo	10 (median)	TG: 11.0/8.2/−2.8; CG: none	NA
Haynes et al[78] (2002) (Good)	Meta-analysis	Dust, paint; meta-analysis of RCTs	NR	4 studies, total subjects = 533	NR	6 to 48 mo	6.7–16.9	Weighted mean change, TG vs CG (95% CI): 2 educational dust-control trials: −0.33 (−1.4 to 0.74); 2 professional dust-control trials: −1.52 (−3.41 to 0.37); all trials: % ≥10 μg/dL in TG vs CG, similar; % ≥15 μg/dL in TG vs CG, 6% vs 14% ($P = .008$); % ≥20 μg/dL in TG vs CG, 2% vs 6% ($P = .024$)	No effect overall; effects seen at higher lead levels
Jordan et al[85] (2003) (Fair)	RCT	Education	NR	594 mothers and 378 of their children	Birth to 36 mo	2 y	<10	TG vs CG: % who maintained BLL <10 μg/dL: 81% vs 73% (P = NS); % with BLL 10–19.99 μg/dL: 15% vs 24% (P = NS); % with BLL >20 μg/dL: 4% vs 2% (P = NS)	No effect
Lanphear et al[86] (1999); Lanphear et al[56] (2000); Lanphear et al[94] (2002) (Fair)	RCT	Education	NR	275	6 mo	48 mo	2.9	Change from age 6 to 24 mo: TG: 2.8/7.3/+5.6 (sic); CG: 2.9/7.8/+6.3 (sic); TG vs CG: (P = NS)	No effect

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TABLE 4 **Continued**

Author (Year) (Quality)	Study Design	Type of Intervention	Years Conducted	*N*	Age	Duration of Follow-up	Baseline BLL, μg/dL	BLL Results, μg/dL (Initial/Final/Change)	Summary of Effect
Lanphear et al[88] (2003) (NA)	Two cross-sectional surveys before and after soil abatement	Soil	1993–1996	198 in first survey, 215 in second survey	6–72 mo	NA (cross-sectional)	5.6	TG: 5.6/3.0/−3.6 ($P = .0001$); CG: 3.0/2.6/−1.4 ($P = .06$); stratifying by age and adjusted for mouthing behavior score and SES: age 36–72 mo, 2.3 μg/dL decline (P = NS), age 6–36 mo, 2.5 μg/dL decline ($P = .03$)	Effect seen only in young children who had not been exposed
Leighton et al[77] (2003) (Good)	Retrospective cohort study	Dust, paint	1994–1997	221	6 mo to 6 y	10–14 mo	20–44	Decline occurred regardless of remediation: 24.3/12.3/−12 ($P < .01$); TG: 24.6/11.6/−53% CG: 23.8/13.9/−41%; remediation effect adjusted for race: 11% (P = NS); effect of remediation tended to be stronger in younger children (10 to <36 mo) vs 36–72 mo ($P = .06$)	No effect
Rhoads et al[74] (1999) (Fair)	RCT	Dust	NR	113; final BLL obtained from 99	6–36 mo	1 y	12	TG: 12.4/10.3/−2.1; CG: 11.6/11.6/+0.1; TG vs CG: −1.9 μg/dL ($P < .05$); adjustment for baseline BLL	+
Schultz et al[87] (1999) (Fair)	Retrospective cohort study	Education	1994	187	3.35 y (mean)	6 mo	20–24	Change in BLL, TG vs CG: −4.2 vs −1.2 ($P < .001$)	+
Strauss et al[83] (2005) (Fair)	Retrospective cohort study	Dust, paint	1993–2002	1179	≤36 mo	3 y	4.5–7.0	TG vs CG BLL reduction, adjusted for time, seasonality, age, and gender; matched on preintervention BLL: TG, 7.07/3.59/−3.48, CG, 6.62/4.28/−2.34 ($P = .015$); matched on preintervention BLL and housing criteria: TG, 7.07/3.57/−3.50, CG, 5.76/3.96/−1.80 ($P = .116$)	+
Swindell et al[80] (1994) (NA)	Retrospective chart review; no comparison group	Dust, paint	1987–1990	132	35 mo (mean; range: 12–91 mo)	2 wk to 6 mo after abatement	26	TG: 26.0/21.2/−4.8 ($P < .001$); TG with baseline <20 μg/dL: 16.7/19.2/+2.5 ($P = .053$); CG: none; stratified by baseline BLL, reductions within 1 y occurred in 97% with baseline ≥30 μg/dL, 81% with baseline 20–29 μg/dL, and 35% with baseline <20 μg/dL	+ only if baseline BLL is ≥20
Taha et al[75] (1999) (Fair)	Retrospective cohort study	Dust, paint	1994	42 eligible data analyzed for 37	6 mo and 6 y	Mean 69 d after abatement	28.8	Adjusted for season and age of child: TG, 28.8/22.8/−6.0 ($P = .05$); CG, 31.1/29.5/−1.6 (P = NS)	+

+ indicates benefit; CG, control group; TG, treatment group; NR, not reported; NS, not significant.

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-milling, or -smelting operations may have a beneficial effect but was not considered within the scope of review (see Appendix 6 for a detailed assessment).

Effect of Nutritional Interventions on BLLs

There is conflicting evidence whether nutritional interventions are an efficacious way to lower children's BLLs. Depending on the nutritional intervention under investigation, findings are limited, preliminary, and somewhat contradictory (Tables 5 and 6; see Appendix 7 for a detailed assessment).

KQ4 and KQ6: Adverse Effects of Screening and Intervention

We identified no substantial new relevant information regarding the adverse effects of screening and interventions for lead toxicity. The most common adverse effects of screening for elevated lead levels remain those identified in the 1996 USPSTF Statement[1] (ie, false-positive results and the associated anxiety, inconvenience, work or school absenteeism, and financial costs of return visits and repeat tests). Adverse effects of environmental interventions may include transient elevation in BLLs, inconvenience associated with abatement work or relocation, and cost/benefit considerations.

Reported adverse effects of treatment with succimer (meso-2,3-dimercaptosuccinic acid [DMSA]) include mild gastrointestinal (vomiting and diarrhea) and systemic symptoms, rashes, transient hyperphosphatasemia, neutropenia, eosinophilia, and elevations in serum transaminases. These effects occurred in up to 10% of cases.[1,16–19,21]

EVIDENCE SYNTHESIS AND CONCLUSIONS

There is no direct evidence that screening for elevated BLLs in asymptomatic children at increased risk for lead exposure will improve clinical outcomes (Table 7). Because there have been no controlled trials that directly evaluated screening for elevated lead levels, this conclusion is based on a chain of evidence constructed from studies of weaker design. First, in young asymptomatic children, BLLs as low as 10 μg/dL, and perhaps lower, are associated with measurable neurodevelopmental dysfunction. Therefore, a relevant threshold level for screening and subsequent intervention cannot be specified on the basis of clinical evidence. Second, the national prevalence of elevated lead levels has declined dramatically in the past 2 decades, although high prevalence persists in some communities, particularly poor urban communities in the Northeast and Midwest. Third, although current interventions (eg, residential lead hazard control and chelation therapy) can reduce BLLs in children identified with levels >25 μg/dL, the quality of evidence supporting their effectiveness is weak, and a beneficial effect on IQ or other clinical outcomes has not yet been demonstrated. In addition, well-designed RCTs do not support beneficial effects and suggest adverse effects of chelation therapy for asymptomatic children with levels <45 μg/dL.

For those children who are screened and found to have initial BLLs <25 μg/dL, there is no evidence regarding the effectiveness of early detection and intervention or of repeated screening to detect additional increases in BLLs. Longitudinal and cross-sectional studies suggest that in children older than 2 years, such levels will decline naturally with time, but elevated levels may persist in children who are chronically exposed.

There is no direct evidence comparing the outcomes of universal screening with the outcomes from targeted screening for elevated lead levels. Recent studies indicate that the prevalence of elevated BLLs in the United States has declined dramatically in the past 2 decades, but local prevalence is highly variable, with >10-fold differences between communities. In a community with a low prevalence of elevated BLLs, universal screening may result in disproportionate risks and costs relative to benefits. The prevalence level at which targeted screening can replace universal screening is a public health policy decision that requires consideration beyond the scientific evidence for effectiveness of early detection, such as available resources, competing public health needs, and costs and availability of alternative approaches to reducing lead exposure. Clinicians can consult their local or state health departments regarding appropriate screening policy for their populations (see Appendix 8 for recommendations from other groups).

In communities from which data suggest that universal screening is not indicated, there may be some children who are at increased risk of BLLs in the range for which individual intervention by chelation therapy or residential lead hazard control has been demonstrated to be effective. In addition to risks from housing, these children may have had exposure to other lead sources such as lead-based hobbies or industries, traditional ethnic remedies, or lead-based pottery. Selective blood lead screening of such high-risk children is appropriate even in low-prevalence communities.

Questionnaires that have been locally validated and are of known and acceptable sensitivity and specificity can assist in identifying those at high risk. In several studies, the CDC[11] and similar questionnaires correctly identified 64% to 87% of urban and suburban children who had BLLs >10 μg/dL. Because of frequent false-positive results in low-prevalence communities, questionnaires may have greater utility in identifying children at low risk of elevated BLLs (negative predictive value) where the population prevalence is low and local risk factors are known. Locale-specific questionnaires that inquire about likely local sources of lead exposure may lead to improved prediction.

There are no controlled trials that have evaluated screening for elevated BLLs in pregnant women, and there are insufficient data to construct an adequate

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TABLE 5 **Nutrition Interventions**

Author (Year)	Nutritional Category	Population	*N*	Initial BLLs	Significant Results	Adverse Effects
Dalton et al[89] (1997)	Calcium, iron, phosphorus	Infants aged 3.6–6 mo in Lawrence, MA; high proportion of low-income families; data collected 1991–1993; majority Latino (>90%)	103	0.12–0.07 μmol/dL	There were no significant differences by treatment group in mean or median change from baseline of serum ferritin, total iron-binding capacity, erythrocyte protoporphyrin, or hematocrit at 4 and 9 mo after enrollment; incidence of iron deficiency was similar for both groups, and no infant developed iron-deficiency anemia during the trial	ND
Gallicchio et al[96] (2002)	Calories, carbohydrates, fat, vitamin C	Children, aged ~1 y, from low-income families, living in urban houses built before 1950; 85% black	205	Mean: 4.0 μg/dL (range: 1–19 μg/dL); 4.9% >10 μg/dL	Statistically significant positive associations ($P < .05$) were found between BLL and calories, total fat, saturated fat, and monounsaturated fat; statistically significant negative associations ($P < .05$) were found between BLL and carbohydrates and vitamin C; after multiple linear regression analyses, statistically significant positive associations were found between BLL and total fat ($P = .03$) as well as BLL and saturated fat ($P = .02$), independent of lead exposure and age of the child; total caloric intake was found to be a marginally significant effect modifier of the association between lead exposure and BLL ($P = .06$)	ND
Hammad et al[98] (1996)	Iron	Children from 9 mo to 5 y old cared for at University of Maryland (Baltimore, MD) Pediatric Ambulatory Center; low-income, inner-city families	299	NA	Average BLL was 11.4 μg/dL; after adjusting for confounders using multiple linear regression models, a negative association between BLL and dietary iron intake was found ($P = .03$); no association was found between BLL and serum iron	ND
Haynes et al[95] (2003)	Calcium, iron	Children living in Rochester, NY, and were 5–7 mo old at baseline visit; low-income families (same participants as in Lanphear et al[94])	275 (245 at 24 mo, 239 with adequate blood samples)	NA	Calcium intake was inversely associated with children's BLL ($P = .03$) in a multivariate model that included VDR Fok1 genotype as an independent variable	ND
Lanphear et al[94] (2002)	Iron, calcium, vitamin C, vitamin D	Children living in Rochester, NY, and were 5–7 mo old at baseline visit; low-income families (same participants as in Haynes et al[95])	249	2.9 μg/dL (95% CI: 2.7 to 3.1)	At 24 mo of age, BLLs were 7.5 μg/dL; 82 (33%) had BLLs ≥10 μg/dL, 32 (13%) had BLLs ≥15 μg/dL, and 14 (6%) had BLLs ≥20 μg/dL; dietary iron intake was inversely associated with BLLs ($P = .03$) during first year of life; calcium intake was not associated with BLL concentration	ND
Lee et al[34] (2005)	Calories, fat, thiamine, pyridoxine, vitamin E, ascorbic acid, folate, calcium, phosphorus, iron	Women 20–49 y old from NHANES III	4394 (3716 had complete data for all variables in study)	NA	Average BLL of reproductive-age women was 1.78 μg/dL; inverse associations ($P < .05$) between BLL and thiamine and serum folate; positive associations ($P < .05$) between BLL and iron, pyridoxine intake, and folate	ND
Lucas et al[97] (1996)	Calories, fat	Children aged 9–6 y, cared for at University of Maryland Pediatric Ambulatory Center; low-income, inner-city families	296	NA	Average BLL was 11.4 μg/dL; after adjusting for confounders using multiple linear regression models, significant positive associations with BLL were found independently for total caloric intake ($P = .01$) and dietary fat ($P = .05$)	ND

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TABLE 5 **Continued**

Author (Year)	Nutritional Category	Population	*N*	Initial BLLs	Significant Results	Adverse Effects
Markowitz et al[84] (1996)	Iron	Moderately lead-poisoned children referred to Montefiore Medical Lead Clinic (Bronx, NY) from 1986–1992 with BLLs 25–55 μg/dL; low-income, inner-city families, living in pre-1960 housing; 2/3 Hispanic, 1/3 black	79	NA	BLLs declined 27% on average over 6 mo; 2/3 <25 μg/dL, 7% <15 μg/dL; iron status did not account for change in BLLs	ND
Markowitz et al[90] (2004)	Calcium	Children aged 1–6 referred to Montefiore Medical Center with BLLs between 10 and 44 μg/dL	88	10–44 μg/dL	No significant differences between BLLs in either group; calcium supplementation of 1800 mg/d for 3 or 6 mo did not reduce BLLs	Abdominal-pain complaints occurred infrequently in both groups
Sargent et al[91] (1999)	Calcium, iron, phosphorus	Infants aged 3.6–6 mo in Lawrence, MA; high proportion of low-income families; data collected 1991–1993; majority Latino (>90%)	103; complete laboratory data collected for 81 (78.6%) of original random assignment	<25 μg/dL	There was no significant difference between groups in the mean ratio of urinary calcium to creatinine, serum calcium and phosphorus, or change in iron status (serum ferritin, total iron-binding capacity); at month 4, the median increase from baseline BLLs in the treatment group was 57% of the increase for the control group ($P = .039$), but this effect weakened after month 4 through the final 9th month of the trial; because the effect did not last, cannot conclude that calcium glycerophosphate supplement prevented lead absorption	Ten children distributed evenly between groups had at least 1 urine sample with a ratio of urinary calcium to creatinine above the age-related norm; 2 had repeat elevated levels (1 in each group); 1 in control group had elevated serum calcium level; 13 had low serum ferritin concentrations (5 control, 8 treatment)
Schell et al[92] (2004)	Calcium, ferritin, iron, protein, supplements, vitamin D, zinc	Mother/infant pairs of low SES in Albany County, NY, from APILS 1992–1998	169	1.6–10 μg/dL (at birth)	By 6 mo, mean BLLs significantly increased from birth to 2.3 μg/dL ($P < .001$); none were ≥10 μg/dL; by 12 mo, mean BLLs significantly increased from 6 mo to 5.1 μg/dL ($P < .001$), and 18% were ≥10 μg/dL; observed significant inverse relationships between infant's 6-mo BLL and intake of zinc ($P = .003$), iron ($P = .015$), and calcium ($P < .001$); at 12 mo, low iron intake continued to be associated with higher BLLs ($P = .041$), although zinc and calcium did not; protein had a paradoxical effect (associated with lower BLL at 6 mo ($P = .001$) but higher BLL at 12 mo; serum vitamin D and ferritin were not associated with BLLs, nor was vitamin supplement use	ND
Schell et al[93] (2003)	Calcium, ferritin, iron, supplements, vitamin D, zinc	Mother/infant pairs of low SES in Albany County, NY, from APILS 1992–1998	220	1.58 μg/dL (neonates)	Mother's BLLs were strongly and positively related to neonate's BLLs ($P < .001$); for the anthropometric measures of maternal nutritional status, variables measuring gain in weight and arm circumference were negatively related to neonate BLLs ($P < .001$); dietary intakes in iron ($P = .003$) and vitamin D ($P = .038$) were negatively related to neonates' BLLs; the effects of zinc varied substantially; calcium was negatively related to BLLs before controlling for age, education index, etc ($P = .042$) but not after controlling for these variables; serum ferritin, serum vitamin D, and supplements were not significantly related to BLLs of neonates; black mothers and newborns had significantly higher BLLs than whites ($P < .001$) except in the second trimester	ND

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TABLE 5 **Continued**

Author (Year)	Nutritional Category	Population	*N*	Initial BLLs	Significant Results	Adverse Effects
Simon and Hudes[99] (1999)	Ascorbic acid	Probability sample of US population from the NHANES III, 1988–1994 without a history of lead poisoning; adults and youths	4213 youths aged 6–16 and 15 365 adults aged ≥17	ND	Twenty-two youths (0.5%) had elevated BLLs, and 57 (0.4%) adults had elevated BLLs; serum ascorbic levels ranged from 0 to 170 μmol/L, with the mean for the youths 55 μmol/L and the adults 43 μmol/L; after controlling for the effects of age, race, gender, income level, and dietary energy, fat, calcium, iron, and zinc intake, youths in the highest serum ascorbic acid tertile had an 89% decreased prevalence of elevated BLLs compared with youths in the lowest serum ascorbic acid tertile ($P = .002$); adults in the highest 2 serum ascorbic acid tertiles had a 65%–68% decreased prevalence of elevated BLLs compared with adults in the lowest serum ascorbic acid tertile ($P = .03$); as a continuous predictor, serum ascorbic acid level was independently associated with decreased BLLs among adults ($P < .001$) but not among youths	ND
Zierold and Anderson[100] (2004)	Many, not described	Data from Wisconsin Childhood Lead Poisoning Prevention Program from 1996–2000; children aged 0–6	111 196	Mean: 5.29 μg/dL	For those in the special nutrition program, mean BLLs declined over the 4-y time period from 7.89 to 5.29 μg/dL (average BLL decline of 0.64 μg/dL per y); for the comparison group, mean BLLs declined over the 4-y time period from 5.51 to 3.70 μg/dL (average BLL decline of 0.42 μg/dL per y); the difference between the groups was not statistically significant ($P = .25$); BLLs of black children in the special nutrition program had a significantly quicker decline compared with white children ($P = .03$)	ND

ND indicates not described; NHANES, National Health and Nutritional Survey; APILS, Albany Pregnancy Infancy Lead Study.

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TABLE 6 Summary of Nutrition Interventions

Studies	Ascorbic Acid	Calcium	Calories	Carbohydrates	Fat	Ferritin	Folate	Folate (Serum)	Iron	Multiple, Not Described	Phosphorus	Protein	Pyridoxine	Supplements	Thiamine	Vitamin D	Zinc
RCTs																	
Dalton et al[89] (1997)	—	NS	—	—	—	—	—	—	NS	—	NS	—	—	—	—	—	—
Markowitz et al[90] (2004)	—	NS	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Sargent et al[91] (1999)	—	NS	—	—	—	—	—	—	NS	—	NS	—	—	—	—	—	—
Prospective Cohort Studies																	
Gallicchio et al[96] (2002)	N	—	P	N	P	—	—	—	—	—	—	—	—	—	—	—	—
Haynes et al[95] (2003)	—	N[a]	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Lanphear et al[94] (2002)	—	NS	—	—	—	—	—	—	N	—	—	—	—	—	—	—	—
Markowitz et al[84] (1996)	—	—	—	—	—	—	—	—	NS	—	—	—	—	—	—	—	—
Schell et al[93] (2003)	—	NS	—	—	—	NS	—	—	N	—	—	—	—	NS	—	N (dietary); NS (serum)	Varied
Schell et al[92] (2004)	—	NS	—	—	—	NS	—	—	N	—	—	Varied	—	NS	—	NS (serum)	NS
Retrospective cohort study (with comparison group)																	
Zierold and Anderson[100] (2004)	—	—	—	—	—	—	—	—	—	NS	—	—	—	—	—	—	—
Cross-sectional studies																	
Hammad et al[98] (1996)	—	—	—	—	—	—	—	—	N	—	—	—	—	—	—	—	—
Lee et al[34] (2005)	—	—	—	—	—	—	P	N	P	—	—	—	P	—	N	—	—
Lucas et al[97] (1996)	—	—	P	—	P	—	—	—	—	—	—	—	—	—	—	—	—
Simon and Hudes[99] (1999)	N	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

— indicates not evaluated; N, negative/inverse relationship; NS, not significant/no relationship; P, positive relationship.

[a] Calcium intake was inversely associated with children's BLL ($P = .03$) in a multivariate model that included VDR Fok 1 genotype as an independent variable. No significant effect modification of calcium intake on BLL by genotype was found ($P = .49$). No significant association was observed when the polymorphism was not included (Lanphear et al[94]).

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TABLE 7 **Summary of Evidence**

KQ	Findings
Children	
KQ1	
Is there direct evidence that screening for lead results in improved health outcomes (ie, cognitive changes, behavioral problems, learning disorders)?	There is no direct evidence from controlled studies of screening.
KQ2	
What is the prevalence of elevated lead in children?	The prevalence of BLLs ≥10 μg/dL among children aged 1–5 y in the United States has declined from 9% in 1988–1991 to 1.6% 1999–2002.
Are there population-level risk factors that identify children at higher risk for elevated lead levels?	Population-level risk factors among children include age <5 y, urban residence, low income, low parental educational attainment, pre-1950 housing, and recent immigration. Mean BLLs among black children remain significantly higher than Mexican American children and non-Hispanic white children.
KQ3	
Can screening tests accurately detect elevated BLLs?	BLL is more sensitive and specific than free EP levels but can be affected by environmental lead contamination and laboratory analytic variation. In 1 study of 47 230 suburban and rural children, 4.7% had an elevated EP level, whereas only 0.6% had elevated an BLL. Capillary sampling has false-positive rates of 3%–9% and false-negative rates of 1%–8%, compared with venous BLLs.
How accurate are questionnaires (or other tools) for risk-factor assessment at various BLLs?	The sensitivity and specificity of questionnaires vary considerably with the prevalence of elevated BLL in the population surveyed and the cutoff BLL (10 vs 15 μg/dL). One study found that rental status, lead-contaminated floor dust, and poor housing conditions were associated with elevated BLLs, suggesting that housing characteristics can be used to identify homes in which a lead hazard may exist before or during occupancy.
What is the optimal frequency for screening?	Not addressed in this review.
What is the optimal frequency for repeat testing?	Not addressed in this review.
KQ5	
Do interventions for elevated BLLs result in improved health outcomes or BLLs?	We identified no evidence that treatment, lead abatement, or education improved neurocognitive outcome in asymptomatic children with mildly to moderately increased lead levels. In 1 trial of succimer, there was no benefit or slight harm. Some interventions have small, inconsistent, or unsustained effects on BLLs in high-risk children.
KQ4 and KQ6	
What are the adverse effects of screening and treatment?	See text.
KQ7	
What are cost-effectiveness issues?	Not addressed in this review.
Pregnant women	
KQ1	
Is there direct evidence that screening in asymptomatic pregnant women for lead results in improved health outcomes?	There is no direct evidence from controlled studies of screening that screening improves maternal hypertension, cognitive changes in offspring, or perinatal outcomes.
KQ2	
What is the prevalence of elevated BLLs in pregnant women?	In 1992, 2 large surveys of low-income pregnant women found 0% and 6% with BLLs >15 μg/dL. A longitudinal study of pregnant women in Boston found that umbilical cord-blood levels declined 82% between 1980 and 1990.
Are there population-level risk factors that identify pregnant women at higher risk for elevated BLLs (ie, geography, racial/ethnicity, SES, age)?	Ethnic background, country of origin, and immigrant status of birth mothers have been shown to be associated with prenatal lead exposure in newborns. Cigarette smoking, maternal age, and alcohol intake have been found to increase umbilical cord-blood lead levels.
KQ3	
Can screening tests accurately detect elevated BLLs?	See KQ3 for children, above.
How accurate are questionnaires (or other tools) for risk-factor assessment at various BLLs?	We found 1 study of a 4-question prenatal survey developed by the CDC that had a sensitivity of 75.7% and a negative predictive value of 93.1%.
What is the optimal frequency for screening? What is the optimal frequency for repeat testing?	Not addressed in this review.
KQ5	
Do interventions for elevated BLLs result in improved health outcomes?	We identified no evidence that treatment, lead abatement, or education improved neurocognitive outcome in asymptomatic children with mildly to moderately increased lead levels. In 1 trial of succimer, there was no benefit or slight harm.
KQ4 and KQ6	
What are the adverse effects of screening and treatment?	See text.
KQ7	
What are cost-effectiveness issues?	Not addressed in this review.

EP indicates erythrocyte protoporphyrin.

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chain of evidence demonstrating benefit. The prevalence of levels >15 μg/dL seems to be quite low in pregnant women. There is some evidence that mildly elevated BLLs during pregnancy are associated with small increases in antepartum blood pressure, but there is only limited evidence that these levels have important adverse effects on reproductive outcomes. An extensive literature search failed to identify studies that have evaluated screening or intervention for lead exposure in pregnant women. There are potentially important adverse effects of chelation therapy on the fetus and of residential lead hazard control on both the pregnant woman and fetus if they are not performed according to established standards. Although removal to a lead-free environment would theoretically be effective in reducing lead exposure, it has not been specifically evaluated in pregnancy.

Community-based interventions for the primary prevention of lead exposure are likely to be more effective, and may be more cost-effective, than office-based screening, treatment, and counseling.[28] Evaluating the effectiveness of community-based interventions and recommendations regarding their use are important areas of future research.

APPENDIX 1. KQs AND CRITICAL KQs

Members of the USPSTF and AHRQ identified an analytic framework (Fig 1) and KQs for updating the USPSTF guidelines for lead screening.

KQs for children were stated as follows:

- KQ1: Is there direct evidence that screening for lead results in improved health outcomes (ie, cognitive changes, behavioral problems, learning disorders)?
- KQ2: What is the prevalence of elevated lead in children? Are there population-level risk factors that identify children at higher risk for elevated lead levels (ie, geography, race/ethnicity, socioeconomic status [SES], age)?
- KQ3: Can screening tests accurately detect elevated BLLs? What is the accuracy of using questionnaires (or other tools) for risk-factor assessment at various BLLs? What is the optimal frequency for screening? What is the optimal frequency for repeat testing?
- KQ4: What are the adverse effects of screening?
- KQ5: Do interventions (ie, counseling families to reduce lead exposure, nutritional interventions, residential lead hazard-control techniques, chelation therapy) for elevated lead levels result in improved health outcomes?
- KQ6: What are the adverse effects of interventions?
- KQ7: What are cost-effectiveness issues?

KQs for pregnant women were stated as follows:

- KQ1: Is there direct evidence that screening in asymptomatic pregnant women for lead results in improved health outcomes (ie, cognitive changes in offspring; perinatal outcomes including birth weight, preterm delivery, etc; maternal blood pressure)?
- KQ2: What is the prevalence of elevated lead in asymptomatic pregnant women? Are there population-level risk factors that identify pregnant women at higher risk for elevated lead levels (ie, geography, racial/ethnicity, SES, age)?
- KQ3: Can screening tests accurately detect elevated BLLs? What is the accuracy of using questionnaires (or other tools) for risk-factor assessment at various BLLs?
- KQ4: What are the adverse effects of screening?
- KQ5: Do interventions (ie, counseling families to reduce lead exposure, nutritional interventions, residential lead hazard-control techniques, chelation therapy) for elevated lead levels result in improved health outcomes?
- KQ6: What are the adverse effects of the interventions?
- KQ7: What are cost-effectiveness issues?

Members of the USPSTF and AHRQ identified KQ1 and KQ5 for children and pregnant women as critical KQs. For these critical KQs, we used USPSTF methods to systematically abstract information about the design, results, and internal validity of each study and included only those studies we rated to be of fair quality or better.[29] We conducted a selected review of the literature that addressed KQ2, KQ3, KQ4, and KQ6. The cost-effectiveness of screening would be examined only in the presence of adequate evidence of intervention efficacy. We did not examine KQ7 because of the lack of evidence of improved clinical outcomes for KQ5. We reviewed the populations of asymptomatic children and pregnant women separately.

APPENDIX 2. CRITERIA FOR GRADING THE QUALITY OF INDIVIDUAL STUDIES

The Methods Work Group for the third USPSTF developed a set of criteria to evaluate the quality of individual studies. At its September 1999 quarterly meetings, the USPSTF accepted the criteria and definitions of quality categories relating to internal validity.

Presented below are a set of minimal criteria for each study design and a general definition of 3 categories: good, fair, and poor. These specifications are not meant to be rigid rules but, rather, are intended to be general guidelines, and individual exceptions, when explicitly explained and justified, can be made. In general, a good study is one that meets all criteria well. A fair study is one that does not meet (or it is not clear that it meets) at

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least 1 criterion but has no major limitations. Poor studies have at least 1 major limitation.

Systematic Reviews

Criteria include:

- comprehensiveness of sources considered/search strategy used;
- standard appraisal of included studies;
- validity of conclusions; and
- recency and relevance (especially important for systematic reviews).

Definition of ratings from above-listed criteria:

Good: Recent, relevant review with comprehensive sources and search strategies; explicit and relevant selection criteria; standard appraisal of included studies; and valid conclusions.

Fair: Recent, relevant review that is not clearly biased but lacks comprehensive sources and search strategies.

Poor: Outdated, irrelevant, or biased review without systematic search for studies, explicit selection criteria, or standard appraisal of studies.

Case-Control Studies

Criteria include:

- accurate ascertainment of cases;
- nonbiased selection of cases/controls, with exclusion criteria applied equally to both;
- response rate;
- diagnostic testing procedures applied equally to each group;
- measurement of exposure accurate and applied equally to each group; and
- appropriate attention to potential confounding variable.

Definition of ratings from above-listed criteria:

Good: Appropriate ascertainment of cases and nonbiased selection of case and control participants; exclusion criteria applied equally to cases and controls; response rate ≥80%; diagnostic procedures and measurements accurate and applied equally to cases and controls; and appropriate attention to confounding variables.

Fair: Recent, relevant, without major apparent selection or diagnostic workup bias but with response rate <80% or attention to some but not all important confounding variables.

Poor: Major selection or diagnostic workup biases, response rates <50%, or inattention to confounding variables.

RCTs and Cohort Studies

Criteria include:

- initial assembly of comparable groups (for RCTs: adequate randomization, including first concealment and whether potential confounders were distributed equally among groups; for cohort studies: consideration of potential confounders with either restriction or measurement for adjustment in the analysis and consideration of inception cohorts);
- maintenance of comparable groups (includes attrition, crossovers, adherence, contamination);
- important differential loss to follow-up or overall high loss to follow-up;
- measurements that are equal, reliable, and valid (includes masking of outcome assessment);
- clear definition of interventions;
- important outcomes considered; and
- analysis (adjustment for potential confounders for cohort studies, or intention-to-treat analysis for RCTs).

Definition of ratings from above-listed criteria:

Good: Meets all criteria: comparable groups are assembled initially and maintained throughout the study (follow-up at least 80%); reliable and valid measurement instruments are used and applied equally to the groups; interventions are spelled out clearly; important outcomes are considered; and appropriate attention is paid to confounders in analysis. In addition, for RCTs, intention-to-treat analysis is used.

Fair: Any or all of the following problems occur, without the fatal flaws noted in the "poor" category below: Generally comparable groups are assembled initially but some question remains whether some (although not major) differences occurred in follow-up; measurement instruments are acceptable (although not the best) and generally applied equally; some but not all important outcomes are considered; and some but not all potential confounders are accounted for. Intention-to-treat analysis is performed for RCTs.

Poor: Any of the following fatal flaws exist: groups assembled initially are not close to being comparable or maintained throughout the study; unreliable or invalid measurement instruments are used or not applied at all equally among groups (including not masking outcome assessment); and key confounders are given little or no attention. For RCTs, intention-to-treat analysis is lacking.

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Diagnostic-Accuracy Studies

Criteria include:

- screening test relevant, available for primary care, adequately described;
- study uses a credible reference standard, performed regardless of test results;
- reference standard interpreted independently of screening test;
- handles indeterminate results in a reasonable manner;
- spectrum of patients included in study;
- appropriate sample size; and
- administration of reliable screening test.

Definition of ratings from above-listed criteria:

Good: Evaluates relevant available screening test; uses a credible reference standard; interprets reference standard independently of screening test; reliability of test is assessed; has few or handles indeterminate results in a reasonable manner; includes large number (>100) of broad-spectrum patients with and without disease.

Fair: Evaluates relevant available screening test; uses reasonable although not best standard; interprets reference standard independent of screening test; has a moderate sample size (50–100 subjects) and a "medium" spectrum of patients.

Poor: Has fatal flaw such as using inappropriate reference standard; screening test is improperly administered; biased ascertainment of reference standard; very small sample size of very narrow selected spectrum of patients.

APPENDIX 3. DETAIL ON PREVALENCE AND RISK

What Is the Prevalence of Elevated Lead in Children?

The prevalence of elevated BLLs in the US continues to decline sharply, primarily because of marked reductions in lead in gasoline, air, dietary sources, and residential paint.[30] In a 1999–2002 national survey of children aged 1 to 5 years, 1.6% had BLLs >10 μg/dL, compared with 9% in a similar survey in 1988–1991.[15] Although the nationwide prevalence of elevated BLLs among children aged 1 to 5 years declined dramatically from 1991–1994 through 1999–2002, the prevalence still varies substantially among different communities and populations, and an estimated 310 000 children remain at risk for exposure to harmful levels of lead.[30]

What Is the Prevalence of Elevated Lead in Asymptomatic Pregnant Women?

BLLs and blood umbilical cord lead levels are frequently used to assess both the mother's and fetus's level of lead exposure and risk. In 1992, 2 large surveys of low-income pregnant women found 0% and 6%[1] with BLLs >15 μg/dL. A study of all women who enrolled in prenatal clinics in Mahoning County, Ohio, from 1990 to 1992 found that 13% of prenatal patients had BLLs >10 μg/dL, with 1% having BLLs >15 μg/dL.[14]

Population mean BLLs in women of childbearing age and pregnant women have decreased over the past 2 decades. Although it was estimated in 1990 that 4.4 million women of childbearing age, and >400 000 pregnant women, had BLLs of >10 μg/dL,[31] a longitudinal study of pregnant women in Boston, Massachusetts, demonstrated that umbilical cord BLLs declined 82% between 1980 and 1990.[32] A recent study of 1109 infants in Quebec, Canada, found a mean cord-blood lead level of 1.5 μg/dL (0.076 μmol/L; 95% CI: 0.074 to 0.079).[33] In a recent review of National Health and Nutrition Examination Survey data of 4394 women of childbearing age, the geometric mean (GM) BLL was 1.78 μg/dL.[34]

Are There Population-Level Risk Factors That Identify Children at Higher Risk for Elevated Lead Levels (ie, Geography, Race/Ethnicity, SES, Age)?

The highest GM BLLs in the United States occur in children aged 1 to 5 years (GM BLL: 1.9 μg/dL) and adults aged >60 years (GM BLL: 2.2 μg/dL), with the lowest levels in youth aged 6 to 19 years (GM BLL: 1.1 μg/dL).[30] Children younger than 5 years are at greater risk for elevated BLLs and lead toxicity because of increased hand-to-mouth activity, increased lead absorption from the gastrointestinal tract, and the greater vulnerability of a developing central nervous system.[35] GM levels are significantly higher in males than in females except among children aged 1 to 5 years.[30]

Correlates of higher BLLs at all ages include minority race/ethnicity, urban residence, low income, low educational attainment, older (pre-1950) housing, and recent immigration.[30,36–40] These factors are associated with increased exposure to important lead sources, including dilapidated housing containing lead-based paint, lead-soldered pipes, household lead dust, and lead in dust and soil from heavy traffic and industry.[1,41] There have been major reductions in the number of US homes with lead-based paint from the estimated 64 million in 1990, but ~24 million housing units still contain substantial lead hazards, with 1.2 million of these units occupied by low-income families with young children.[30,42]

Less frequent sources of household lead exposure include contaminated clothing or materials brought home by workers in lead-using industries, lead-using home businesses or hobbies, lead-based paint and dust contamination in pre-1978 housing that is undergoing remodeling or renovation,[40] dietary intake from lead-contaminated consumer products, drinking water, and lead-based pottery, and traditional ethnic remedies.[3,28,30,43,44]

GM BLLs among black children (2.8 μg/dL) remain

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significantly higher than those among Mexican American children (1.9 μg/dL) and non-Hispanic white children (1.8 μg/dL). Even among low-income families, however, GM BLLs declined significantly from 1991–1994 (3.7 μg/dL) to 1999–2002 (2.5 μg/dL).[30]

A woman of childbearing age with a high BLL risks transmitting lead to her unborn child.[45] Ethnic background, country of origin, and immigrant status of birth mothers, pica behavior, as well as lifestyle and work patterns of pregnant women and age have shown to be associated with prenatal lead exposure in newborns. Multivariate analyses of pregnant women in Quebec revealed that both cigarette smoking (15% increase) and alcohol intake (17% increase) make significant and independent contributions to cord-blood lead concentrations.[46] In a survey of 10 Quebec hospitals, umbilical cord-blood samples were obtained from 1109 newborns. Although BLLs were considered low, a statistically significant relationship was observed between maternal age and smoking during pregnancy in cord-blood lead concentrations.[33]

Mother/infant pairs (159) from a cohort of women receiving prenatal care in Pittsburgh, Pennsylvania, provided blood samples at delivery for lead determination. Alcohol use was associated with relatively greater cord-blood lead levels compared with maternal BLLs. No association was found with cord-blood lead level or maternal BLL with smoking, physical exertion, or calcium consumption.[47]

A recent study in New York City, New York, of pregnant women in their third trimester with an incident BLL of ≥20 μg/dL showed they had newborns with a median incident BLL of 12 μg/dL. In addition, maternal BLLs were directly associated with gestational age and pica behavior. These subjects were more than twice as likely to be foreign-born women.[48]

Neurotoxic Effects of Lead Exposure in Children

High levels of lead can produce serious central and peripheral neurologic complications including acute encephalopathy, which can result in coma, death, or long-term impairment.[1,49,50] Prospective cohort studies across several child populations have suggested that a rise in BLL from 10 to 20 μg/dL is associated with a likely decrement of 2 to 3 points (reported range: −6 to +1) in intelligence test scores (IQ).[1] The variety of test instruments that have been used, and differences in adjustment for important covariates, make direct comparison of these studies difficult, but a consistent negative effect on intellectual development has been reported.

Significant associations have been demonstrated between umbilical BLLs and neurodevelopmental testing at 2 years of age, although the association was not significant at later ages. BLLs at 2 years of age, however, were associated with neurocognitive performance at 10 years of age.[35] A recent analysis of school-aged children demonstrated a stronger cross-sectional inverse association of IQ with contemporary BLLs (mean BLL: 8 μg/dL at 7 years of age) than with baseline BLLs (mean BLL: 26 μg/dL at 24 months of age), suggesting an ongoing adverse effect of lead on cognitive performance among school-aged children.[51]

Previous cross-sectional studies[1] consistently reported small, inverse associations between blood or tooth lead level and reaction (attentional) performance, but studies that evaluated the effect of mildly elevated BLLs on other measures of neurodevelopmental function (eg, behavior, learning disorders, auditory function) produced inconclusive results. These outcomes have been evaluated less thoroughly than IQ, and more recent studies have bolstered an association between childhood lead exposure and disorders of attention and learning and of aggressive and delinquent behavior.[35,49,52,53]

A growing number of human epidemiology studies have reported associations between neurotoxic effects and BLLs once thought to be harmless. Several recent studies have demonstrated an inverse relationship between historical BLLs and subsequent measures of intellectual and cognitive performance at BLLs of <10 μg/dL. The shape of the dose-response curve at levels <10 μg/dL is uncertain, although data suggest that lead-associated cognitive changes may be greater with incremental changes in BLLs in this range.[35,49,53–57] A recent meta-analysis of 7 prospective international cohort studies found evidence of deficits on standard IQ testing among children with maximal BLLs <7.5 μg/dL. A decline of 6.2 IQ points (95% CI: 3.8 to 8.6) was observed as BLLs increased from 1 to 10 μg/dL.[58]

Lead-associated effects on neurobehavioral functioning must be considered relative to other important covariates such as SES, home and parenting environment, and genetic factors.[54] The contribution of childhood lead exposure to the observed variance in cognitive ability (IQ testing) is believed to be in the range of 1% to 4%, whereas social and caregiving factors may be responsible for ≥40%.[52,54] BLLs, however, seem to be associated with a substantial proportion of the known, modifiable variance in children's cognitive ability and incur a substantial social and economic burden among those affected and on the nation.[59,60]

Reproductive Effects of Lead Exposure

The effects of high BLLs on reproductive outcomes have been well described.[1] High paternal BLLs (>40 μg/dL or prolonged levels >25 μg/dL) are associated with impaired fertility, spontaneous abortion, and fetal growth abnormalities (preterm delivery and low birth weight). Maternal BLLs as low as 10 μg/dL have been associated with pregnancy hypertension, spontaneous abortion, and neurobehavioral effects in offspring. Studies that evaluated potential associations between parental lead exposure and congenital malformations in offspring

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have not demonstrated consistent patterns of defects or magnitude of risk and often lack biological indices of exposure at developmentally significant times.[2]

The Mexico City Prospective Lead Study examined the association of maternal prenatal BLLs during pregnancy (range: 7.5–9.0 μg/dL [0.36–0.43 μmol/L]) and child postnatal BLLs (range of median BLL from birth to 48 months: 7.0–10.0 μg/dL [0.34–0.48 μmol/L]) with head circumference in a sample of Latino immigrants living in Los Angeles, California. Multiple regression modeling showed significant negative associations ($P < .05$, 2-tailed) between 6-month head circumference and 36-week maternal BLL and between 36-month head circumference and 12-month BLL, but these were the only significant associations among >50 assessed in this study.[61]

In 272 mother/infant pairs, tibia bone lead was the only lead biomarker clearly related to birth weight (other significant birth weight predictors included maternal nutritional status, parity, education, gestational age, and smoking during pregnancy). Findings suggest that bone lead might be a better biomarker of body lead burden than BLL.[62]

Neurodevelopmental and Cognitive Measures and Lead Effects

Recent observational studies (prospective cohort and cross-sectional) provide limited, preliminary data that prenatal BLLs may be associated with neurodevelopmental delay or impairment. Study design and measurement issues, however, limit interpretation of these studies.

A prospective study of 103 black neonates with low-level (<5 μg/dL) parental lead exposure included a battery of 16 neonatal behavioral assessments 1 to 2 days after birth. No differences were found in 15 of the 16 domains studied, with neonates in the higher-exposure group receiving lower scores on the hand-to-mouth motor activity than did those infants in the lower-exposure group ($P < .05$).[63] A sample of 79 black infants with low-level prenatal parental lead exposure was given the Fagan Test of Infant Intelligence (FTII) battery at 7 months of age.[64] Excluding all but infants with scores in the 5th and 95th percentiles of the FTII ($n = 5$ in both groups) revealed that subjects rated at high risk for impairment on the FTII (lower 5th percentile) were 6 times more likely to be in the highest maternal BLL quartile ($P < .004$). Infants scoring in the lower 15th percentile on FTII score ($n = 12$) were 2 times more likely to be in the high maternal BLL quartile, although significance dropped to $P < .056$.[64] The difference between the mean BLLs in the infants with lowest and highest FTII scores (5th and 95th percentiles) was very small, however (0.44 vs 0.94 μg/dL). Recent evidence suggests that children may demonstrate differences in evoked visual and auditory potentials associated with increased levels of prenatal lead exposure.[65,66]

Other Adverse Effects of Lead Exposure

Higher BLLs (>40 μg/dL) exert detrimental effects on neurologic, cardiovascular, renal, and hepatic function.[1] Subclinical effects on renal function can be observed at lower levels of exposure, and children may be more vulnerable.[67,68]

In a cohort of women in their third trimester of pregnancy, immigrant women were more likely to have elevated BLLs and elevated blood pressure compared with nonimmigrant women. An association between elevated BLL and blood pressure was significant only in the group of immigrants.[69] Past lead exposure was associated with hypertension and elevated blood pressure during pregnancy. Bone lead concentration, however, was not shown to be related to hypertension or elevated BLL in pregnancy.[70]

Among 110 women in their third trimester of pregnancy, those with gestational hypertension showed significantly higher BLLs than those who were normotensive, and BLL was significantly related to blood pressure even after correcting for BMI and age. The lead/ionized-calcium ratio showed a stronger association with blood pressure than with lead alone.[71] A cross-sectional study of 39 pregnant women in their third trimester compared red blood cell (RBC) levels of lead and blood pressure. The study population included 20 women with normal pregnancies, 15 with mild hypertension, and 4 with severe hypertension and preeclampsia. Preeclamptic women were more likely to have an elevated RBC lead level. Rank correlation showed a significant effect of RBC lead level on blood pressure.[72]

APPENDIX 4. DETAIL ON RESIDENTIAL LEAD HAZARD CONTROL ON BLLs

Although newer residential hazard-control methods can effectively reduce exposure to lead paint and lead-contaminated dust,[1] compared with older strategies that often increased lead exposure during the intervention, these newer techniques can still result in an elevation of BLL in a subset of children immediately after lead-control interventions (Tables 3 and 4). In an evaluation of US Department of Housing and Urban Development (HUD)-sponsored lead-control interventions among 14 state and local governments, 81 (9.3%) of 869 children had an elevation of >5 μg/dL. Risk factors associated with postintervention increases were the number of exterior paint deteriorations, the educational level of the female parent or caregiver, and younger age of the child.[73]

Before 1996, retrospective cohort studies, case series, and uncontrolled experiments suggested a modest decline (4–10 μg/dL) in mean BLLs in children with initial BLLs >25 μg/dL. More recent studies of newer lead-

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based paint hazard-control techniques that included an untreated comparison group, however, found more modest beneficial effects[74,75] or no effects.[76,77]

A meta-analysis of 4 RCTs conducted in 1996–2000 found that interventions had no effect on mean BLLs (−0.62 μg/dL; 95% CI: −1.55 to 0.32), but there were significant reductions in the proportion of children who had BLLs >15 μg/dL (6% vs 14%; $P = .008$) and >20 μg/dL (2% vs 6%; $P = .024$) in those in the intervention group compared with controls.[78]

Of these 4 trials, 2 evaluated dust control and 2 evaluated providing education and equipment to families. The earlier of the 2 trials of dust control (1998) evaluated 1-time professional dust control and window-sill-paint sealing in homes of children aged 4 or younger with mean BLLs of 16.9 μg/dL.[76] There were similar reductions in BLLs in the children in the intervention and control groups (−6.2 vs −5.9 μg/dL) 6 months after abatement. In the second randomized trial (1999), conducted in Jersey City, New Jersey, investigators recruited children aged 6 to 36 months who had lead paint in the home. Families ($n = 113$) were randomly assigned to a lead-exposure–reduction group or to an accident-prevention control group. In the lead-exposure–reduction group, staff members visited the home every 2 weeks and spent ~2 hours cleaning up dust. After 1 year, there was a small but statistically significant difference in BLL change between those in the intervention and control groups, adjusted for baseline BLLs (−2.1 vs +0.1 μg/dL; $P < .05$).[74]

A follow-up study in urban children participating in the TLC trial examined the effects of a second professional lead-dust cleaning of homes 18 months after an initial cleaning and therapy commencement.[79] All homes in the Philadelphia, Pennsylvania, site ($n = 165$) of the TLC trial were offered a second professional cleaning. Participation in the follow-up intervention was voluntary rather than randomly assigned. The mean BLL at study initiation was 26 μg/dL. The mean BLL was 15.7 μg/dL at the second cleaning visit, but 6 months later there was no difference in BLLs between children whose homes were cleaned ($n = 73$) and those whose homes were not cleaned ($n = 86$). The report did not stratify results by the original treatment assignment of the subjects (chelation versus placebo), so the effects of the combined interventions cannot be compared with those for an untreated group.

A 2003 retrospective cohort study identified children listed in the New York City child blood lead registry and compared BLLs before and 10 to 14 months after remediation with those of a control group that did not have remediation.[77] Mean BLLs declined significantly from 24.3 to 12.3 μg/dL at follow-up regardless of remediation. After adjusting for confounders, the remediation effect was 11% (P = not significant). Race was identified as the only confounding factor: white and Asian children had an adjusted mean follow-up BLL 30% lower than that of black children ($P < .01$). The effect of remediation seemed to be stronger in younger children (10 to <36 months) than in older children (36–72 months). Another retrospective cohort study that evaluated in-home counseling, combined with professional lead-paint remediation, compared BLLs of children aged 6 months to 6 years with mean BLLs of 28.8 μg/dL with similar children who did not receive the intervention.[75] Follow-up BLL was measured, on average, 69 days after abatement, 172 days after the initial sample. After adjusting for season and age of the child, the treatment-group BLL decreased 6.0 μg/dL from 28.8 to 22.8 μg/dL, and the effect of treatment was significant ($P < .05$). The comparison group mean BLL decreased 1.6 μg/dL from 31.1 to 29.5 μg/dL (P = not significant).

In a retrospective study that measured BLLs in children whose homes were abated from 1987 to 1990, before and after abatement policies in Massachusetts became more stringent in 1988, the mean BLL decreased from 26.0 μg/dL at baseline to 21.2 μg/dL ($P < .001$) measured between 2 weeks to 6 months postabatement. Reductions were only seen, however, among children whose baseline BLLs were >20 μg/dL. This study found no meaningful change in preabatement to postabatement levels by calendar year of intervention.[80] The effect of different housing policies on the risk of subsequent lead exposure in homes where a child with an elevated BLL resided in the past was demonstrated in adjacent geographic regions of 2 northeastern states. Approximately 8 years later, the risk of identifying at least 1 child with an elevated BLL (>10 μg/dL) was 4 times greater in the state with less stringent housing-based lead-poisoning–prevention policies.[81]

A study of 1212 HUD dwellings that received interior treatment for lead hazard control in 13 states from 1994 to 1998 reported a mean 2.8 μg/dL reduction in children's ($n = 240$) BLLs 12 months postintervention (from a median level of 10 μg/dL at baseline).[82] The effect of treatment in these studies was not compared with an untreated population. Another study of HUD dwellings in 4 Massachusetts communities found a significantly larger decline in BLLs between 1993 and 2002 among children in treated homes than among those in untreated homes, matching on preintervention BLL. Children's BLLs decreased from 7.07 and 6.62 μg/dL to 3.59 and 4.28 μg/dL in the treated and untreated homes, respectively ($P = .015$). The study adjusted for time and seasonality to account for the downward trend in BLLs observed among children in the general Massachusetts population, from 5.9 μg/dL in 1994 to 3.2 μg/dL in 2002.[83]

These trials highlight the difficulties of lead-paint hazard control as a method to reduce lead exposure. Poor, inner-city families tend to move frequently, and so treating the current residence may have limited long-term

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benefit to the individual child, although benefit accrues to subsequent children moving into that residence. In the Jersey City study, for example, ~30% of the randomly assigned families moved during the 12-month follow-up period.[74] Residential lead-paint hazard control can be costly and labor-intensive, which limits the availability of intervention, especially in poor communities.[1] Recontamination by nearby lead sources, including soil lead, may occur after lead-paint hazard-control efforts in a dwelling.[1,25] These limitations demonstrate the need for effective comprehensive individual interventions, as well as community-based interventions, to reduce household lead exposure. Unfortunately, available data about programs that use multiple interventions are sparse.[73,84]

APPENDIX 5. DETAIL ON EFFECT OF COUNSELING AND EDUCATION INTERVENTIONS ON BLLs

There have been no controlled studies to evaluate whether counseling families to perform cleaning would be as effective in reducing BLLs as professional cleaning. Two RCTs that administered counseling alone[85] or counseling with the provision of cleaning supplies[86] found no significant effects of the intervention on children's BLLs. A retrospective cohort study of children with BLLs of 20–24 μg/dL found that a 1-time in-home educational visit was associated with a greater reduction in BLL after 6 months, compared with households that did not receive an educational visit (-4.2 vs -1.2 μg/dL; $P < .001$).[87]

APPENDIX 6. DETAIL ON THE EFFECT OF SOIL ABATEMENT ON BLLs

Results of the US Environmental Protection Agency's Three City Urban Soil Lead Abatement Demonstration Project suggest that substantial declines in soil lead cause only modest or no reduction in mildly elevated BLLs.[1,25–27] The small effect is caused at least in part by rapid recontamination with dust lead in households undergoing soil abatement. Cross-sectional surveys before and after soil abatement in the vicinity of a former smelting and milling operation observed a statistically significant reduction in BLLs among children aged 6 to 36 months who had not been exposed to lead-contaminated yards in early childhood. A significant reduction was not seen in children aged 36 to 72 months.[88]

APPENDIX 7. DETAIL ON NUTRITIONAL INTERVENTIONS ON BLLs

Three RCTs[89–91] and 3 prospective cohort studies[92–94] did not find a significant correlation between calcium and BLLs, although 1 prospective cohort study[95] found an inverse association. Fat and caloric intakes were positively associated with BLLs in a prospective cohort study[96] and a cross-sectional study.[97] Carbohydrates had an inverse association according to a prospective cohort study.[96] Two prospective cohort studies[92,93] found that ferritin is not significantly related to BLLs. One cross-sectional study[34] found a positive association with folate and a negative association with serum folate. Iron has not been shown to have an effect on BLLs in 2 RCTs[89,91] and 1 prospective cohort study,[84] although 3 prospective cohort studies[92–94] and a cross-sectional study[98] revealed a negative association, whereas another cross-sectional study showed a positive association.[34] Two RCTs[89,91] found no correlation between BLLs and phosphorus. One cross-sectional study found a positive association between BLLs and pyridoxine.[34] Protein had a paradoxical effect in 1 prospective cohort study, significantly associating with lower BLLs at 6 months but then higher BLLs at 12 months.[92] Two prospective cohort studies showed no relationship between supplement use and BLLs.[92,93] One cross-sectional study found a negative association between BLLs and thiamine.[34] Vitamin C is inversely related with BLLs according to a prospective cohort study.[96] Vitamin C was also inversely associated with BLLs in a cross-sectional study.[99] Dietary vitamin D is also inversely related to BLLs according to a prospective cohort study,[93] whereas serum vitamin D was not correlated with BLLs in 2 prospective cohort studies.[92,93] Two prospective cohort studies yielded different results concerning zinc, showing no association to BLLs,[92] and conflicting results.[93]

Despite the significant relationships between nutrients and children's BLLs in the epidemiologic studies described above, it is noticeable that none of the RCTs found significant correlations.[89–91] Similarly, a 2004 retrospective cohort study that used data from the Wisconsin Childhood Lead Poisoning Prevention Program in children aged 0 to 6 years compared BLLs of children enrolled in the Special Supplemental Nutrition Program for Women, Infants, and Children from 1996 to 2000 with BLLs of children not enrolled in the nutrition program and did not find any significant differences between the 2 groups.[100] Other cohort studies revealed significant association with calories, carbohydrates, fat, iron, vitamin C, and vitamin D,[84,92–96] whereas the cross-sectional studies demonstrated significant associations with ascorbic acid, calories, fat, folate, serum folate, iron, pyridoxine, and thiamine.[34,97–99] Adverse effects were reported in 2 of the 14 studies, both of which were RCTs. A calcium study using a 1800 μg/dL[90] dosage reported abdominal pain in subjects in both the treatment and control groups. A calcium glycerophosphate-supplemented infant formula study reported elevated ratios of urinary calcium to creatinine and low concentrations of serum ferritin, but these effects also occurred in subjects in both the treatment and placebo groups.[91] None of the other studies reported adverse effects.

Concerning pregnancy, a recent review concluded that experimental studies in animals and observational studies of humans provide evidence that calcium sup-

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plementation during the second half of pregnancy may reduce prenatal lead exposure by reducing mobilization of lead from bone.[2]

APPENDIX 8. RECOMMENDATIONS OF OTHER GROUPS

The CDC updated its lead-screening recommendations in 1997 in response to evidence of inadequate screening of children at high risk and concerns regarding appropriate use of limited resources in low-prevalence communities. The revised CDC guidelines provided state public health entities with authority and guidance to develop state and local policies for childhood lead screening. The CDC recommended universal screening in communities without data regarding the prevalence of elevated BLLs adequate for local policy development and in communities where >27% of the housing was built before 1950. Screening of all children receiving Medicaid, Supplemental Food Program for Women, Infants and Children (WIC) or other governmental assistance and in populations where >12% of children aged 1 to 2 years have elevated BLLs was also recommended. Targeted screening is recommended for all other children on the basis of individual risk assessment.[3] This approach is also supported by the American College of Preventive Medicine.[101]

In 1998, the American Academy of Pediatrics recommended that pediatricians (1) provide anticipatory guidance to parents of all infants and children regarding potential risk factors and specific prevention strategies tailored for the family and community, (2) in conjunction with public health authorities, develop and use community-specific risk-assessment questionnaires to guide targeted screening in communities where universal screening is not appropriate, (3) provide lead screening at age 9 to 12 months and consider again at ~24 months after state health department guidelines using individualized targeted or universal screening as recommended, and (4) assess possible lead exposure periodically between 6 months and 6 years of age using community-specific risk-assessment questionnaires (blood lead testing should be considered in children with a history of abuse, neglect, or conditions associated with increased lead exposure), and (5) actively participate in state and local lead-poisoning–prevention activities. Recommendations by the American Academy of Pediatrics regarding the urgency and extent of follow-up differ slightly from those of the CDC and depend on the risk classification and on confirmed venous BLLs.[102] The 1998 recommendation was recently updated to include recent data regarding the prevalence and adverse effects of lead exposure and to provide recommendations for pediatricians and government policy makers.[103]

The American Academy of Family Physicians recommends lead screening at 12 months of age for infants who have the following risk factors: residence in a community with a high or undefined prevalence of BLLs requiring intervention; residence in or frequent visits to a home built before 1950 that has dilapidated paint or has recently undergone or is undergoing renovation or remodeling; close contact to a person who has an elevated BLL; residence near a lead industry or heavy traffic; residence with a person whose hobby or job involves lead exposure; use of lead-based pottery; or use of traditional remedies that contain lead.[104]

Medicaid's Early and Periodic Screening, Diagnostic, and Treatment Program requires that all children be considered at risk and must be screened for lead poisoning. The Centers for Medicare and Medicaid Services requires that all children receive a screening blood lead test at 12 and 24 months of age. Children between the ages of 36 and 72 months must receive a screening blood lead test if they have not been screened previously for lead poisoning. At this time, states may not adopt a statewide plan for screening children for lead poisoning that does not require lead screening for all Medicaid-eligible children.[30,105]

Studies of provider behavior before and after the 1997 revision of the CDC recommendations demonstrate that blood lead screening and follow-up of children is often inadequate.[106,107]

Recently, the CDC Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) reaffirmed its support for state and local decision-making based on local data and conditions regarding the appropriate lead-screening recommendations. The ACCLPP also acknowledged the limitations of screening and other forms of secondary prevention, and advocated an increased local and national focus on housing-based primary prevention of lead exposure.[28]

No national organizations currently recommend screening pregnant women for elevated BLLs. Some state organizations have developed local policies regarding lead screening. In 1995, the New York State Department of Health and American College of Obstetricians and Gynecologists District II developed lead-poisoning–prevention guidelines that mandate anticipatory guidance for pregnant women, risk assessment and risk-reduction counseling, and childhood lead-poisoning–prevention education.[108]

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