Prevention of Lead Toxicity in US Children

Bruce P. Lanphear, MD, MPH; Kim N. Dietrich, PhD; Omer Berger, MD

During the past 2 decades, the proportion of US children who have blood lead concentrations of 10 μg/dL or higher declined by over 80% after the elimination of leaded gasoline and lead solder from canned foods, and a ban on leaded paint used in housing and other consumer products. Fatalities and symptomatic lead poisoning are now rare. Residential lead hazards, which are exceedingly difficult to control, are currently the major source of lead intake for children. Undue lead exposure has retreated into 2 major risk groups; impoverished children who live in older, poorly maintained rental housing and more affluent children who live in older housing undergoing renovation. Despite the dramatic decline in children's blood lead levels, lead toxicity remains epidemic among impoverished children who live in older rental housing, especially those who live in the northeastern and midwestern regions of the United States. There are increasing data linking lead exposure with other systemic effects including delinquency, dental caries, and learning problems. Moreover, there is evidence indicating that there is no discernible threshold for lead-associated cognitive deficits. Thus, it is increasingly important to shift our efforts toward the primary prevention of childhood lead exposure from residential hazards. This article reviews the epidemiology and control of childhood lead exposure, focusing especially on steps necessary to shift toward primary prevention.

KEY WORDS: abatement; blood lead concentration; children; lead hazard control; paint; primary prevention; screening

Ambulatory Pediatrics 2003;3:27–36

In the past, lead poisoning was diagnosed by a constellation of signs and symptoms. Children with symptomatic lead poisoning presented with abdominal cramps (lead colic), persistent vomiting, anorexia, or obscure neurologic problems such as headache and visual disturbance.[1] At blood lead concentrations of 70 μg/dL or higher, encephalopathy, coma, and death often ensued.[2] In the era before chelation therapy, 28% to 45% of lead-poisoned children who presented with signs or symptoms of encephalopathy died.[2–3] Over 25% of children with acute encephalopathy sustained overt brain damage, such as mental retardation and seizures.[4–5]

The major problem of lead toxicity in US children has shifted from overt lead poisoning to subclinical effects. Fatal lead poisoning has become rare in the United States. The mortality rate from lead poisoning declined precipitously with the advent of chelation, a pharmacologic therapy that binds lead in the vascular system to make it soluble so that it can be excreted in the urine.[6] In March 2000, after a decade without a fatality linked to lead poisoning, a 2-year-old Sudanese immigrant died in New Hampshire from ingestion of lead-based paint.[7] This article will review the epidemiology and control of childhood lead exposure, focusing especially on the rationale and steps necessary to shift toward primary prevention.

From the Cincinnati Children's Environmental Health Center, Departments of Pediatrics and Environmental Health, Children's Hospital Medical Center, and the University of Cincinnati, Cincinnati, Ohio.

Address correspondence to Bruce P. Lanphear, MD, MPH, Division of General and Community Pediatrics, Children's Hospital Medical Center, 3333 Burnet Ave, Cincinnati, OH 45229-3039 (e-mail: bruce.lanphear@chmcc.org).

Received for publication May 16, 2002; accepted September 9, 2002.

EPIDEMIOLOGY OF LEAD EXPOSURE

Over the past 2 decades, blood lead levels among children in the United States have declined dramatically.[8] This has been due, in part, to the elimination of leaded gasoline. Before 1977, the allowable level of lead in gasoline was 0.78 g/L. In 1977, it was lowered to 0.026 g/L in the United States, but over 50 countries continue to use leaded gasoline.[9] In 1976, the Consumer Product Safety Commission (CPSC) restricted the allowable level of lead in residential paint to 0.06%.[10] The proportion of canned foods using lead solder was reduced, from over 90% in 1978 to less than 5% in 1988.[11] Finally, there was a large decline in the percentage of housing painted with lead-based paint.[12] It is difficult to quantify the blood lead decline attributable to specific sources, but the combined effect led to a rapid decline in children's blood lead levels.

Despite the reduction in children's blood lead concentration, lead toxicity remains epidemic in some populations. Using data from the Third National Health and Nutrition Examination Survey (NHANES III), conducted from 1991 to 1994, the Centers for Disease Control and Prevention estimated that 890 000 (4.4%) of the preschool children in the United States have a blood lead concentration of 10 μg/dL or higher.[8] But in some cities, especially in the northeastern and midwestern United States, over 20% of preschool children have blood lead levels exceeding 10 μg/dL.[13–16] Racial disparity in blood lead levels is pronounced. Black children have significantly higher blood lead levels than do white children.[17–19] Among black children aged 1 to 5 years who lived in housing built before 1946, 22% have blood lead levels

≥10 μg/dL.[8] Latino children have blood lead concentrations lower than those of black children but higher than those of white children.[8]

Racial disparity in blood lead levels persists even after controlling for other sources of environmental lead exposure.[16,18] Racial disparity in blood lead levels is due, in large part, to differences in environmental exposures. But differences in calcium intake or metabolism may also account for some of the racial disparity in blood lead levels.[19–21] Black children ingest, on average, lower amounts of calcium than white children,[19] and low dietary calcium intake is associated with higher blood lead levels.[19,20] Nonetheless, in a study that controlled for environmental exposures and dietary calcium and iron intakes, black children's blood lead levels were still 50% higher than white children's.[18]

Age of housing is a major predictor for childhood lead poisoning.[13,14,22] In a national survey of housing conducted in 1998, Jacobs and coworkers[12] estimated that over 25 million (27%) of US housing units contain 1 or more lead hazards. But the prevalence was higher in older housing. For housing built from 1978 to 1998, 1% contained lead hazards, whereas the prevalence of residential hazards increased to 10% of housing built between 1960 and 1978, 51% of housing built between 1940 and 1959, and 67% of housing units built before 1940.[12] Over 30% of housing units with lead-based paint contained 1 or more lead hazards even when the paint was in good condition. The prevalence of housing units with lead hazards was higher in the northeastern and midwestern regions of the United States, in rental housing, and in lower-income housing.[12]

EVOLUTION OF A DISEASE

The epidemic of childhood lead poisoning in the United States was preventable. In the early 1900s, 2 Australian physicians, Drs L. Gibson[23] and A. J. Turner,[24] described childhood lead poisoning as a distinct entity. In 1904, Gibson showed that painted walls and railings were the source of lead after collecting wipe samples of various surfaces in the homes of lead-poisoned children. Gibson[23] believed that because most children with lead poisoning bite their nails or suck their fingers, educational efforts could help prevent lead poisoning. Four years later, with no evidence that education led to a decline in cases of lead poisoning, Turner[24] concluded, "Prevention is easy. Paint containing lead should never be employed ... where children, especially young children, are accustomed to play."

Unfortunately, the Lead Industry Association, a trade group of lead manufacturers, successfully fought the promulgation of regulations in the United States for over 50 years.[25] Many European countries banned leaded paint in 1921, but it was not banned in the United States until 1978.[25] Ultimately, regulations to eliminate the use of lead

Sequelae of Lead Poisoning

In the early 1900s, it was believed that if a patient survived lead poisoning, he or she would recover completely.[26,27] In 1943, Byers and Lord[28] reported that the effects of childhood lead poisoning were not limited to symptoms associated with acute lead poisoning. They found that 19 of 20 children who had "recovered" from lead poisoning failed high school or had behavioral problems. In 1979, Needleman and his coworkers[29] found that children with higher dentine lead concentrations were more likely to be rated unfavorably by teachers on the dimensions of distractibility, persistence in work, organizational ability, dependence, impulsivity, daydreaming, and ability to follow directions. In a follow-up study, they reported that higher dentine lead levels were associated with lower reading scores, lower class rank, and increased absenteeism in young adulthood.[30] Children in the group with higher lead levels were 5.8 times more likely to have a reading disability and 7.4 times more likely to drop out of school than children in the group with lower levels.

Experimental studies, in both rodents and nonhuman primates, have since documented lead-related deficits at low-level lead exposure and established these to be direct effects of lead.[31–33] Moreover, the preponderance of epidemiologic studies show persistent, deleterious effects of low-level lead exposure on brain function.[34–42] It has been estimated that there is a 2.5- to 3-point IQ decrement associated with an increase in blood lead concentration from 10 to 20 μg/dL.[43,44]

The current definition of an elevated blood lead concentration of 10 μg/dL or higher, as defined by the World Health Organization[43] and the US Centers for Disease Control[45] (CDC), was based on adverse outcomes from numerous cross-sectional and prospective studies. The CDC[45] recognized that there was no discernable threshold for the adverse effects of lead exposure, but set 10 μg/dL as an action level. Unfortunately, this action level is often misconstrued as evidence that there are no adverse effects below 10 μg/dL. Indeed, many pediatricians consider blood lead concentrations below 10 μg/dL to be normal. But contemporary children have a body lead burden that is 10 to 100 times higher than that of preindustrial humans.[46]

There is increasing evidence that lead-associated cognitive deficits occur at blood lead levels below 10 μg/dL.[47–50] Schwartz[47] and Schwartz and Otto[48] reported that lead-associated cognitive deficits and hearing loss occur at blood lead levels below 10 μg/dL. Others have found that lead-related cognitive deficits occur among children who have blood lead levels below 10 or 5 μg/dL.[49,50] Collectively, these data indicate that there is no threshold for the adverse consequences of childhood lead exposure.

Lead-associated cognitive deficits associated with each 1 μg/dL increase in blood lead level are greater at blood

els.[47] In an analysis of children in NHANES III, the lead-associated reading deficit increased, from −1.0 point per 1 μg/dL increase in blood lead level for the entire sample to −1.7 point per 1 μg/dL increase for the subgroup with blood lead levels below 5 μg/dL.[49] Although a biological explanation for this finding is not apparent, children with lower blood lead concentrations may have fewer other environmental insults (and therefore less confounding) than more heavily exposed children.[51]

Behavioral Problems

There is no "behavioral signature" of low-level lead toxicity, but a consistent pattern of lead-induced abnormalities is emerging.[52] Although the development of antisocial, delinquent behavior during childhood and adolescence is a product of multiple interacting socioheredity variables,[53,54] there is increasing evidence that lead toxicity plays a role in its epigenesis.[55–59] Concurrent blood lead levels greater than 10–15 μg/dL have been associated with suboptimal ratings on aggressive and antisocial behaviors.[56,57] Needleman and coworkers[58] found that adolescents with higher bone lead concentrations had higher T-scores on the Delinquency and Aggressive clusters of the Achenbach Child Behavior Checklist. Dietrich and coworkers[59] reported that higher blood lead levels in childhood were associated with 4.5 more episodes of antisocial and delinquent behaviors that posed a risk of arrest in the prior 12 months. Existing data suggest that attention, executive functions, and behavioral conduct are affected by lead exposure.[60–62] But, despite some features that are consistent with attention deficit hyperactivity disorder, evidence linking lead exposure with the clinical diagnosis of this disorder is weak.[63,64]

Developmental Vulnerability

Young children and fetuses are especially vulnerable to the adverse effects of environmental neurotoxicants.[65] Critical processes occur in the central nervous system during fetal development and early childhood, including synaptogenesis, myelination, and programmed apoptosis.[66] Some investigators have found that blood lead levels in early childhood are better predictors of cognitive deficits,[35] whereas others have reported that blood lead levels in older childhood are better predictors.[34,38,39] Blood lead concentration in early childhood tracks closely with subsequent blood lead levels.[34,38] Thus, the larger effects observed in older children may be due to chronicity of exposure.[52] Although the question of whether children are more vulnerable to the toxic effects of lead exposure during the first 2 years of life is unresolved, they do ingest more lead and may absorb it more efficiently than older children and adults.[18,67] Thus, efforts to prevent lead toxicity must occur before birth or in early infancy.[68]

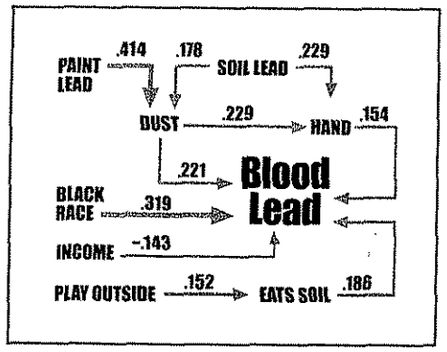
RATIONALE FOR SHIFTING TO PRIMARY PREVENTION

Despite the dramatic decline in children's blood lead of lead in their environments.[13–16,18] There is also considerable evidence that lead is a systemic toxin.[69–78] It has been estimated, for example, that for every 1 μg/dL decline in the population mean blood lead level, there would be 635 000 fewer persons with hypertension, 3200 fewer with myocardial infarctions, 1300 fewer with strokes, and 3300 fewer deaths annually in the United States.[69] Survivors of childhood lead poisoning are twice as likely to die from cardiovascular disease compared with the general population.[70] Dental caries, linked with lead exposure in both experimental and epidemiologic studies, have been estimated to account for over 2.5 million cases of tooth decay in US children.[71,72] Other major problems are linked with lead exposure, including spontaneous abortions and prematurity,[73,74] impaired motor development,[75,76] growth retardation,[77] and delayed nerve conduction.[78] Moreover, there is no discernable threshold for the cognitive deficits linked with childhood exposure.[47–50] Indeed, lead-associated cognitive deficits appear to be greater at blood lead levels below 10 μg/dL.[47,49,50] Finally, investigators of a randomized controlled trial of succimer (*meso* 2, 3-dimercapto-succinic acid, or DMSA) did not find any neurobehavioral benefit of chelation for children who had blood lead levels between 20 and 44 μg/dL.[79] Collectively, the results of these studies argue that our efforts to prevent impairments associated with low-level lead exposure should emphasize primary prevention. This contrasts with current practices and policies that rely almost exclusively on secondary prevention efforts.

The costs to prevent childhood lead poisoning from residential hazards are substantial. It has been estimated, for example, that the first-year cost to reduce residential lead hazards in federally owned or federally assisted housing is $458 million. The Department of Housing and Urban Development[80] (HUD) has estimated that the overall benefit, defined as increase in lifetime earnings of children who are protected from the detrimental effects of lead exposure, is $1.54 billion—a net benefit of $1.08 billion. This estimate does not include recent findings indicating that the drop in IQ is greater for each 1 μg/dL increase in blood lead level at levels below 10 μg/dL.[47–50] Nor does it include other anticipated benefits, such as reductions in rates of cardiovascular disease, tooth decay, and delinquent behaviors.[69–78]

PREVENTION OF LEAD EXPOSURE

The steps to prevent childhood lead exposure are, in theory, simple. The first step is to identify major sources of lead exposure. Second, because lead is ubiquitous—it can be found in house dust and residential soil throughout the country—it is necessary to identify unacceptable or hazardous levels of lead in sources that children encounter. The third step is to conduct screening to identify housing or products that contain lead hazards. Screening children to identify those with undue lead exposure is important, but screening should be used as a safety net, not as



Pathways of lead exposure using structural equation analysis are graphically illustrated. The strength of the associations is shown by the coefficients and the width of each arrow. Adapted from Environ Res. 1997;74:67–73.

sures. Finally, regulations and policies are needed to identify lead hazards and implement the interventions.

Sources of Lead Exposure

Paint is the major source of childhood lead poisoning in the United States.[18,22,81–85] Paint that was used on both the interior and exterior of houses through the 1950s, and continuing to some extent through the 1970s, often contained high concentrations of lead.[81] Over 80% of children with blood lead levels greater than 50 μg/dL were reported to ingest paint chips or broken plaster.[82] Children with blood lead levels above 55 μg/dL were 10 times more likely to have paint chips observable on abdominal radiographs than children who have blood lead levels below this value.[83] The majority of preschool children with blood lead levels over 25 μg/dL were reported to put paint chips in their mouths.[84] For children with lower blood lead levels, paint is the major source of lead-contaminated house dust in the urban setting[85] (Figure).

Although lead-based paint is the major source of lead intake for children, lead-contaminated house dust and residential soil are the major pathways by which children are exposed to lead[85–91] (Figure). In 1904, Gibson[23] proposed that ingestion of disintegrating paint and lead-contaminated house dust was the primary mechanism for lead exposure. In the 1970s and 1980s, Sayre and coworkers[88] and Charney and coworkers[89] showed that normal mouthing behaviors led to ingestion of lead-contaminated house dust. The primary source of lead for the majority of children with blood lead levels between 10 and 25 μg/dL is house dust contaminated by lead-based paint that is damaged or in disrepair.[18,85–87] Scraping, sanding, or construction during painting, renovation, and abatement also increases lead contamination of house dust.[92,93]

Lead-contaminated soil is an important source of lead intake for children living in urban areas.[86,87,90,91,94–96] Soil ingestion, reported to occur in 26% of urban children, is

every 1000–parts per million (ppm) increase in soil lead concentration, but this estimate did not account for other sources of lead intake. In a randomized controlled trial of soil abatement, Aschengrau and coworkers[91] reported a 1.12 to 1.35 μg/dL decrease in blood lead level for every 1000-ppm reduction in soil lead concentration. In a pooled analysis of 12 studies, there was an estimated 3.8 μg/dL increase in blood lead concentration for every 1000-ppm increase in soil lead concentration.[87] The variation in the reported relationship of lead-contaminated soil is due to a number of factors, including the age of children studied, adjustment for the contribution of lead intake from other sources, and mouthing behaviors.

The US Environmental Protection Agency[97] (EPA) standard for lead in water is 15 μg/L (parts per billion [ppb]) in residential water and 20 ppb in public drinking fountains. In a study in Glasgow, tap water was the main correlate of elevated maternal blood lead concentrations, accounting for 76% of maternal blood lead levels above 10 μg/dL.[98] In a prospective study of 248 children followed from 6 to 24 months of age, children who were exposed to a water lead levels of >5 ppb had blood lead concentrations that were 1.0 μg/dL higher than those of children exposed to water lead levels of <5 ppb.[18] Thus, water is not a trivial source of lead intake for young children in many communities. Indeed, as predicted, water is becoming an increasingly important source of childhood lead exposure as other sources of lead intake decline.[99]

There are other sources of lead intake for US children, such as folk medicines,[100] ceramics,[101,102] cosmetics,[103] and lead brought from the work site into the home by a parent.[104,105] Products such as crayons and miniblinds that are contaminated with lead receive a lot of attention. These products probably constitute a small source of lead intake for children. Still, it is easier to eliminate children's exposure to lead by preventing the introduction of new sources of lead into children's environments. Furthermore, because lead exposure is cumulative and there is no apparent threshold for the adverse effects of lead exposure, all sources of lead exposure must eventually be eliminated.

Ingestion and Absorption

There is large variation in the ingestion and absorption of lead during the first 2 years of life. Children's blood lead levels rise rapidly between 6 and 12 months of age, peak between 18 and 36 months of age, and then gradually decline.[18,22] The peak in children's blood lead levels is due to the confluence of normal mouthing behaviors and increasing mobility.[18] Lead-contaminated floor dust is a source of lead intake throughout early childhood, but lead-contaminated dust on windowsills is not a major source of intake until the second year of life, when children stand upright.[18] Soil

and iron.[19,67,106–109] In experimental studies, calcium-deficient animals absorb more lead.[106,107] In an observational study in humans, children with a lower calcium intake had a higher blood lead concentration.[19] Children who had a lower iron intake or who were iron deficient also had higher blood lead levels.[18,108–110] Collectively, these data suggest that a child's dietary intake of calcium and iron is inversely related to his or her blood lead levels. Still, data from randomized controlled trial of calcium-supplemented formula, Sargent and his coworkers[111] found no difference in blood lead levels among children.

In the experimental setting, fasting has been shown to modify lead absorption in adults. There is a 10-fold increase in the absorption of ingested lead among fasting volunteers compared with those who have recently eaten.[112,113] The effect of fasting or frequent snacking on blood lead concentration in childhood has not been quantified.

Screening Children to Prevent Undue Lead Exposure

Universal screening, recommended by the CDC in 1991, is controversial.[114,115] The vast majority of pediatricians recognize lead as a toxicant, but less than half perform blood screening for excessive exposure to lead.[114] There are numerous reasons for pediatricians' paradoxic behavior. First, due to the focal distribution of lead exposure, few children are identified as having an elevated blood lead level in some clinics or communities.[13–16,115] Second, many pediatric clinics do not have a phlebotomist available. Third, there are uncertainties about the safety and efficacy of lead hazards controls or educational interventions.[68] Thus, some pediatricians and public health officials have vigorously opposed universal screening. In 1997 and 1998, the CDC[116] and the American Academy of Pediatrics Committee on Environmental Health[17] shifted away from universal screening and recommended targeted blood lead screening.

The only rationale for screening children for undue lead exposure is to prevent lead poisoning or lead toxicity. Several criteria must be met to justify screening. First, screening must prevent or reduce children's exposure and the adverse consequences of lead exposure. That is, there must be an intervention that is proven to be effective at reducing children's blood lead concentrations. Second, screening must be acceptable for families. Finally, the recommended intervention must be available to families with children that are identified through screening.

Theoretically, detecting a child with a modestly elevated blood lead level allows a physician to provide guidance or treatment to prevent further elevations. Indeed, in studies published in the early 1970s, Sachs and colleagues[82] and Sachs[118] concluded that widespread screening led to a reduction in the proportion of children having blood lead levels of 50 μg/dL or higher, from 8.5% in 1967 to 3.8% in 1968. But these studies were never replicated to ascertain their validity for the current era of lead poisoning. Studies such as this led to the current fixation on screening children to prevent lead exposure.

To reduce children's exposure to lead, physicians often recommend targeted dust control, increasing dietary calcium and iron intakes, washing children's hands, and keeping soil out of children's mouths.[45] Unfortunately, with the exception of dust control, there is little evidence that these interventions reduce children's blood lead levels.[68,119,120] The efficacy of lead hazard control is equally uncertain for children who have blood lead levels below 30 μg/dL.[68]

Most state or local health departments are mandated to conduct environmental testing to identify lead hazards and to conduct interventions for children who have blood lead levels consistently higher than 15 or 20 μg/dL. Unfortunately, environmental inspections are often delayed or never completed.[121,122] In New Jersey, a study found that abatement was not conducted as required for 118 (24%) of 488 children who had blood lead levels between 30 and 44 μg/dL.[121] If a screening test identifies children who require an intervention that is not available, then the test is of limited value.[123]

Numerous studies have shown that family or community characteristics can be used in the clinic setting to identify children who are at increased risk for having elevated blood lead levels.[13–16,124–126] In general, these studies have shown that older age of housing, poorer housing condition, rental status, black race, and poverty are all significant predictors of undue lead exposure.[13–16,124–126] These criteria, however, are highly predictive only in high-prevalence areas.

Interventions to reduce children's blood lead levels that are triggered by an elevated blood lead value are unlikely to benefit children who are older than 2 to 3 years. Bornschein and coworkers (R. Bornschein, unpublished data, 2001) have found a significant correlation of the level of lead-contaminated floor dust with children's blood lead levels until they reach 24 months of age. Thereafter, prior blood lead level is the best predictor of a child's blood lead concentration, and floor dust lead levels are no longer a significant predictor. Thus, although it is important to refine screening strategies to identify children with undue lead exposure, it is more critical to expand our efforts to identify and eliminate residential lead hazards and other sources of lead before children are unduly exposed. Screening children should therefore be a safety net to determine when prevention efforts have failed to protect children.

Residential Standards

Under section 403 of Title X, the US Congress mandated the EPA to promulgate health-based lead standards. There are at least 4 reasons to develop residential lead standards.[68] First, residential standards are necessary to screen housing and identify lead hazards before a child is unduly exposed. The alternative, to wait until a child is unduly exposed, is no longer defensible. Second, stan-

The Effect of Lead Hazard Controls on Children's Blood Lead Levels From Controlled Trials

Trial	Type of Lead Hazard Control	RCT[a]	Sample Size, N	Baseline Blood Lead Level, μg/dL	Child Age, mo	Change in Blood Lead Level, μg/dL
Charney et al	dust control	no	49	≥30	15–72	−6.9*
Staes et al	paint stabilization	no	185	≥25	<72	−4.0*
Schultz (1998)	multifactorial	no	413	20–24	40	−3.1*
Aschengrau et al	paint abatement	no	91	3–22	<48	+6.5*
Aschengrau (1998)	low-cost repairs	yes	41	16.9	24.5	+1.1†
Hilts (1995)	dust control	yes	111	4–26	6–70	+0.3†
Lanphear (1996)	dust control	yes	94	1–31	12–31	+0.6†
Lanphear (1998)	dust control	yes	276	1–12.5	24	−0.5†
Rhoads (1998)	dust control	yes	113	12	20.3	−1.9†

[a]RCT indicates randomized controlled trial.
*$P < .05$.
†Difference not statistically significant by 2-sided significance test, $P < .05$.

testing is not done, the major source(s) of environmental lead exposure may be overlooked. More importantly, it is clear that attempts to reduce lead exposure can actually result in increased contamination of a child's environment and blood lead concentration. Clearance dust tests should therefore be conducted after remodeling or renovation, abatement, or a lead hazard control to protect children. Finally, standards serve as a benchmark to compare the effectiveness and duration of various lead hazard controls. Unfortunately, if standards remain voluntary, they are unlikely to be implemented and will not protect children from undue lead exposure.

Most lead standards have been driven by what is *believed* to be feasible to attain, not because they have been shown to protect children. In 1976, the Consumer Product Safety Commission (CPSC) set the residential paint lead concentration at 0.06% because there was evidence that paint could be manufactured with this smaller amount of contamination.[10] Similarly, data used to estimate the safe level of lead in water may not adequately protect pregnant women and children.[18,97] In 1988, HUD[127] established a postabatement floor standard of 200 μg/ft² because there was evidence that this level was feasible to attain, not because it was demonstrated to be safe or protective. In 1992, Congress mandated the EPA to set a health-based standard for residential lead hazards. The residential standards promulgated by the EPA[128] were, once again, based on what was believed to be feasible to attain rather than on empiric data shown to protect children.[68] Unfortunately, these standards dictate the levels of lead contamination considered "normal" or "low," and they provide an illusion of safety.

In 2001, the EPA[128] promulgated residential lead standards of 40 μg/ft² for floors and 250 μg/ft² for windowsills. Data from epidemiologic studies show that 5% of children have a blood lead level ≥10 μg/dL at a median floor dust lead level of 5 μg/ft².[86,87] At a floor standard of 50 μg/ft², 20% of children are estimated to have a blood

EPA lead standards for floors do not adequately protect children.

Hazards of Lead Hazard Controls

Lead poisoning is often regarded as a preventable disease. But the safety and efficacy of measures intended to control or reduce residential lead hazards are uncertain. Interventions to prevent or control childhood lead exposure (called lead hazard controls) have far too often been shown to result in an increase in children's blood lead levels.[129–134] There is some evidence that lead hazard controls, including paint abatement and stabilization of painted surfaces, can reduce lead exposure for children who have blood lead levels ≥30 μg/dL[135,136] (Table). But, with the exception of dust control, it is uncertain if lead hazard controls are safe or beneficial for children with lower blood lead levels[68] (Table).

Studies conducted since the issuance of the HUD 1988 postabatement dust lead standards show that paint abatement can cause children's blood lead levels to rise. Swindell et al[131] found that, for children who had blood lead levels below 20 μg/dL, there was a 2.5 μg/dL increase in blood lead concentration after abatement. In a controlled study of children with a baseline blood lead below 22 μg/dL, Aschengrau and colleagues[132] reported a 6.5 μg/dL increase in blood lead level for children whose residences underwent paint abatement. Presumably, this rise in blood lead levels after abatement or renovation is due to lead contamination from removal or scraping of leaded paint.[129,130,133,134] Collectively, these studies indicate that the levels of lead-contaminated dust generated by lead hazard controls are sufficient to result in undue lead exposure and absorption for children who have blood lead levels below 25 to 30 μg/dL.[68]

Control of Lead Exposure

Several trials have evaluated the effect of dust control on children's blood lead levels (Table). In a meta-analysis

ıge in Blood ead Level, µg/dL
−6.9*
−4.0*
−3.1*
+6.5*
+1.1†
+0.3†
+0.6†
−0.5†
−1.9†

lead concentrations ≥15 and ≥20 µg/dL in the experimental groups compared with the control groups, indicating some benefit of dust control for children with higher blood lead levels.[119] Still, because the adverse effects of lead are persistent and appear to occur at blood lead levels below 10 µg/dL, it is insufficient to rely on dust control to prevent childhood lead exposure.

There are few controlled trials evaluating the effect of lead hazard controls on children's blood lead levels, but there is evidence that they can result in dramatic and sustained reductions in the amount of lead in dust. Dust lead levels immediately after abatement were 8.5, 8.0, and 21 µg/ft² for floors, interior windowsills, and window troughs, respectively—representing reductions of over 80% compared with preabatement levels.[137] In a study of over 2600 housing units, postabatement dust lead levels were 12, 31, and 32 µg/ft² for floors, windowsills, and window troughs, respectively.[138] Because these levels were achieved with dust clearance testing set at 100 µg/ft² or higher, it is likely that floor dust lead levels below 10 µg/ft² can routinely be achieved. It is also clear that we will fail to protect children if we do not lower the standards.

Steps to Eliminate Lead Toxicity

A comprehensive strategy for the primary prevention of childhood lead poisoning should include several components.

1) Establish empirically based residential lead standards. Promulgation of empirically derived, health-based residential lead standards is essential. These standards must be required and enforced; "voluntary" standards will not protect the majority of children from undue lead exposure.
2) Improve screening programs. Housing should be screened before a child is unduly exposed, after lead hazard controls or renovation.[68] Screening housing units by using dust samples and a visual inspection should be incorporated into housing codes. Rental housing could be targeted to enhance the cost-effectiveness of a screening program.[12–14] Dust sampling should be required before approval of federal subsidies for housing. Exceptions for screening could be made for housing units that do not contain leaded paint or were built after 1978.
3) Conduct trials to prove that lead hazard controls protect children. Once residential lead hazards are identified, it is essential to have safe and effective methods to eliminate them. Too often, we have relied on expert opinion about what is safe or effective. Evidence that lead hazard controls are safe and efficacious must initially rely on their effect on children's blood lead levels. Thereafter, dust lead levels and other environmental measures could be used to evaluate lead hazard controls. Lead hazard controls need to be assessed in trials that are experimental in design or include a control group to plementation, various types of lead hazard controls (including full abatement and less permanent solutions), and interventions that combine education and environmental controls. Specific components of lead hazard controls, such as wet versus dry scraping to remove leaded paint, should also be tested in randomized trials.
4) Devise a strategy to identify and target residential lead hazards. National, state, and community surveys of housing need to be conducted to identify and prioritize the elimination of residential lead hazards. There should be plans for the remediation of lead-contaminated housing, including the gradual elimination of lead hazards during renovation or demolition of older housing. Lead-safe work practices should be adopted and taught to home owners, contractors, painters, and persons who maintain housing.

Despite dramatic reductions in children's blood lead levels, childhood lead exposure remains a major public health problem. For too long, we have simply passed out brochures or instructed mothers to "clean their houses better" to reduce their child's risk of lead poisoning. As foretold by Turner[24] in 1908, educational efforts alone are inadequate to prevent undue lead exposure in children unless lead-based paint is eliminated. The current strategy to prevent lead poisoning largely ignores existing scientific evidence indicating that our efforts should emphasize primary prevention with environmental controls to make lead-contaminated paint inaccessible before a child is unduly exposed. Most federal agencies involved in prevention of lead poisoning acknowledge that primary prevention is preferable, yet our efforts continue to focus on screening children for elevated blood lead levels and controlling lead hazards only after a child has been unduly exposed. Until we shift our efforts toward the primary prevention of childhood lead exposure, we will inadvertently but knowingly continue to use children as biologic indicators of substandard housing.[139]

REFERENCES

1. Weitzman M, Glotzer D. Lead poisoning. *Pediatr Rev.* 1992; 13:461–468.
2. Greengard J, Adams B, Berman E. Acute lead encephalopathy in young children. *J Pediatr.* 1965;66:707.
3. Christian JR, Celewycz BS, Andelman SH. A three year study of lead poisoning in Chicago. *Am J Public Health.* 1964;54: 1241–1245.
4. Chisolm JJ Jr, Harrison HE. The exposure of children to lead. *Pediatrics.* 1956;18:943–957.
5. Perlstein MA, Attala R. Neurologic sequelae of plumbism in children. *Clin Pediatr.* 1966;5:292–298.
6. Hammond P. The effects of chelating agents on the tissue distribution and excretion of lead. *Toxicol Appl Pharmacol.* 1971; 18:296–310.
7. Centers for Disease Control and Prevention. Fatal pediatric lead poisoning—New Hampshire, 2000. *MMWR.* 2001;50: 457–459.
8. Pirkle JL, Kaufmann RB, Brody DJ, et al. Exposure of the US population to lead, 1991–1994. *Environ Health Perspect.* 1998;11:745–750.
9. The global dimensions of lead poisoning. Washington, DC:

10. Committee on Toxicology, Assembly of Life Sciences, National Research Council. Recommendations for the prevention of lead poisoning in children. *Nutr Rev.* 1976;34:321–327.
11. Bolger PM, Carrington CD, Capar SG, Adams MA. Reductions in dietary lead exposure in the United States. *Chem Spec Bioavail.* 1991;3:31–36.
12. Jacobs DR, Friedman W, Clickner RP, et al. The prevalence of lead-based paint hazards in US Housing. *Environ Health Perspect.* In press.
13. Sargent JD, Brown MJ, Freeman JL, et al. Childhood lead poisoning in Massachusetts communities: its association with sociodemographic and housing characteristics. *Am J Public Health.* 1995;85:528–534.
14. Lanphear BP, Byrd RS, Auinger P, Schaffer S. Community characteristics associated with elevated blood lead levels in children. *Pediatrics.* 1998;101:264–271.
15. Brown MJ, Shenassa E, Matte TD, Catlin SN. Children in Illinois with elevated blood lead levels, 1993–1998, and lead-related pediatric hospital admissions in Illinois, 1993–1997. *Public Health Rep.* 2000;115:532–536.
16. Lanphear BP, Weitzman M, Eberly S. Racial differences in environmental exposures to lead. *Am J Public Health.* 1996; 86:1460–1463.
17. Lin-Fu JS. Vulnerability of children to lead exposure and toxicity. *N Engl J Med.* 1973;289:1289–1293.
18. Lanphear BP, Hornung R, Ho M, et al. Environmental lead exposure during early childhood. *J Pediatr.* 2002;140:40–47.
19. Mahaffey KR, Gartside PS, Glueck CJ. Blood lead levels and dietary calcium intake in 1- to 11-year-old children: the second National Health and Nutrition Examination Survey, 1976 to 1980. *Pediatrics.* 1986;78:257–262.
20. Blake KCH, Mann M. Effect of calcium and phosphorus on the gastrointestinal absorption of ^{203}Pb in man. *Environ Res.* 1983;30:188–194.
21. Abrams SA, O'Brien KO, Liang LK, Stuff JE. Differences in calcium absorption and kinetics between black and white girls aged 15–16 years. *J Bone Miner Res.* 1995;10:829–833.
22. Clark CS, Bornschein RL, Succop P, et al. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. *Environ Res.* 1985;38: 46–53.
23. Gibson JL. A plea for painted railings and painted rooms as the source of lead poisoning amongst Queensland children. *Australas Med Gazette.* 1904;23:149–153.
24. Turner AJ. Lead poisoning in childhood. *Australas Med Congress (Melbourne).* 1908:2–9.
25. Reich P. *The Hour of Lead: A Brief History of Lead Poisoning in the Untied States,* Washington DC: Environmental Defense Fund; 1992.
26. McKhann CF. Lead poisoning in children. *Am J Dis Child.* 1926;32:386–392.
27. McKhann CF. Lead poisoning in children: the cerebral manifestations. *Arch Neurol Psychiatry.* 1932;27:294–298.
28. Byers RK, Lord EE. Late effects of lead poisoning on mental development. *Am J Dis Child.* 1943:66:471–484.
29. Needleman HL, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med.* 1979;300:689–695.
30. Needleman HL, Schell A, Bellinger D, et al. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. *N Engl J Med.* 1990;322:83–88.
31. Rice DC. Lead-induced changes in learning: evidence for behavioral mechanisms from experimental animal studies. *Neurotoxicology.* 1993;14:167–178.
in monkeys. *Environ Health Perspect.* 2000;108(suppl 3):405–408.
34. Baghurst PA, McMichael AJ, Wigg NR, et al. Environmental exposure to lead and children's intelligence at the age of seven years. *N Engl J Med.* 1992:327:1279–1284.
35. Bellinger DC, Stiles KM, Needleman HL. Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study. *Pediatrics,* 1992;90:855–861.
36. Bellinger D, Sloman J, Leviton A, et al. Low-level lead exposure and children's cognitive function in the preschool years. *Pediatrics.* 1991;87:219–227.
37. Needleman HL, Jackson RJ. Lead toxicity in the 21st century: will we still be treating it? *Pediatrics.* 1992;89:678–680.
38. Dietrich KN, Berger OG, Succop PA, et al. The developmental consequences of low to moderate prenatal and postnatal lead exposure: intellectual attainment in the Cincinnati Lead Study cohort following school entry. *Neurotoxicol Teratol.* 1993;15: 37–44.
39. Factor-Litvak P, Wasserman G, Kline JK, et al. The Yugoslavia prospective study of environmental lead exposure. *Environ Health Perspect.* 1999;107:9–15.
40. Wasserman GA, Liu X, Lolacono NJ, et al. Lead exposure and intelligence in 7-year-old children: the Yugoslavia Prospective Study. *Environ Health Perspect.* 1997;105:956–962.
41. Schnaas L, Rothenberg SJ, Perroni E, et al. Temporal pattern in the effect of postnatal blood lead level on intellectual development of young children. *Neurotoxicol Teratol.* 2000;22: 805–810.
42. Fulton M, Raab G, Thomson G, et al. Influence of blood lead on the ability and attainment of children in Edinburgh. *Lancet.* 1987;1:1221–1226.
43. World Health Organization, International Programme on Chemical Safety. *Environmental Health Criteria 165—Inorganic Lead.* Geneva, Switzerland: WHO; 1995.
44. Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children. *JAMA.* 1990;263:673–678.
45. Centers for Disease Control. *Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control.* Atlanta, Ga: US Dept of Health and Human Services; October 1991.
46. Patterson CC. Contaminated and natural lead environments of man. *Arch Environ Health.* 1965;11:344–360.
47. Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. *Environ Res.* 1994;65:42–55.
48. Schwartz J, Otto D. Lead and minor hearing impairment. *Arch Environ Health.* 1991;46:300–305.
49. Lanphear BP, Dietrich KN, Auinger P, Cox C. Cognitive deficits associated with blood lead levels < 10 μg/dl in US children and adolescents. *Public Health Rep.* 2000;115:521–529.
50. Lanphear BP, Canfield RC, Henderson CR, et al. Environmental lead exposure and children's intelligence at blood lead concentrations below 10 micrograms per deciliter [abstract]. *Pediatr Res.* 2001;49:16A.
51. Rothman KJ, Poole C. A strengthening programme for weak associations. *Int J Epidemiol.* 1988;17:955–959.
52. Bellinger D, Dietrich KN. Low-level lead exposure and cognitive function in children. *Pediatr Ann.* 1994;23:600–605.
53. Moffitt TE. Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychol Rev.* 1993;100:674–701.
54. Moffitt TE. Measuring children's antisocial behaviors. *JAMA.* 1996;275:403–404.
55. Denno D. *Biology and Violence.* New York, NY: Cambridge

58.
59.
60.
61.
62.
63.
64.
65
66
67
68
69
70
7
7
7
7
7

58. Needleman HL, Reiss JA, Tobin MJ, *et al.* Bone lead levels and delinquent behavior. *JAMA.* 1996;275:363–369.
59. Dietrich K, Ris M, Succop P, et al. Early exposure to lead and juvenile delinquency. *Neurotoxicol Teratol.* 2001;23:511–518.
60. Fergusson DM, Fergusson JE, Horwood LJ, Kinzett NG. A longitudinal study of dentine lead levels, intelligence, school performance and behaviour, part I: dentine lead levels and exposure to environmental risk factors. *J Child Psychol Psychiatry.* 1988;29:781–792.
61. Yule Q, Lansdown R, Millar IB, Urbanowicz MA. The relationship between blood lead concentrations, intelligence and attainment in a school population: a pilot study. *Dev Med Child Neurol.* 1981;25:567–576.
62. Silva PA, Hughes P, Williams S, Faed JM. Blood lead, intelligence, reading attainment and behaviour in eleven-year-old children in Dunedin, New Zealand. *J Child Psychiatry.* 1988; 29:43–52.
63. David OJ, Clark J, Voeller K. Lead and hyperactivity. *Lancet.* 1972;2:900–903.
64. Gittleman R, Eskenazi B. Lead and hyperactivity revisited: an investigation of non-disadvantaged children. *Arch Gen Psychiatry.* 1983;40:827–833.
65. Dietrich KN. Environmental chemicals and child development. *J Pediatr.* 1999;134:7–9.
66. Dobbing, J. The later development of the brain and its vulnerability. In: Davis JA, Dobbing J, eds. *Scientific Foundations of Paediatrics.* 2nd ed. London, England: Heinemann; 1981.
67. Ziegler EE, Edwards BB, Jensen RL, et al. Absorption and retention of lead by infants. *Pediatr Res.* 1978;12:29–34.
68. Lanphear BP. The paradox of lead poisoning prevention. *Science.* 1998;281:617–618.
69. Schwartz J. Lead, blood pressure, and cardiovascular disease in men. *Arch Environ Health.* 1995;50:31–37.
70. McDonald JA Potter NU. Lead's legacy? Early and late mortality of 454 lead- poisoned children. *Arch Environ Health.* 1996;51:116–121.
71. Watson GE, Davis BA, Raubertas RF, et al. Influence of maternal lead ingestion on caries in rat pups. *Nat Med.* 1997;3: 1024–1025.
72. Moss ME, Lanphear BP, Auinger P. Association of dental caries and blood lead levels among the US population. *JAMA.* 1999;281:2294–2298.
73. Hertz-Picciotto I. The evidence that lead increases the risk for spontaneous abortion. *Am J Ind Med.* 2000;38:300–309.
74. Borja-Aburto VH, Hertz-Picciotto I, Rojas Lopez M, *et al.* Blood lead levels measured prospectively and risk of spontaneous abortion. *Am J Epidemiol.* 1999;150:590–597.
75. Dietrich KN, Berger OG, Succop PA. Lead exposure and the motor developmental status of urban six-year-old children in the Cincinnati Prospective Study. *Pediatrics.* 1993;91:301–307.
76. Wasserman GA, Musabegovic A, Liu X, et al. Lead exposure and motor functioning in 4(1/2)-year-old children: the Yugoslavia prospective study. *J Pediatr.* 2000;137:555–561.
77. Ballew C, Khan LK, Kaufmann R, et al. Blood lead concentration and children's anthropometric dimensions in the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994. *J Pediatr.* 1999;134:623–630.
78. Schwartz J, Landrigan PJ, Feldman RG, et al. Threshold effect in lead-induced peripheral neuropathy. *J Pediatr.* 1988;112: 12–17.
79. Rogan WJ, Dietrich KN, Ware JH, *et al.* The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. *N Engl J Med.* 2001;344:1421–1426.
80. *Regulatory Impact Analysis of the Proposed Rule on Lead Based Paint in Federally Owned Residential Property.* Washof children in Cincinnati, Ohio. *Chem Spec Bioavail.* 1991;3: 163–171.
82. Sachs HK, Blanksma LA, Murray EF, O'Connell MJ. Ambulatory treatment of lead poisoning: report of 1,155 cases. *Pediatrics.* 1970;46:389–396.
83. McElvaine MD, DeUngria EG, Matte TD, et al. Prevalence of radiographic evidence of paint chip ingestion among children with moderate to severe lead poisoning, St Louis, Missouri, 1989 through 1990. *Pediatrics.* 1992;89:740–742.
84. Shannon MW, Graef JW. Lead intoxication in infancy. *Pediatrics.* 1992;89:87–90.
85. Lanphear BP, Roghmann KJ. Pathways of lead exposure in urban children. *Environ Res.* 1997;74:67–73.
86. Lanphear BP, Weitzman M, Winter NL, et al. Lead-contaminated house dust and urban children's blood lead levels. *Am J Public Health.* 1996;86:1416–1421.
87. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels: a pooled analysis of 12 epidemiologic studies. *Environ Res.* 1998;79:51–68.
88. Sayre JW, Charney E, Vostl J, et al. House and hand dust as a potential source of childhood lead exposure. *Am J Dis Child.* 1974;127:167–170.
89. Charney E, Sayre JW, Coulter M. Increased lead absorption in inner city children: where does the lead come from? *Pediatrics.* 1980;65:226–231.
90. Weitzman M, Aschengrau A, Bellinger D, et al. Lead-contaminated soil abatement and urban children's blood lead levels. *JAMA.* 1993;269:1647–1654.
91. Aschengrau A, Beiser A, Bellinger D, et al. The impact of soil lead abatement on urban children's blood lead levels: phase II results from the Boston Lead-In-Soil Demonstration Project. *Environ Res.* 1994;67:125–148.
92. Centers for Disease Control and Prevention. Children with elevated blood lead levels attributed to home renovation and remodeling activities–New York, 1993–1994. *MMWR Morb Mortal Wkly Rep.* 1997;45:1120–1123.
93. Farfel MR, Chisolm JJ. Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. *Am J Public Health.* 1990;80:1240–1245.
94. Duggan MJ, Inskip MJ. Childhood exposure to lead in surface dust and soil: a community health problem. *Public Health Rev.* 1985;13(1–2):1–54.
95. Landrigan PJ, Gehlbach SH, Rosenblum BF, et al. Epidemic lead absorption near an ore smelter: the role of particulate lead. *N Engl J Med.* 1975;292:123–129.
96. Gulson BL, Davis JJ, Mizon KJ, et al. Lead bioavailability in the environment of children: blood lead levels in children can be elevated in a mining community. *Arch Environ Health.* 1994;49:326–331.
97. US Environmental Protection Agency. Drinking water regulations—maximum contaminant level goals and national primary drinking water regulations for lead and copper; Final Rule. 56. *Federal Register* 26460–26564 (1991).
98. Watt GCM, Britton A, Gilmour WH, et al. Is lead in tap water still a public health problem? An observational study in Glasgow. *BMJ.* 1996;313:979–981.
99. Levin R, Schock MR, Marcus A. Exposure to lead in US drinking water. *Trace Subst Environ Health.* 1989;319–344.
100. Gellert GA, Wagner GA, Maxwell RM, et al. Lead poisoning among low-income children in Orange County, California: a need for regionally differentiated policy. *JAMA.* 1993;270:69–71.
101. Klein M, Namer R, Harpur E, Corbin R. Earthenware containers as a source of fatal lead poisoning. *N Engl J Med.* 1970; 283:669–672.
102. Matte TD, Proops D, Palazuelos E, et al. Acute high-dose lead [illegible] from beverage contaminated by traditional Mexican

the Third World to the First World. *Environ Health Perspect.* 1991;94:121–123.
104. Baker EL, Folland DS, Taylor TA, et al. Lead poisoning in children of lead workers: home contamination with industrial dust. *N Engl J Med.* 1977;296:260–261.
105. Roscoe RJ, Gittleman JL, Deddens JA, et al. Blood lead levels among children of lead-exposed workers: a meta-analysis. *Am J Ind Med.* 1999;36:475–481.
106. Mahaffey KR, Goyer R, Haseman JK. Dose-response to lead ingestion in rats fed low dietary calcium. *J Lab Clin Med.* 1973;82:92–100.
107. Fullmer C. Intestinal calcium and lead absorption: effects of dietary lead and calcium. *Environ Res.* 1991;54:159–169.
108. Wright RO, Shannon MW, Wright RJ, Hu H. Association between iron deficiency and low-level lead poisoning in an urban primary care clinic. *Am J Public Health.* 1999;89:1049–1053.
109. Yip R, Norris TN, Anderson AS. Iron status of children with elevated blood lead concentrations. *J Pediatr.* 1981;98:922–925.
110. Hammad TA, Sexton M, Langenberg P. Relationship between blood lead and dietary iron intake in preschool children: a cross-sectional study. *Ann Epidemiol.* 1996;6:30–33.
111. Sargent JD, Dalton MA, O'Connor GT, et al. Randomized trial of calcium glycerophosphate-supplemented infant formula to prevent lead absorption. *Am J Clin Nutr.* 1999;69:1224–1230.
112. Maddaloni M, Lolacono N, Manton W, et al. Bioavailability of soilborne lead in adults, by stable isotope dilution. *Environ Health Perspect.* 1998;106(suppl):1589–1594.
113. Rabinowitz MB, Kopple JD, Wetherill GW. Effect of food intake and fasting on gastrointestinal lead absorption in humans. *Am J Clin Nutr.* 1980;33:1784–1788.
114. Campbell JR, Schaffer SJ, Szilagyi PG, et al. Blood lead screening practices among US pediatricians. *Pediatrics.* 1996; 98:372–377.
115. Harvey B. Should blood lead screening recommendations be revised? *Pediatrics.* 1994;93:201–204.
116. Centers for Disease Control and Prevention. *Screening Young Children for Lead Poisoning.* Atlanta, Ga: CDC; 1997.
117. Committee on Environmental Health. Screening for elevated blood lead levels. *Pediatrics.* 1998;101:1072–1078.
118. Sachs HK. Effect of a screening program on changing patterns of lead poisoning. *Environ Health Perspect.* 1974;41–45.
119. Haynes E, Lanphear BP, Tohn E, et al. The effect of dust controls on children's blood lead concentrations: a systematic review. *Environ Health Perspect.* 2001;110:103–107.
120. Centers for Disease Control and Prevention. *Managing Elevated Blood Lead Levels Among Young Children: Recommendations From the Advisory Committee on Childhood Lead Poisoning Prevention.* Atlanta, Ga: CDC; 2002.
121. Rich DQ, Rhoads GG, Wartenberg D, Sweatlock J. The effects of home lead abatements on childhood blood lead levels: a retrospective study. *Environ Health.* 2001;2001;9–15.
122. National Center for Lead-Safe Housing. *State Policies and Practices for Case Management and Environmental Investigation for Lead-Poisoned Children.* November 2001.
123. Rose G. *The Strategy of Preventive Medicine.* New York, NY: Oxford Medical Publications, Oxford University Press; 1992.
124. Haan MN, Gerson M, Zishka BA. Identification of children at risk for lead poisoning: an evaluation of routine pediatric blood lead screening in an HMO-insured population. *Pediatrics.* 1996;97:79–83.
125. Binns HJ, LeBailly SA, Fingar AR, Saunders S. Evaluation of risk assessment questions used to target blood lead screening in Illinois. *Pediatrics.* 1999;103:100–106.
126. Schaffer SJ, Szilagyi PG, Weitzman M. Lead poisoning risk determination in an urban population through the use of a standardized questionnaire. *Pediatrics.* 1994;93:159–163.
127. Department of Housing and Urban Development. Lead-based paint hazard elimination in public and Indian housing. 51 *Federal Register* 27774–27809 (1986).
128. US Environmental Protection Agency. Lead: identification of dangerous levels of lead: Final Rule. 66 *Federal Register* 1206–1240 (2001) (codified at 40 CFR §745).
129. Amitai Y, Brown MJ, Graef JW, Cosgrove E. Residential deleading: effects on the blood lead levels of lead-poisoned children. *Pediatrics.* 1991;88:893–897.
130. Rey-Alvarez S, Menke-Hargrave T. Deleading dilemma: pitfall in the management of childhood lead poisoning. *Pediatrics.* 1987;79:214–217.
131. Swindell SL, Charney E, Brown MJ, Delaney J. Home abatement and blood lead changes in children with class III lead poisoning. *Clin Pediatr.* 1994;33:536–541.
132. Aschengrau A, Beiser A, Bellinger D, et al. Residential lead-based-paint hazard remediation and soil lead abatement: their impact among children with mildly elevated blood lead levels. *Am J Public Health.* 1997;87:1698–1702.
133. Chisolm JJ. Removal of lead paint from old housing: the need for a new approach. *Am J Public Health.* 1986;26:236–237.
134. Marino PE, Landrigan PJ, Graef J, et al. A case report of lead paint poisoning during renovation of a Victorian farmhouse. *Am J Public Health.* 1990;80:1183–1185.
135. Charney E, Kessler B, Farfel M, Jackson D. Childhood lead poisoning: a controlled trial of the effect of dust-control measures on blood lead levels. *N Engl J Med.* 1983;309:1089–1093.
136. Staes C, Matte T, Copley CG, et al. Retrospective study of the impact of lead-based paint hazard remediation on children's blood lead levels in St Louis, Missouri. *Am J Epidemiol.* 1994; 139:1016–1026.
137. Farfel MR, Rohde C, Lees PSJ, et al. *Lead-Based Paint Abatement and Repair and Maintenance Study in Baltimore: Findings Based on Two Years of Follow-up.* Washington, DC: US Environmental Protection Agency; 1998.
138. Galke W, Clark S, Wilson J, et al. Evaluation of the HUD lead hazard control grant program. *Environ Res.* 2001;86:149–156.
139. Barltrop D. Children and lead. *Am J Dis Child.* 1974;127:165–166.