POLICY STATEMENT Organizational Principles to Guide and Define the Child Health Care System and/or Improve the Health of all Children

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Prevention of Childhood Lead Toxicity

COUNCIL ON ENVIRONMENTAL HEALTH

abstract

Blood lead concentrations have decreased dramatically in US children over the past 4 decades, but too many children still live in housing with deteriorated lead-based paint and are at risk for lead exposure with resulting lead-associated cognitive impairment and behavioral problems. Evidence continues to accrue that commonly encountered blood lead concentrations, even those below 5 μg/dL (50 ppb), impair cognition; there is no identified threshold or safe level of lead in blood. From 2007 to 2010, approximately 2.6% of preschool children in the United States had a blood lead concentration ≥5 μg/dL (≥50 ppb), which represents about 535 000 US children 1 to 5 years of age. Evidence-based guidance is available for managing increased lead exposure in children, and reducing sources of lead in the environment, including lead in housing, soil, water, and consumer products, has been shown to be cost-beneficial. Primary prevention should be the focus of policy on childhood lead toxicity.

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OVERVIEW AND INTRODUCTION

Primary prevention, reducing or eliminating the myriad sources of lead in the environment of children before exposure occurs, is the most reliable and cost-effective measure to protect children from lead toxicity. Very high blood lead concentrations (eg, >100 μg/dL) can cause significant overt symptoms, such as protracted vomiting and encephalopathy, and even death. Low-level lead exposure, even at blood lead concentrations below 5 μg/dL (50 ppb), is a causal risk factor for diminished intellectual and academic abilities, higher rates of neurobehavioral disorders such as hyperactivity and attention deficits, and lower birth weight in children. No effective treatments ameliorate the permanent developmental effects of lead toxicity. Reducing lead exposure from residential lead hazards, industrial sources, contaminated foods or water, and other consumer products is an effective way to prevent or control childhood lead exposure. Lead poisoning prevention education directed at hand-washing or dust control fails to reduce children's blood lead concentrations. However, pediatricians and parents should be aware of measures to reduce the toxic effects of lead on children, including the promulgation of regulations to screen or test older housing units for lead hazards

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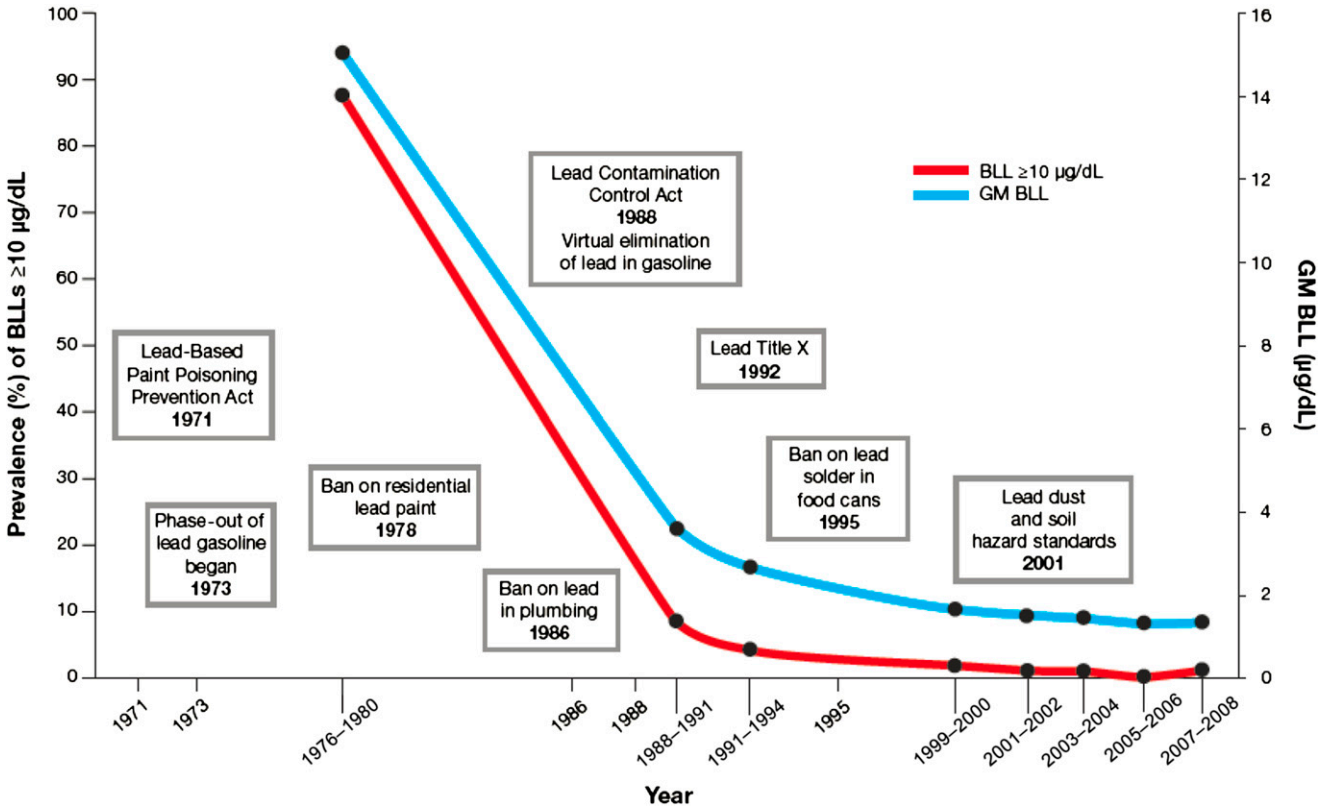

FIGURE 1
Timeline of lead poisoning prevention policies and blood lead levels in children aged 1–5 years, by year—NHANES, United States, 1971–2008. BLL, blood lead level; GM BLL, geometric mean blood lead level. Adapted from Brown et al.[1]

before occupancy and after major renovation and abatement; revision of federal standards to reduce allowable levels of lead in settled house dust, water, soil, cosmetics, and other consumer products; and enhanced protection for children who live in lead-contaminated communities or near lead-emitting industries.

SCOPE OF THE PROBLEM

Over the past 4 decades, blood lead concentrations among US children have declined dramatically since the elimination of lead from gasoline, paints, and other consumer products[1] (Fig 1, Table 1). From 1976 to 1980, blood lead concentrations among US children declined more sharply than anticipated after the phase-out of leaded gasoline.[2] In 1978, the US Consumer Product Safety Commission (CPSC) restricted the allowable content of lead in residential paint to 0.06% (600 ppm); in 2008, it was lowered to 0.009% (90 ppm).[3,4] There have also been significant reductions in tap water lead concentrations since the US Environmental Protection Agency (EPA) promulgated the Lead and Copper Rule.[5,6] Finally, use of lead solder in canned foods and other consumer products was banned. It is difficult to accurately apportion the decline in blood lead concentrations to specific sources, but the combined effect of these regulations clearly led to the dramatic reductions in children's blood lead concentrations.[1] The key to preventing lead toxicity in children is to reduce or eliminate persistent sources of lead exposure in their environment.

Prevention of low-level lead toxicity has historically focused on anticipatory guidance, screening children's blood for lead after exposure, and iron or calcium supplementation to reduce lead absorption.[7] Unfortunately, studies that evaluated the efficacy of parent education or provision of cleaning equipment to families failed to show significant reductions in children's blood lead concentrations.[8] Similarly, calcium and iron supplementation have not consistently been shown to be efficacious in reducing blood lead concentrations of children.[9,10] Collectively, these studies indicate that the focus of prevention should be on reducing the sources of childhood lead exposures rather than identifying children who have already been unduly exposed or attempting to ameliorate the toxic effects of lead exposure.

In 2005, the American Academy of Pediatrics (AAP) recognized that blood lead concentrations below 10 µg/dL (100 ppb) may impair cognition; no threshold for the

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toxic effects of lead was identified.[7] The AAP adopted a blood lead concentration >10 μg/dL (>100 ppb) as the "level of concern" recommended by the Centers for Disease Control and Prevention (CDC), which indicated the need for closer medical and public health management.[7] Extensive and compelling evidence now indicates that lead-associated cognitive deficits and behavioral problems can occur at blood lead concentrations below 5 μg/dL (50 ppb). In 2012, the US National Toxicology Program of the National Institutes of Health reported that, after other risk factors are accounted for, blood lead concentrations <5 μg/dL (<50 ppb) are strongly associated with intellectual deficits, diminished academic abilities, attention deficits, and problem behaviors (Table 2).[11] In that same year, the Advisory Committee on Childhood Lead Poisoning Prevention of the CDC concluded that there is no safe level of lead exposure and adopted the use of a reference value of ≥5 μg/dL (≥50 ppb) (based on the 97.5th percentile of blood lead concentrations from the National Health and Nutrition Examination Survey [NHANES]) to be used as a trigger to guide clinical and public health interventions.[12]

Low-level elevations in children's blood lead concentrations, even at concentrations below 5 μg/dL (50 ppb), can result in decrements in cognitive functions, as measured by IQ scores and academic performance.[13,14] For a given level of exposure, lead-associated IQ decrements are proportionately greater at the lowest blood lead concentrations. The IQ decrement associated with an increase in blood lead concentration from <1 μg/dL (<10 ppb) to 30 μg/dL (300 ppb) was 9.2 IQ points, but the decrement associated with an increase in blood lead concentration from <1 μg/dL (<10 ppb) to 10 μg/dL (100 ppb) was 6.2 IQ points.[14] The population impact of lead on intellectual abilities is substantial. Despite the dramatic reductions in blood lead levels, lead toxicity accounts for an estimated total loss of 23 million IQ points among a 6-year cohort of contemporary US children.[15]

Focusing efforts on children who have blood lead concentrations ≥5 μg/dL (≥50 ppb) is efficient but will fail to preserve the majority of lost IQ points in US children. The *prevention paradox* refers to the concept that most disease or disability occurs in low- to moderate-risk groups. Children who have blood lead concentrations ≥5 μg/dL (≥50 ppb) will, on average, experience

TABLE 1 Federal Lead Poisoning Prevention Policies

Policy or Legislation	Year	Comment
Lead Based Paint Poisoning Prevention Act	1971	First major lead-based paint legislation; addressed lead-based paint in federal housing.
Phase Out Lead in Gasoline	1973	US EPA regulated a phase-out of lead in gasoline.
Ban on Residential Paint	1978	CPSC banned lead paint in residential properties.
Safe Drinking Water Act	1986	US EPA banned use of lead pipes and lead solder in plumbing.
Housing and Community Development Act	1987	Highlighted the danger to children of lead-contaminated dust.
Lead Contamination Control Act	1988	Authorized CDC to make grants to state and local programs to screen children and to provide for education about lead poisoning.
Residential Lead-Based Paint Hazard Reduction Act, Title X	1992	Established primary prevention of lead poisoning as a national strategy.
Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing	1995, 2012	HUD established guidelines for evaluating and controlling residential lead-based paint hazards.
Ban Lead Solder in Food Cans	1995	FDA amended food additive regulations to ban lead solder from food cans.
Lead Safe Housing Rule	1999, 2012	Regulation issued by HUD setting forth new requirements for lead-based paint notification, evaluation, and remediation.
Hazard Standards for Lead in Paint, Dust and Soil	2001	US EPA established a definition of a lead-based paint hazard and standards for paint, dust, and soil in children's play areas.
Consumer Product Safety Improvement Act	2008	CPSC lowered the cap on lead in paint from 0.06% to 0.0009% and incorporated the Lead-Free Toy Act, setting limit on lead content in toys.
Lead Renovation, Repair and Paint Rule	2010	US EPA required contractors working on homes built before 1978 to be certified and follow lead safe guidelines.

TABLE 2 Effects of Low-Level Lead Exposure on Academic and Intellectual Abilities, Puberty, Kidney Function, Postnatal Growth, Hearing, and Other Health Endpoints

Blood Lead Concentration	Evidence Level	Health Effect
<5 μg/dL	Sufficient	Decreased academic achievement
		Lower IQ scores
		Attention-related behavior problems
		Antisocial behaviors
	Limited	Delayed puberty
		Decreased kidney function in children ≥12 y of age
<10 μg/dL	Sufficient	Delayed puberty
		Reduced postnatal growth
		Decreased hearing
	Limited	Hypersensitivity by skin prick test
	Inadequate	Asthma and eczema
		Cardiovascular effects
		Kidney function <12 y of age

From the US Department of Health and Human Services, National Institute of Environmental Health Sciences, 2012.

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a lead-associated IQ deficit of 6.1 points, an IQ deficit much larger than that of children who have lower blood lead concentrations (Fig 2). Still, if the focus is only on reducing exposures for children who have a blood lead concentration ≥5 μg/dL (≥50 ppb), we will fail to preserve more than 20 million (>80% of total) of the 23 million IQ points lost among US children with lower lead exposure because there are so many more children who have low to moderate blood lead concentrations (Fig 2). No therapeutic interventions currently exist for low blood lead concentrations; therefore, prevention of exposure is paramount. For these reasons, this statement focuses heavily on how pediatricians can help *prevent* lead exposure in children.

Elevated blood lead concentrations can result in the development of behavioral problems in children, including inattention, impulsivity, aggression, and hyperactivity.[16–18] In a nationally representative study of 8- to 15-year-old US children, Froehlich et al[17] found that having a blood lead concentration >1.3 μg/dL (>13 ppb) was associated with an elevated risk for attention-deficit/hyperactivity disorder (ADHD). Children with a blood lead concentration in the lowest tertile (<0.7 μg/dL, or <7 ppb) exhibited, on average, 1 symptom of ADHD, whereas children with a blood lead concentration in the highest tertile (>1.3 μg/dL, or >13 ppb) exhibited 3 symptoms. Some critics have argued that these "subtle" shifts in behavioral symptoms are inconsequential, but this shift in the population distribution of ADHD symptoms led to an increase in the percentage of children who met criteria for ADHD from 5% to 13%. Approximately 1 in 5 cases of ADHD among US children have been attributed to lead exposure.[17]

Antisocial behaviors, including conduct disorder, delinquency, and criminal behaviors, can result from

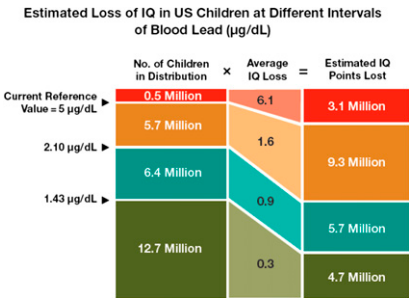

FIGURE 2
Prevention paradox. The majority of IQ points lost due to lead exposure occur in children who have low to moderate blood lead levels. Using the current reference value of 5 μg/dL, we will protect only 3.1 million IQ points (about 13% of the total). Adapted from Bellinger.[15]

a variety of risk factors, but there is substantial evidence that lead toxicity is 1 of the major risk factors for their development.[16,19–22] Needleman et al[16] found that adolescents who had higher bone lead concentrations had higher scores for delinquency and aggression. In a meta-analysis of 16 studies, Marcus et al[22] concluded that lead exposure, measured via blood lead or bone lead concentrations, was a risk factor for conduct disorder. In 2 prospective longitudinal studies, higher childhood blood lead or tooth lead concentrations resulted in higher rates of self-reported delinquent behaviors and arrests or convictions.[20,21] Reyes[23] concluded that the reduction in population mean blood lead concentrations was the major risk factor associated with the decline in severe violent behaviors over the past 3 decades.

Limited evidence implicates lead exposure in diminished kidney function in adolescents at low levels of exposure.[11] Using the NHANES, Fadrowski et al[24] found that, among 769 adolescents with a median blood lead concentration of 1.5 μg/dL (15 ppb), a doubling of the concentration led to a significant reduction in the glomerular filtration rate. It is not clear whether chronic, low-level lead exposure in childhood or adolescence is sufficient to result in chronic renal failure or whether it is the cumulative effect of a variety of risk factors that ultimately results in the development of chronic renal failure. Still, this study is consistent with others linking lead exposure with chronic renal failure in adults.[11]

Lead can cause spontaneous abortion, low birth weight, and reduced growth in children. In a case–control study of pregnant women in Mexico City with blood lead concentrations that ranged from 1.3 μg/dL (13 ppb) to 29 (290 ppb) μg/dL, the odds for spontaneous abortion increased by 1.8 for every 5-μg/dL (50-ppb) increase in maternal blood lead concentration.[25] Early studies that examined the association of prenatal lead exposure and low birth weight or preterm birth, measured via either maternal or cord blood lead concentrations, found inconsistent results. However, in a large cohort involving more than 34 000 live births, investigators found that a 5-μg/dL (50-ppb) increase in blood lead concentrations was associated with a 61-g decrement in birth weight.[26] The National Toxicology Program concluded that maternal blood lead concentrations <5 μg/dL (<50 ppb) are associated with lower birth weight.

PREVENTING LEAD TOXICITY

Despite historical reductions in children's blood lead concentrations, preventing childhood lead toxicity remains a major public health priority in the United States. Many children who live in older, poorly maintained housing or older housing that undergoes renovation are at high risk for lead exposure. In the NHANES conducted from 2007 to 2010, approximately 2.6% of preschool children in the United States had a blood lead concentration ≥5 μg/dL (≥50 ppb), which represents about 535 000 US children 1 to 5 years of age.[12] Children who lived in older housing units experienced an increased risk

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for having a blood lead concentration in excess of 5 μg/dL (50 ppb); 15% of US children who lived in housing units built before 1950 had a blood lead concentration ≥5 μg/dL (≥50 ppb), whereas 4.2% of children who lived in housing built between 1950 and 1978 had a blood lead concentration ≥5 μg/dL (≥50 ppb), compared with 2.1% of children who lived in housing units built after 1978.[27] No treatments have been shown to be effective in ameliorating the permanent developmental effects of lead toxicity.[28] Finally, the economic costs of childhood lead toxicity are substantial. Despite the historical reductions in blood lead concentrations, it has been estimated that the annual cost of childhood lead exposure in the United States is $50 billion.[29] For every $1 invested to reduce lead hazards in housing units, society would benefit by an estimated $17 to $221, a cost–benefit ratio that is comparable with the cost–benefit ratio for childhood vaccines.[30]

The key to preventing lead toxicity in children is identification and elimination of the major sources of lead exposure. Primary prevention of lead exposure is now widely recognized as the optimal strategy because of the irreversible effects of low-level lead toxicity.[7,12] The primary prevention approach contrasts with practices and policies that too often have relied predominantly on detection of lead exposure only after children develop elevated blood lead concentrations.

SOURCES AND VARIABILITY OF LEAD EXPOSURE

Lead ingestion and absorption are dynamic during the first 2 years of life. Blood lead concentrations of children who live in lead-contaminated environments typically increase rapidly between 6 and 12 months of age, peak between 18 and 36 months of age, and then gradually decrease.[31] The peak in children's blood lead concentrations stems from the confluence of normal mouthing behaviors and increasing mobility.[31] Younger children also absorb lead more efficiently than older children and adults.[32] Iron deficiency can also increase the absorption of lead.[33]

A large number of housing units in the United States contain lead-based paint. In a national survey of housing conducted in 2011, it was estimated that 37 million (35%) of 106 million housing units contain lead-based paint.[34] Lead-based paint is the most common, highly concentrated source of lead exposure for children who live in older housing.[35] Paint that was used on both the interior and exterior of houses through the 1950s contained higher concentrations of lead than that of houses built in later years.[34,35] The lead concentration in paint and other media can be measured by using a hand-held instrument called the x-ray fluorescence (XRF) spectrum analyzer or by chemically analyzing paint chips.

The US Department of Housing and Urban Development (HUD) defines lead-based paint as an XRF reading ≥1 μg/cm^2 or 5000 ppm of lead in a paint chip.[36] The presence of *lead*-based *paint* is not as predictive of childhood lead exposure as a *lead paint hazard*. A lead paint hazard is defined by the EPA as "any condition that causes exposure to lead from contaminated dust, lead-contaminated soil, or lead-contaminated paint that is deteriorated, or the presence of accessible (or chewable) surfaces, friction surfaces or impact surfaces that would result in adverse human health effects."[37]

Age of the housing is a major determinant of lead paint hazards. For housing built from 1978 to 1998, 2.7% contained one or more lead paint hazards, whereas the prevalence of residential hazards increased to 11.4% of housing built from 1960 to 1977, 39% of housing built from 1940 to 1959, and 67% of housing units built before 1940.[34] Federal regulations for defining a lead paint hazard in house dust are obsolete. Federal agencies have set environmental lead standards to protect children from having a blood lead concentration ≥10 μg/dL (≥100 ppb), but it is now recognized that there is no safe level of lead exposure. Therefore, because the current standards for lead in house dust, water, and soil remain too high to protect children,[31,38] the percentage of housing that contains one or more lead paint hazards described above is an underestimate.

Lead-based paint is the major source of lead, but ingestions of lead-contaminated house dust and residential soil are the major pathways for exposure (Fig 3).[35–42] House dust, which can be contaminated by small particles of lead-based paint or track-in of lead-contaminated soil, is a major pathway of lead exposure for children who live in older, poorly maintained housing.[40] Ingestions of lead-contaminated house dust and soil are also the primary pathways of exposure for children who live in homes that were recently abated or renovated.[43–45]

Sampling house dust for lead hazards involves using a special wipe to sample a specified area, such as the floor, which is readily accessible to a child, or a window sill or window trough.[36] Windows are often more heavily contaminated than floors because exterior paints often contained higher concentrations of lead, and window troughs can act as reservoirs. Sampling house dust for lead is used to screen older housing units that may contain lead hazards at the time of purchase or rental and before occupancy; to conduct a full risk assessment that involves extensive sampling of settled dust in housing units that failed a lead hazard screen or where there is a high probability of a lead hazard;

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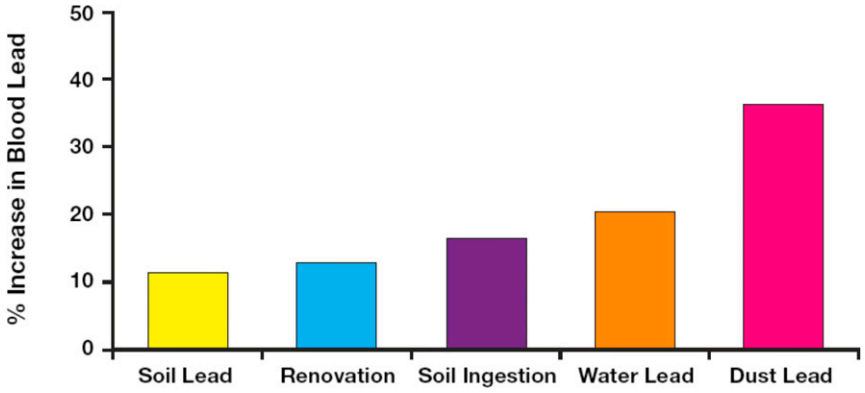

FIGURE 3
Contribution of lead exposure to children's blood lead concentrations. Adapted from Lanphear et al[31] and Spanier et al.[45]

TABLE 3 Common Sources of Lead Exposure

Source	Comment
House paint used before 1978 but especially before 1960	Deteriorated paint releases fine lead dust during home renovation.
Toys and furniture painted before 1976	
Painted toys made outside the United States	
Lead bullets, fishing sinkers, certain weights	Exposures often occur during practice in firing ranges.
Plumbing, pipes, and faucets	Lead leaches into drinking water when the pipes are connected with lead solder.
Soil contaminated by lead	Often in soil near highways and in yard of houses with exterior lead paint.
Hobbies involving soldering such as stained glass, jewelry making, pottery glazing, and miniature lead figures	Always check the labels.
Children's paint sets and art supplies	Always check the labels.
Pewter pitchers and ceramic dinner ware	
Storage batteries	
Parental occupation	Auto repair, mining, battery manufacture, pipe fitting and plumbing, welding, firing range use, ship building, painting, construction.
Folk remedies	Greta and Azarcon, Hispanic traditional medicines; Ghasard, an Indian folk medicine; and Ba-baw-saw, a Chinese herbal remedy, contain lead.
Cosmetics	Examples include Swad brand Sindoor, a cosmetic product used by traditional Hindus; Tiro, an eye cosmetic from Nigeria.
Candy from Mexico	Ingredient tamarind may contain lead.
Toy jewelry	A child died in 2006 after swallowing a metal heart charm that came with a purchase of shoes made by Reebok.

and to conduct clearance testing after repair or renovation of painted surfaces and after lead abatement, to verify that the housing unit is safe for occupancy (Table 3).[38]

Lead-contaminated soil is an important source of lead intake for children.[40,41] Lead-contaminated soil can directly contribute to children's blood lead concentrations via soil ingestion and indirectly from soil tracked indoors on shoes, which then contaminates house dust (Fig 3). Former mine and smelter communities present a particular risk to children for the ingestion of lead-contaminated soil, but lead in urban soil also is often heavily contaminated from the past use of leaded gasoline and paints. Other sources of lead in soil include weathering of lead-based exterior paint and nearby renovation or demolition activity. Soil testing is usually performed in areas where children play and the foundation perimeter. The EPA standards are 400 μg of lead per gram of soil for play areas and 1200 μg/g for the foundation perimeter.[37] Children's blood lead concentrations increase by approximately 3.8 μg/dL (38 ppb) for every 1000-ppm increase in soil lead concentration.[40]

Water is an important but often overlooked source of exposure for children, especially for infants who are formula fed.[5,46,47] Water typically contributes to approximately 20% of a child's blood lead concentrations if the water lead concentration exceeds 5 ppb (Fig 3).[31] The contribution of lead from water can be much higher for some children, especially for infants who ingest large quantities of tap water.[5,46,47] Children who reside in communities with lead service lines and inadequate anticorrosion control are also at increased risk for elevated blood lead concentrations.[48]

Phasing out leaded gasoline and creating stricter national air lead standards led to large reductions in the contribution of airborne lead to children's blood lead concentrations. Still, in some communities, such as those surrounding regional airports, airborne lead is an important source of lead exposure. Airborne lead is ingested primarily after it settles in house dust and soil where children play. Current sources of airborne lead include lead battery recycling operations, piston engine aircraft, and incinerators.[49] The contributions of airborne lead to children's blood lead concentrations are proportionately greater at the lower levels of exposure than at higher levels.[49]

Other sources of lead intake for children have been identified, such as nutritional supplements and folk medicines, ceramic dishware, and cosmetics[50–52] (Table 3).

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Lead brought into the home from a worksite by a parent can also be a major source of exposure for some children.[53] Consumer products such as children's toys, lunch boxes, crayons, and lipstick that are contaminated with lead have received a great deal of attention. These products constitute a small source of lead intake for most children, but they can be the major source for an individual child. Moreover, because lead exposure is cumulative and there is no apparent threshold for the adverse effects of lead exposure, all sources of lead exposure should be eliminated. It is the responsibility of the relevant federal agencies, such as the CPSC and the Food and Drug Administration (FDA), to promulgate and enforce standards that will protect children from lead-contaminated consumer products.

TABLE 4 Federal Standards for Lead in House Paint, House Dust, Soil, Water, Air, and Candy

Source	Standard
1. Lead-based paint (XRF)	1 μg/cm^2
2. Paint containing lead applied after August 14, 2009	90 ppm by wt
3. Testing (full risk assessment) for dust lead hazards (by wipe sampling)	
a. Floors	40 μg/ft^2
b. Interior window sills	200 μg/ft^2
4. Screening test for dust levels (by wipe sampling) to determine whether a full risk assessment is indicated	
a. Floors	25 μg/ft^2
b. Interior window sills	125 μg/ft^2
5. Dust lead clearance levels after abatement (by wipe sampling)	
a. Floors	40 μg/ft^2
b. Interior window sills	250 μg/ft^2
6. Bare residential soil	
a. Children's playground area	400 μg/g
b. Yard other than play area	1200 μg/g
7. Drinking water systems	
Exceeded if lead is above this concentration in >10% of a drinking water system's tap water samples	15 ppb (0.015 mg/L)
8. Candy likely to be consumed by small children	0.1 ppb
9. National Ambient Air Quality Standards: http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_history.html	0.15 μg/m^3

Other state or local standards may vary, and the most protective standard applies. FDA has not set a standard for lead in cosmetics.
1–7, adapted from HUD.[36]
8, from FDA Guidance for Industry, November 2006.

RESIDENTIAL STANDARDS FOR LEAD IN PAINT, DUST, AND WATER

Lead in Paint and Dust

Under section 403 of Title X, the US Congress mandated the EPA to promulgate residential health-based lead standards that are designed to protect children from lead toxicity.[37] Standards are necessary to identify lead hazards before a child is unduly exposed and to identify the source of lead exposure for children who have blood lead concentrations ≥5 μg/dL (≥50 ppb).[31] Unless performed carefully, attempts to reduce lead exposure, such as abatement, repair, or renovation, can result in increased contamination and elevation in a child's blood lead concentration.[43–45] Dust clearance tests, which involve collecting dust from floors or windows of a home by using a lead-free material that resembles a baby wipe, should be conducted after extensive repair, renovation, or abatement of older housing units to determine whether the housing intervention was sufficient to protect children from lead hazards, especially in housing units built before 1960.[27,34] Property owners are required to disclose possible presence of lead-based paint in properties built before 1978 and are required to provide the blue pamphlet from the EPA, HUD, and Consumer Product Safety Commission titled "Protect Your Family From Lead in Your Home" at the time of rental or sale.

Most existing lead standards fail to protect children (Table 4). In 1978, the CPSC set the maximum paint lead concentration at 0.06% (600 ppm), because there was evidence that paint could be manufactured with this lower level of contamination.[3] Similarly, the EPA's action level of 15 ppb of lead in water, which is used to regulate water systems in the United States, is routinely (but erroneously) used as a health-based standard; it was not intended as a health-based standard, nor does it adequately protect children or pregnant women from adverse effects of lead exposure.[5,31] In 1988, the HUD established a postabatement floor dust standard of 200 μg/ft^2 because there was evidence that it was feasible to attain, not because it was demonstrated to be safe or protective. In 2001, the EPA promulgated residential lead standards of 40 μg/ft^2 for floors and 250 μg/ft^2 for window sills.[37] Unfortunately, these standards, which failed to protect children from having a blood lead concentration ≥10 μg/dL (≥100 ppb) when they were first promulgated, dictate the levels of lead contamination considered "normal" or "low," and they provide an illusion of safety.[38,40] At a floor standard of 40 μg/ft^2, the current EPA standard for floors, 50% of children were estimated to have a blood lead concentration ≥5 μg/dL (≥50 ppb); 5% of children have a blood lead concentration ≥5 μg/dL (≥50 ppb) at a median floor dust lead level of 1.5 μg/ft^2 (Fig 4).[42]

Scraping, sanding, or construction during painting, repair, renovation, or abatement of older housing can result in lead contamination of a child's environment.[41,43–45,54] In a controlled study of children with baseline blood lead concentrations

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<22 μg/dL (<220 ppb), Aschengrau et al[41] reported a 6.5-μg/dL (65-ppb) *increase* in blood lead concentrations for children whose homes had undergone paint abatement. Clark et al[44] reported that 6-month-old infants were 11 times more likely to have a ≥5 μg/dL (≥50 ppb) increase in blood lead concentrations after abatement compared with older children. Spanier et al[45] reported that routine renovation of older housing was associated with a 12% higher mean blood lead concentration. These studies indicate that the levels of lead-contaminated dust generated by lead hazard control work or housing renovations can result in excessive lead exposure and absorption for children unless there is sufficient cleanup and clearance testing after the work is completed. The HUD has published technical guidelines and regulations for workers involved in lead-based paint abatement or remediation of housing.[36]

In 1992, the US Congress mandated the EPA to promulgate regulations to protect children from lead exposure resulting from housing repairs and renovation.[37] In 2011, the EPA finalized recommendations for the Lead Renovation, Repair and Painting Rule.[54] Unfortunately, the EPA failed to recommend the validated wipe-sampling method for clearance testing. Instead, it used an unvalidated cloth test, which should not be confused with the validated wipe sampling test. The white cloth test assumes that if dust is visible on a white cloth (ie, the "white glove test"), it contains a lead hazard; conversely, if there is no visible dust, it does not contain a lead hazard.[54] Although it would be valuable to have a quick test to identify the presence of a lead hazard, the white cloth test is not a validated tool and is not a reliable way to quantify the presence of a lead hazard.

Lead hazard control work can result in sizable reductions in the magnitude of dust lead loading when proper procedures are followed and cleanup and postwork clearance testing are performed. In 1 study, dust lead levels (measured as micrograms of lead per area) immediately after professional abatement were 8.5 μg/ft^2, 8.0 μg/ft^2, and 21 μg/ft^2 for floors, interior window sills, and window troughs, respectively, representing reductions of more than 80% compared with preabatement levels.[55] In another study of more than 2600 housing units, postabatement dust lead levels were 12 μg/ft^2, 31 μg/ft^2, and 32 μg/ft^2 for floors, window sills and window troughs, respectively.[56] These levels were achieved with dust clearance testing set at 100 μg/ft^2 or higher, but floor dust lead levels below 5 μg/ft^2 can be achieved by following a specific protocol. In 1 unpublished study of more than 160 housing units built before 1978, 1 group found that it is possible to routinely meet floor lead levels below 5 μg/ft^2 after housing renovations costing an average of $5600 (B. Lanphear, MD, MPH, Simon Fraser University, unpublished data).

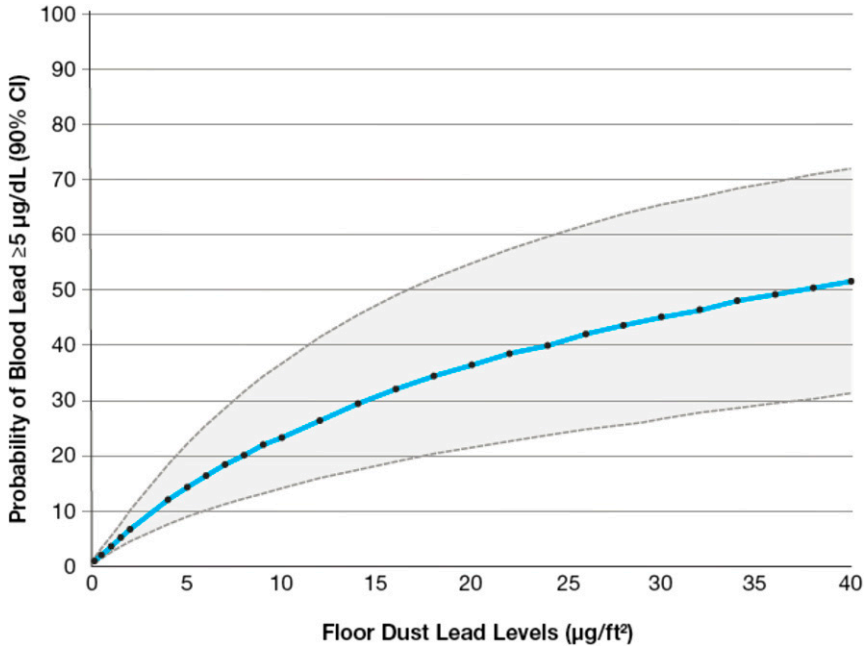

FIGURE 4
Estimated probability of blood lead concentrations ≥5 μg/dL for children living in pre-1978 housing by floor dust lead level, NHANES, 1999–2004. CI, confidence interval. Adapted from Dixon et al.[42]

Lead in Water

The primary sources of lead in water, which can be dissolved or particulate, consist of lead service lines, lead solder, and brass fittings that contain high concentrations of lead.[5] Plumbing installed before 1986, the year a federal ban was issued on using lead pipe and lead solder and a maximum lead content of 8% by weight for brass plumbing was established, is more likely to contain higher concentrations of lead.[5] Lead services lines that are being replaced, are undergoing maintenance, or are damaged can release particles of lead that can be ingested.[57] Partial service line replacement, which is sometimes performed to minimize the cost of service line repair by water authorities, fails to reduce lead exposure.[57] Proper maintenance and ultimately full replacement of water service lines will be necessary to eliminate lead intake from water, but it must be performed with proper precautions. In the interim,

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water filters that are certified by the National Sanitation Foundation for lead removal can effectively reduce water lead concentrations. The EPA recommends running the cold water of residential units for up to 2 minutes to flush the lead leached from pipes out of the plumbing system, but flushing is useful only in housing units without lead service lines.[58–61] In housing units without lead service lines, and where the primary source is brass fittings or lead-soldered joints, a 1-minute flush may be sufficient, depending on the length of plumbing; for housing units with lead service lines, flushing may *increase* lead exposure, again depending on the length of the lead service lines.[58–61]

Drinking fountains in older schools can be an important source of lead exposure.[5] Unfortunately, there are no regulations for evaluating lead contamination of school drinking fountains in most states.

Implementation of the Lead and Copper Rule has significantly reduced tap water lead levels. In 1991, the US EPA set an action level for lead in water of 15 μg/L or (15 ppb).[6] Communities in which >10% of water samples taken from various taps throughout the system exceed 15 ppb are considered to be out of compliance and are required by the EPA to take action to reduce lead levels using corrosion control methods or replacement of lead service lines. The action level is used as an administrative tool to evaluate community-level exposure; it is not a health-based standard. The maximum contaminant level goal, the value the EPA deems acceptable for health, is 0.

Testing Asymptomatic Children for Elevated Blood Lead Concentrations

In the primary care office, primary prevention begins with education and counseling. Ideally, environmental assessments, such as screening older housing units, occurs before a child is born so that parents can identify and hire trained workers to abate environmental lead exposure hazards.[12] It is especially important to conduct an environmental assessment for lead if a family resides in a housing unit built before 1960 that has undergone recent renovation, repair, or painting or if it is poorly maintained.

Screening questionnaires frequently used in the primary care setting fail to identify children who have elevated blood lead concentrations,[62] but they may be useful as a tool to identify lead hazards in children who have a blood lead concentration ≥5 μg/dL (≥50 ppb). In addition, public health agencies often use other methods of targeting children who should be screened with a blood lead test on the basis of community and residential characteristics, such as older housing. Blood lead surveillance data can be used to identify cities, communities, or housing units at higher than typical risk for lead poisoning. Technologies using geographic information system–based analyses and surveillance from electronic medical records are important tools to identify at-risk children who should have their blood lead concentration measured.

In 1991, the CDC recommended universal blood lead testing for all children.[63] In 2005, the AAP recommended that states and cities formulate their own lead screening recommendations on the basis of local data because of the wide variation in lead exposure.[7] The AAP, consistent with the CDC, recommended universal screening of children's blood for lead if they lived in communities with more than 27% of housing built before 1950 or a prevalence of blood lead concentrations ≥10 μg/dL in children 12 to 36 months old of 12% or greater.[7,12,63,64] Screening is not efficient after 36 months of age unless specific high-risk factors are identified; the likelihood of a child having a blood lead concentration >10 μg/dL after 36 months of age is low.[65] These recommendations now need to be updated to conform to with our new understanding of lead toxicity.[11,12]

A detailed evaluation and follow-up of children who have blood lead concentrations <10 μg/dL (<100 ppb) is now indicated. Current federal regulations for clinical laboratory testing through the Clinical Laboratory Improvement Amendments of 1988[66] permit an allowable laboratory error in blood lead proficiency testing programs of ±4 μg/dL (±40 ppb) for blood lead concentrations ≤20 μg/dL (≤200 ppb). This range of error can result in children being misclassified and cause additional anxiety or false comfort when blood lead concentrations within the margin of error erroneously are interpreted as going up or down. The majority of laboratories analyzing blood lead reference materials routinely achieved laboratory error of ±2 μg/dL (±20 ppb) at blood lead concentrations ≤20 μg/dL (≤200 ppb).[67] Changing the allowable laboratory error to tighter performance requirements, such as ±2 μg/dL (±20 ppb), could decrease misclassification of children and lead to better allocation of health care resources.

Case Management of Children With a Blood Lead Concentration at or Above Reference Value

The AAP is adopting the current reference value of ≥5 μg/dL (≥50 ppb) for case management.[12] The CDC recommended that the 97.5th percentile of blood lead concentrations derived from the combination of the 2 most recent cycles of NHANES data be used to identify children who have unacceptably high exposure and to set public health goals.[12] The CDC will reconsider the reference value for children's blood lead concentrations every 4 years.[12]

After confirmatory testing, it is important to monitor children who have blood lead concentrations

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TABLE 5 AAP Recommendations on Management of Childhood Lead Exposure and Poisoning

Lead Level	Recommendation
<5 μg/dL (<50 ppb)	1. Review laboratory results with family. For reference, the geometric mean blood lead concentration for US children 1–5 y old is <2 μg/dL (<20 ppb); 2.5% have a blood lead concentration ≥5 μg/dL (≥50 ppb). 2. Repeat the blood lead concentration in 6–12 mo if the child is at high risk for lead exposure or if risk profile increases. Follow all local and state lead screening recommendations. 3. For children initially screened before 12 mo of age, consider retesting in 3–6 mo for children at high risk; lead exposure may increase as mobility increases. 4. Perform routine assessment of nutrition and physical and mental development and assess risk factors for iron deficiency. 5. Provide anticipatory guidance about common sources of environmental lead exposure: paint in homes or child care facilities built before 1960, soil near roadways, take-home exposures related to adult occupations, and imported spices, cosmetics, folk remedies, and cookware.
5–14 μg/dL (50–140 ppb)	1. Perform steps as described above for blood lead concentrations <5 μg/dL (<50 ppb). 2. Retest venous blood lead concentration within 1–3 mo to verify that the lead concentration is not rising. If it is stable or decreasing, retest the blood lead concentration in 3 mo. Refer patient to local health authorities if such resources are available. Most states require elevated blood lead concentrations be reported to the state health department. Contact the CDC at 800-CDC-INFO (800-232-4636) or www.cdc.gov/nceh/lead or the National Lead Information Center at 800-424-LEAD (5323) for resources regarding lead poisoning prevention and local childhood lead poisoning prevention programs. 3. Take a careful environmental history to identify potential sources of exposures (see #5 above) and provide preliminary advice about reducing or eliminating exposures. Take care to consider other children who may be exposed. 4. Provide nutritional counseling related to calcium and iron. Encourage the consumption of iron-enriched foods (eg, cereals, meats). Encourage families to sign up for the Special Supplemental Nutrition Program for Women, Infants, and Children, if eligible. 5. Screen for iron sufficiency with adequate laboratory testing (complete blood cell count, ferritin, C-reactive protein) and provide treatment per AAP guidelines. Consider starting a multivitamin with iron. 6. Perform structured developmental screening evaluations at child health maintenance visits, because lead's effect on development may manifest over years.
15–44 μg/dL (150–440 ppb)	1. Perform steps as described above for blood lead concentrations 5–14 μg/dL (50–140 ppb). 2. Confirm the blood lead concentration with repeat venous sample within 1–4 wk. 3. Abdominal radiography should be considered for children who have a history of pica for paint chips or excessive mouthing behaviors. Gut decontamination may be considered if leaded foreign bodies are visualized on radiography. Any treatment of blood lead concentrations in this range should be provided in consultation with an expert. Contact local pediatric environmental health specialty unit (www.pehsu.net or 888-347-2632) or local or regional Poison Control Center (www.aapcc.org or 800-222-1222) for guidance.
>44 μg/dL (>440 ppb)	1. Follow guidance for blood lead level 15–44 μg/dL (150–440 ppb) as listed above. 2. Confirm the blood lead concentration with repeat venous lead level within 48 h. 3. Consider hospitalization or chelation therapy (managed with the assistance of an experienced provider). Safety of the home or child care facility with respect to lead hazards, isolation of the lead source, family social situation, and chronicity of the exposure are factors that may influence management. Contact your regional pediatric environmental health specialty unit or Poison Control Center or the CDC for assistance.

Modified from Pediatric Environmental Health Specialty Unit. Medical Management of Childhood Lead Exposure and Poisoning (http://www.pehsu.net/_Library/facts/medical-mgmnt-childhood-lead-exposure-June-2013.pdf).

≥5 μg/dL (≥50 ppb). The pediatrician should inform the local or state health department and request an inspection of the child's house to identify and remediate any lead hazards (Table 4). Screening children for iron deficiency and insufficient dietary calcium intake is also important.[7] A detailed description of the diagnosis and treatment of significant lead toxicity (ie, ≥45 μg/dL [≥450 ppb]) is beyond the scope of this policy statement, but guidance is available in an earlier publication of the AAP[7] and through the Pediatric Environmental Health Specialty Units Web site (www.pehsu.net) (Table 5). Children who have elevated blood lead concentrations need to be monitored until environmental investigations and remediation are complete and blood lead concentrations decline.[12]

The AAP recognizes that environmental investigations will typically be conducted by local or state health or environmental departments to identify sources of lead exposure for a child who has a blood lead concentration ≥5 μg/dL (≥50 ppb). In many cases, however, the pediatrician can provide clues about possible sources of lead intake by taking a careful history.

Case management involves a thorough investigation of potential sources of lead poisoning in a child's environment, including paint, house dust, water, and soil. Case management also includes a questionnaire and visual inspection for other potential sources of lead exposure, including antique furniture, toys, ethnic folk remedies, and consumer products such as imported food, cosmetics, and ceramics.[12,50–52] It can include testing deteriorated paint on furniture, such as

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a crib, taking dust samples from child care settings or a family member's house, and taking soil samples from a child's play area.

SUMMARY AND RECOMMENDATIONS

Lead toxicity results in substantial, population-level effects on children's intellectual abilities, academic abilities, problem behaviors, and birth weight. Pediatricians may be well equipped to advocate for more stringent regulations to reduce sources of lead exposure and prevent childhood lead exposure. The AAP recognizes the importance of a variety of educational, enforcement, and environmental actions to reduce the number of children who are exposed to lead hazards and concur with recent detailed recommendations for prioritization of primary prevention of lead toxicity.[7,12,68–70] The AAP offers the following recommendations for government as well as pediatricians, other health care providers, and public health officials.

Recommendations for Government

1. The federal government should expand the resources currently offered by the HUD to local and state governments for lead hazard control work.

2. The federal government should provide both financial and nonfinancial resources and technical guidance through the CDC, the EPA, and the HUD to state and local public health agencies as well as environmental and housing agencies engaged in childhood lead poisoning prevention efforts.

3. The US EPA and HUD should review their protocols for identifying and mitigating residential lead hazards (eg, lead-based paint, dust, and soil) and lead-contaminated water from lead service lines or lead solder and revise downward the allowable levels of lead in house dust, soil, paint, and water to conform with the recognition that there are no safe levels of lead.

4. The federal government should resume and expand its vital role in providing federal public health leadership in childhood lead poisoning prevention work through the CDC. Allocation of additional resources would be necessary to accomplish this goal.

5. The Centers for Medicare & Medicaid Services, which is responsible for regulating clinical laboratory testing through the Clinical Laboratory Improvement Amendments of 1988,[69] should expeditiously revise current regulations for allowable laboratory error permitted in blood lead proficiency testing programs from ±4 μg/dL (±40 ppb) to ±2 μg/dL (±20 ppb) for blood lead concentrations ≤20 μg/dL (≤200 ppb).[12] In the future, when feasible, allowable laboratory error permitted in blood lead proficiency testing programs should be reduced even more, to ±1 μg/dL (±10 ppb) for blood lead concentrations ≤20 μg/dL (≤200 ppb).

6. The federal government should continue to conduct the NHANES and provide national data on trends in blood lead concentrations. These newer data should be used by the CDC to periodically formulate a new reference value and guide clinical and public health interventions.

7. The federal government should continue to regularly survey children and adolescents in the NHANES for ADHD and conduct disorder by using validated diagnostic surveys from the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* to examine the association of lower blood lead concentrations with these conditions.

8. Local or state governments, in consultation with pediatricians, should develop policies and regulations requiring the remediation of lead-contaminated housing and child care facilities, including the elimination of lead hazards during transfer of rental units or renovation or demolition of older housing.

9. State and local governments should collect, analyze, and publish blood lead test results performed in their jurisdictions and should regularly publish reports of age of housing and other risk factors for children having blood lead concentrations ≥5 μg/dL (≥50 ppb). These reports should be readily available to pediatricians, health care providers, and the public.

10. Federal, state, and local governments should provide resources for environmental evaluations and case management of children who have blood lead concentrations ≥5 μg/dL (≥50 ppb), in conjunction with the child's primary care provider.

11. State and local governments should take steps to ensure that water fountains in schools do not exceed water lead concentrations of 1 ppb.

Recommendations for Pediatricians, Health Care Providers, and Public Health Officials

1. Pediatricians are in a unique position to work with public health officials to conduct surveys of blood lead concentrations among a randomly selected,

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representative sample of children in their states or communities at regular intervals to identify trends in blood lead concentrations. These periodic surveys are especially important for children who live in highly contaminated communities, such as smelter communities or regions with a historically high prevalence of lead exposure.

2. Pediatricians, heath care providers, and public health officials should routinely recommend individual environmental assessments of older housing,[12] particularly if a family resides in a housing unit built before 1960 that has undergone recent renovation, repair, or painting or that has been poorly maintained.

3. Pediatricians and public health officials should advocate for the promulgation and enforcement of strict legal standards based on empirical data that regulate allowable levels of lead in air, water, soil, house dust, and consumer products. These standards should address the major sources of lead exposure, including industrial emissions, lead paint in older housing, lead-contaminated soil, water service lines, and consumer products.

4. Pediatricians should be familiar with collection and interpretation of reports of lead hazards found in house dust, soil, paint, and water, or they should be able to refer families to a pediatrician, health care provider, or specialist who is familiar with these tools.

5. Pediatricians, women's health care providers, and public health officials should be familiar with federal, state, local, and professional recommendations or requirements for screening children and pregnant women for lead poisoning.[12,68,69]

6. Pediatricians and other primary care providers should test asymptomatic children for elevated blood lead concentrations according to federal, local, and state requirements. Immigrant, refugee, and internationally adopted children also should be tested for blood lead concentrations when they arrive in the United States because of their increased risk.[71,72] Blood lead tests do not need to be duplicated, but the pediatrician or other primary care provider should attempt to verify that screening was performed elsewhere and determine the result before testing is deferred during the office visit.

7. Pediatricians and other primary care health providers should conduct targeted screening of children for elevated blood lead concentrations if they are 12 to 24 months of age and live in communities or census block groups with ≥25% of housing built before 1960 or a prevalence of children's blood lead concentrations ≥5 μg/dL (≥50 ppb) of ≥5%.

8. Pediatricians and other primary care providers should test children for elevated blood lead concentrations if they live in or visit a home or child care facility with an identified lead hazard or a home built before 1960 that is in poor repair or was renovated in the past 6 months.[7,12]

9. Pediatricians and primary care providers should work with their federal, state, and local governments to ensure that a comprehensive environmental inspection is conducted in the housing units of children who have blood lead concentrations ≥5 μg/dL (≥50 ppb) and that they receive appropriate case management.

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ABBREVIATIONS

AAP: American Academy of Pediatrics
ADHD: attention-deficit/hyperactivity disorder
CDC: Centers for Disease Control and Prevention
CPSC: Consumer Product Safety Commission
EPA: Environmental Protection Agency
FDA: US Food and Drug Administration
HUD: Department of Housing and Urban Development
NHANES: National Health and Nutrition Examination Survey
XRF: x-ray fluorescence

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