PEDIATRICS®
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Interpreting and Managing Blood Lead Levels of Less Than 10 µg/dL in Children and Reducing Childhood Exposure to Lead: Recommendations of the Centers for Disease Control and Prevention Advisory Committee on Childhood Lead Poisoning Prevention

Helen J. Binns, Carla Campbell, Mary Jean Brown and for the Advisory Committee on Childhood Lead Poisoning Prevention

Pediatrics 2007;120;e1285-e1298
DOI: 10.1542/peds.2005-1770

SPECIAL ARTICLE

Interpreting and Managing Blood Lead Levels of Less Than 10 μg/dL in Children and Reducing Childhood Exposure to Lead: Recommendations of the Centers for Disease Control and Prevention Advisory Committee on Childhood Lead Poisoning Prevention

Helen J. Binns, MD[a], Carla Campbell, MD[b], Mary Jean Brown, ScD, RN[c], for the Advisory Committee on Childhood Lead Poisoning Prevention

[a] Feinberg School of Medicine, Northwestern University, Chicago, Illinois; [b]Children's Hospital of Philadelphia, Philadelphia, Pennsylvania; [c] Division of Environmental and Emergency Health Services, National Center for Environmental Health/Agency for Toxic Substances and Disease Registry, Center for Disease Control and Prevention, Atlanta, Georgia

The authors have indicated they have no financial relationships relevant to this article to disclose.

ABSTRACT

Lead is a common environmental contaminant. Lead exposure is a preventable risk that exists in all areas of the United States. In children, lead is associated with impaired cognitive, motor, behavioral, and physical abilities. In 1991, the Centers for Disease Control and Prevention defined the blood lead level that should prompt public health actions as 10 μg/dL. Concurrently, the Centers for Disease Control and Prevention also recognized that a blood lead level of 10 μg/dL did not define a threshold for the harmful effects of lead. Research conducted since 1991 has strengthened the evidence that children's physical and mental development can be affected at blood lead levels of <10 μg/dL. In this report we provide information to help clinicians understand blood lead levels < 10 μg/dL, identify gaps in knowledge concerning lead levels in this range, and outline strategies to reduce childhood exposures to lead. We also summarize scientific data relevant to counseling, blood lead screening, and lead-exposure risk assessment. To aid in the interpretation of blood lead levels, clinicians should understand the laboratory error range for blood lead values and, if possible, select a laboratory that achieves routine performance within ±2 μg/dL. Clinicians should obtain an environmental history on all children they examine, provide families with lead-prevention counseling, and follow blood lead screening recommendations established for their areas. As circumstances permit, clinicians should consider referral to developmental programs for children at high risk for exposure to lead and more frequent rescreening of children with blood lead levels approaching 10 μg/dL. In addition, clinicians should direct parents to agencies and sources of information that will help them establish a lead-safe environment for their children. For these preventive strategies to succeed, partnerships between health care providers, families, and local public health and housing programs should be strengthened.

www.pediatrics.org/cgi/doi/10.1542/peds.2005-1770

doi:10.1542/peds.2005-1770

Drs Brown, Campbell, and Binns contributed equally to this work.

The material in this report originated in the Division of Environmental and Emergency Health Services, National Center for Environmental Health/Agency for Toxic Substances and Disease Registry.

Key Words
blood lead levels, lead poisoning, screening, prevention

Abbreviations
BLL—blood lead level
CDC—Centers for Disease Control and Prevention
ACCLPP—Advisory Committee on Childhood Lead Poisoning Prevention
EPA—Environmental Protection Agency

Accepted for publication Apr 30, 2007

Address correspondence to Mary Jean Brown, ScD, RN, Division of Environmental and Emergency Health Services, National Center for Environmental Health/Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention, Atlanta, GA 30341. E-mail: mbrown6@cdc.gov

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275); published in the public domain by the American Academy of Pediatrics

Lead is a common environmental contaminant, and exposure to lead is a preventable risk in all areas of the United States. Lead is associated with negative outcomes for children, including impaired cognitive, motor, behavioral, and physical abilities.[1–4] In 1991, the Centers for Disease Control and Prevention (CDC) defined the blood lead level (BLL) that should prompt public health actions as 10 μg/dL.[5] Concurrently, the CDC recognized that a BLL of 10 μg/dL did not define a threshold for the harmful effects of lead. Research conducted since 1991 has strengthened the evidence that the physical and mental development of children can be affected at BLLs of <10 μg/dL.[1,3]

In 2002 to 2004, a workgroup of the CDC Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) reviewed the scientific literature regarding adverse health effects associated with BLLs of <10 μg/dL, including 23 published reports that analyzed 16 separate populations with IQ or general cognitive index outcomes and 12 publications related to other health outcomes. In its 2005 report, the workgroup concluded that an inverse association exists between BLLs and cognitive function, with no evidence of a weaker association in populations with lower BLLs.[1] The direct evidence for this inverse association was strongest in a study conducted in Rochester, New York, which included children who were born in 1994 or 1995, enrolled at 6 months of age, and monitored for 5 years.[6] The majority of children studied had BLLs of <10 μg/dL throughout the study period. The IQ/BLL relationship was described most accurately by a nonlinear negative association, with a decrease in IQ of >7 points over the first 10 μg/dL increase in lifetime average BLL. On the basis of the evidence, the workgroup concluded that a causal association between lead exposure and impaired cognitive functioning was most likely. However, the potential for residual confounding, particularly by social factors, made the strength and shape (ie, linear or nonlinear) of this association across BLLs uncertain. In addition, the workgroup concluded that children with BLLs of <10 μg/dL should not be classified as "lead poisoned." The report noted that no safe BLL in children has been identified.[1]

Two studies published subsequently reported negative effects of BLLs of <10 μg/dL on developmental outcomes.[7,8] One study, which included participants from the Rochester cohort[6] and from 6 other prospective studies of children with peak BLLs across a range of values, reaffirmed an inverse association between low BLLs and IQ.[7] Those studies accounted for key potential confounders, including maternal IQ, Home Observation for Measurement of the Environment Inventory score (which is a measure of the quality and quantity of stimulation and support available to a child in the home environment), maternal education, and birth weight.

Although the ACCLPP previously reviewed case management for children with BLLs of ≥10 μg/dL,[2] this is the first ACCLPP report to summarize scientific information relevant to clinical management for children with BLLs of <10 μg/dL. This report also outlines recommendations from the ACCLPP to reduce childhood exposure to lead. Information on assessments of environmental history and prevention strategies to decrease exposure to lead was published previously[2,3] and is not included in this report.

METHODS

The ACCLPP provides advice and guidance to the US Department of Health and Human Services and the CDC regarding new scientific knowledge and technological developments and their practical implications for preventing childhood lead poisoning and recommends improvements as needed. ACCLPP members are selected on the basis of their expertise in childhood lead poisoning prevention, screening, diagnosis, and medical management. ACCLPP liaisons represent federal agencies and organizations with particular interest and expertise in childhood lead poisoning prevention.

In October 2003, the ACCLPP formed another workgroup, consisting of 3 pediatricians and a CDC health scientist, to review the scientific literature regarding clinical management options for BLLs of <10 μg/dL and to outline recommendations for clinical care providers. On the basis of its analysis, the workgroup developed draft recommendations that were reviewed and then adopted by the ACCLPP in February 2006.

RESULTS

Historic Trends in Children's BLLs in the United States

Since 1976, BLLs in US children 1 to 5 years of age have decreased substantially (Table 1), primarily as a result of policies that have reduced the dispersal of lead into the environment.[9–12] However, many US children continue to be exposed to lead, primarily in their homes.[13] Overt clinical symptoms of lead intoxication are uncommon in the United States, and lead evaluation and management strategies typically are intended to reduce the negative effects of lead on central nervous system development in children who are clinically asymptomatic. Because no safe BLL has been defined,[1] small reductions in population-level exposures to lead would likely affect substantial numbers of children and could be expected to reduce

TABLE 1 **BLLs of US Children 1 to 5 Years of Age**

Year	Proportion With BLLs of ≥10 μg/dL, %	Geometric Mean BLL, μg/dL
1976–1980[10]	88.2	15.0
1991–1994[11]	4.4	2.7
1999–2002[12]	1.6	1.9

the number of children with adverse health outcomes associated with lead exposure.[14]

BLL Measurements

As with any biological test, BLL measurements have inherent uncertainties resulting from imprecise analytic techniques and preanalytic variables (eg, the specimen collection process). However, the imprecision/measurement value ratio, particularly at BLLs of <10 μg/dL, is relatively high. The degree of inherent error in BLL analyses varies according to the analytic method used but, whichever method is used, laboratory performance depends on the procedures and skills of the laboratory team.[15,16] Federal regulations allow laboratories that perform BLL testing to operate with a total allowable error of ±4 μg/dL or ±10%, whichever is greater. Consequently, at BLLs of ≤10 μg/dL, a laboratory might operate within an error range of 8 μg/dL and still meet federal proficiency standards. For example, an actual BLL of 7 μg/dL could be reported as any value ranging from 3 to 11 μg/dL and still remain within the allowable error limit. A study of duplicate testing of identical blood samples (all with mean BLLs of <10 μg/dL) at 8 laboratories reported all results as <10 μg/dL and within 3 μg/dL of the overall mean for that specimen value.[17] A study conducted in 2001 indicated that the majority of BLL laboratories can achieve routine performance of ±2 μg/dL at concentrations of ≤10 μg/dL without difficulty.[18]

BLL test reliability also depends on adherence to blood collection techniques that reduce sample contamination. Collection of capillary blood from a finger-stick into a lead-free collection device is an accepted method for obtaining a screening test sample,[19–23] and contamination by lead from the skin surface can be minimized if a protocol for proper capillary specimen collection is followed. (A complimentary videotape or DVD, *CDC Guidelines for Collecting and Handling Blood Lead Samples: 2004*, may be obtained from the National Center for Environmental Health, Division of Laboratory Sciences, Lead and Multielement Proficiency Program.) However, because BLLs determined from capillary blood samples vary from those determined from simultaneously drawn venous samples, elevated capillary BLL results should be confirmed with blood samples drawn through venipuncture. Multiple studies have reported on the uncertainty introduced through collection of capillary blood samples, rather than samples obtained through venipuncture, at thresholds of 10 or 15 μg/dL,[19–23] but none has examined the sensitivity or specificity of capillary blood sample collection methods at thresholds of <10 μg/dL.

Children's BLL Patterns

BLLs increase quickly after acute exposure and then gradually (over weeks) reach equilibrium with body stores of lead. Lead is distributed unevenly within the human body; in children, ~70% is stored in the bone compartment.[24–26] The residence time of lead in bone can be decades.[27] Therefore, elevated BLLs decline within a few weeks to months after acute exposure. However, for children with chronic lead exposure and presumably greater bone lead stores, the decline in BLLs can take much longer.[28] Although bone lead levels can provide information regarding past absorption of lead, measurements of lead levels in bone by using x-ray fluorescence instruments are available for research purposes only.

The BLL of a newborn infant reflects closely that of the mother.[29] In 1999 to 2002, the geometric mean BLL for US women 20 to 59 years of age was 1.2 μg/dL, with 0.3% having BLLs of ≥10 μg/dL.[12] Typically, as infants become more active and increase their environmental exposure, BLLs increase. Longitudinal studies of lead-exposed children have confirmed an increase in BLLs beginning in late infancy, with peak BLLs being reached at 18 to 36 months of age.[6,30–32] No studies have examined BLL patterns specifically for children with peak BLLs of <10 μg/dL, although certain studies included children with levels that low. A study of children born in 1994 and 1995, in which >50% of the children had peak BLLs of <10 μg/dL, reported an expected pattern of mean BLLs of 3.4 μg/dL at 6 months of age, 9.7 μg/dL at 24 months of age, and 5.8 μg/dL at 61 months of age.[6] A study of children born in Boston, Massachusetts, in 1979 to 1981 identified a mean BLL of 7.2 μg/dL at birth, and subsequent BLLs for those children remained relatively constant (6.2 μg/dL at 6 months of age, 6.8 μg/dL at 24 months of age, and 6.4 μg/dL at 57 months of age).[33–35] In both studies, higher levels of lead in home environmental samples were associated directly with higher BLLs in children.[34,36] In addition, the Boston study demonstrated an association between the occurrence of home renovation and increased BLLs.[34] The BLL patterns for individual children with BLLs of <10 μg/dL vary, depending on environmental exposures.[28] More research is needed to understand more thoroughly age-related patterns for BLLs that remain at <10 μg/dL. Even if additional research data become available, however, laboratory uncertainty might interfere with a clinician's ability to detect patterns for individual children.

Once a high BLL has been established for a child, the time required for the BLL to decline to <10 μg/dL can range from months to years, depending on the duration and dose of exposure. For example, for a group of children who started with BLLs of 10 to 14 μg/dL and received case management services, the mean time required for 50% to achieve BLLs of <10 μg/dL was 9 months.[37] The time needed for BLLs of <10 μg/dL to decline in response to interventions is not known.

Multiple studies have confirmed that BLL measurements vary seasonally. For example, a study conducted in Boston reported that BLLs were highest in late June and lowest in March.[38] A study performed in Milwaukee,

Wisconsin, indicated that BLLs were higher in the summer than in the winter.[39] Some of the variability (higher BLLs in summer) might result from increased exposure to lead in dust and soil in the summer months.[40] BLL values for urban children are predicted to be 1 to 2 μg/dL higher in the summer months than in the winter months.[41]

Association of BLL Patterns With Developmental Outcomes

Although BLLs peak in early childhood, when young children are especially vulnerable to lead, negative effects are associated with lead exposure at any age. Multiple studies have examined the effects of lead on children's developmental outcomes; in those studies, the ages at which BLLs were measured varied, as did the range of ages over which BLLs were averaged.[1–4] Statistically significant associations between average BLLs over a specific period (eg, 0–5 years) and various adverse health outcomes have been identified.[6,42–44] Other studies reported statistically significant associations with a single lead measurement at a specific age (eg, before birth, at 24 months, or at 6.5 years) or with a peak measurement.[6,30,45] Concurrent BLLs (ie, those measured close to the time of neurodevelopmental testing) might demonstrate stronger associations with neurodevelopmental abilities, compared with other BLL measurements.[6–8,31,46]

Lead has a continuing negative association with IQ as children reach elementary school age. For children who participated in a trial of chelation therapy, a subsequent data analysis indicated that BLLs measured concurrently with developmental testing were associated more closely with children's cognitive abilities than were peak levels measured at ~2 years of age.[47] This association was stronger when children were tested at 7 years of age, compared with 5 years of age, which underscores the continuing need to reduce lead exposure after 5 years of age.

Strategies to Enhance Children's Positive Developmental Outcomes

Although lead is a risk factor for developmental and behavioral problems, its presence does not indicate that these problems will necessarily occur. No characteristic developmental pattern is attributable solely to the effects of lead, and measures of the effects of lead on children are imperfect. For an individual child, neurobehavioral test performance might indicate clinically significant impairments related to lead exposure but might not fully capture the array of negative outcomes caused by lead.[14] The effects of lead at levels approaching 10 μg/dL might not be recognizable to the child's family or clinician and might not be identified through neurobehavioral testing. However, lead exposure might assume greater importance for children with other environmental, genetic, biological, social, or demographic developmental risk factors. The effects of exposure to lead at lower levels might not be evident in testing of individual children and are best evaluated on a community-wide basis.[14]

Multiple factors influence a child's development, including how the child is treated by parents and other adult caregivers. The child's family and personal psychosocial experiences are associated strongly with performance on neurodevelopmental measures and account for a greater proportion of the explained variance in these measures than do BLLs of <10 μg/dL.[2,42,44,48] A child's BLL measurement is estimated to account for 2% to 4% of the variance in neurodevelopmental measures (~4%–8% of the explained variance).[2,42,49]

All children, regardless of their BLLs, benefit from parental nurturing. For example, a child's language skills are enhanced by the amount of language addressed to the child (more is better), combined with a predominant pattern of positive feedback.[50] This pattern of parenting for children <3 years of age was associated with enhanced language and cognitive skills when children were tested in the third grade.[51] Therefore, parents might help counteract the negative effects of lead by providing a nurturing enriched environment during development. Studies examining the effects of lead have attempted to control for this psychosocial factor by including measures such as the Home Observation for Measurement of the Environment Inventory score.[7] Although no studies have evaluated specifically the effects of early intervention programs on cognitive or behavioral outcomes in relationship to children's BLLs, several laboratory studies that applied a nurturing environment to very young animals during lead acquisition demonstrated the beneficial effect of the social environment in ameliorating lead-related negative developmental outcomes.[52,53]

Early enrichment programs, although not tested specifically in relation to BLLs, have been effective in improving the cognitive development and social competence of young children, particularly infants from families with low levels of social or economic resources.[54] Research demonstrates that children whose development has been delayed or who are at high risk for delay benefit most from interventions applied at an early age.[55–57]

Strategies to Prevent and to Reduce Exposure to Lead

Major Sources of Exposure

The CDC and the American Academy of Pediatrics recommend that preventive care for every child should include assessment of environmental history and identification of the occupational lead exposure of household members.[2,3,5] The major sources of lead exposure among US children are lead-contaminated dust, deteriorated lead-based paint, and lead-contaminated soil.[36,58] Typically, lead contamination of water contributes less to a child's lead burden than do home and soil sources.[58] If additives to water (eg, those used in disinfection pro-

cesses) are changed, however, then the contribution to lead contamination may be greater.[59] The extent of lead paint hazards (ie, the presence of lead in an accessible condition, such as deteriorated lead-based paint or lead-contaminated dust or soil) on interior and exterior surfaces and in soil is associated with BLLs in children.[58] Children also are exposed to nonhousing lead sources (eg, lead in foods, cosmetics, pottery, folk remedies, and toys).[2,3,60]

Home-Related Lead Exposure

An estimated 4.1 million homes in the United States (25% of US homes with children <6 years of age) have a lead-based paint hazard.[13] An estimated 68% of US homes built before 1940 have lead hazards, as do 43% of homes built between 1940 and 1959 and 8% of homes built between 1960 and 1977; estimates are higher for homes in the Northeast and Midwest and for homes in which young children reside.[13] Despite considerable attention and resources from federal, state, and local agencies and advocacy groups, publicly available funding has not been able to provide sufficient resources to eliminate all lead paint hazards from US homes. Publicly funded home inspections are most often limited to homes of children with elevated BLLs; the BLL threshold value that prompts an inspection varies according to the state and municipality.[61] Even when a child's elevated BLL triggers an inspection, public funding for repairs to reduce or to eliminate identified lead hazards typically is not available.

Since 1991, lead hazard-control grant programs through the US Department of Housing and Urban Development Office of Healthy Homes and Lead Hazard Control have provided funding for local and state agencies to reduce lead and other environmental hazards in privately owned, low-income housing. In 2005, the Office of Healthy Homes and Lead Hazard Control allocated $139 million for this purpose, administered through 7 different grant types. Other federal programs provide funding to eliminate lead-based paint hazards in federally assisted housing. The focus of these programs typically is on housing rehabilitation and remediation of lead hazards after children with elevated BLLs are identified, but Department of Housing and Urban Development-funded local programs now include primary prevention interventions that control or eliminate lead before children are exposed.

The CDC is working with the Department of Housing and Urban Development, the US Environmental Protection Agency (EPA), state and local health department lead poisoning prevention grant recipients, and child health and environmental justice advocates to promote primary prevention strategies to reduce exposure to lead.[1,62,63] In addition to their traditional role of providing services to children with elevated BLLs, CDC-funded state and local lead poisoning prevention programs have been charged with implementing housing-based primary prevention strategies in their jurisdictions; this involves developing responses to local risks, with a focus on identifying and remedying housing-based lead hazards. The ACCLPP recommendations for essential elements for state and local primary prevention plans have been published previously,[62] as have strategies that have been implemented at the state and local levels to address the problem.[63] As the ACCLPP noted, implementation of state and local primary prevention plans requires (1) targeting of the highest-risk areas, populations, and activities; (2) fostering of political will for jurisdictions to provide adequate levels of funding; (3) expansion of resources for housing remediation and identification and correction of lead hazards; and (4) establishment of a regulatory infrastructure to create and to maintain lead-safe housing and to support the use of lead-safe construction practices.[62,64] (State prevention plans are available at www.cdc.gov/nceh/lead.)

Certain state and local health departments initiate case management services and home inspections when BLLs reach 10 μg/dL. As more primary prevention strategies are implemented, the number of health departments that pursue home inspections when BLLs reach 10 μg/dL will likely increase. Certain communities have developed online registries to help parents identify homes that are lead-safe or that have lead hazards.[65]

Steps to Identify and Safely Reduce Lead-Based Paint Hazards in Homes

Lead-based paint hazards in homes are important sources of lead exposure. Preventive actions can be implemented to identify and to address these hazards. Tenants can request copies of all lead testing reports for housing sites from landlords at any time. The landlords should have been provided with such information when they purchased the building; compliance with tenant requests for copies of all lead testing reports is required by federal law.[66] In addition, federal regulations require sellers and landlords (1) to disclose the possible presence of lead-based paint in any pre-1978 property and (2) to provide information on known lead-based paint and lead-based paint hazards (eg, by providing the results of any previous evaluations of the property for lead) at the time final agreements are signed for the purchase or rental of most housing built before 1978.[66] Prospective buyers or renters have the opportunity to arrange for a lead inspection or risk assessment by a qualified professional at their own expense; buyers have up to 10 days to check for lead. Furthermore, the law requires sellers, landlords, and renovators to provide buyers, renters, and individuals hiring renovators with an EPA-approved pamphlet (ie, *Protect Your Family From Lead in Your Home*).[67] To protect their children from lead, parents might choose not to buy or to rent a property or might choose to negotiate remediation of identified lead haz-

ards. However, landlords or homeowners might not know whether their property has any lead-based paint or lead hazards.

Lead-based paint hazards are likely to be present in older homes; all homes built before 1978 should be presumed either to have a lead hazard present or to contain intact lead-based paint unless a licensed lead inspector has determined otherwise. Lack of a deteriorated surface decreases the likelihood of lead-contaminated dust being present but does not ensure its absence. Knowledge of the general characteristics of lead-based paint and lead-based paint hazards and their control might help parents to understand their home better (Appendix 1).[68–72]

Screening for lead dust hazards through dust-wipe testing (ie, standardized collection of dust through wiping of surfaces and measurement of lead collected) can help identify areas of concern. Because lead is not distributed uniformly within a home, wipe testing neither ensures the absence of lead hazards at locations in the home that were not tested nor ensures future protection from lead dust hazards if lead-painted surfaces subsequently deteriorate or are disturbed. Potential sources of future contamination include lead-containing paint on areas disturbed by impact/friction (eg, windows, doors, and floors) and interior migration of lead-contaminated exterior dust and soil.[69] However, identifying lead dust hazards in the home is a first step toward protecting children and might help parents lower dust lead levels in their homes.[73] Proper training is recommended for individuals collecting dust wipes, to focus tests on areas at highest risk.[62] Parents or property owners who wish to perform dust-wipe sampling may consult their local health or housing departments for advice regarding sampling procedures, interpretation of results, and additional actions based on results.

For a lead-safe environment to be established in older buildings, repair of lead hazards and careful attention to maintenance are necessary. However, local ordinances typically do not require action until a child's BLL is elevated, and property owners might be unaware of lead hazards or ignore them. Primary prevention is possible only if the focus on safety in older housing is increased and lead hazards are repaired proactively, before a child is exposed. In all pre-1978 properties, owners should use lead-safe work techniques when performing routine maintenance, to decrease the likelihood of lead hazards developing in a home.

Home renovation or repair is known to be a risk factor for increasing or elevated BLLs, principally through exposure to the dust residue generated during the work.[34,74–76] All contractors who perform repair and renovation work in older housing should be trained in lead-safe work practices and comply with any state and local requirements governing work with lead paint hazards.[77] Property owners performing work themselves should seek expert advice and training to protect themselves and their families.[78,79] Lead-safe work practices include (1) relocating families when the work warrants, (2) minimizing the amount of dust created, (3) containing dust in the work area, (4) cleaning up completely, (5) disposing of waste safely, and (6) performing clearance testing (ie, testing of dust for lead after site cleanup) to ensure that residual lead levels do not exceed EPA standards.[80] Families with young children should be restricted from work areas until clearance testing has been performed and the area has been judged to be safe.

In previous evaluation studies, lead dust clearance standards were not low enough to protect children from increased exposure to lead-contaminated dust after lead hazard remediation; as a result, BLLs of children with preremediation BLLs of <25 μg/dL increased after home repairs.[81] In 2001, the EPA lead dust clearance standards were lowered to 40 μg/ft^2 for floors, 250 μg/ft^2 for windowsills, and 400 μg/ft^2 for window wells.[80] No studies have evaluated whether these lower clearance levels protect children with BLLs of <10 μg/dL adequately from ongoing lead exposure. A cross-sectional study estimated that 20% of children with current exposure to floor dust lead at 40 μg/ft^2 would have BLLs of ≥ 10 μg/dL.[82]

A study conducted in 1994 to 1999 in 14 US cities, involving 2682 pre-1978 homes, demonstrated reductions in dust lead levels and decreases in children's BLLs when lead-safe work practices were used during remediation efforts.[68,83,84] The study applied lead dust clearance standards substantially less stringent than those currently in place, although clearance floor dust lead levels were generally low (geometric mean: 16 μg/ft^2).[85] Of the 869 children in that study who were tested within 4 months before home lead remediation and ~7 weeks after remediation, 81 (9.3%) had clinically significant increases (≥ 5 μg/dL) in BLLs; infants, children of less-educated mothers, and children from homes with greater numbers of preintervention exterior lead hazards were at greatest risk.[86] Dust lead levels at clearance were not associated significantly with increases in BLLs. The study listed multiple types of exposures (eg, other homes and parental job exposures) that might have accounted for increased BLLs, but they were not evaluated systematically. Although lead remediation work reduced overall dust lead levels and BLLs, the finding that >9% of children had increases in BLLs of ≥ 5 μg/dL underscores the need to maintain a high level of vigilance to ensure that children are protected when homes or apartments undergo renovation and repair.

Educational Strategies

Lead-exposure–prevention strategies for children with BLLs of <10 μg/dL typically focus on education and promotion of home cleanliness, without further identifying lead hazards or repairing them. Providing low-

income parents with lead-related education via videotape in a pediatric office was demonstrated to be effective in increasing knowledge and parental reports of compliance with lead-prevention actions in the home.[87] No studies have evaluated office-based education with accompanying in-home strategies or used children's BLLs as the outcome measure for an office-based education strategy.

Studies of children at high risk that applied intervention strategies in the home or community demonstrated the failure of education and nonprofessional cleaning conducted alone (ie, in the absence of other measures to reduce lead exposure) in preventing the development of BLLs of ≥10 μg/dL.[2,88–90] Few studies used prospective designs that included control groups. One study indicated that a highly intensive education program, delivered by community members, that started at birth and lasted for >3 years (28 sessions) decreased the risk of BLLs of ≥10 μg/dL by 34%, but this result was not statistically significant.[91] Repeated in-home lead-prevention education, even when accompanied by complimentary supplies of cleaning materials, was ineffective in decreasing the incidence of elevated BLLs.[92,93] A review of 4 studies[89] involving caregiver education[93,94] and professional house cleaning[95,96] indicated that such low-cost interventions reduced the overall proportions of children with BLLs of ≥15 or ≥20 μg/dL but the effect on mean BLLs was not statistically significant ($P > .05$).

Intensive cleaning regimens reduce BLLs; in 1 study, biweekly professional cleaning resulted in a 17% decrease in mean BLLs after 1 year.[95] However, the benefit of such intense and repeated cleaning was limited to homes without carpets.[97] Intensive cleaning can be used without subjecting children to a risk of increased lead exposure resulting from unsafe repair methods (ie, those not in compliance with lead-safe work practices). A single intensive cleaning does reduce levels of lead in dust by 32% to 93%, depending on the surfaces tested and the starting lead concentrations,[98] but reaccumulation occurs within 3 to 6 months.[99,100]

A study that involved children with BLLs of 15 to 19 μg/dL compared the effects of nurse home visits (5 visits in 1 year) accompanied by lead dust tests with those of usual care (1 or 2 visits by an outreach worker in 1 year).[73] After 1 year, dust lead levels were significantly lower ($P < .05$) in homes where lead dust tests had been conducted during intervention than in usual-care homes. This finding suggests that dust testing might help parents better understand lead hazards and take action to decrease them. However, changes in dust lead levels were not mirrored by changes in BLLs in this group of children with elevated BLLs.

BLL-Screening Strategies

The CDC[101] and the American Academy of Pediatrics[3] have recommended that health care providers conduct BLL tests for children enrolled in Medicaid and those identified as being at risk on the basis of the state or local screening plan or risk assessment process. Federal policy requires that all children enrolled in Medicaid receive BLL-screening tests at 12 and 24 months of age and that BLL screening be performed for children 36 to 72 months of age who have not been screened previously.[102] Despite this, BLL-screening rates for Medicaid-enrolled children have been low (<20%)[103] and in certain areas remain ~20%.[104] In 1997, the CDC requested that state and local health officials use local community-wide data (eg, BLL prevalence, housing age, and poverty status) to develop plans for BLL screening for their jurisdictions and provide them to clinicians.[101] These plans recommend either universal or targeted BLL screening (state and local screening plans are available at www.cdc.gov/nceh/lead/grants/contacts/CLPPP%20Map.htm).

Targeted screening strategies enable clinicians to assess risks for individual children and to recommend BLL testing for the subset of children in the jurisdiction who are thought to be at increased risk for lead exposure. The CDC recommends that risk evaluations be conducted on the basis of factors such as residence in a specific geographic area, membership in a group at high risk, answers on a personal risk assessment questionnaire (which might include local factors such as cultural practices and use of products such as herbal remedies, traditional cosmetics, and imported spices), and other risk factors relevant to the jurisdiction.[101]

The CDC recommends that locally developed targeted risk assessment and BLL-screening strategies be applied at 1 and 2 years of age.[101] Children 36 to 72 months of age who have been identified as being at risk and who have not been screened previously also should receive a BLL test.[101] For clinicians in areas that lack a state or local screening plan, the CDC recommends that BLL testing be performed for all children at 1 and 2 years of age and for children 36 to 72 months of age who have not been screened previously.[101]

Because lead exposure might change with a child's developmental progress (eg, walking or reaching window sills) or as a result of external factors (eg, family relocation or home remodeling), 2 routine screenings are recommended (at ~1 and ~2 years of age). Among children in Chicago at high risk with BLLs of <10 μg/dL at 1 year of age, 21% had BLLs of ≥10 μg/dL when tested again at ≥2 years of age.[102] That report does not change current CDC recommendations regarding ages for routine BLL testing. However, certain local health departments (eg, those in Chicago, IL; New York, NY; and Philadelphia, PA) recommend BLL screening at younger ages or more frequently.[105–107] For example, those departments recommend BLL testing starting at 6 to 9 months of age in high-risk areas, BLL testing at more-frequent intervals (eg, every 6 months) for chil-

TABLE 2 **Sensitivity and Specificity of Lead-Risk-Assessment Questionnaires in Predicting BLLs of ≥10 μg/dL Among Patient Samples in the United States (1994–2003)**

Location	Sample Characteristics	Proportion of Study Sample With BLLs of ≥10 μg/dL	Type of Lead Exposure Risk-Assessment Questions	With Cutoff Value of ≥10 μg/dL	
				Sensitivity	Specificity
Alaska[108]	Medicaid	0.6	Modified	0.83	0.39
California[109]	Medicaid	2.0	CDC	0.46	0.74
Suburban Chicago, IL[110]	Private practices	2.2	CDC	0.69	0.70
			Modified	0.86	0.53
Arizona[111]	Navajo reservation	2.2	CDC	0.43	0.74
New York[112]	Rural	2.3	CDC	0.25	0.49
			Modified	0.50	NR
Denver, CO[113]	Community health centers	2.9	Modified	0.60	0.36
Illinois[114]	Low-risk zip codes	3.5	Modified	0.75	0.39
Wisconsin[115]	HMO clinic A	5.4	CDC	0.77	0.37
			Modified	1.00	0.42
Ohio[116]	Mixed sample	5.6	CDC	0.85	0.42
			Modified	0.92	0.57
San Francisco, CA[117]	Mixed sample	5.8	CDC	0.87	0.75
California[118]	Public clinics	6.1	CDC	0.30	0.80
			Modified	0.90	0.37
New York[119]	Rural	8.4	CDC	0.75	0.31
			Modified	0.88	0.44
Vermont[120]	Birth certificate cohort	9.0	CDC	0.63	0.57
Minnesota[121]	HMO	11.8	Modified	0.90	0.17
			Modified, brief	0.77	0.48
Illinois[114]	High-risk zip codes	12.1	Modified	0.74	0.27
Vermont[120]	Medicaid	14.9	CDC	0.67	0.50
Wisconsin[115]	HMO clinic B	16.8	CDC	0.64	0.32
			Modified	0.91	0.43
Massachusetts[122]	Urban, high-risk	21.8	CDC	0.70	0.32
Philadelphia, PA, area[123]	Privately insured	29.1	CDC	0.40	0.60
Rochester, NY[124]	Primarily Medicaid	28[a]	CDC	0.70	0.49

NR indicates not reported; HMO, health maintenance organization.
[a] Data are not available to add a decimal place.

dren <2 years of age, or the provision of additional education and more-rapid follow-up BLL testing for children <12 months of age with BLLs of 6 to 9 μg/dL.

Personal Lead-Risk–Assessment Questionnaires

The effectiveness of personal risk assessment questionnaires in identifying children with elevated BLLs has been documented in the scientific literature (Table 2.)[108–124] However, no studies have evaluated the performance of these questionnaires at cutoff levels of <10 μg/dL or their effectiveness in directing counseling or in identifying lead hazards in the home. When used for consecutive samples of patients in clinical settings, the sensitivity of such questionnaires in identifying children with BLLs of ≥10 μg/dL varied considerably according to population.[108–127] In certain studies, the sensitivity improved if higher cutoff levels were used in the analysis[102,114,118,119] or if the questions used were developed specifically for the population being tested.* In general, to identify ~80% of children with BLLs of ≥10 μg/dL, a blood test needed to be performed for more than one half of the children whose risk factors for lead exposure were assessed by using a questionnaire. Multiple studies in populations with low† or high[122,123] prevalence for elevated BLLs concluded that risk assessment questionnaires were not effective in their clinical settings.

Future Research Needs

Additional study is needed to assess the effects of BLLs of <10 μg/dL on children. Such research should entail monitoring of large diverse populations, with careful attention to potential confounders and measurements of social factors. Additional research also is needed to evaluate the effectiveness of strategies to decrease exposure to lead. This should include research on the effectiveness of strategies applied in the medical office and the home and interventions provided through medical, public health, and environmental means.

BLL-screening strategies should be evaluated to determine the most-appropriate ages for screening and the utility of screening strategies applied at the community level. Evaluations of lead surveillance strategies should test ways to identify changing patterns of environmental risks and subpopulations exposed to established and emerging sources of lead. In addition, better ways to

*Refs 113, 116, 117, 119, 120, and 122.

†Refs 108, 109, 111, 113, 126, and 127.

alert public and clinical health care professionals of changes in exposure sources and patterns and to enhance their responses to such changes through increased surveillance and BLL monitoring of populations identified as being at increased risk for exposure should be identified. Additional studies might provide data that can be used to improve laboratory methods and performance monitoring. This would require the development of criteria to evaluate individual laboratories and mechanisms to provide this information to clinicians.

SUMMARY OF RECOMMENDATIONS

Recommendations for Clinicians

1. Provide anticipatory guidance to parents of all young children regarding sources of lead and help them identify sources of lead in their child's environment. Obtain an environmental and family occupational history and educate parents about the most common sources of childhood lead exposure for their child and in their community. Encourage parents to identify lead hazards and sources in their homes and to reduce their child's potential for exposure to lead, including the safe implementation of control measures before BLLs increase. Warn parents about the dangers posed by unsafe renovation methods and urge them to be cognizant of the possibility of new and reemerging sources of lead in children's environments. Direct parents to local, state, and federal agencies and organizations for information, particularly concerning methods to identify lead hazards and to repair them safely (Appendix 2).
2. Help parents to understand the uncertainty of BLL values and potential reasons for their fluctuation, including errors introduced by the sampling methods and laboratory-, age-, and season-related exposures.
3. Assess all children for developmental and behavioral status and seek additional evaluation and therapy to reduce developmental or behavioral problems, as necessary. Consider the potential influences of lead when conducting developmental screening. For children with multiple developmental risk factors that might include lead exposure, consider more-frequent developmental surveillance or conduct more-extensive developmental evaluations.
4. Discuss with parents the potential impact of lead on child development and promote strategies that foster optimal development, including encouraging parents to influence their child's development positively by providing nurturing and enriching experiences. For all children from families with low levels of economic and social resources who are living in areas where exposure to lead is likely, promote participation in early enrichment programs regardless of the child's BLL.
5. Whenever possible, use laboratories that can achieve routine performance of ± 2 μg/dL for BLL analysis. Evaluate laboratory performance by reviewing the laboratory's quality control chart or statistical quality control summary.
6. Review office procedures and policies to ensure that lead-exposure risk assessment or BLL screening is performed for all children as required by state or local health officials or as recommended by the CDC. Consider the child's age, season of testing, and exposure history when deciding when to obtain follow-up BLL tests. For a child whose BLL is approaching 10 μg/dL, more-frequent BLL screening (ie, more often than annually) might be appropriate, particularly if the child is <2 years of age, was tested at the start of warm weather (when BLLs tend to increase), or is at high risk for lead exposure.
7. Perform a diagnostic BLL test for all children suspected of having lead exposure or an elevated BLL and institute the recommended management guidelines if a child's BLL increases to ≥ 10 μg/dL.
8. Become informed about lead-exposure–prevention strategies of local or state health departments and partner with public health agencies, community groups, and parents to work toward establishing lead-safe environments in homes and schools for all children and reducing exposure to lead from all sources. Advocate for the expansion of services that foster primary lead poisoning prevention.

Recommendations for Government Agencies

1. Increase efforts to resolve lead-based paint hazards safely before children are exposed.
2. Expand services that promote primary lead poisoning prevention and develop systems that enable clinicians and parents to learn about such services.
3. Develop and implement strategies to encourage the safe elimination of lead hazards in properties, using trained workers and lead-safe work practices, in compliance with federal, state, and local regulations.
4. Establish jurisdictional policies that mandate ensuring lead safety in housing and enforce these mandates.
5. Develop and apply systematic approaches to prevent exposure to even small amounts of lead in food or consumer products, particularly when safer alternatives are available.
6. Promote implementation of state and local primary prevention plans that target areas, populations, and activities of highest risk; expand resources for hous-

ing remediation; identify and correct lead hazards; and establish a regulatory infrastructure to create and to maintain lead-safe housing and to support the use of lead-safe construction practices.

7. Expand the availability of and promote the use of early enrichment programs for all children from families with low levels of economic and social resources who are living in areas where exposure to lead is likely.
8. Promote and fund additional research to evaluate the effects of lead at BLLs of <10 μg/dL and to evaluate strategies to identify and to reduce exposure or the potential for exposure to lead, including strategies applied in medical offices and in homes.

APPENDIX 1. TIPS FOR REDUCING LEAD-BASED PAINT AND LEAD-BASED PAINT HAZARDS

- The concentration of lead is generally highest in lead-based paint on exterior surfaces.
- Among interior surfaces, windows are most likely to have the highest lead content.
- Interior surfaces can become contaminated from exterior sources or common areas.
- Lead-based paint on impact/friction surfaces (eg, windows, doors, and floors) deteriorates as paint is disturbed during use.
- Lack of a deteriorated surface does not ensure the absence of lead-contaminated dust, although it decreases the risk.
- Renovation, remodeling, and repainting can increase dust lead levels significantly.
- Vacuum methods (using a traditional vacuum or a high-energy particulate air-filtered vacuum) do not decrease lead levels on soiled carpets or upholstery enough to achieve safe levels.
- Creating smooth cleanable surfaces helps achieve lower dust lead levels.
- Treatments addressing lead-contaminated exterior dust/soil and building exterior lead hazards can contribute to lower dust lead levels in entryway and home interior locations.
- Safely addressing interior, exterior, and soil lead hazards in an integrated manner is most beneficial in establishing lasting, lead-safe environments.

ACKNOWLEDGMENTS

The ACCLPP membership included chairperson Carla Campbell, MD, (Children's Hospital of Philadelphia, Philadelphia, PA); executive secretary Mary Jean Brown, ScD, RN (Division of Environmental and Emergency Health Services, National Center for Environmental Health/Agency for Toxic Substances and Disease Reg-

APPENDIX 2 **Guide to Resources for Parents**

Agency/Organization	Specific Resources	Contact Information
National Lead Information Center	Multiple general publications	Telephone: 800–424-LEAD (800–424–5323); fax: 585–232–3111; Web site: www.epa.gov/lead/pubs/nlic.htm; e-mail: see Web site
US Consumer Product Safety Commission	*Protect Your Family From Lead in Your Home*; product-specific information; multiple reports, recall notices, and safety alerts	Telephone: 800–638–2772 (800–638–8270 for hearing and speech impaired); fax: 301–504–0124, 301–504–0025; Web site: www.cpsc.gov; e-mail: info@cpsc.gov
CDC, Lead Poisoning Prevention Branch	Questions and answers; fact sheets; lead screening plans by state and area; links to other sources	Telephone: 770–488–3300; fax: 770–488–3635; Web site: www.cdc.gov/nceh/lead; e-mail: leadinfo@cdc.gov
US Department of Housing and Urban Development, Office of Healthy Homes and Lead Hazard Control	*Lead Paint Safety: A Field Guide for Painting, Home Maintenance, and Renovation Work*; *Caution: Lead Paint, Handle With Care*; *Lead Paint Can Poison: Learn the Facts*; other materials	Telephone: 202–755–1785; fax: 202–755–1000; Web site: www.hud.gov/offices/lead; e-mail: lead_regulations@hud.gov
EPA, Office of Pollution Prevention and Toxics	*Lead in Your Home: A Parent's Reference Guide*; *Reducing Lead Hazards When Remodeling Your Home*; *Protect Your Family From Lead in Your Home*; *Is There Lead in My Drinking Water?*; *Lead Poisoning and Your Children*; other materials	Telephone: 202–566–0500; fax: 202–566–0469; Web site: www.epa.gov/lead; e-mail: see Web site
Alliance for Healthy Homes	*Lead Safety Tips for Tenants*; *Lead-Safe Painting and Renovation*; links to other sources	Telephone: 202–543–1147; fax: 202–543–4466; Web site: www.afhh.org; e-mail: afhh@afhh.org
National Center for Healthy Housing	*A Guide to Working Safely With Residential Paint*; links to other sources	Telephone: 410–992–0712; fax: 410–715–2310; Web site: www.centerforhealthyhousing.org; e-mail: nchh@enterprisefoundation.org
National Library of Medicine, National Institutes of Health	Links to other sources, including Spanish-language materials	Web site: http://sis.nlm.nih.gov/enviro/lead.html

istry, CDC, Atlanta, GA); members Magaly C. Angeloni, MBA (Rhode Island Department of Public Health, Providence, RI), Valerie Charlton, MD (California Department of Health, Richmond, CA), Walter S. Handy, Jr, PhD (Cincinnati Health Department, Cincinnati, OH), Ing Kang Ho, PhD (University of Mississippi Medical Center, Jackson, MS), Valarie Johnson (Urban Parent to Parent, Rochester, NY), Linda Kite, MBA (Healthy Homes Collaborative, Los Angeles, CA), Jessica Leighton, PhD (New York City Department of Health and Mental Hygiene, New York, NY), George G. Rhoads, MD (University of Medicine and Dentistry of New Jersey, Piscataway, NJ), Catherine M. Slota-Varma, MD (deceased; Medical College of Wisconsin, Milwaukee, WI), Wayne R. Snodgrass, MD, PhD (University of Texas Medical Branch, Galveston, TX), Kevin U. Stephens, Sr, MD, JD (New Orleans Department of Health, New Orleans, LA), Helen J. Binns, MD (member in 2002–2004; Feinberg School of Medicine, Northwestern University, Chicago, IL), Kimberly M. Thompson, ScD (member in 2002–2005; Harvard University, Boston, MA); nonvoting federal members Phyllis Stubbs-Wynn, MD (Maternal and Child Health Bureau, Health Resources and Services Administration, Washington, DC), Michael Bolger, PhD (US Food and Drug Administration, Washington, DC), David Jacobs, PhD (member in 1996–2004; US Department of Housing and Urban Development, Washington, DC), Warren Friedman, PhD (US Department of Housing and Urban Development, Washington, DC), Jacqueline E. Mosby, MPH (EPA, Washington, DC), Walter Rogan, MD (National Institute of Environmental Health Sciences, Washington, DC), Robert J. Roscoe, MS (National Institute for Occupational Safety and Health, CDC, Cincinnati, OH), Lori E. Saltzman, MS (US Consumer Product Safety Commission, Washington, DC); and nonvoting liaison representatives Steve M, Hays (American Industrial Hygiene Association, Nashville, TN), Ezatollah Keyvan-Larijani, MD, DrPH (Council of State and Territorial Epidemiologists, Baltimore, MD), Pat McLaine, MPH (representative in 1998–2005; National Center for Healthy Housing, Columbia, MD), Jonathan Wilson, MPP (National Center for Healthy Housing, Columbia, MD), Benjamin Gitterman, MD (American Public Health Association, Washington, DC), Routt Reigart II, MD (representative in 1997–2004; American Academy of Pediatrics, Charleston, SC), George C. Rodgers, Jr, MD, PhD (American Association of Poison Control Centers, Georgetown, IN), Jan Towers, PhD (American Academy of Nurse Practitioners, Gettysburg, PA), Anne M. Wengrovitz, MPH (Alliance for Healthy Homes, Washington, DC), and Calvin B. Johnson, MD (American State and Territorial Health Officials, Harrisburg, PA).

Helpful suggestions were provided by Patrick J. Parsons, PhD (Lead Poisoning/Trace Elements Laboratory, Wadsworth Center, New York State Department of Health, Albany, NY).

REFERENCES

1. Centers for Disease Control and Prevention. *Preventing Lead Poisoning in Young Children*. Atlanta, GA: Centers for Disease Control and Prevention; 2005
2. 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: Centers for Disease Control and Prevention; 2002.Availableat:www.cdc.gov/nceh/lead/CaseManagement/caseManage_main.htm. Accessed September 10, 2007
3. American Academy of Pediatrics, Committee on Environmental Health. Lead exposure in children: prevention, detection, and management. *Pediatrics.* 2005;116:1036–1046
4. Bellinger DC. Lead. *Pediatrics.* 2004;113:1016–1022
5. Centers for Disease Control and Prevention. *Preventing Lead Poisoning in Young Children*. Atlanta, GA: Centers for Disease Control and Prevention; 1991
6. Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 μg per deciliter. *N Engl J Med.* 2003;348:1517–1526
7. Lanphear BP, Hornung R, Khoury J, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect.* 2005; 113:894–899
8. Téllez-Rojo MM, Bellinger DC, Arroyo-Quiroz C, et al. Longitudinal associations between blood lead concentrations lower than 10 μg/dL and neurobehavioral development in environmentally exposed children in Mexico City. *Pediatrics.* 2006;118(2). Available at: www.pediatrics.org/cgi/content/full/118/2/e323
9. Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG. Chronological trend in blood lead levels between 1976 and 1980. *N Engl J Med.* 1983;308:1373–1377
10. Pirkle JL, Brody DJ, Gunter EW, et al. The decline in blood lead levels in the United States: the National Health and Nutrition Examination Surveys (NHANES). *JAMA.* 1994;272: 284–291
11. Centers for Disease Control and Prevention. Update: blood lead levels: United States, 1991–1994 [published correction appears in *MMWR Morb Mortal Wkly Rep*. 1997;46:607]. *MMWR Morb Mortal Wkly Rep.* 1997;46:141–146
12. Centers for Disease Control and Prevention. Blood lead levels: United States, 1999–2002. *MMWR Morb Mortal Wkly Rep.* 2005;54:513–516
13. Jacobs DE, Clickner RP, Zhou JY, et al. The prevalence of lead-based paint hazards in US housing. *Environ Health Perspect.* 2002;110:A599–A606
14. Bellinger DC. What is an adverse effect? A possible resolution of clinical and epidemiological perspectives on neurobehavioral toxicity. *Environ Res.* 2004;95:394–405
15. Sargent JD, Johnson L, Roda S. Disparities in clinical laboratory performance for blood lead analysis. *Arch Pediatr Adolesc Med.* 1996;150:609–614
16. Roda SM, Greenland RD, Bornschein RL, Hammond PB. Anodic stripping voltammetry procedure modified for improved accuracy of blood lead analysis. *Clin Chem.* 1988;34:563–567
17. Johanputra NK, Jones R, Guckler G, et al. Accuracy and reproducibility of blood lead testing in commercial laboratories. *Arch Pediatr Adolesc Med.* 1998;152:548–553
18. Parsons PJ, Geraghty C, Verostek MF. An assessment of contemporary atomic spectroscopic techniques for the determi-

nation of lead in blood and urine matrices. *Spectrochim Acta [B].* 2001;56:1593–1604
19. Schlenker TL, Fritz CJ, Mark D, et al. Screening for pediatric lead poisoning: comparability of simultaneously drawn capillary and venous blood samples. *JAMA.* 1994;271:1346–1348
20. Parsons PJ, Reilly AA, Esernio-Jenssen D. Screening children exposed to lead: an assessment of the capillary blood lead fingerstick test. *Clin Chem.* 1997;43:302–311
21. Sargent JD, Dalton MA. Rethinking the threshold for an abnormal capillary blood lead screening test. *Arch Pediatr Adolesc Med.* 1996;150:1084–1088
22. Holtrop TG, Yee HY, Simpson PM, Kauffman RE. A community outreach lead screening program using capillary blood collected on filter paper. *Arch Pediatr Adolesc Med.* 1998;152:455–458
23. Schoenfeld DJ, Cullen MR, Rainey PM, et al. Screening for lead poisoning in an urban pediatric clinic using samples obtained by fingerstick. *Pediatrics.* 1994;94:174–179
24. Barry PS, Mossman DB. Lead concentrations in human tissues. *Br J Ind Med.* 1970;27:339–351
25. Schroeder HA, Tipton IH. The human body burden of lead. *Arch Environ Health.* 1968;17:965–978
26. Leggett RW. An age-specific kinetic model of lead metabolism in humans. *Environ Health Perspect.* 1993;101:598–616
27. Hu H, Rabinowitz M, Smith D. Bone lead as a biological marker in epidemiologic studies of chronic toxicity: conceptual paradigms. *Environ Health Perspect.* 1998;106:1–8
28. Manton WI, Angle CR, Stanek KL, Reese YR, Kuehnemann TJ. Acquisition and retention of lead by young children. *Environ Res.* 2000;82:60–80
29. Schell LM, Denham M, Stark AD, et al. Maternal blood lead concentration, diet during pregnancy, and anthropometry predict neonatal blood lead in a socioeconomically disadvantaged population. *Environ Health Perspect.* 2003;111:195–200
30. Dietrich KN, Ris MD, Succop PA, Berger OG, Bornschein RL. Early exposure to lead and juvenile delinquency. *Neurotoxicol Teratol.* 2001;23:511–518
31. Dietrich K, Berger O, Succop P. Lead exposure and the motor developmental status of urban six-year-old children in the Cincinnati Prospective Study. *Pediatrics.* 1993;91:301–307
32. Baghurst PA, Robertson EF, McMichael AJ, Vimpani GV, Wibb NR, Roberts RR. The Port Pirie Cohort Study: lead effects on pregnancy outcome and early childhood development. *Neurotoxicology.* 1987;8:395–402
33. Rabinowitz M, Leviton A, Needleman H. Variability of blood lead concentrations during infancy. *Arch Environ Health.* 1984;39:74–77
34. Rabinowitz M, Leviton A, Needleman H, Bellinger D, Waternaux C. Environmental correlates of infant blood lead levels in Boston. *Environ Res.* 1985;38:96–107
35. Bellinger D, Sloman J, Leviton A, Rabinowitz M, Needleman HL, Waternaux C. Low-level lead exposure and children's cognitive function in the preschool years. *Pediatrics.* 1991;87:219–227
36. Lanphear BP, Hornung R, Ho M, Howard CR, Eberly S, Knauf K. Environmental lead exposure during early childhood. *J Pediatr.* 2002;140:40–47
37. Roberts JR, Reigart JR, Ebeling M, Hulsey TC. Time required for blood lead levels to decline in nonchelated children. *J Toxicol Clin Toxicol.* 2001;39:153–160
38. US Environmental Protection Agency. *Seasonal Rhythms of Blood-Lead Levels: Boston, 1979–1983*. Washington, DC: US Environmental Protection Agency; 1995. Publication EPA 747-R-94-003
39. US Environmental Protection Agency. *Seasonal Trends in Blood Lead Levels in Milwaukee: Statistical Methodology*. Washington, DC: US Environmental Protection Agency; 1996. Publication EPA 747-R-95-010
40. Yiin LM, Rhoads GG, Lioy PJ. Seasonal influences on childhood lead exposure. *Environ Health Perspect.* 2000;108:177–182
41. Laidlaw MAS, Mielke HW, Filippelli GM, Johnson DL, Gonzales CR. Seasonality and children's blood lead levels: developing a predictive model using climatic variables and blood lead data from Indianapolis, Indiana, Syracuse, New York, and New Orleans, Louisiana (USA). *Environ Health Perspect.* 2005;113:793–800
42. Wasserman GA, Liu X, Popovac D, et al. The Yugoslavia Prospective Lead Study: contributions of prenatal and postnatal lead exposure to early intelligence. *Neurotoxicol Teratol.* 2000;22:811–818
43. Pocock S, Smith M, Baghurst P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. *BMJ.* 1994;309:1189–1197
44. Baghurst PA, McMichael AJ, Wigg NR, et al. Environmental exposure to lead and children's intelligence at the age of seven years: the Port Pirie Cohort Study. *N Engl J Med.* 1992;327:1279–1284
45. Bellinger D, Stiles K, Needleman H. Low-level lead exposure, intelligence, and academic achievement: a long-term follow-up study. *Pediatrics.* 1992;90:855–891
46. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 μg/dL in US children and adolescents. *Public Health Rep.* 2000;115:521–529
47. Chen A, Dietrich KN, Ware JH, Radcliffe J, Rogan WJ. IQ and blood lead from 2 to 7 years of age: are the effects in older children the residual of high blood lead concentrations in 2-year-olds? *Environ Health Perspect.* 2005;113:597–601
48. Koller K, Brown R, Spurgeon A, Levy L. Recent developments in low-level lead exposure and intellectual impairment in children. *Environ Health Perspect.* 2004;114:987–994
49. Needleman H, Gatsonis C. Low-level lead exposure and the IQ of children. *JAMA.* 1990;263:673–678
50. Hart B, Risley TR. American parenting of language-learning children: persisting differences in family-child interactions observed in natural home environments. *Dev Psychol.* 1992;28:1096–1105
51. Walker D. Prediction of school outcomes based on early language production and socioeconomic factors. *Child Dev.* 1994;65:606–621
52. Schneider JS, Lee MH, Anderson DW, Zuck L, Lidsky TI. Enriched environment during development is protective against lead-induced neurotoxicity. *Brain Res.* 2001;896:48–55
53. Guilarte TR, Toscano CD, McGlothan JL, Weaver SA. Environmental enrichment reverses cognitive and molecular deficits induced by developmental lead exposure. *Ann Neurol.* 2003;53:50–56
54. Ramey CT, Ramey SL. Prevention of intellectual disabilities: early interventions to improve cognitive development. *Prev Med.* 1998;27:224–232
55. Glascoe FP. Early detection of developmental and behavioral problems. *Pediatr Rev.* 2000;21:272–280
56. Anderson LM, Shinn C, Fullilove MT, et al. The effectiveness of early childhood development programs: a systematic review. *Am J Prev Med.* 2003;24(suppl):32–46
57. Campbell FA, Pungello EP, Miller-Johnson S, Burchinal M, Ramey CT. The development of cognitive and academic abilities: growth curves from an early childhood educational experiment. *Dev Psychol.* 2001;37:231–242
58. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to chil-

dren's blood lead levels: a pooled analysis of 12 epidemiologic studies. *Environ Res.* 1998;79:51–68
59. Centers for Disease Control and Prevention. Blood lead levels in residents of homes with elevated lead in tap water: District of Columbia, 2004. *MMWR Morb Mortal Wkly Rep.* 2004;53:268–270
60. Manton WE, Angle CR, Krogstrand KLS. Origin of lead in the United States diet. *Environ Sci Technol.* 2005;39:8995–9000
61. National Center for Healthy Housing. *Another Link in the Chain Update: State Policies and Practices for Case Management and Environmental Investigation for Lead-Poisoned Children*. Columbia, MD: National Center for Healthy Housing; 2001. Available at: www.afhh.org/res/res_pubs/Link_in_Chain_Update.pdf. Accessed September 10, 2007
62. Centers for Disease Control and Prevention. *Preventing Lead Exposure in Young Children: A Housing-Based Approach to Primary Prevention of Lead Poisoning*. Atlanta, GA: Centers for Disease Control and Prevention; 2004. Available at: www.cdc.gov/nceh/lead/Publications/Primary%20Prevention%20Document.pdf. Accessed September 10, 2007
63. Centers for Disease Control and Prevention. *Building Blocks for Primary Prevention: Protecting Children From Lead-Based Paint Hazards*. Atlanta, GA: Centers for Disease Control and Prevention; 2005. Available at: www.cdc.gov/nceh/lead/Building%20Blocks%20June%202005.pdf. Accessed September 10, 2007
64. Brown MJ, Gardner J, Sargent JD, Swartz K, Hu H, Timperi R. The effectiveness of housing policies in reducing children's lead exposure. *Am J Public Health.* 2001;91:621–624
65. US Department of Housing and Urban Development. LeadSafeHomes.info: mapping a healthy future. Available at: www.leadsafehomes.info. Accessed September 10, 2007
66. Lead: requirements for disclosure of known lead-based paint and/or lead-based paint hazards in housing. 42 USC §4852d (1992)
67. Lead-based paint poisoning prevention in certain residential structures: disclosure requirements for sellers and lessors. 40 CFR §745.107 (2005)
68. National Center for Healthy Housing, University of Cincinnati, Department of Environmental Health. *Evaluation of the HUD Lead-Based Paint Hazard Control Grant Program: Final Report*. Cincinnati, OH: National Center for Healthy Housing; 2004. Available at: www.centerforhealthyhousing.org/HUD_National_Evaluation_Final_Report.pdf. Accessed September 10, 2007
69. Clark S, Menrath W, Chen M, et al. The influence of exterior dust and soil lead on interior dust lead levels in housing that had undergone lead-based paint hazard control. *J Occup Environ Hyg.* 2004;1:273–282
70. Dixon SL, Wilson JW, Clark CS, Galke WA, Succop PA, Chen M. The influence of common area lead loadings and lead hazard control on dust lead loadings in multiunit buildings. *J Occup Environ Hyg.* 2005;2:659–666
71. Binns HJ, Gray KA, Chen T, et al. Evaluation of landscape coverings to reduce soil lead hazards in urban residential yards: the Safer Yards Project. *Environ Res.* 2004;96:127–138
72. Yiin LM, Rhoads GG, Rich DQ, et al. Comparison of techniques to reduce residential lead dust on carpet and upholstery: the New Jersey Assessment of Cleaning Techniques Trial. *Environ Health Perspect.* 2002;110:1233–1237
73. Brown MJ, McLaine P, Dixon S, Simon P. A randomized, community-based trial of home visiting to reduce blood lead levels in children. *Pediatrics.* 2006;117:147–153
74. 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
75. Reissman DB, Matte TD, Gurnitz KL, Kaufmann RB, Leighton J. Is home renovation or repair a risk factor for exposure to lead among children residing in New York City? *J Urban Health.* 2002;79:502–511
76. US Environmental Protection Agency. *Lead Exposure Associated With Renovation and Remodeling Activities: Phase III: Wisconsin Childhood Blood-Lead Study*. Washington, DC: US Environmental Protection Agency; 1999. Publication EPA 747-R-99-002
77. US Environmental Protection Agency. *EPA Model Renovation Training Course Minimizing Lead-Based Paint Hazards During Renovation, Remodeling, and Painting*. Washington, DC: US Environmental Protection Agency; 2000. Publication EPA 747-B-00-005/6
78. US Department of Housing and Urban Development. *Lead Paint Safety: A Field Guide for Painting, Home Maintenance, and Renovation Work*. Washington, DC: US Department of Housing and Urban Development; 2001. Available at: www.hud.gov/offices/lead/training/LBPguide.pdf. Accessed September 10, 2007
79. US Environmental Protection Agency. *Lead in Your Home: A Parent's Reference Guide*. Washington, DC: US Environmental Protection Agency; 1998. Publication EPA 747-B-98-002. Available at: www.epa.gov/lead/pubs/leadrev.pdf. Accessed September 10, 2007
80. US Environmental Protection Agency. Lead: identification of dangerous levels of lead: final rule. *Fed Regist.* 2001;66:1205–1240
81. Aschengrau A, Beiser A, Bellinger D, Copenhafer D, Weitzman D. 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
82. 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
83. Galke W, Clark S, McLaine P, et al. National evaluation of the US Department of Housing and Urban Development Lead-Based Paint Hazard Control Grant Program: study methods. *Environ Res.* 2005;98:315–328
84. Galke W, Clark S, Wilson J, et al. Evaluation of the HUD Lead Hazard Control Grant Program: early overall findings. *Environ Res.* 2001;86:149–156
85. Dixon SL, Wilson JW, Succop PA, et al. Residential dust lead loading immediately after intervention in the HUD Lead Hazard Control Grant Program. *J Occup Environ Hyg.* 2004;1:716–724
86. Clark S, Grote JA, Wilson J, et al. Occurrence and determinants of increases in blood lead levels in children shortly after lead hazard control activities. *Environ Res.* 2004;96:196–205
87. Kersten HB, Moughan B, Moran MM, Spector ND, Smals LE, DeLago CW. A videotape to improve parental knowledge of lead poisoning. *Ambul Pediatr.* 2004;4:344–347
88. US Environmental Protection Agency. *Basis for Educational Recommendations on Reducing Childhood Lead Exposure*. Washington, DC: US Environmental Protection Agency; 2000. Publication EPA 747-R-00-001. Available at: www.epa.gov/opptintr/lead/pubs/reduc_pb.pdf. Accessed September 10, 2007
89. Haynes E, Lanphear BP, Tohn E, Farr N, Rhoads GG. The effect of interior lead hazard controls on children's blood lead concentrations: a systematic evaluation. *Environ Health Perspect.* 2002;110:103–107
90. Sandel M, Phelan K, Wright R, Hynes HP, Lanphear BP. The effects of housing interventions on child health. *Pediatr Ann.* 2004;33:474–478
91. Jordan DM, Yuse BL, Robinson LL, Hannan P, Deinard AS. A randomized trial of education to prevent lead burden in chil-

dren at high risk for lead exposure: efficacy as measured by blood lead monitoring. *Environ Health Perspect.* 2003;111: 1947–1951
92. Lanphear BP, Howard C, Eberly S, et al. Primary prevention of childhood lead exposure: a randomized trial of dust control. *Pediatrics.* 1999;103:772–777
93. Lanphear BP, Eberly S, Howard CR. Long-term effect of dust control on blood lead concentrations. *Pediatrics.* 2000;106(4). Available at: www.pediatrics.org/cgi/content/full/106/4/e48
94. Lanphear BP, Winter NL, Apetz L, Eberly S, Weitzman M. A randomized trial of the effect of dust control on children's blood lead levels. *Pediatrics.* 1996;98:35–40
95. Rhoads GG, Ettinger AS, Weisel CP, et al. The effect of dust control on blood lead in toddlers: a randomized trial. *Pediatrics.* 1999;103:551–555
96. Aschengrau A, Hardy S, Mackey P, Pultinas D. The impact of low technology lead hazard reduction activities among children with mildly elevated blood lead levels. *Environ Res.* 1998; 79:41–50
97. Liin LM, Lioy PJ, Rhoads GG. Impact of home carpets on childhood lead intervention study. *Environ Res.* 2003;92: 161–165
98. Ettinger AS, Bornschein RL, Farfel M, et al. Assessment of cleaning to control lead dust in homes of children with moderate lead poisoning: Treatment of Lead-Exposed Children Trial. *Environ Health Perspect.* 2002;110:A773–A779
99. Campbell C, Schwarz DF, Rich D, Dockery D. Effect of a follow-up professional home cleaning on serial dust and blood lead levels in urban children. *Arch Environ Health.* 2003; 58:771–780
100. Tohn ER, Dixon SL, Wilson JW, Galke WA, Clark CS. An evaluation of one-time professional cleaning in homes with lead-based paint hazards. *Appl Occup Environ Hyg.* 2003;18: 138–143
101. Centers for Disease Control and Prevention. *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials*. Atlanta, GA: Centers for Disease Control and Prevention; 1997
102. Advisory Committee on Childhood Lead Poisoning Prevention. Recommendations for blood lead screening of young children enrolled in Medicaid: targeting a group at high risk. *MMWR Recomm Rep.* 2000;49(RR-14):1–13
103. US General Accounting Office. *Lead Poisoning: Federal Health Care Programs Are Not Effectively Reaching At-Risk Children.* Washington, DC: US General Accounting Office; 1999. Publication GAO/HEHS-99-18
104. Kemper AR, Cohn LM, Fant KE, Dombkowski KJ. Blood lead testing among Medicaid-enrolled children in Michigan. *Arch Pediatr Adolesc Med.* 2005;159:646–650
105. Steinsapir C, Leighton J, Nagin D, Ehrlich J. Childhood lead poisoning: prevention and management. *City Health Inf.* 2004; 23(5):23–28
106. Philadelphia Department of Public Health, Childhood Lead Poisoning Prevention Program. *Recommendations for the Screening and Management of Young Children Potentially Exposed to Lead*. Philadelphia, PA: Philadelphia Department of Public Health; 1997
107. Chicago Department of Public Health. *Blood Lead Testing Guidelines for Chicago*. Chicago, IL: Department of Public Health; 1999. Available at: www.ci.chi.il.us/webportal/COCWebPortal/COC_EDITORIAL/ChicagoBLLTestingGuidelines.pdf. Accessed September 10, 2007
108. Robin LF, Beller M, Middaugh JP. Statewide assessment of lead poisoning and exposure risk among children receiving Medicaid services in Alaska. *Pediatrics.* 1997;99(4). Available at: www.pediatrics.org/cgi/content/full/99/4/e9
109. Centers for Disease Control and Prevention. Blood lead levels among children in a managed-care organization: California, October 1992–March 1993. *MMWR Morb Mortal Wkly Rep.* 1995;44:627–635
110. Binns HJ, LeBailly SA, Poncher J, Kinsella TR, Saunders SE, Pediatric Practice Research Group. Is there lead in the suburbs? Risk assessment in Chicago suburban pediatric practices. *Pediatrics.* 1994;93:164–171
111. Kazal LA Jr. The failure of CDC screening questionnaire to efficiently detect elevated lead levels in a rural population of children. *J Fam Pract.* 1997;45:515–518
112. Muniz MA, Dundas R, Mahoney MC. Evaluation of a childhood lead questionnaire in predicting elevated blood lead levels in a rural community. *J Rural Health.* 2003;19:15–19
113. France EK, Gitterman BA, Melinkovich P, Wright RA. The accuracy of a lead questionnaire in predicting elevated pediatric blood lead levels. *Arch Pediatr Adolesc Med.* 1996;150: 958–963
114. 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
115. Rooney BL, Hayes EB, Allen BK, Strutt PJ. Development of a screening tool for prediction of children at risk for lead exposure in a Midwestern clinical setting. *Pediatrics.* 1994;93: 183–187
116. Striph KB. Prevalence of lead poisoning in a suburban practice. *J Fam Pract.* 1995;41:65–71
117. Tejeda DM, Wyatt DD, Rostek BR, Solomon WB. Do questions about lead exposure predict elevated lead levels? *Pediatrics.* 1994;93:192–194
118. Snyder DC, Mohle-Boetani JC, Palla B, Fenstersheib M. Development of a population-specific risk assessment to predict elevated blood lead levels in Santa Clara County, California. *Pediatrics.* 1995;96:643–648
119. Schaffer SJ, Kincaid MS, Endres N, Weitzman M. Lead poisoning risk determination in a rural setting. *Pediatrics.* 1996; 97:84–90
120. Paulozzi LJ, Shapp J, Drawbaugh RE, Carney JK. Prevalence of lead poisoning among two-year-old children in Vermont. *Pediatrics.* 1995;96:78–81
121. Rolnick SJ, Nordin J, Cherney LM. A comparison of costs of universal versus targeted lead screening for young children. *Environ Res.* 1999;80:84–91
122. Dalton MA, Sargent JD, Stukel TA. Utility of a risk assessment questionnaire in identifying children with lead exposure. *Arch Pediatr Adolesc Med.* 1996;150:197–202
123. Casey R, Wiley C, Rutstein R, Pinto-Martin J. Prevalence of lead poisoning in an urban cohort of infants with high socioeconomic status. *Clin Pediatr (Phila).* 1994;33:480–484
124. 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
125. Bronson MA, Tilden RL, Renier CM. Community-based screening for childhood lead poisoning: identification of risk factors and susceptible populations in Duluth. *Minn Med.* 1999;82:25–29
126. 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
127. Schonfeld DJ, Rainey PM, Cullen MR, Showalter DR, Cicchetti DV. Screening for lead poisoning by fingerstick in suburban pediatric practices. *Arch Pediatr Adolesc Med.* 1995;149: 447–450

Interpreting and Managing Blood Lead Levels of Less Than 10 µg/dL in Children and Reducing Childhood Exposure to Lead: Recommendations of the Centers for Disease Control and Prevention Advisory Committee on Childhood Lead Poisoning Prevention

Helen J. Binns, Carla Campbell, Mary Jean Brown and for the Advisory Committee on Childhood Lead Poisoning Prevention

Pediatrics 2007;120;e1285-e1298
DOI: 10.1542/peds.2005-1770