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**POLICY STATEMENT**

# Lead Exposure in Children: Prevention, Detection, and Management

**Committee on Environmental Health**

## ABSTRACT

Fatal lead encephalopathy has disappeared and blood lead concentrations have decreased in US children, but approximately 25% still live in housing with deteriorated lead-based paint and are at risk of lead exposure with resulting cognitive impairment and other sequelae. Evidence continues to accrue that commonly encountered blood lead concentrations, even those less than 10 µg/dL, may impair cognition, and there is no threshold yet identified for this effect. Most US children are at sufficient risk that they should have their blood lead concentration measured at least once. There is now evidence-based guidance available for managing children with increased lead exposure. Housing stabilization and repair can interrupt exposure in most cases. The focus in childhood lead-poisoning policy, however, should shift from case identification and management to primary prevention, with a goal of safe housing for all children.

**Key Words:** child • lead • environmental exposure • chelation therapy • succimer • cognition • clinical trials • housing • prevention • behavior

**Abbreviations:** CDC, Centers for Disease Control and Prevention • AAP, American Academy of Pediatrics • EPA, Environmental Protection Agency • CNS, central nervous system • EP, erythrocyte protoporphyrin • EDTA, ethylenediaminetetraacetic acid • TLC, Treatment of Lead-Exposed Children • HUD, Department of Housing and Urban Development

## BACKGROUND

In 1991, when 1 in 11 US children had a blood lead concentration greater than 10 µg/dL, both the Centers for Disease Control and Prevention (CDC) and the American Academy of Pediatrics (AAP) recommended that all US children have their blood lead concentration measured at around 1 and 2 years of age, when concentrations increase and then peak. By 1997, the median blood lead concentration in the United States had decreased, and screening in some areas with newer housing turned up few cases of elevated blood lead concentration. The CDC and AAP then began to recommend screening only those children with a greater chance of having an elevated blood lead concentration—those in older housing, those who had a sibling or playmate with an elevated blood lead concentration, or those who had lived in or visited a structure that might contain deteriorated, damaged, or recently remodeled lead-painted surfaces. Screening of all children eligible for Medicaid, among whom were found 80% of those with increased blood lead concentration,[1] continued to be recommended and had been required by Health Care Financing Administration (now the Centers for Medicare and Medicaid Services) regulation since 1989.

This new policy statement replaces the 1998 statement and includes discussion of new data, including:

- Reliable estimates of the percentage of the US homes containing lead hazards[2];
- Results from a large clinical trial showing that chelation in children with moderately elevated blood lead concentrations does not improve cognitive or neuropsychologic test scores[3];
- Documentation of unacceptably low screening rates among Medicaid-eligible children[4];
- Further confirmation of the link between lead exposure in early childhood and delinquent behavior during adolescence[5,6]; and
- New data showing inverse associations between blood lead concentrations less than 10 µg/dL and IQ.[7,8]

The best approach to lead poisoning is to prevent exposure in the first place, but it will be years before that goal is realized. In the meantime, case finding, case management, and prevention of additional exposure will still be required. This document considers relevant aspects of the epidemiology, clinical toxicology, prevention, and treatment of lead exposure in young children and provides recommendations for pediatricians as well as public health authorities.

## DECLINE OF LEAD POISONING IN THE UNITED STATES

Lead is an element and occurs naturally, but blood lead concentrations are quite low in the absence of industrial activities.[9] In the United States, there were historically 2 major sources of industrially derived lead for children: airborne lead, mostly from the combustion of gasoline containing tetraethyl lead; and leaded chips and dust, mostly from deteriorating lead paint. Both contribute to soil lead. A steep decrease in exposure to airborne lead in the United States has occurred since 1980. Federal legislation in the 1970s removed lead from gasoline and decreased smokestack emissions from smelters and other sources, causing blood lead concentrations in children to decrease. From 1976 to 1980, before the regulations had their full effect, US children 1 to 5 years of age had a median blood lead concentration of 15 µg/dL.[10] In 1988–1991, the median was 3.6 µg/dL[11]; in 1999, the median was 1.9 µg/dL.[12] Although concentrations have decreased in all children, black children and poor children continue to have higher blood lead concentrations. Airborne lead should no longer be a source of community exposure in the United States, but individual counties sometimes still exceed airborne lead regulations, and continued vigilance is warranted. Individual children may still be exposed to airborne lead in fumes or respirable dust resulting from sanding or heating old paint, burning or melting automobile batteries, or melting lead for use in a hobby or craft.

## SOURCES OF LEAD EXPOSURE

### Lead Paint, Dust, and Soil

The source of most lead poisoning in children now is dust and chips from deteriorating lead paint on interior surfaces.[13] Children who developed lead encephalopathy with blood lead concentrations more than 100 µg/dL often had chips of lead paint visible on abdominal plain films. Children who live in homes with deteriorating lead paint, however, can achieve blood lead concentrations of 20 µg/dL or greater without frank pica.[14] The use of leaded paint on interior surfaces ceased in the United States by the mid-1970s. However, in 1998, of the 16.4 million US homes with ≥1 child younger than 6 years, 25% still had significant amounts of lead-contaminated deteriorated paint, dust, or adjacent bare soil ("lead hazard").[2] Dust and soil are also a final resting place for airborne lead from gasoline and dust from paint. Lead in dust and soil can recontaminate cleaned houses[15] and contribute to elevating blood lead concentrations in children who play on bare, contaminated soil.[16]

### Transplacental Exposure and Lead in Human Milk

Lead crosses the placenta, and the blood lead concentration of the infant is similar to that of the mother.[17] The source of lead in the infant's blood seems to be a mixture of approximately two thirds dietary and one third skeletal lead, as shown by studies that exploited the differences in lead isotopes stored in the bones of women

migrating from Europe to Australia.[18] Although lead appears in human milk, the concentration is closer to plasma lead and much lower than blood lead, so little is transferred. Because infant formula and other foods for infants also contain lead, women with commonly encountered blood lead concentrations who breastfeed their infants expose them to slightly less lead than if they do not breastfeed.[19] In Mexico, giving women supplemental calcium during lactation resulted in a small (less than 2 µg/dL) decrease in the mother's blood lead concentration, presumably by decreasing skeletal resorption.[20] Theoretically, this could diminish transfer of lead through breast milk even further. In the United States, however, where calcium intake may be higher, calcium supplementation does not prevent bone loss during lactation[21] and, thus, might not affect lead transfer at all.

**Other Sources**

Lead plumbing (in Latin, "plumbus" = lead) has contaminated drinking water for centuries, and lead in water can contribute to elevated blood lead concentrations in children.[13] In 2003–2004, some tap water in Washington, DC, was found to exceed Environmental Protection Agency (EPA) regulations. This was thought to be caused by a change in water disinfection procedures, which increased the water's ability to leach lead from connector pipes between the water mains and interior plumbing in old houses. The extent of this problem in Washington and other cities is not yet known. Affected families are drinking filtered or bottled water until the pipes can be replaced. (Most bottled water is not fluoridated; its consumption may lead to marginal fluoride intakes in children.) Much more about lead in drinking water is available on the EPA Web site (www.epa.gov/safewater/lead/index.html).

Table 1 includes questions about less common sources of lead exposure, which include hobbies, contaminated work clothes, ceramics, cosmetics, imported canned foods, etc. Such questions may be useful if a child has an elevated blood lead concentration but no exposure to leaded dust or soil. They have not been validated for the purpose of deciding whether to screen.

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**TABLE 1.** Suggested Clinical Evaluation for Lead Exposure

The lead concentration of blood for transfusion is not routinely measured. After exchange transfusion in the extremely low birth weight infant, 90% of the infant's blood is donor blood. Bearer et al[22] recommended that only units with lead concentrations of less than 0.09 µmol/L be used in these patients, on the basis of their adaptation of the World Health Organization tolerable weekly intake from ingestion to intravenous injection. Approximately one third of the units of blood that they measured were above this concentration. The effect of lead in transfused blood used in older children has not been considered.

# TOXICITY OF LEAD

**Subclinical Effects**

At the levels of lead exposure now seen in the United States, subclinical effects on the central nervous system (CNS) are the most common effects. The best-studied effect is cognitive impairment, measured by IQ tests. The strength of this association and its time course have been observed to be similar in multiple studies in several countries.[23] In most countries, including the United States, blood lead concentrations peak at approximately 2 years of age and then decrease without intervention. Blood lead concentration is associated with lower IQ scores as IQ becomes testable reliably, which is at approximately 5 years of age.[23] The strength of the association is similar from study to study; as blood lead concentrations increase by 10 µg/dL, the IQ at 5 years of age and later

decreases by 2 to 3 points. Canfield et al[7] recently extended the relationship between blood lead concentration and IQ to blood lead concentrations less than 10 µg/dL. They observed a decrease in IQ of more than 7 points over the first 10 µg/dL of lifetime average blood lead concentration. Bellinger and Needleman[8] subsequently reported a similarly steep slope in a reanalysis of data from their study of children with blood lead concentrations similar to those in the Canfield et al study. To confirm the adverse effects of lead on IQ at these concentrations, however, more children whose blood lead concentration has never been more than 10 µg/dL should be studied. A reanalysis of the primary data from several of the prospective studies is underway to help resolve this issue. At the moment, however, these data have not yet been incorporated into policy, and the CDC[16] and AAP[24] both currently use 10 µg/dL (Table 2) as the blood lead concentration of concern.

| View this table: [in this window] [in a new window] | **TABLE 2.** Summary of Recommendations for Children With Confirmed (Venous) Elevated Blood Lead Concentrations[16] |
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Other aspects of brain or nerve function, especially behavior, also may be affected. Teachers reported that students with elevated tooth lead concentrations were more inattentive, hyperactive, disorganized, and less able to follow directions.[25,26] Additional follow-up of some of those children[25] showed higher rates of failure to graduate from high school, reading disabilities, and greater absenteeism in the final year of high school.[27] Elevated bone lead concentrations are associated with increased attentional dysfunction, aggression, and delinquency.[28] In children followed from infancy with blood lead measurements, self-reported delinquent behavior at 15 to 17 years of age increased with both prenatal and postnatal lead exposure,[5] and bone lead, thought to represent cumulative dose, is higher in adjudicated delinquents.[6] These data imply that the effects of lead exposure are long lasting and perhaps permanent. Subclinical effects on both hearing[29] and balance[30] may occur at commonly encountered blood lead concentrations.

Although there are reasonable animal models of low-dose lead exposure and cognition and behavior,[31] the mechanisms by which lead affects CNS function are not known. Lead alters very basic nervous system functions, such as calcium-modulated signaling, at very low concentrations in vitro,[32] but it is not yet clear whether this process or some other one yet to be examined is the crucial one. Lead interferes detectably with heme synthesis beginning at blood lead concentrations of approximately 25 µg/dL.[33] Both aminolevulinate dehydratase, an early step enzyme, and ferrochelatase, which completes the heme ring, are inhibited. Ferrochelatase inhibition is the basis of an erstwhile screening test for lead poisoning that measures erythrocyte protoporphyrin (EP), the immediate heme precursor. Because it is insensitive to the lower concentrations of blood lead that are of concern now, the test is obsolete for that use; however, EP measurement is still used clinically in managing children with higher blood lead concentrations.

### Clinical Effects

Children with blood lead concentrations greater than 60 µg/dL may complain of headaches, abdominal pain, loss of appetite, and constipation and display clumsiness, agitation, and/or decreased activity and somnolence. These are premonitory symptoms of CNS involvement and may rapidly proceed to vomiting, stupor, and convulsions.[34] Symptomatic lead toxicity should be treated as an emergency. Although lead can cause clinically important colic, peripheral neuropathy, and chronic renal disease in adults with occupational exposures, these symptoms are rare in children.

**Reversibility**

In an influential 1994 study, 154 children who were 13 to 87 months old and had blood lead concentrations between 25 and 55 µg/dL were given chelation with ethylenediaminetetraacetic acid (EDTA) and therapeutic iron when clinically indicated and then followed for 6 months. Those whose blood lead concentrations decreased the most had improved cognitive test scores independent of whether they had been given iron or chelation therapy.[35] An Australian study[36] of 375 children with longer follow-up, however, found only small and inconsistent improvement in the IQs of children whose blood lead concentrations decreased the most. A large (780-children) randomized trial of the use of succimer in children with blood lead concentrations of 20 to 44 µg/dL, the Treatment of Lead-Exposed Children (TLC)[3] Trial, showed no benefit on cognitive or neuropsychologic testing despite an abrupt but transient decrease in the treated children's blood lead concentrations. The children were randomly assigned at approximately 2 years of age and followed with cognitive, neuropsychologic, and behavioral tests until they were approximately 5 years of age. The large size of the trial permits confident exclusion of a drug-related improvement of 2 IQ points or more. Additional follow-up at 7 years of age with more sophisticated testing still showed no advantage for the succimer-treated children.[37]

Because blood lead concentrations decreased as the children in the TLC Trial got older regardless of whether they had chelation, Liu et al[38] used the TLC data to attempt to replicate the reported relationship between decreasing blood lead concentrations and improved cognitive test scores. Test scores were unrelated to decreasing blood lead concentrations at 6 months' follow-up, but results from following the children for 36 months, when they were approximately 5 years of age, showed improved test scores with greater decreases in blood lead concentration but only in the placebo group. Additional research on whether some effective intervention can be isolated to account for this phenomenon is needed. There remains no evidence that chelation will reverse cognitive impairment, and the predominance of data is consistent with a noncausal association between decreasing blood lead concentrations and improved cognitive test scores.

## COSTS OF CHILDHOOD LEAD POISONING AND BENEFITS OF PREVENTION

**Cost-Benefit Analyses**

The removal of lead from gasoline cost money, and it will cost more money to remove lead from housing. If childhood lead exposure, however, affects cognitive function and its consequences, such as graduating from high school, then it is plausible that it will affect social function, employment, and earnings. Several groups have estimated the long-term dollar costs of childhood lead exposure, assuming that the effect of lead on IQ is linear and permanent; they also assume a specific economic value of increased IQs. Grosse et al[39] estimated the economic benefit of the 25-year secular downward trend in childhood lead exposure in the cohort of children 2 years of age in 2000. The estimated increase in earnings for the 3.8 million children would be between $110 billion and $319 billion over their lifetimes, compared with what they would have earned if they had been exposed to 1975 lead levels. Landrigan et al[40] estimated the lifetime costs for each year's cohort of children currently exposed to lead to be $43 billion. On the cost side, Needleman[41] estimated a $10 billion cost for deleading the estimated 2 million lead-contaminated houses that existed in 1990. In 2002, a more reliable estimate is that there are 4 million such lead-contaminated houses,[2] and when adjusting for inflation (with the Consumer Price Index inflation calculator [www.bls.gov/cpi]), Needleman's estimate becomes approximately $28 billion in 2002. Combining these estimates leads to the conclusion that removing lead paint is cost-effective if it prevents even two thirds of lead exposure for any single year's cohort of 2-year-olds. Similarly, a presidential task force estimated that the net nationwide benefit of interim control of lead hazards in the nation's pre-1960 housing would be $1 billion to $9 billion over 10 years. The benefit of abating the hazards permanently would be $21 billion to $38 billion. Such quantitation allows planning and setting priorities to be done more transparently and allows comparisons to estimates of the cost for lead-abatement programs and other preventive activities. Although these are exemplary numbers in simplified analyses, all parts of which could be

challenged, they illustrate the rationale for viewing lead exposure as a problem that should be solved, even on economic grounds.

**Federal Strategy to Prevent Lead Poisoning**

The President's Task Force on Environmental Health Risks and Safety Risks to Children was formed in 1997 by executive order. It consists of government officials from the EPA, the Department of Health and Human Services, the Consumer Product Safety Commission, the Department of Housing and Urban Development (HUD), and others. One of its first projects was to formulate a plan to eliminate childhood lead poisoning,[42] a goal that was incorporated into the Healthy People 2010 goals for the nation (www.healthypeople.gov/Document/HTML/Volume1/08Environmental.htm#_Toc490564710). For the first time, the strategy concentrated on primary prevention and was directed at housing. It did not require that a lead-poisoned child first be identified before a house was considered eligible for participation (the principle of primary prevention). The core of the strategy is a grant-based program administered by the HUD that would accelerate the pace at which in-place management of lead hazards would occur in US homes. The strategy projected that more than 20 million houses could be remediated in the decade from 2000–2010, making lead-safe housing available to a large majority of US children. The strategy also included continued screening, especially among Medicaid-eligible children, enforcement of existing statutes and regulations, and research, especially on the effectiveness of in-place management of lead hazards. The HUD plans periodic evaluations and progress reports, which can be tracked on its Web site (www.hud.gov/offices/lead).

## DIAGNOSTIC MEASURES

The diagnosis of lead poisoning or increased lead absorption depends on the measurement of blood lead concentration. This is best performed by using a venous sample, but a carefully collected finger-stick sample can be used. Most blood lead measurements are now performed because the child meets some general eligibility criteria (screening) and not because they are at especially high risk of exposure or have symptoms suggestive of lead poisoning (diagnosis).

**Screening**

Between 1991 and 1997, both the AAP and CDC recommended universal screening, that is, that all children have their blood lead concentration measured, preferably when they are 1 and 2 years of age. Because the prevalence of elevated blood lead concentrations has decreased so much, a shift toward targeted screening has begun,[43] and the criteria for and implementation of targeted screening continues to develop. As of early 2005, the situation is as follows. All Medicaid-eligible children must be screened.[4] Medicaid will reimburse 2 screenings, one at 1 year of age and one at 2 years of age. Most children with elevated blood lead concentrations are Medicaid eligible, and most Medicaid-eligible children have not been screened.[4] The Advisory Committee on Childhood Lead Poisoning Prevention has proposed criteria by which a state could acquire an exemption from this requirement, and the proposal is under consideration in the Secretary of Health and Human Services' office. Until such exemptions are granted, both the CDC[4] and AAP support universal screening of Medicaid-eligible children. The thinking behind the availability of exemptions is not primarily to decrease the number of screenings performed but rather to increase it among groups in which increased lead absorption will be found. Children whose families participate in any assistance program but who, for whatever reason, are not eligible for Medicaid should also be screened.

For children not eligible for Medicaid, several states and some municipalities have developed targeted screening recommendations or policies using suggestions made by the CDC,[43] their own data, or some combination of the 2. All practitioners should determine if such recommendations are in place where they practice. Appropriate

contacts at state and city health departments with CDC-funded programs are listed on the CDC Web site (www.cdc.gov/nceh/lead/grants/contacts/CLPPP%20Map.htm).

The approach to screening children who are not eligible for Medicaid and who live in areas in which health authorities have not made locale-specific recommendations is less clear. Although targeted screening may be desirable, well-validated tools with which to achieve it are not yet in place.[44] In the absence of policy, current recommendations support screening all children who are not enrolled in Medicaid and who live in areas in which local authorities have not issued specific guidance.

There are now many case reports of children who are recent immigrants, refugees, or international adoptees who have elevated (sometimes very elevated) blood lead concentrations.[45] Such children should be screened on arrival in the United States.

**Diagnostic Testing**

Some experienced clinicians measure the blood lead concentration in children with growth retardation, speech or language dysfunction, anemia, and attentional or behavioral disorders, especially if the parents have a specific interest in lead or in health effects from environmental chemicals. However, a persistent elevation of blood lead concentration into school age is unusual, even if peak blood lead concentration at 2 years of age was high and the child's housing has not been abated. This is probably because hand-to-mouth activity decreases and the child's body mass increases. Thus, a low blood lead concentration in a school-aged child does not rule out earlier lead poisoning. If the question of current lead poisoning arises, however, the only reliable way to make a diagnosis is with a blood lead measurement. Hair lead concentration gives no useful information and should not be performed.[46] Radiograph fluorescence measurement of lead in bone is available in a few research centers and has been used in children as young as 11 years with acceptable validity for research purposes,[47] but it has no clinical utility as yet.

## MANAGEMENT OF CHILDREN WITH ELEVATED BLOOD LEAD CONCENTRATIONS

In 2002, the national Advisory Committee on Childhood Lead Poisoning Prevention published a monograph, "Managing Elevated Blood Lead Levels Among Young Children."[16] The goal of the monograph was to provide an evidence-based, standard approach to management usable throughout the United States. Anyone involved with the management of children with elevated blood lead concentrations needs access to it. This section is consistent with the monograph.

The management of children with elevated blood lead concentrations is determined primarily by how high the concentration is (Table 2). Children with concentrations less than 10 µg/dL are not currently considered to have excess lead exposure. Children with concentrations 10 µg/dL or greater should have their concentrations rechecked; if many children in a community have concentrations greater than 10 µg/dL, the situation requires investigation for some controllable source of lead exposure. Children who ever have a concentration greater than 20 µg/dL or persistently (for more than 3 months) have a concentration greater than 15 µg/dL require environmental and medical evaluation.

**Residential Lead Exposure**

Most children with elevated blood lead concentrations live in or regularly visit a home with deteriorating lead paint on interior surfaces. Some children eat paint chips, but pica is not necessary to achieve blood lead concentrations of 20 µg/dL or greater.[14] Children can ingest lead-laden dust through normal mouthing behaviors by simply placing their hand or an object in their mouth. This also happens when children handle food during eating.[48–4950] There is increasing evidence that professional cleaning, paint stabilization, and removal and

replacement of building components can interrupt exposure. Cooperation with the health department in investigating and decreasing the source is necessary. Although some authorities insist that moving children to unleaded housing or removal of all lead paint from their current housing is the only acceptable solution,[51] alternative housing is rarely available and extensive on-site removal of leaded paint can raise the concentration in house dust and resident children.[52]

Lead in soil is higher around houses with exterior lead paint and in places where there has been a smokestack or other point source or heavy traffic. Soil concentrations are related to blood lead concentrations but not as closely as are interior dust lead concentrations.[13] Soil can be tested for lead content, and the EPA has guidelines for testing on its Web site (www.epa.gov/lead/leadtest.pdf). Lead should no longer be a problem in municipal water supplies, but wells, old pipes from the municipal supply to the house (as has been the case in Washington, DC), or soldered joints may add lead to water (see www.epa.gov/safewater/lead/index.html).

**Other Sources**

Some children will have persistently elevated blood lead concentrations without access to lead paint, bare soil, or lead in their drinking water. Their exposure may come from any of the sources listed in Table 3. Blood lead concentrations should decrease as the child passes approximately 2 years of age, and a stable or increasing blood lead concentration beyond that age is likely to be caused by ongoing exposure.

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| **View this table:** [in this window] [in a new window] | **TABLE 3.** Sources of Lead Exposure and Prevention Strategies[59] |

The recommended approach to environmental investigation of a child with an elevated blood lead concentration consists of (1) an environmental history, such as the one shown in Table 1, (2) an inspection of the child's primary residence and any building in which they spend time regularly, (3) measurement of lead in deteriorated paint, dust, bare soil, or water as appropriate, (4) control of any immediate hazard, and (5) remediation of the house, which may require temporary relocation of the child. If new or lead-safe housing is an option for the family, it offers a simple and permanent solution. These situations can be frightening for the families. Involving the family and providing them with information as it is obtained is the right thing to do and may help lessen anxiety.

Although intense regimens of professional cleaning decrease children's blood lead concentrations, providing families with instructions and cleaning materials does not. Washing children's hands has intuitive appeal, but no data support its role in decreasing exposure. Suggested prevention strategies are listed in Table 3.

**Medical Management**

If the blood lead concentration is greater than 45 µg/dL and the exposure has been controlled, treatment with succimer should begin. A pediatrician experienced in managing children with lead poisoning should be consulted; these pediatricians can be found through state health department lead programs, through pediatric environmental health specialty units (www.aoec.org/pehsu.htm), at hospitals that participated in the largest clinical trial of succimer,[3] or by calling the local poison control center or the AAP Committee on Environmental Health. The most common adverse effects of succimer listed on the label are abdominal distress, transient rash, elevated hepatocellular enzyme concentrations, and neutropenia. The drug is unpleasant to administer because of a strong "rotten-egg" odor, and 40% of the families on active drug compared with 26% on placebo found the drug difficult to administer.[53] The succimer label provides dosages calculated both by body surface area and by

weight, but the equivalent dose by both methods would occur in a child approximately 5 years of age. For the younger children typically given the drug, body surface area calculations give higher doses, which are those that are recommended.[54]

Although chelation therapy for children with blood lead concentrations of 20 to 44 µg/dL can be expected to lower blood lead concentrations, it does not reverse or diminish cognitive impairment or other behavioral or neuropsychologic effects of lead.[3] There are no data supporting the use of succimer in children whose blood lead concentrations are less than 45 µg/dL if the goal is to improve cognitive test scores.

Children with symptoms of lead poisoning, with blood lead concentrations higher than 70 µg/dL, or who are allergic or react to succimer will need parenteral therapy with EDTA and hospitalization. Guidelines for these circumstances are beyond the scope of this statement, but the same consultation as described above is recommended. There are academic centers that use D-penicillamine, another oral chelator used in Wilson disease, for lead poisoning. Its safety and efficacy, however, have not been established,[55] and the AAP Committee on Drugs considers it to be a third-line drug for lead poisoning.[56]

**Dietary Intervention**

The Advisory Committee on Childhood Lead Poisoning Prevention reviewed the evidence for dietary intervention in lead-exposed children.[16] They concluded that there are no trial data supporting dietary interventions aimed specifically at preventing lead absorption or modulating the effects of lead. However, there are laboratory and clinical data suggesting that adequate intake of iron, calcium, and vitamin C are especially important for these children. Adequate iron and calcium stores may decrease lead absorption, and vitamin C may increase renal excretion. Although there is epidemiologic evidence that diets higher in fat and total calories are associated with higher blood lead concentrations at 1 year of age,[57] the absence of trial data showing benefits and the caloric requirements of children at this age preclude recommending low-fat diets for them.

**Psychological Assessment**

The Advisory Committee on Childhood Lead Poisoning Prevention reviewed the evidence for psychological assessment and intervention in lead-exposed children.[16] Despite data from several large epidemiologic studies suggesting that moderate exposure to lead produces specific deficits in attention or executive functions, visual-spatial skills, fine-motor coordination, balance, and social-behavioral modulation,[58] there is no specific "signature" syndrome yet identified. In addition, although 2-year-olds tend to have the highest blood lead concentrations, they will usually not have detectable cognitive damage, which can be expected to become more apparent at 4 years of age and later. It seems reasonable to manage children whose blood lead concentration is 20 µg/dL or greater at its peak as having a higher risk of developmental delay and behavior abnormalities.[16] Because the effects emerge later, after the child's blood lead concentration will have decreased, the child's record must be kept open even after the blood lead concentration has decreased.

Although there is not specific literature supporting the use of enrichment programs in lead-poisoned children, programs aimed at children with delay from another cause should be effective in lead-poisoned children.

## RECOMMENDATIONS FOR PEDIATRICIANS

1. Provide anticipatory guidance to parents of all infants and toddlers about preventing lead poisoning in their children. In particular, parents of children 6 months to 3 years of age should be made aware of normal mouthing behavior and should ascertain whether their homes, work, or hobbies present a lead

hazard to their toddler. Inform parents that lead can be invisibly present in dust and can be ingested by children when they put hands and toys in their mouths.

2. Inquire about lead hazards in housing and child care settings, as is done for fire and safety hazards or allergens. If suspicion arises about the existence of a lead hazard, the child's home should be inspected. Generally, health departments are capable of inspecting housing for lead hazards. Expert training is needed for safe repair of lead hazards, and pediatricians should discourage families from undertaking repairs on their own. Children should be kept away from remediation activities, and the house should be tested for lead content before the child returns.
3. Know state Medicaid regulations and measure blood lead concentration in Medicaid-eligible children. If Medicaid-eligible children are a significant part of a pediatrician's practice or if a pediatrician has an interest in lead poisoning, he or she should consider participating in any deliberations at the state and local levels concerning an exemption from the universal screening requirement.
4. Find out if there is relevant guidance from the city or state health department about screening children not eligible for Medicaid. If there is none, consider screening all children. Children should be tested at least once when they are 2 years of age or, ideally, twice, at 1 and 2 years of age, unless lead exposure can be confidently excluded. Pediatricians should recognize that measuring blood lead concentration only at 2 years of age, when blood lead concentration usually peaks, may be too late to prevent peak exposure. Earlier screening, usually at 1 year of age, should be considered where exposure is likely. A low blood concentration in a 1-year-old, however, does not preclude elevation later, so the test should be repeated at 2 years of age. Managed health care organizations and third-party payers should fully cover the costs of screening and follow-up. Local practitioners should work with state, county, or local health authorities to develop sensitive, customized questions appropriate to the housing and hazards encountered locally.
5. Be aware of any special risk groups that are prevalent locally, such as immigrants, foreign-born adoptees, refugees, or children whose parents work with lead or lead dust in their occupation or hobby and, of course, those who live in, visit, or work on old houses.
6. In areas with old housing and lead hazards, encourage application for HUD or other moneys available for remediation.
7. Keep current with the work of the national Advisory Committee on Childhood Lead Poisoning Prevention and any relevant local committees. Although there is now evidence that even lower blood lead concentrations may pose adverse effects to children, there is little experience in the management of excess lead exposure in these children. Although most of the recommendations concerning case management of children with blood lead concentrations of 15 µg/dL should be appropriate for children with lower concentrations, tactics that decrease blood lead concentrations might be expected to be less and less effective as they are applied to children with lower and lower blood lead concentrations.

## RECOMMENDATIONS FOR GOVERNMENT

1. Identify all children with excess lead exposure, and prevent further exposure to them. The AAP supports the efforts of individual states to design targeted screening programs, even for Medicaid children. However, the goal must be to find all children with excess exposure and interrupt that exposure, not simply to screen less. To do this, state and local government activities must focus on the children who are most at risk, which requires more and better data about the prevalence of elevated blood lead concentrations in specific communities. Prevalence estimates based on convenience samples or clinic attendees are not reliable and should not be used as the basis of policy.
2. Realize that case-finding per se will not decrease the risk of lead poisoning. It must be coupled with public health programs including environmental investigation, transitional lead-safe housing assistance,

and follow-up for individual cases. Lead-screening programs in high-risk areas should be integrated with other housing and public health activities and with facilities for medical management and treatment.

3. Continue commitment to the Healthy People 2010 goal of eliminating lead poisoning by 2010. The AAP supports the current plan with emphasis on lead-safe housing. Continued monitoring and commitment will be necessary. Research findings on low-cost methods of remediating housing have become controversial. The federal government should support impartial scientific and ethical inquiry into the best way to carry out the needed research.
4. Minimize the further entry of lead into the environment. Regulations concerning airborne lead should be enforced, use of lead in consumer products should be minimized, and consideration should always be given to whether a child might come into contact with such a product.
5. Encourage scientific testing of the many simple, low-cost strategies that might decrease lead exposure. Examples include hand-washing and use of high chairs. Exploration of innovative, low-technology tactics should be encouraged, perhaps through the use of special study sections or review groups. Educational resources for parents and landlords need to be developed and tested.
6. Require coverage of lead testing for at-risk children by all third-party payers by statute or regulation.
7. Fund studies to confirm or refute the finding that blood lead concentrations of less than 10 µg/dL are associated with lower IQ. The next important step in lead research is conducting of studies in which confounding by socioeconomic factors is not so strong. Funding of studies in this area needs to be given high priority, as was done in the early 1980s when the question of effects of blood lead concentrations less than 20 µg/dL was raised.
8. Gather the nationally representative data necessary for a rational public health response to the problem of childhood lead poisoning. The federal government should continue measuring children's blood lead concentrations in the National Health and Nutrition Surveys to allow national estimates of exposure and should periodically resurvey housing to measure progress in the reduction of lead-paint hazards. In addition, state governments can improve monitoring of trends among screened children by supporting electronic reporting of blood lead test results to the CDC.

## Committee on Environmental Health, 2004–2005

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**TABLE 1.** Suggested Clinical Evaluation for Lead Exposure

| |
|---|
| Medical history |
| Ask about |
| Symptoms |
| Developmental history |
| Mouthing activities |
| Pica |
| Previous blood lead concentration measurements |
| Family history of lead poisoning |
| Environmental history |
| Paint and soil exposure |
| What is the age and general condition of the residence or other structure in which the child spends time? |
| Is there evidence of chewed or peeling paint on woodwork, furniture, or toys? |
| How long has the family lived at that residence? |
| Have there been recent renovations or repairs to the house? |
| Are the windows new? |
| Are there other sites at which the child spends significant amounts of time? |
| What is the condition/make-up of indoor play areas? |
| Do outdoor play areas contain bare soil that may be contaminated? |
| How does the family attempt to control dust and dirt? |
| Relevant behavioral characteristics of the child |
| To what degree does the child exhibit hand-to-mouth activity? |
| Does the child exhibit pica? |
| Are the child's hands washed before meals and snacks? |
| Exposures to and behaviors of household members |
| What are the occupations of adult household members? |
| What are the hobbies of household members? (Fishing, working with ceramics or stained glass, and hunting are examples of hobbies that involve risk for lead exposure.) |
| Are painted materials or unusual materials burned in household fireplaces? |
| Miscellaneous |
| Does the home contain vinyl miniblinds made overseas and purchased before 1997? |
| Does the child receive or have access to imported food, cosmetics, or folk remedies? |
| Is food prepared or stored in imported pottery or metal vessels? |

| |
|---|
| Does the family use imported foods in soldered cans? |
| Nutritional history |
| Take a dietary history |
| Evaluate the child's iron status by using the appropriate laboratory tests |
| Ask about history of food stamps or participation in the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) |
| Physical examination |
| Pay particular attention to the neurologic examination and the child's psychosocial and language development |

**TABLE 2.** Summary of Recommendations for Children With Confirmed (Venous) Elevated Blood Lead Concentrations[16]

| Blood Lead Concentration | Recommendations |
|---|---|
| 10–14 µg/dL | Lead education |
| | Dietary |
| | Environmental |
| | Follow-up blood lead monitoring |
| 15–19 µg/dL | Lead education |
| | Dietary |
| | Environmental |
| | Follow-up blood lead monitoring |
| | Proceed according to actions for 20–44 µg/dL if |
| | A follow-up blood lead concentration is in this range at least 3 months after initial venous test; or |
| | Blood lead concentration increases |
| 20–44 µg/dL | Lead education |
| | Dietary |
| | Environmental |
| | Follow-up blood lead monitoring |
| | Complete history and physical examination |
| | Lab work |
| | Hemoglobin or hematocrit |
| | Iron status |

| | |
|---|---|
| | Environmental investigation |
| | Lead hazard reduction |
| | Neurodevelopmental monitoring |
| | Abdominal radiography (if particulate lead ingestion is suspected) with bowel decontamination if indicated |
| 45–69 µg/dL | Lead education |
| | Dietary |
| | Environmental |
| | Follow-up blood lead monitoring |
| | Complete history and physical examination |
| | Lab work |
| | Hemoglobin or hematocrit |
| | Iron status |
| | Free EP or ZPP |
| | Environmental investigation |
| | Lead hazard reduction |
| | Neurodevelopmental monitoring |
| | Abdominal radiography with bowel decontamination if indicated |
| | Chelation therapy |
| ≥70 µg/dL | Hospitalize and commence chelation therapy |
| | Proceed according to actions for 45–69 µg/dL |

| Not Recommended at Any Blood Lead Concentration |
|---|
| Searching for gingival lead lines |
| Evaluation of renal function (except during chelation with EDTA) |
| Testing of hair, teeth, or fingernails for lead |
| Radiographic imaging of long bones |
| X-ray fluorescence of long bones |

ZPP indicates zinc protoporphyrin.

**TABLE 3.** Sources of Lead Exposure and Prevention Strategies[59]

| Source | Prevention Strategy |
|---|---|

| | |
|---|---|
| Environmental | |
| Paint | Identify and abate |
| Dust | Wet mop (assuming abatement) |
| Soil | Restrict play in area, plant ground cover, wash hands frequently |
| Drinking water | Flush cold-water pipes by running the water until it becomes as cold as it will get (a few seconds to 2 minutes or more; use cold water for cooking and drinking |
| Folk remedies | Avoid use |
| Cosmetics containing additives such as kohl or surma | Avoid use |
| Old ceramic or pewter cookware, old urns/kettles | Avoid use |
| Some imported cosmetics, toys, crayons | Avoid use |
| Contaminated mineral supplements | Avoid use |
| Parental occupations | Remove work clothing at work; wash work clothes separately |
| Hobbies | Proper use, storage, and ventilation |
| Home renovation | Proper containment, ventilation |
| Buying or renting a new home | Inquire about lead hazards |
| Lead dust in carpet | Cover or discard |
| Host | |
| Hand-to-mouth activity (or pica) | Frequent hand washing; minimize food on floor |
| Inadequate nutrition | Adequate intake of calcium, iron, vitamin C |
| Developmental disabilities | Enrichment programs |