

Planet

Pediatric Lead Assessment Network Education Training



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- Ohio is among the top five states with the highest number of housing units with lead-based paint (US EPA)
- Ohio has the fourth-highest percentage of tested children under age 6 with elevated blood lead levels (CDC 2007)

Why is lead poisoning an issue in Ohio?

Notes:

According to the U.S. Environmental Protection Agency (USEPA). Ohio is among the top five states in number of homes with lead-based paint. Other states are New York, California, Pennsylvania and Illinois.

Ohio has 1.5 million homes built before 1950 that definitely have lead paint inside and outside. An additional 1 million homes were built between 1950 and 1978 before lead paint was banned, so there is high probability they contain lead paint.

A 2007 report by the Centers for Disease Control and Prevention (CDC) looked at data from 43 states and determined Ohio ranked fourth for elevated blood lead levels (EBLs) in children.

II. Sources of Lead and Health Effects

- Deteriorated paint
- Soil
- Occupations
- Cultural sources
- Hobbies



What are the common points of childhood exposure to lead hazards?

Notes:

Ninety-five percent of lead poisoning results from lead-paint dust created by deteriorated or sanded lead-based paint.

- Vinyl mini-blinds (imported)
- Jewelry
- Some painted toys
- Lead-glazed pottery
- Metal, pewter, brass, crystal
- Imported canned foods
- Folk remedies and cosmetics (e.g., surma)
- Hobbies (e.g., stained glass, indoor firing range)
- Fishing sinkers and bullets
- Occupational (take-home lead)

Health Effects



- Central Nervous System
- Kidney
- Blood
- Gastrointestinal (GI)
- Reproductive

How does lead affect child health and development?

Notes:

- a. CDC level of concern stands at 10 $\mu\text{g}/\text{dL}$, but recent studies show lead has serious health effects on children at lower concentrations. There is no safe or normal blood lead level.
- b. Low lead levels affect the child's central nervous system, development and IQ.
- c. Children with higher levels of lead poisoning have some degree of constipation. As lead levels rise, loss of appetite and nausea may occur.

Possible health issues at levels < 45 $\mu\text{g}/\text{dL}$:

- Apathy
- Reduction in IQ
- Irritability
- Hyperactivity
- Aggression
- Loss of new skills
- Language deficiency
- Hearing loss
- Poor muscle coordination

Sources of Lead Exposure

- **Occupational**
 - Plumbers, pipe fitters
 - Lead miners/smelters
 - Auto repairers
 - Glass manufacturers
 - Shipbuilders
 - Printers
 - Plastic manufacturers
 - Lead smelters and refiners
 - Police officers
 - Steel welders or cutters
 - Construction workers
 - Rubber product manufacturers/Gas station attendants
 - Battery manufacturers/rendering
 - Bridge reconstruction workers
 - Firing range instructors, staff, visitors
- **Environmental**
 - Lead-containing paint
 - Soil/dust near lead industries, roadways, lead-painted homes
 - Plumbing leachate
 - Ceramicware
 - Leaded gasoline
- **Hobbies and Related Activities**
 - Glazed pottery making
 - Target shooting at firing ranges
 - Lead soldering (e.g., electronics)
 - Painting
 - Preparing lead shot, fishing sinkers
 - Stained-glass making
 - Car or boat repair
 - Home remodeling
 - NASCAR racing
- **Substance Use**
 - Folk remedies
 - Health foods
 - Cosmetics
 - Moonshine whiskey

Food may contain lead from the environment or from containers. Agricultural vehicles are not required to use unleaded gasoline; consequently, lead can be deposited on and retained by crops, particularly leafy vegetables. Acidic foods have been found to leach lead from lead solder in imported cans and lead glazes used in making pottery and ceramicware. Water from leaded pipes, soldered plumbing or water coolers is another potential source of lead exposure. Stationary or point sources of lead include mines and smelters.

Several **folk remedies** used in this country have been shown to contain large amounts of lead. Two Mexican folk remedies are azarcon and greta, which are used to treat "empacho," a colic-like illness. Azarcon and greta are also known as liga, Maria Luisa, alarcon, coral and rueda. Lead-containing remedies and cosmetics used by some Asian communities are chuifong tokuwan, pay-looah, ghasard, bali goli and kandu. Middle Eastern remedies and cosmetics include alkohl, kohl, surma, saott and cebagin.

Materials	Sources	Uses
Azarcon, greta	Mexico	GI symptoms
Paylooah	Southeast Asia	Fever, rash
Surma, kohl, kajal	India, Pakistan, Middle East	Medicinal or decorative cosmetics for eyes, skin
Ghasard, bali, goli, kandu	India	GI symptoms
Saott, cabgain (teething powder)	Saudi Arabia	Pain reliever
Folk remedy of powdered rock	United Arab Emirates	Relief of colic
Calcium supplements – bonemeal, dolomite	United States	Dietary supplement
Lozeena	Iraq	Food coloring, orange powder
Metal urns/kettles	Various	Boiling water
Gunshot pellets	Various	Accidental exposure
Foreign bodies: fishing sinkers, bullets, curtain weights	Various	Accidental exposure

In addition to these **environmental sources**, many occupations, hobbies and other activities result in potential exposures to high levels of lead and can put the entire family at risk of lead poisoning. Sources of lead exposure are listed above. Lead-glazed pottery, particularly if it is imported, is a potential source of exposure that is often overlooked. Even "safe" ceramicware can become harmful; dishwashing may chip or wear off the protective glaze and expose lead-containing pigments.

Inorganic lead enters the body primarily through inhalation and ingestion and does not undergo biologic transformation. In contrast, **organic lead**, found primarily in gasoline as tetraethyl lead, enters the body through inhalation and skin contact and is metabolized in the liver. In 1976 and in 1984, federal regulation drastically reduced the amount of lead in gasoline, and today organic lead in gasoline is not as great an environmental concern in the United States as it is in other countries, where it remains a serious hazard.

Taken From

Case Studies in Environmental Medicine (CSEM) – Lead Toxicity

Agency for Toxic Substances & Disease Registry (ATSDR) 2007

Introduction	<p>Lead serves no useful purpose in the human body, but its presence in the body can lead to toxic effects, regardless of exposure pathway.</p> <ul style="list-style-type: none">• Lead toxicity can affect every organ system.• On a molecular level, proposed mechanisms for toxicity involve fundamental biochemical processes. These include lead's ability to inhibit or mimic the actions of calcium (which can affect calcium-dependent or related processes) and to interact with proteins (including those with sulfhydryl, amine, phosphate and carboxyl groups) (ATSDR, 2005). <p>It must be emphasized that <u>there may be no threshold</u> for developmental effects on children.</p> <ul style="list-style-type: none">• The practicing health care provider can distinguish overt clinical symptoms and health effects that come with high exposure levels on an individual basis.• However, lack of overt symptoms does not mean “no lead poisoning.”• Lower levels of exposure have been shown to have many subtle health effects.• Some researchers have suggested that lead continues to contribute significantly to socio-behavioral problems such as juvenile delinquency and violent crime (Needleman 2002, Nevin 2000).• It is important to prevent all lead exposures. <p>While the immediate health effect of concern in children is typically neurological, it is important to remember that childhood lead poisoning can lead to health effects later in life including renal effects, hypertension, reproductive problems, and developmental problems with their offspring (see below). The sections below describe specific physiologic effects associated with major organ systems and functions.</p>
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Neurological Effects	<p>The nervous system is the most sensitive target of lead exposure.</p> <ul style="list-style-type: none">• There may be no lower threshold for some of the adverse neurological effects of lead in children.• Neurological effects of lead in children have been documented at exposure levels once thought to cause no harmful effects (<10 µg/dL) (Canfield 2003; CDC 1997a).• Because otherwise asymptomatic individuals may experience neurological effects from lead exposure, clinicians should have a high index of suspicion for lead exposure, especially in the case of children.
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Children	<p>In children, acute exposure to very high levels of lead may produce encephalopathy and other accompanying signs of</p> <ul style="list-style-type: none">• ataxia• coma• convulsions• death• hyperirritability• stupor <p>The BLLs associated with encephalopathy in children vary from study to study, but BLLs of 70-80 µg/dL or greater appear to indicate a serious risk (ATSDR 2005).</p> <ul style="list-style-type: none">• Even without encephalopathy symptoms, these levels are associated with increased incidences of lasting neurological and behavioral damage (ATSDR 2005). <p>Children suffer neurological effects at much lower exposure levels.</p> <ul style="list-style-type: none">• Neurological effects may begin at low (and, relatively speaking, more widespread) BLLs, at or below 10 µg/dL in some cases, and it may not be possible to detect them on clinical examination.• Some studies have found, for example, that for every 10 µg/dL increase in BLL, children's IQ was found to be lower by four to seven points (Yule <i>et al.</i>, 1981; Schroeder <i>et al.</i>, 1985; Fulton <i>et al.</i>, 1987; Landsdown <i>et al.</i>
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	<p>1986; Hawk <i>et al.</i> 1986; Winneke <i>et al.</i> 1990 as cited in AAP 1993).</p> <ul style="list-style-type: none"> • There is a large body of evidence that associates decrement in IQ performance and other neuropsychological defects with lead exposure. • There is also evidence that attention deficit hyperactivity disorder (ADHD) and hearing impairment in children increase with increasing BLLs, and that lead exposure may disrupt balance and impair peripheral nerve function (ATSDR 2005). • Some of the neurological effects of lead in children may persist into adulthood.
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<p>Adults</p>	<p>There can be a difference in neurological effects between an adult exposed to lead as an adult, and an adult exposed as a child when the brain was developing.</p> <ul style="list-style-type: none"> • Childhood neurological effects, including ADHD, may persist into adulthood. Lead-exposed adults may also experience many of the neurological symptoms experienced by children, although the thresholds for adults tend to be higher. <p>Lead encephalopathy may occur at extremely high BLLs, <i>e.g.</i>, 460 µg/dL. (Kehoe 1961 as cited in ATSDR 2005)</p> <ul style="list-style-type: none"> • Precursors of encephalopathy, such as dullness, irritability, poor attention span, muscular tremor, and loss of memory may occur at lower BLLs. <p>Less severe <i>neurological and behavioral effects</i> have been documented in lead-exposed workers with BLLs ranging from 40 to 120 µg/dL. (ATSDR 2005) These effects include</p> <ul style="list-style-type: none"> • decreased libido • depression/mood changes, headache • diminished cognitive performance • diminished hand dexterity • diminished reaction time • diminished visual motor performance • dizziness • fatigue
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	<ul style="list-style-type: none"> • forgetfulness • impaired concentration • impotence • increased nervousness • irritability • lethargy • malaise • paresthesia • reduced IQ scores • weakness <p>There is also some evidence that lead exposure may affect adults' postural balance and peripheral nerve function. (ATSDR 1997a, b; Arnvig <i>et al.</i> 1980; Haenninen <i>et al.</i> 1978; Hogstedt <i>et al.</i> 1983; Mantere <i>et al.</i> 1982; Valciukas <i>et al.</i> 1978 as cited in ATSDR 1999)</p> <p>Slowed nerve conduction and forearm extensor weakness (wrist drop), as late signs of lead intoxication, are more classic signs in workers chronically exposed to high lead levels</p>
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<p>Renal Effects</p>	<p>Many studies show a strong association between lead exposure and renal effects. (ATSDR 1999)</p> <ul style="list-style-type: none"> • Acute high dose lead-induced impairment of proximal tubular function manifests in aminoaciduria, glycosuria, and hyperphosphaturia (a Fanconi-like syndrome). These effects appear to be reversible (ATSDR 1999). • However, continued or repetitive exposures can cause a toxic stress on the kidney, if unrelieved, may develop into chronic and often irreversible lead nephropathy (<i>i.e.</i>, chronic interstitial nephritis). <p>The lowest level at which lead has an adverse effect on the kidney remains unknown.</p> <ul style="list-style-type: none"> • Most documented renal effects for occupational workers have been observed in acute high-dose exposures and high-to-moderate chronic exposures (BLL > 60 µg/dL).
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- Currently, there are no early and sensitive indicators (e.g., biomarkers) considered predictive or indicative of renal damage from lead. (ATSDR 2000) Serum creatinine and creatinine clearance are used as later indicators.
- However, certain urinary biomarkers of the proximal tubule (e.g., NAG) show elevations with current exposures, even at BLLs less than 60 µg/dL; and some population-based studies show accelerated increases in serum creatinine or decrements in creatinine clearance at BLLs below 60 µg/dL. (Staessen *et al.* 1992; Kim *et al.* 1996; Payton *et al.* 1994; Tsaih *et al.* 2004)

Latent effects of lead exposure that occurred years earlier in childhood may cause some chronic advanced renal disease or decrement in renal function.

- In children, the acute lead-induced renal effects appear reversible with recovery usually occurring within two months of treatment. (Chisolm *et al.* 1976)
- Treatment of acute lead nephropathy in children appears to prevent the progression to chronic interstitial nephritis. (Weeden *et al.* 1986)

It should be noted that lead-induced end-stage renal disease is a relatively rare occurrence in the U.S. population.

- Renal disease can be asymptomatic until the late stages and may not be detected unless tests are performed.
- Because past or ongoing excessive lead exposure may also be a causative agent in kidney disease associated with essential hypertension (ATSDR 1999), primary care providers should follow closely the renal function of patients with hypertension and a history of lead exposure. (See “*Hypertension Effects*” section).

Lead exposure is also believed to contribute to “*saturnine gout*,” which may develop because of lead-induced hyperuricemia due to decreased renal excretion of uric acid.

- In one study, more than 50% of patients suffering from lead nephropathy also suffered from gout. (Bennett 1985 as cited in ATSDR 2000)
- Saturnine gout is characterized by less frequent attacks than primary gout. Lead-associated gout may occur in pre-menopausal women, an

	<p>uncommon occurrence in non lead-associated gout. (Goyer 1985, as cited in ATSDR 2000)</p> <ul style="list-style-type: none"> • A study by Batuman et al (1981) suggests that renal disease is more frequent and more severe in saturnine gout than in primary gout.
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<p>Hematological Effects</p>	<p>Lead inhibits the body's ability to make hemoglobin by interfering with several enzymatic steps in the heme pathway.</p> <ul style="list-style-type: none"> • Specifically, lead decreases heme biosynthesis by inhibiting <i>d</i>-aminolevulinic acid dehydratase (ALAD) and ferrochelatase activity. • Ferrochelatase, which catalyzes the insertion of iron into protoporphyrin IX, is quite sensitive to lead. • A decrease in the activity of this enzyme results in an increase of the substrate, erythrocyte protoporphyrin (EP), in the red blood cells (also found in the form of ZPP—bound to zinc rather than to iron). • Also associated with lead exposure is an increase in blood and plasma <i>d</i>-aminolevulinic acid (ALA) and free erythrocyte protoporphyrins (FEP) (EPA 1986a as cited in ATSDR 1999). <p>EPA estimated the threshold BLL for a decrease in hemoglobin to be 50 µg/dL for occupationally exposed adults and approximately 40 µg/dL for children, although other studies have indicated a lower threshold (<i>e.g.</i>, 25 µg/dL) for children. (EPA 1986b as cited in ATSDR 1999; ATSDR 1999)</p> <ul style="list-style-type: none"> • Recent data indicate that the EP level, which has been used in the past to screen for lead toxicity, is not sufficiently sensitive at lower levels of blood lead and is therefore not as useful a screening test as previously thought (see the “<i>Laboratory Evaluation</i>” section for further discussion of EP testing.). <p>Lead can induce <u>two types of anemia</u>, often accompanied by basophilic stippling of the erythrocytes. (ATSDR 1999)</p> <ul style="list-style-type: none"> • Acute high-level lead exposure has been associated with hemolytic anemia. • Frank anemia is not an early manifestation of lead exposure and is evident only when the BLL is significantly elevated for prolonged periods.
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	<ul style="list-style-type: none"> • In chronic lead exposure, lead induces anemia by both interfering with heme biosynthesis and by diminishing red blood cell survival. • The anemia of lead intoxication is hypochromic, and normo- or microcytic with associated reticulocytosis. <p>The heme synthesis pathway, on which lead has an effect, is involved in many other processes in the body including neural, renal, endocrine, and hepatic pathways.</p> <ul style="list-style-type: none"> • There is a concern about the meaning of and possible sequelae of these biochemical and enzyme changes at lower levels of lead.
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<p>Endocrine Effects</p>	<p>Studies of children with high lead exposure have found that a strong inverse correlation exists between BLLs and vitamin D levels.</p> <ul style="list-style-type: none"> • Lead impedes vitamin D conversion into its hormonal form, 1, 25-dihydroxyvitamin D, which is largely responsible for the maintenance of extra- and intra-cellular calcium homeostasis. • Diminished 1, 25-dihydroxyvitamin D, in turn, may impair cell growth, maturation, and tooth and bone development. • In general, these adverse effects seem to be restricted to children with chronically high BLLs (most striking in children with BLLs > 62 µg/dL) and chronic nutritional deficiency, especially with regard to calcium, phosphorous, and vitamin D (Koo <i>et al.</i> 1991 as cited in ATSDR 1999). • However, Rosen <i>et al.</i> (1980) noted that in lead-exposed children with blood lead levels of 33-55 µg/dL, 1, 25-dihydroxyvitamin D levels were reduced to levels comparable to those observed in children with severe renal insufficiency. • Lead appears to have a minimal, if any, effect on thyroid function.
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<p>Gastrointestinal Effects</p>	<p>In severe cases of lead poisoning, children or adults may present with severe cramping abdominal pain, which may be mistaken for an acute abdomen or appendicitis.</p>
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<p>Cardiovascular (Hypertension) Effects</p>	<p>Hypertension is a complex condition with many different causes and risk factors, including age, weight, diet, and exercise habits.</p> <ul style="list-style-type: none"> • Lead exposure is one factor of many that may contribute to the onset and development of hypertension. • Although low to moderate lead level exposures (BLL<30 µg/dL) show only a low degree of association with hypertension, higher exposures (primarily occupational) increase the risk for hypertensive heart disease and cerebrovascular disease as latent effects. • One study found that adults who experienced lead poisoning as children had a significantly higher risk of hypertension 50 years later (relative to control adults without childhood lead exposure). (Hu, 1991, as cited in ATSDR 2000) The association has been shown in population-based studies with BLLs below 10 µg/dL. Data supports an association between lead exposure and elevations in blood pressure. (Victory <i>et al.</i> 1988; Schwartz 1995 as cited in ATSDR 2000; Korrick <i>et al.</i> 1999; Hu <i>et al.</i> 1996) • It is estimated that, on a population basis, blood lead can account for a 1% to 2% variance in blood pressure. (ATSDR 2000) This could increase the incidence of hypertension a substantial amount, due to the high prevalence of hypertension of all causes in general populations.
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<p>Reproductive Effects</p>	<p>Reproductive effects examined in the literature include sperm count, fertility, and pregnancy outcomes. While several studies have implicated lead as contributing to reproductive and developmental effects, these effects have not been well-established at low exposure levels.</p> <p>Male Reproductive Effects</p> <p>Recent reproductive function studies in humans suggest that current occupational exposures decrease sperm count totals and increase abnormal sperm frequencies (Alexander <i>et al.</i> 1996; Gennart <i>et al.</i> 1992; Lerda 1992; and Lin <i>et al.</i> 1996 as cited in ATSDR 2000; Telisman <i>et al.</i> 2000).</p> <ul style="list-style-type: none"> • Effects may begin at BLLs of 40 µg/dL. (ATSDR 2005)
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	<ul style="list-style-type: none"> • Long-term lead exposure (independent of current lead exposure levels) also may diminish sperm concentrations, total sperm counts, and total sperm motility (Alexander <i>et al.</i> 1996 as cited in ATSDR 2000). • It is unclear how long these effects may last in humans after lead exposure ceases. <p>Fertility</p> <p>It is not currently possible to predict fertility outcomes based on current BLLs or past lead exposure levels. (ATSDR 2000)</p> <p>Pregnancy Outcomes</p> <p>The effect of low-level lead exposures on pregnancy outcomes is not clear. Thus it appears that at higher (<i>e.g.</i>, occupational) exposure levels, the evidence is clearer for an association between lead and adverse pregnancy outcomes. This association becomes equivocal when looking at women exposed to lower environmental levels of lead. The data concerning exposure levels are incomplete, probably a result of far greater exposures than are currently found in lead industries.</p> <ul style="list-style-type: none"> • Some studies of women living near smelters versus those living some distance away <u>did show</u> increased frequency of spontaneous abortions (Nordstrom <i>et al.</i> 1979) and miscarriages and stillbirths (Baghurst <i>et al.</i> 1987; McMichael <i>et al.</i> 1986). • In contrast, Murphy <i>et al.</i> (1990) evaluated past pregnancy outcomes among women living in the vicinity of a lead smelter and <u>did not</u> find an increase in spontaneous abortion risk among the lead exposed group versus the unexposed group. • Women with BLL 5-9 µg/dL were two to three times more likely to have a spontaneous abortion than were women with BLL lesser than 5 µg/dL. (Borja-Aburto <i>et al.</i> 1999).
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<p>Developmental Effects</p>	<p>Developmental effects examined in the literature include pregnancy outcomes (<i>e.g.</i>, premature births and low birth weights), congenital abnormalities, and post birth effects on growth or neurological development.</p>
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	<ul style="list-style-type: none"> • Increasing evidence indicates that lead, which readily crosses the placenta, adversely affects fetus viability as well as fetal and early childhood development. • Prenatal exposure to low lead levels (e.g., maternal BLLs of 14 µg/dL) may increase the risk of reduced birth weight and premature birth (ATSDR 1999). • Although lead is an animal teratogen, most human studies have not shown a relationship between lead levels and congenital malformations. • A study by Needleman <i>et al.</i> (1984) correlated increased prenatal lead exposure with increased risk for minor congenital abnormalities (e.g., minor skin abnormalities and undescended testicles). • No association between prenatal lead exposure and major congenital abnormalities has been found (Ernhart <i>et al.</i> 1985, 1986; McMichael <i>et al.</i> 1986). • In a retrospective study, a higher proportion of learning disabilities were found among school-aged children with biological parents who were lead poisoned as children 50 years previously (Hu 1991).
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<p>Other Potential Effects</p>	<p>Lead has been linked to problems with the development and health of bones. At high levels, lead can result in slowed growth in children.</p> <ul style="list-style-type: none"> • Studies have shown increased likelihood of osteoporosis (weakened bones later in life) in animals exposed to lead. A review of this issue can be found in Puzas (1992). Although this link has not been established in humans, it is likely that upon closer examination of lead-exposed individuals, lead will be shown to be a new risk factor for the disease. • Research currently underway may provide more information about potential impacts of lead on osteoporosis (bone health) in the future. <p>Current available data are not sufficient to determine the carcinogenicity of lead in humans.</p> <ul style="list-style-type: none"> • EPA has classified elemental lead and inorganic lead compounds as Group 2B: probable human carcinogens. (ATSDR 1999) This classification is based in part on animal studies, which have been criticized because the
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doses of lead administered were extremely high (ATSDR 1999).

- The National Toxicology Program classifies lead and lead compounds as “reasonably anticipated to be a carcinogen” (NTP 2004).
- Information regarding the association of occupational exposure to lead with increased cancer risk is generally limited. This is because these occupational exposure studies, which primarily examined lead smelters, involved confounding exposures to other chemicals, including arsenic, cadmium, antimony, and toxicants from worker smoking habits (Cooper 1976 and IARC 1987).

Researchers are currently investigating the impacts of lead on dental health.

- One study found pre- and perinatal exposure to lead increased prevalence of caries in rat pups by almost 40% (Watson 1997).
- Human epidemiological studies suggesting an association between lead exposure and caries although this has not been well-established (Bowen 2001).



Interpreting and Managing Blood Lead Levels <10 μg/dL in Children and Reducing Childhood Exposures to Lead

Recommendations of CDC's Advisory Committee on Childhood Lead Poisoning Prevention

Please note: An erratum has been published for this article. To view the erratum, please click [here](#).

Prepared by
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Abstract

Lead is a common environmental contaminant, and exposure to lead is a preventable risk that exists in all areas of the United States. Lead is associated with negative outcomes in children, including impaired cognitive, motor, behavioral, and physical abilities. In 1991, CDC defined the blood lead level (BLL) that should prompt public health actions as 10 μg/dL. Concurrently, CDC also 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 children's physical and mental development can be affected at BLLs <10 μg/dL.

This report summarizes the findings of a review of clinical interpretation and management of BLLs <10 μg/dL conducted by CDC's Advisory Committee on Childhood Lead Poisoning Prevention. This report provides information to help clinicians understand BLLs <10 μg/dL, identifies gaps in knowledge concerning lead levels in this range, and outlines strategies to reduce childhood exposures to lead. In addition, this report summarizes scientific data relevant to counseling, blood lead screening, and lead exposure risk assessment.

To aid in the interpretation of BLLs, 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 local and patient circumstances permit, clinicians should consider early referral to developmental programs for children at high risk for exposure to lead and consider more frequent rescreening of children with BLLs approaching 10 μg/dL, depending on the potential for exposure to lead, child age, and season of testing. 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.

Introduction

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 in children, including impaired cognitive, motor, behavioral, and physical abilities (1--4). In 1991, CDC defined the blood lead level (BLL) that should prompt public health actions as 10 μ g/dL. Concurrently, CDC also recognized that a BLL of 10 μ g/dL did not define a threshold for the harmful effects of lead (5). Research conducted since 1991 has strengthened the evidence that children's physical and mental development can be affected at BLLs <10 μ g/dL (1,3).

During 2002--2004, a workgroup of CDC's Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) reviewed the scientific literature regarding adverse health effects associated with BLLs <10 μ g/dL, including 23 published reports that analyzed 16 separate populations with Intelligence Quotient (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, that included children born in 1994 and 1995, enrolled at age 6 months, and followed for 5 years (6). The majority of children studied had BLLs <10 μ g/dL throughout the study period. The IQ and blood lead level relationship was most accurately described by a nonlinear negative association, with a decrease in IQ of more than seven points over the first 10 μ g/dL increase in lifetime average blood lead concentration. 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 (i.e., linear or nonlinear) of this association across BLLs uncertain. In addition, the workgroup concluded that children with BLLs <10 μ g/dL should not be classified as "lead poisoned." The report noted that no safe level for blood lead in children has been identified (1).

Two studies published subsequently have reported negative effects of BLLs <10 μ g/dL on developmental outcomes (7,8). One study, which included participants from the Rochester cohort (6) and from six other past prospective studies of children with peak BLLs across a range of values, reaffirmed an inverse association between lead at low levels and IQ (7). In these studies, key potential confounders were accounted for, including maternal IQ, the Home Observation for Measurement of the Environment Inventory (HOMEI) 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 ACCLPP has previously reviewed case management of children with BLLs \geq 10 μ g/dL (2), this is the first ACCLPP report to summarize scientific information relevant to clinical management of children with BLLs <10 μ g/dL. This report also outlines recommendations from ACCLPP to reduce childhood exposure to lead. Information on assessing an environmental history and prevention strategies to decrease exposures to lead have been published previously (2,3) and are not included in this report.

Methods

ACCLPP provides advice and guidance to the U.S. Department of Health and Human Services and CDC regarding new scientific knowledge and technologic 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, blood lead 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, ACCLPP formed another workgroup comprising three pediatricians and a CDC health scientist to review the scientific literature regarding clinical management options for BLLs <10 μ g/dL and to outline recommendations for clinical care providers. On the basis of its analysis, the workgroup developed draft recommendations that were reviewed and later adopted by ACCLPP in February 2006.

Results

Historic Trends in Children's BLLs in the United States

Since 1976, BLLs in U.S. children aged 1--5 years 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 U.S. 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 will likely affect substantial numbers of children, and can be expected to reduce the number of children affected by adverse health outcomes associated with lead exposure (14).

Blood Lead Measurements

As with any biologic test, blood lead measurements entail inherent uncertainties as a result of imprecise analytic techniques and preanalytic variables (e.g., the specimen collection process). However, the ratio of imprecision to measurement value, particularly at BLLs <10 μ g/dL, is relatively high. The degree of inherent error in blood lead analysis varies by analytic methodology 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 blood lead testing to operate with a total allowable error of ± 4 μ g/dL or $\pm 10\%$, whichever is greater. Consequently, at BLLs ≤ 10 μ g/dL, a laboratory might operate within an error range of 8 μ g/dL and still meet federal proficiency standards. For example, an actual value of blood lead at 7 μ g/dL could be reported as being any value ranging from 3 μ g/dL to 11 μ g/dL and still remain within the allowable error limit. A study of duplicate testing of identical blood samples (all with a mean blood lead value <10 μ g/dL) at eight laboratories reported all results as <10 μ g/dL and within 3 μ g/dL of the overall mean for that specimen value (17). A study conducted in 2006 indicated that the majority of blood lead laboratories can achieve routine performance of ± 2 μ g/dL at concentrations of ≤ 10 μ g/dL without difficulty (18).

Blood lead test reliability also depends on adhering to blood collection techniques that reduce sample contamination. Collection of capillary blood from a fingerstick into a lead-free collection device is an accepted method for obtaining a screening test (19--23) and contamination by lead from the skin surface can be minimized if a protocol for proper capillary specimen collection is followed (24).[†] However, because lead levels from a capillary blood sample will vary from those of a simultaneously drawn venous sample, elevated capillary results should be confirmed with blood drawn by venipuncture. Multiple studies have reported on the uncertainty introduced by collecting capillary blood rather than venipuncture at thresholds of 10 μ g/dL or 15 μ g/dL (19--23), but none has examined the sensitivity or specificity of capillary methods at thresholds <10 μ g/dL.

Children's BLL Patterns

BLLs increase quickly after an acute exposure, then gradually (over weeks) reach equilibrium with body stores of lead. Lead is distributed unevenly within the human body; in children, approximately 70% is stored in the bone compartment (25--27). The residence time of lead in bone can be decades (28). Thus, an elevated BLL will decline within a few weeks to months after an acute exposure. However, for those children with chronic lead exposure and, presumably higher bone lead stores, the decline in BLL can take much longer (29). Although bone lead levels can provide information regarding past absorption of lead, measurements of lead in bone using X-ray fluorescence instruments are available for research purposes only.

A newborn infant's BLL closely reflects that of the mother (30). During 1999--2002, the geometric mean BLL for U.S. women aged 20--59 years was 1.2 μ g/dL, with 0.3% having a BLL ≥ 10 μ g/dL (12). Typically, as infants become more active and increase their environmental exposures, BLLs increase. Longitudinal studies of lead-exposed children have confirmed an increase in BLLs beginning in late infancy, with a peak level reached at age 18--36 months (6,31--33). No studies have examined blood lead patterns specifically for children with peak levels <10 μ g/dL, although certain studies have included children with levels this low. A study of children born during 1994--1995 in which >50% of the children had peak BLLs <10 μ g/dL reported an expected pattern in mean BLLs of 3.4 μ g/dL at age 6 months, 9.7 μ g/dL at age 24 months, and 5.8 μ g/dL at age 61 months (6). A study of children born in Boston during 1979--1981 identified mean BLLs of 7.2 μ g/dL at birth, and subsequent BLLs in these children remained relatively constant (6.2 μ g/dL at age 6 months, 6.8 μ g/dL at age 24 months, and 6.4 μ g/dL at age 57 months) (34--36). In both studies, higher levels of lead in home environmental samples were associated directly with higher BLLs in children (35,37). In addition, the Boston study demonstrated an association between the occurrence of home renovation and increased BLLs (35). The blood lead pattern for individual children with BLLs <10 μ g/dL varies depending on their environmental exposures (29). More research is needed to better understand age-related patterns for BLLs that remain <10 μ g/dL. However, in clinical practice, even should additional research data become available, laboratory uncertainty might interfere with a clinician's ability to detect patterns for individual children.

Once a high BLL has been established in a child, the time required for the BLL to decline to <10 μ g/dL can range from months to years, depending on the duration and dose of exposure. For example, for a group of children starting at a BLL of 10--14 μ g/dL and receiving case management services, the mean time required

for 50% to achieve a BLL <math><10 \mu\text{g}/\text{dL}></math> was 9 months (38). How much time is needed for BLLs <math><10 \mu\text{g}/\text{dL}></math> to decline in response to interventions is unknown.

Multiple studies have confirmed that blood lead measurements vary seasonally. For example, a study conducted in Boston reported that BLLs were highest in late June and lowest in March (39). A Milwaukee study indicated that BLLs were higher in the summer than in the winter (40). Some of the variability (higher blood lead in summer) might result from increased exposure to lead in dust and soil in summer months (41). Blood lead values for urban children are predicted to be 1–2 mg/dL higher in the summer than winter months (42).

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 development outcomes; in these 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 have been identified between average BLLs over a specific period (e.g., 0–5 years) and various adverse health outcomes (6,43–45); other studies have reported statistically significant associations with a single lead measurement at a specific age (e.g., prenatal, 24 months, and 6.5 years) or with a peak measurement (6,31,46). Concurrent BLLs (i.e., those measured close to the time of neurodevelopmental testing) might demonstrate stronger associations with neurodevelopmental abilities than other blood lead measures (6–8,32,47).

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 was a peak level at approximately age 2 years (48). This association was stronger when children were tested at age 7 years than at age 5 years, which underscores the continuing need to reduce lead exposures after age 5 years.

Strategies to Enhance Children's Positive Developmental Outcomes

Although lead is a risk factor for developmental and behavior 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. Thus, for an individual child, neurobehavioral test performance might indicate clinically-significant impairments related to lead exposures but might not fully capture the array of negative outcomes caused by lead (14). The effects of lead at levels approaching 10 $\mu\text{g}/\text{dL}$ might not be recognizable to either the child's family or clinician or be identified through neurobehavioral testing. However, lead exposure might assume greater importance for children with other environmental, genetic, biologic, social, or demographic developmental risks factors. Effects of exposures to lead at lower levels might not be evident in testing of individual children but are best evaluated on a communitywide basis (14).

Multiple factors influence a child's development, including how the child is treated by parents or other adult caregivers. The child's family and personal psychosocial experiences are strongly associated with performance on neurodevelopment measures and account for a greater proportion of the explained variance in these measures than BLLs <math><10 \text{mg}/\text{dL}></math> (2,43,45,49). A child's blood lead measurement is estimated to account for 2%–4% of variance in neurodevelopment measures (approximately 4%–8% of the explained variance) (2,43,50).

All children benefit from parental nurturing, regardless of their BLL. 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 (51). This pattern of parenting of children under age 3 years has been associated with enhanced language and cognitive skills when children were tested in the third grade (52). Thus, parents might help counteract the negative effects of lead by providing a nurturing and enriched environment during development. Studies to examine effects of lead have attempted to control for this psychosocial factor by including measures such as the HOMEI score (7). Although no studies have specifically evaluated 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 on ameliorating lead-related negative developmental outcomes (53,54).

Early enrichment programs, although not tested specifically in relation to BLLs, have been effective in improving cognitive development and social competence of young children, particularly infants from families with low levels of social or economic resources (55). 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 (56--58).

Strategies to Prevent and Reduce Exposure to Lead

CDC and the American Academy of Pediatrics (AAP) recommend that preventive care for every child should include obtaining an environmental history and identifying occupational lead exposure of household members (2,3,5). The major sources of lead exposure among U.S. children are lead-contaminated dust, deteriorated lead-based paint, and lead-contaminated soil (37,59). Typically, lead contamination of water contributes less to a child's lead burden than home and soil sources (59); however, if additives to water (e.g., those used in disinfection processes), are changed, the contribution of lead contamination might be greater (60). The extent of lead paint hazards (i.e., 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 increased BLLs in children (59). Children also are exposed to nonhousing lead sources (e.g., lead in foods, cosmetics, pottery, folk remedies, and toys) (2,3,61).

Home-Related Lead Exposure

An estimated 4.1 million homes in the United States (25% of U.S. homes with children aged <6 years) have a lead-based paint hazard (13). An estimated 68% of U.S. homes built before 1940 have lead hazards, as do 43% of those built during 1940--1959 and 8% of those built during 1960--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 U.S. homes. Publicly funded home inspections are most often limited to homes of children with elevated BLLs; the blood lead threshold value that prompts an inspection varies by state or municipality (62). In addition, even when a child's elevated BLL triggers an inspection, public funding for repairs to reduce or eliminate identified lead hazards typically is not available.

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

CDC is working with HUD, the U.S. Environmental Protection Agency (EPA), state and local health department lead poisoning prevention grantees, and child health and environmental justice advocates to promote primary prevention strategies to reduce exposure to lead (1,63,64). 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 implementation of housing-based primary prevention strategies in their jurisdictions. This moves beyond their traditional role of providing services to children with elevated BLLs and involves developing responses to local risks and a focus on identifying and remediating housing-based lead hazards. ACCLPP recommendations for essential elements for state and local primary prevention plans have been published previously (63), and strategies that have been implemented at the state and local levels to address the problem also have been outlined previously (64). As ACCLPP noted, implementation of state and local primary prevention plans will require 1) targeting the highest risk areas, populations, and activities; 2) fostering political will for jurisdictions to provide an adequate level of funding; 3) expanding resources for housing remediation; identification and correction of lead hazards; and 4) establishing a regulatory infrastructure to create and maintain lead-safe housing and to support the use of lead-safe construction work practices (63,65). Links to state and local health department web sites, which include their primary prevention plans, are available at <http://www.cdc.gov/nceh/lead/grants/contracts/CLPPP%20map.htm>.

Certain state and local health departments initiate case management services and home inspections when BLLs reach 10 mg/dL. As more primary prevention strategies are implemented, the number of health departments pursuing 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

(66).

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 address these hazards. Tenants can request a copy of all lead testing reports for housing sites from landlords at any time. Their landlord should have been provided with such information when they purchased the building; compliance with a tenant request for a copy of all lead testing reports is required by federal law (67). 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 at the time final agreements are signed on the purchase or rental of most housing built before 1978 (e.g., by providing results of any past evaluations of the property for lead) (67). 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. Further, the law requires sellers, landlords, and renovators to provide buyers, renters, and those hiring renovators with an EPA-approved pamphlet, "Protect Your Family from Lead in Your Home" (68). To protect their children from lead, parents might choose not to buy or rent a property or to negotiate remediation of identified lead hazards. 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 general characteristics of lead-based paint and lead-based paint hazards and their control might help parents to understand their home better ([Box](#)) (69--73).

Screening for lead dust hazards through dust wipe testing (i.e., standardized collection of dust by wiping 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 absence of lead hazards at locations in the home that were not tested, nor does it ensure 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 (e.g., windows, doors, and floors) and the interior migration of lead-contaminated exterior dust and soil (70). However, identifying lead dust hazards in the home is a first step toward protecting children and might help parents lower lead dust levels in their homes (74). Proper training is recommended for those collecting dust wipes to focus tests on areas at highest risk (63). 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 further actions based on results.

For a lead-safe environment to be established in older buildings, repair of lead hazards and careful attention to maintenance is 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 implementing 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 exposures to the dust residue generated during the work (35,75--77). 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 (78). Property owners doing work themselves should seek expert advice and training to protect themselves and their families (79,80). 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 (i.e., testing of dust for lead after site clean up) to ensure that residual lead levels do not exceed EPA standards (81). Families with young children should be restricted from work areas until clearance testing has been performed and the area has been judged safe.

In previous evaluation studies, lead dust clearance standards were not low enough to protect children from increased exposures to lead-contaminated dust after lead hazard remediation; as a result, after home repairs, BLLs of children with prerediation BLLs <25 μg/dL increased (82). In 2001, the EPA's lead dust clearance standards were lowered to 40 μg/ft² for floors, 250 μg/ft² for windowsills, and 400 μg/ft² for window wells (81). No studies have evaluated if these lower clearance levels protect children whose BLLs are <10

them to clinicians (102). These plans recommend either universal or targeted blood lead screening. State and local screening plans are available at <http://www.cdc.gov/nceh/lead/grants/contacts/CLPPP%20Map.htm>.

Targeted screening strategies enable clinicians to assess risks for individual children and recommend blood lead testing for a subset of children in the jurisdiction thought to be at increased risk for lead exposure. CDC recommends that risk evaluations be conducted on the basis of such factors as residence in a geographic area, membership in a group at high risk, answers to a personal-risk assessment questionnaire (which might include local factors such as cultural practices or products, such as herbal remedies, traditional cosmetics or imported spices), or other risk factors relevant to the jurisdiction (102).

CDC recommends that locally developed targeted risk assessment and blood lead screening strategies be applied at ages 1 and 2 years (102). Children aged 36--72 months who have been identified as being at risk and who have not been screened previously also should receive a blood lead test (102). For clinicians in areas that lack a state or local screening plan, CDC recommends that a blood lead test be performed on all children at ages 1 and 2 years and on children aged 36--72 months who have not been screened previously (102).

Because lead exposures might change with a child's developmental progress (e.g., walking or reaching window sills) or as a result of external factors (e.g., family relocation or home remodeling), two routine screenings are recommended (at approximately ages 1 and 2 years). Among children in Chicago at high risk with BLLs $<10 \mu\text{g/dL}$ at age 1 year, 21% had a BLL of $\geq 10 \mu\text{g/dL}$ when tested again at age ≥ 2 years (103). This report does not change current CDC recommendations in ages for routine blood lead testing. However, certain local health departments (e.g., those in Chicago, Illinois; New York, New York; and Philadelphia, Pennsylvania) recommend blood lead screening at younger ages or more frequently (106--108). For example, these departments recommend BLL testing starting at ages 6--9 months in high risk areas, blood lead testing at more frequent intervals (e.g., every 6 months) for children aged <2 years, or the provision of additional education and more rapid follow-up blood lead testing for children aged <12 months with BLLs 6--9 $\mu\text{g/dL}$.

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) (109--125). However, no studies have evaluated the performance of these questionnaires at cut-off levels $<10 \mu\text{g/dL}$ or their effectiveness in directing counseling or in identifying lead hazards in the home. When applied in consecutive samples of patients in clinical settings, the sensitivity of such questionnaires to identify children with BLLs $\geq 10 \mu\text{g/dL}$ varies considerably by population (109--128). In certain studies, the sensitivity improved if higher cut-off levels were used in the analysis (103, 115, 119, 120) or if the questions used were developed specifically for the population tested (113, 116, 117, 119, 120, 122). In general, to identify approximately 80% of children with BLLs $\geq 10 \mu\text{g/dL}$, a blood test had to be performed for more than half of those children whose risk factors for lead exposure were assessed using a questionnaire. Multiple studies in populations with low (109, 110, 112--114, 127, 128) or high (123, 124) prevalence for elevated BLLs concluded that risk assessment questionnaires were not effective in their clinical settings.

Future Research Needs

Further study is needed to assess the effects of BLLs $<10 \mu\text{g/dL}$ on children. Such research will entail following large and 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 lower exposures to lead. This should include research on the effectiveness of strategies applied in the medical office and home and those that provide interventions through medical, public health, and environmental means.

Blood lead 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 should be identified to alert public and clinical health-care professionals of changes in exposure sources and patterns and to enhance their response to such changes by increased surveillance and blood lead monitoring of populations identified as being at increased risk for exposure. Additional studies might provide data that can be used to improve laboratory methods and performance monitoring. This will require developing criteria to evaluate individual laboratories and mechanisms to provide this information to clinicians.

Summary of Recommendations

For Clinicians

- 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 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 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 and safely repair lead hazards ([Appendix](#)).
- Help parents to understand the uncertainty of a blood lead value and potential reasons for its fluctuation, including error introduced by the sampling methods and laboratory-, age-, and season-related exposures.
- Assess all children for developmental and behavior status and seek further 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, which might include lead exposures, consider more frequent developmental surveillance or conduct more extensive developmental evaluations.
- Discuss with parents the potential impact of lead on child development and promote strategies that foster optimum development, including encouraging parents to influence their child's development positively by providing nurturing and enriching experiences. For all children from economically and socially low-resource families living in areas where exposure to lead is likely, promote participation in early enrichment programs regardless of the child's BLL.
- Whenever possible, utilize laboratories that can achieve routine performance of $\pm 2 \mu\text{g/dL}$ for blood lead analysis. Evaluate laboratory performance by reviewing the laboratory's quality control chart or statistical quality control summary.
- Review office procedures and policies to ensure that lead exposure risk assessment or blood lead screening is performed on all children as required by state or local health officials or as recommended by CDC. Consider the child's age, season of testing, and exposure history when deciding when to obtain follow-up blood lead tests. For a child whose BLL is approaching $10 \mu\text{g/dL}$, more frequent blood lead screening (i.e., more than annually) might be appropriate, particularly if the child is aged <2 years old, was tested at the start of warm weather when BLLs tend to increase, or is at high risk for lead exposures.
- Perform a diagnostic blood lead test on all children suspected of having lead exposure or an elevated BLL and institute the recommended management guidelines if a child's BLL increases to $\geq 10 \mu\text{g/dL}$.
- 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 the reduction of exposure to lead from all sources. Advocate for the expansion of services that foster lead poisoning primary prevention.

For Government Agencies

- Increase efforts to resolve lead-based paint hazards safely before children are exposed.
- Expand services that promote lead poisoning primary prevention and develop systems that enable clinicians and parents to learn about such services.
- 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.
- Establish jurisdictional policies that mandate ensuring lead safety in housing and enforce these mandates.
- Develop and apply systematic approaches to prevent exposures to even small amounts of lead in food or consumer products, particularly when safer alternatives are available.
- Promote implementation of state and local primary prevention plans that target areas, populations, and activities of highest risk; foster political will; expand resources for housing remediation; identify and correct lead hazards; and establish a regulatory infrastructure to create and maintain lead-safe housing and support the use of lead-safe construction work practices.
- Expand the availability of and promote the use of early enrichment programs for all children from economically and socially low-resource families living in areas where exposure to lead is likely.

- Promote and fund research that will further evaluate the effects of lead in blood at levels <math><10 \mu\text{g}/\text{dL}</math> and evaluate strategies to identify and reduce exposure or the potential for exposure to lead, including strategies applied in medical offices and in homes.

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* A list of members of this committee appears on page 16 of this issue.

† A complimentary video or DVD entitled, "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 at e-mail ncehdls@cdc.gov.

Advisory Committee on Childhood Lead Poisoning Prevention

Membership List, October 2004--February 2006

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* Member 2002--2004. .

† Member 2002--2005.

§ Member 1996--2004.

¶ Representative 1998--2005.

** Representative 1997--2004.

Table 1

TABLE 1. Blood lead levels (BLLs) of children aged 1–5 years — National Health and Nutrition Examination Survey, United States, selected years

Year	% with BLL ≥10 μg/dL	Geometric mean BLL (μg/dL)
1976–1980	88.2	15.0
1991–1994	4.4	2.7
1999–2002	1.6	1.9

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Table 2

TABLE 2. Sensitivity and specificity of lead risk assessment questionnaires to predict blood lead levels (BLLs) of ≥10 μg/dL among patient samples — United States, 1994–2003

Location	Sample characteristics	Prevalence in study sample of % BLLs ≥10 μg/dL	Type of lead exposure risk assessment questions	At cut-off value of ≥10 μg/dL	
				Sensitivity	Specificity
Alaska*	Medicaid	0.6	Modified	0.83	0.99
California†	Medicaid	2.0	CDC	0.46	0.74
Suburban Chicago‡	Private practices	2.2	CDC	0.69	0.70
			Modified	0.86	0.59
Arizona§	Navajo Reservation	2.2	CDC	0.43	0.74
New York**	Rural	2.3	CDC	0.25	0.49
			Modified	0.50	NR††
Denver¶¶	Community health centers	2.9	Modified	0.60	0.36
Illinois¶¶¶	Low-risk ZIP codes	3.5	Modified	0.75	0.39
Wisconsin¶¶¶¶	HMO Clinic A	5.4	CDC	0.77	0.37
			Modified	1.00	0.42
Ohio†††	Mixed sample	5.6	CDC	0.85	0.42
			Modified	0.92	0.57
San Francisco¶¶¶¶	Mixed sample	5.8	CDC	0.87	0.75
California¶¶¶¶¶	Public clinics	6.1	CDC	0.30	0.80
			Modified	0.90	0.37
New York¶¶¶¶¶	Rural	8.4	CDC	0.75	0.31
			Modified	0.88	0.44
Vermont†††††	Birth certificate cohort	9.0	CDC	0.63	0.57
Minnesota¶¶¶¶¶	HMO	11.8	Modified	0.90	0.17
			Modified brief	0.77	0.48
Illinois¶¶	High-risk ZIP codes	12.1	Modified	0.74	0.27
Vermont†††††	Medicaid	14.9	CDC	0.67	0.50
Wisconsin¶¶¶¶	HMO Clinic B	16.8	CDC	0.64	0.32
			Modified	0.91	0.43
Massachusetts†††††	Urban, high risk	21.8	CDC	0.70	0.32
Philadelphia area¶¶¶¶¶	Privately insured	29.1	CDC	0.40	0.60
Rochester, New York††††††	Primarily Medicaid	28.9/99	CDC	0.70	0.49

* Source: Robin LF, Beller M, Middaugh JP. Statewide assessment of lead poisoning and exposure risk among children receiving Medicaid services in Alaska. *Pediatrics* 1997;99:e9. Available at <http://www.pediatrics.org/cgi/content/full/99/4/e9>.

† Source: CDC. Blood lead levels among children in a managed-care organization—California, October 1992–March 1993. *MMWR* 1995;44:627–35.

‡ Source: Binns HJ, LeBailey SA, Ponder J, Kinsella TR, Saunders SE, Pediatric Practice Research Group. Is there lead in the suburbs? Risk assessment in Chicago suburban pediatric practices. *Pediatrics* 1994;93:164–71.

§ Source: 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;46:515–8.

** Source: Muriz 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;19:15–9.

†† Not reported.

¶¶ Source: France EK, Gitterman SA, Melnikovich P, Wright RA. The accuracy of a lead questionnaire in predicting elevated pediatric blood lead levels. *Arch Pediatr Adolesc Med* 1996;150:958–63.

¶¶¶ Source: Binns HJ, LeBailey SA, Fingar AR, Saunders S. Evaluation of risk assessment questions used to target blood lead screening in Illinois. *Pediatrics* 1999;103:100–6.

¶¶¶¶ Source: 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;93:183–7.

††† Source: Striph KB. Prevalence of lead poisoning in a suburban practice. *J Fam Pract* 1995;41:65–71.

¶¶¶¶ Source: Tejada DM, Wyatt DD, Rostek BR, Solomon WB. Do questions about lead exposure predict elevated lead levels? *Pediatrics* 1994;93:192–4.

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¶¶¶¶¶ Data not available to add a decimal place.

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Box

BOX. Tips to reduce lead-based paint and lead-based paint hazards

- Concentration of lead is generally highest in lead-based paint on exterior surfaces.
- Among interior surfaces, windows are most likely to have highest lead content.
- Interior surfaces can become contaminated from exterior sources or common areas.
- Lead-based paint on impact/friction surfaces (e.g., windows, doors, floors) deteriorates as paint is disturbed during use.
- Lack of a deteriorated surface does not ensure absence of lead-contaminated dust, although it lowers the risk.
- Renovation, remodeling, and repainting can significantly increase lead dust levels.
- Vacuum methods (using a traditional vacuum or a high-energy particulate air [HEPA] filtered vacuum) will not lower lead levels on soiled carpets or upholstery far 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 will contribute to lower lead dust in entryway and home interior locations.
- Safely addressing interior, exterior, and soil lead hazards in an integrated manner will be most beneficial in establishing lasting, lead-safe environments.

[Return to top.](#)

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**Questions or messages regarding errors in formatting should be addressed to mmwrq@cdc.gov.

Date last reviewed: 10/10/2007

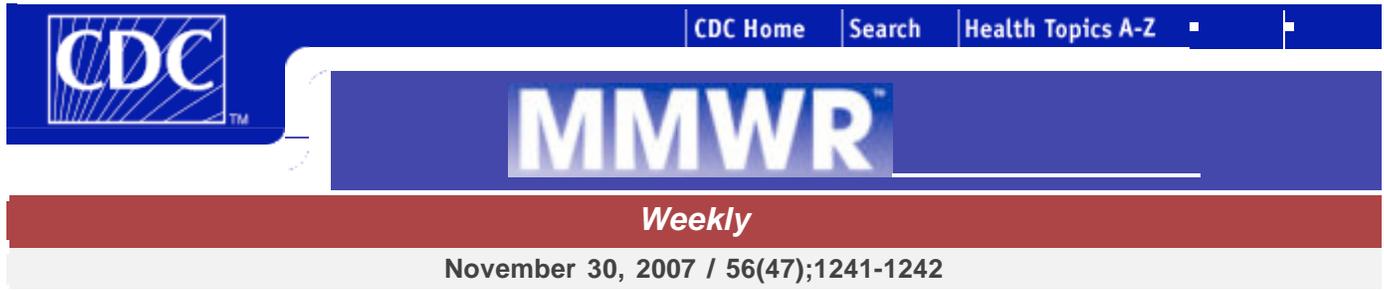
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 Centers for Disease Control and Prevention
 1600 Clifton Rd, MailStop E-90, Atlanta, GA
 30333, U.S.A.



Department of Health
 and Human Services



Errata: Vol. 56, No. RR-8

Errors occurred in the *MMWR Recommendations and Reports*, "[Interpreting and Managing Blood Lead Levels <10 µg/dL in Children and Reducing Childhood Exposures to Lead: Recommendations of CDC's Advisory Committee on Childhood Lead Poisoning Prevention.](#)"

On page 4, in the first column, the sentence at the top of the page should read, "Blood lead values for urban children are predicted to be 1--2 µg/dL higher in the summer than winter months (42)."

Also on page 4, in the second column, the second sentence of the first full paragraph should read, "The child's family and personal psychosocial experiences are strongly associated with performance on neurodevelopment measures and account for a greater proportion of the explained variance in these measures than BLLs <10 µg/dL (2,43,45,49)."

On page 5, in the first column, the first sentence of the first full paragraph should read, "Certain state and local health departments initiate case management services and home inspections when BLLs reach 10 µg/dL."

On page 7, in the second column, the second sentence should read, "One study indicated that a highly intensive education program starting at birth and lasting for ≥3 years (28 sessions) delivered by community members lowered the risk of BLLs ≥10 µg/dL 34%, but this result was not statistically significant (92)."

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Did you know Ohio requires a lead test for at-risk children?

Ohio Revised Code, Section 3742 requires a blood lead test for children at risk of lead poisoning. Ohio Administrative code specifies who is considered at risk.

Who is at risk for lead poisoning?

1. All Medicaid consumers

Test all children at 1 and 2 years of age. Test children 3-6 years of age if never tested.

It's Ohio law and a federal requirement to test children on Medicaid!

2. Children residing in high-risk ZIP codes

All children living in high-risk ZIP codes must have a documented test twice between 9 and 36 months, with 12 months between tests unless clinically indicated sooner, or at least once if age 3-6 years but without a previous documented test.

It's Ohio law to test children living in high risk Zip Code areas!

3. Children who are at risk as determined by responses to the Risk Assessment Questionnaire in low-risk ZIP code areas. Ask key questions twice between 9 and 36 months, with 12 months between assessments unless clinically indicated sooner.

Does your child?

- Live in or regularly visit a house built before 1950? This includes a day care center, preschool or home of a babysitter or relative.
- Live in or visit a house that has peeling, chipping, dusting or chalking paint?
- Live in or visit a house built before 1978 with recent, ongoing or planned renovation/remodeling?
- Have a sibling or playmate who has or did have lead poisoning?
- Frequently come in contact with an adult who has a hobby or works with lead? Examples are construction, welding, pottery, painting and casting ammunition.

Test, its Ohio law!

Parents have a right to refuse a test. 3701-30-04 Ohio Revised Code; Religious exception:

(A) The provisions of this chapter requiring blood lead screening tests of all children at risk of lead poisoning do not apply if the parents of the child object thereto on the grounds that such screening conflicts with their religious tenets and practices.

(B) Objection to a blood lead screening test shall be documented in the child's medical record.

3701-30-01 **Definitions.**

- (A) "Board of health" means the board of health of a city or general health district or the authority having the duties of a board of health under section 3709.05 of the Revised Code.
- (B) "Certified nurse practitioner" means a registered nurse who holds a valid certificate of authority issued under Chapter 4723. of the Revised Code that authorizes the practice of nursing as a certified nurse practitioner.
- (C) "Child at risk of lead poisoning" means any child under six years of age who meets one or more of the following:
 - (1) Is Medicaid eligible in accordance with Chapter 5111. of the Revised Code;
 - (2) Lives in a high risk zip code as designated by the Director;
 - (3) Lives in or regularly visits a residential unit, child day-care facility, or school built before 1950;
 - (4) Lives in or regularly visits a residential unit built before 1978 that has peeling, chipping, dusting, or chalking paint;
 - (5) Lives in or regularly visits a residential unit built before 1978 with recent ongoing or planned renovation/remodeling;
 - (6) Has a sibling or playmate that has or did have lead poisoning; or
 - (7) Frequently comes in contact with an adult who has a lead-related hobby, or occupation.
- (D) "Child day-care facility" means each area of any of the following in which child day-care, as defined in section 5104.01 of the Revised Code, is provided to children under six years of age:
 - (1) A child day-care center, type A family day-care home, or type B family day-care home as defined in section 5104.01 of the Revised Code;
 - (2) A type C family day-care home authorized to provide child day-care by Sub. H.B. 62 of the 121st general assembly, as amended by Am. Sub. S.B. 160 of the 121st general assembly and Sub. H.B. 407 of the 123rd assembly; or
 - (3) A preschool program or school child program as defined in section 3301.52 of the Revised Code.
- (E) "Clearance examination" means an examination to determine whether the lead hazards in a residential unit, child day-care facility, or school have been sufficiently controlled. A clearance examination includes a visual assessment, collection and analysis of environmental samples.

- (F) "Clinical nurse specialist" means a registered nurse who holds a valid certificate of authority issued under Chapter 4723. of the Revised Code that authorizes the practice of nursing as a clinical nurse specialist.
- (G) "Director" means the director of the Ohio department of health, the director's designee, or the director's authorized agent.
- (H) "Lead abatement" means a measure or set of measures designed for the single purpose of permanently eliminating lead hazards. "Lead abatement" includes all of the following:
- (1) Removal of lead-based paint and lead-contaminated dust;
 - (2) Permanent enclosure or encapsulation of lead-based paint;
 - (3) Replacement of surfaces or fixtures painted with lead-based paint;
 - (4) Removal or permanent covering of lead-contaminated soil;
 - (5) Preparation, cleanup, and disposal activities associated with lead abatement;
- "Lead abatement" does not include any of the following:
- (a) Preventative treatments performed pursuant to section 3742.41 of the Revised Code;
 - (b) Implementation of interim controls;
 - (c) Activities performed by a property owner on a residential unit to which both of the following apply:
 - (i) It is a freestanding single-family home used as the property owner's private residence;
 - (ii) No child under six years of age who has lead poisoning resides in the unit.
- (I) "Lead-based paint" means any paint or other similar surface-coating substance containing lead at or in excess of the level that is hazardous to human health as set forth in rule 3701-32-19 of the Administrative Code.
- (J) "Lead-contaminated dust" means surface dust that contains an area or mass concentration of lead at or in excess of the level that is hazardous to human health as set forth in rule 3701-32-19 of the Administrative Code.

- (K) "Lead-contaminated soil" means soil that contains lead at or in excess of the level that is hazardous to human health as set forth in rule 3701-32-19 of the Administrative Code.
- (L) "Lead-contaminated water pipes" means water pipes containing lead materials resulting in contamination of the water supply with lead at or in excess of the level that is hazardous to human health as set forth in rule 3701-32-19 of the Administrative Code.
- (M) "Lead hazard" means material that is likely to cause lead exposure and endanger an individual's health as set forth in rule 3701-32-19 of the Administrative Code. "Lead hazard" includes lead-based paint, lead-contaminated dust, lead-contaminated soil, and lead-contaminated water pipes.
- (N) "Lead poisoning" means a confirmed level of lead in human blood of ten micrograms per deciliter or greater.
- (O) "Manager" means a person, who may be the same person as the owner, responsible for the daily operation of a residential unit, child day-care facility, or school.
- (P) "Physician" means an individual authorized under Chapter 4731. of the Revised Code to practice medicine and surgery or osteopathic medicine and surgery.
- (Q) "Primary health care provider" means any person or government entity that provides well child health care services, such as annual examinations and immunizations to children under six years of age. "Primary health care provider" includes, but is not limited to, physicians, certified nurse practitioners, clinical nurse specialists, local health departments, medical clinics, offices and hospitals.
- (R) "Public health lead investigation" means an investigation conducted by a public health lead investigator in accordance with rule 3701-30-07 of the Administrative Code.
- (S) "Public health lead investigator" means an employee of the director or a designated board of health who is:
- (1) A licensed lead risk assessor in the state of Ohio; and
 - (2) A registered sanitarian, registered sanitarian-in-training, or a licensed leak risk assessor employed by a board of health and who conducted environmental lead investigations on or before April 7, 2003 in accordance with authority delegated by the director pursuant to section 3742.13 of the Revised Code in effect prior to April 7, 2003.
- (T) "Public health lead risk assessment" means a lead risk assessment conducted by a public health lead investigator in accordance with rule 3701-30-08 of the Administrative Code.

- (U) "Residential unit" means a dwelling or any part of a building being used as an individual's private residence.
- (V) "School" means a public or nonpublic school in which children under six years of age receive education.

Effective: 04/01/2004

R.C. 119.032 review dates: 04/01/2009

CERTIFIED ELECTRONICALLY

Certification

03/12/2004

Date

Promulgated Under: 119.03
Statutory Authority: 3742.50
Rule Amplifies: 3742.01
Prior Effective Dates: None

3701-30-02

Primary Health Care Provider Responsibility.

Primary health care providers of children under six years of age shall do the following:

- (A) Determine if the child has had a blood lead screening test. If the child has had a blood lead screening test, determine at what age the child was tested and the blood lead screening test result.
- (B) If the child has not had a blood lead screening test and is between the ages of nine months and seventy-two months, determine if the child is at risk of lead poisoning as defined in paragraph (C) of rule 3701-30-01 of the Administrative Code.
- (C) If any child under six years of age is determined to be at risk of lead poisoning but has not had a blood lead screening test or has had a blood lead screening test but the results are not available, the primary health care provider shall order a blood lead screening test. It is recommended that a child at risk of lead poisoning have a blood lead screening test at the time of the child's one and two year well child visits and annually thereafter as medically indicated.
- (D) The primary health care provider shall make a good faith effort to obtain results of all blood lead screening tests performed on a child at risk of lead poisoning.
- (E) Nothing in this rule is intended to preclude a primary health care provider from following the procedures in Chapter 5101. of the Revised Code for medicaid eligible children or from ordering blood lead screening tests on a child less than nine months of age or greater than six years of age.

Effective 04/01/2004

R.C. 119.032 review dates: 04/01/2009

CERTIFIED ELECTRONICALLY

Certification

03/12/2004

Date

Promulgated Under: 119.03
Statutory Authority: 3742.50
Rule Amplifies: 3742.30
Prior Effective Dates: None

Record-keeping and reporting requirements.

- (A) Except as provided in paragraph (C) of this rule, any clinical laboratory that performs any analysis of human blood on a child under sixteen years of age and residing in Ohio to detect or determine levels of lead shall collect and report to the director all of the following information on a form prescribed by the director:
- (1) Child's name and parent's or guardian's name;
 - (2) Child's street and mailing address, including the city, state, county and zip code;
 - (3) Child's social security number, date of birth, gender, race and ethnicity;
 - (4) Telephone number, with area code, where the parents or guardians can be reached;
 - (5) Specimen matrix (blood);
 - (6) Analyte (lead);
 - (7) Procedure used to obtain the specimen and the date it was obtained;
 - (8) Physician's or healthcare provider's first name, last name, address, and telephone number;
 - (9) Child's medicaid number, if any;
 - (10) Clinical laboratory improvement amendments of 1998 (CLIA) number of the laboratory performing the analysis; and
 - (11) The accession number, the date the sample was analyzed, and the test result in micrograms per deciliter.
- (B) Any physician or healthcare provider requesting analysis of blood of a child under sixteen years of age and residing in Ohio to detect or determine levels of lead shall complete each request for analysis with the information required in paragraphs (A) (1) to (A) (9) of this rule of the Administrative Code.
- (C) The clinical laboratory analyzing human blood to detect or determine levels of lead shall report the information required in paragraphs (A) (1) to (A) (11) of this rule, to the director in a format prescribed by the director by electronic transfer, unless otherwise authorized by the director. All electronic transfers of information shall be transmitted to the director within seven calendar days of obtaining the result.
- (D) The director shall forward any test result required to be reported by a clinical laboratory which indicates the presence of lead in any child under sixteen years of age and residing in Ohio to the appropriate local board of health approved by

the director pursuant to section 3742.34 of the Revised Code within ten business days of receiving the information.

- (E) Any clinical laboratory that performs any analysis of human blood to detect or determine levels of lead in a person sixteen years of age or older and residing in Ohio shall comply with the requirements in rule 3701-32-14 of the Administrative Code.

Effective: 04/01/2004

R.C. 119.032 review dates: 04/01/2009

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Certification

03/12/2004

Date

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Rule Amplifies: 3742.09
Prior Effective Dates: None



HIGH RISK ZIP CODES REQUIRING BLOOD LEAD TESTING

for Children Ages 6 to 72 months, as Ohio Law designates

Ohio Department of Health
Bureau of Child and Family Health Services
Ohio Childhood Lead Poisoning Prevention Program (OCLPPP)
Revised January 2004

ADAMS	45133	...CLARK	45387	...CUYAHOGA	44121
ALLEN	45801		45424		44122
	45804		45503		44123
	45805		45504		44125
ASHLAND	44691		45505		44126
	44805		45506		44127
	44903	CLERMONT	none		44128
ASHTABULA	44004	CLINTON	45177		44135
	44030		45385		44144
	44062	COLUMBIANA	43920	DARKE	45308
ATHENS	45701		43968		45321
	45732		44413		45331
AUGLAIZE	none		44441		45337
BELMONT	43906	COSHOCTON	44601		45338
	43912		43812		45348
	43917	CRAWFORD	43302		45362
	43935		44807		45380
	43943		44818		45382
BROWN	none		44820	DEFIANCE	none
BUTLER	45005		44827	DELAWARE	43015
	45011		44833	ERIE	44811
	45013		44854		44870
	45015		44865	FAIRFIELD	43113
	45036		44875		43130
	45040	CUYAHOGA	44882	FAYETTE	43115
	45042		44887		43143
	45044		44028		43145
	45050		44094		43153
	45056		44102		45123
	45069		44103	FRANKLIN	43085
	45231		44104		43140
	45246		44105		43201
	45327		44106		43202
CARROLL	none		44107		43203
CHAMPAIGN	43078		44108		43204
	45365		44109		43205
CLARK	43078		44110		43206
	43140		44111		43207
	43153		44112		43209
	45314		44113		43210
	45324		44114		43211
	45373		44115		43212
			44117		43213
			44118		
			44119		
			44120		



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Revised January 2004

...FRANKLIN	43214 43215 43217 43219 43221 43222 43223 43224 43227 43228 43230 43231 43232	...HAMILTON	45217 45219 45220 45221 45223 45224 45225 45226 45227 45229 45231 45232 45236 45237 45238 45239 45242 45246	...HURON	44811 44854 44865
FULTON	43502 43521 43567 43570			JACKSON	45601
GALLIA	none			JEFFERSON	43901 43910 43917 43935 43943 43952 43953 43964
GEAUGA	44021 44062 44077 44094 44231 44491	HANCOCK	43316 43359 43516 44817 44830 45840 45843 45872	LAKE	44077 44094
GREENE	43153 45177 45314 45324 45385 45387 45420 45424 45431	HARDIN	43345 43347 43358 45843	LAWRENCE	45638
GUERNSEY	43725	HARRISON	43901 43910 44683	LICKING	43055 43056
HAMILTON	45013 45202 45203 45204 45205 45206 45207 45208 45209 45210 45211 45212 45213 45214 45215 45216	HENRY	43502 43511 43516 43567	LOGAN	43311 43345 43347 43358 45365
		HIGHLAND	45123 45133 45612	LORAIN	44028 44035 44044 44052 44055
		HOCKING	43130 45601 45732	LUCAS	43402 43460 43551 43602 43604 43605 43606 43607 43608 43609 43610 43611 43612 43613 43614 43615
		HOLMES	43812 44624 44627		
		HURON	44807		



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Revised January 2004

...LUCAS	43620 43624	...MIAMI	45424	PICKAWAY	43113 43115 43143 43145 43164 45601
MADISON	43140 43143 43153 45314	MONROE	none		
MAHONING	44405 44413 44420 44436 44471 44502 44503 44504 44505 44506 44507 44509 44510 44511 44512 44555 44601	MONTGOMERY	45005 45042 45324 45325 45327 45338 45342 45345 45402 45403 45404 45405 45406 45407 45408 45409 45410 45414 45417 45418 45419 45420 45424 45426 45427 45431	PIKE	45133 45601 45612
MARION	43302 43337 44833			PORTAGE	44231 44240 44266 44491 44601
MEDINA	44028 44044 44203 44321	MORGAN	45732	PREBLE	45042 45056 45321 45325 45327 45338 45345 45382
MEIGS	45701 45760 45769	MORROW	43015 44833 44903	PUTNAM	43516
MERCER	45348 45388 45846	MUSKINGUM	43056 43701	RICHLAND	44805 44827 44833 44854 44865 44875 44902 44903 44905 44906
MIAMI	45308 45318 45337 45339 45356 45359 45365 45373 45380	NOBLE	none	ROSS	43113 43115 43145 43164 45123 45601 45612
		OTTAWA	43420	SANDUSKY	43420 43431 43435 43551 44811
		PAULDING	none		
		PERRY	45732		



HIGH RISK ZIP CODES REQUIRING BLOOD LEAD TESTING

for Children Ages 6 to 72 months, as Ohio Law designates

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SANDUSKY	44830 44883	...SUMMITT	44311 44313 44314 44320 44321	WILLIAMS	43502 43543 43570
SCIOTO	45638 45662			WOOD	43402 43413 43431 43460 43511 43516 43551 43605 43609 43614 44817 44830 45872
SENECA	43316 43420 44807 44811 44818 44830 44854 44882 44883 45840	TRUMBULL	44062 44231 44420 44436 44446 44483 44484 44485 44491 44504 44505 44509 44510		
SHELBY	45318 45356 45365 45380 45388	TUSCARAWAS	44622 44624 44683	WYANDOT	43302 43316 43323 43337 43351 43359 44882 44883 45843
STARK	44601 44618 44624 44646 44647 44662 44667 44702 44703 44704 44705 44706 44707 44708 44710 44714	UNION	43302 43345 43358		
		VAN WERT	45891		
		VINTON	45601		
		WARREN	45005 45036 45040 45042 45044 45050 45069 45177 45327 45342		
SUMMIT	44146 44203 44221 44223 44240 44301 44302 44303 44304 44305 44306 44307 44308 44310	WASHINGTON	45750		
		WAYNE	44203 44606 44618 44624 44627 44662 44667 44691		

The Lead Risk Model used to determine the high risk zip codes was developed by The Ohio State University, Center for Biostatistics.

2000 Census data and 2001 blood lead data were used to locate hot census tracts, which were then overlaid with zip code boundaries. A zip code with any part of a hot census tract is considered to be at high risk.

The variables used in the Lead Risk Model included:

- At least 12% of children tested in that census tract have BLL 10µg/dL or higher (2001 blood lead data)
- Housing environment
- Demographic characteristics
- Socioeconomic
- Housing density & % public assistance

* Based on Ohio Department of Administrative Services 2001 zip code shape file.

GUIDELINES for IMPROVING BLOOD LEAD TESTING RATES

REMEMBER 2 TESTS ARE REQUIRED PRIOR TO A CHILD'S 37 MONTH OLD BIRTHDAY.

You can improve your rate of blood lead screening for your Medicaid patients by following these guidelines which focus on identifying children for screening, creating opportunities for screening, obtaining specimens, and following-up on results. Note that if you provide services for a Medicaid managed care plan, you may have additional guidelines related to lead screening.

IDENTIFYING PATIENTS for BLOOD LEAD SCREENING

- A. **Mandatory screening-** All 12- and 24-months-old Medicaid eligible children **must** have a blood lead screening test (regardless of ZIP Code or exposure to lead), as stated in the EPSDT rule 5101:3-14-03.
- B. A Risk Assessment Questionnaire **is not** an acceptable substitute for the blood lead screening test.
- C. **Every Medicaid eligible child between the ages of 36- and 72-months of age** must have a blood lead screening test unless you have documentation that the child has been previously screened for lead poisoning.
- D. **Use a Medical Record reminder-** Flag the medical records of all Medicaid eligible children in your practice--**for example, using a red sticker--** as a reminder of the requirement of a blood lead screening test at 12- and 24-months of age.
- E. A **provider will be considered to be in compliance** with the blood lead testing requirement at 12 months of age if the child receives a blood lead screening test between 9 months and 21, inclusive. If a blood lead test has been performed earlier than 9 months, the child must receive another blood lead screening test sometime during the 9 to 21 month period.
- F. A **provider will be considered in compliance** with the blood lead testing requirement at 24 months of age, if the child receives a blood lead screening test between 22 months and 36 months, inclusive.
- G. There **should be a 12 month interval between the first blood lead screening test** (that is, the screening during the 9 to 21 month period) **and the second blood lead screening test** (that is, the screening during the 22 to 36 month period). Please make sure child is at least 22 months of age before ordering a blood test for their 24 month test.

CREATING OPPORTUNITIES for BLOOD LEAD SCREENING

- A. **Schedule a HealthChek (EPSDT) or comprehensive well child exam** (to include a blood lead test) during a current exam; blood lead tests **can be drawn or ordered during a well or sick care visit for 12- and 24-month-old children.**

- B. The patient's health plan should send reminders when a child is approaching their 1st and 2nd birthdays, stressing the need for a HealthChek or comprehensive well child exam, including a history and physical and assessment, immunization update, and a blood lead screening test.

OBTAINING BLOOD LEAD SPECIMENS

- A. Ohio law requires that **laboratories report all blood lead test results** to the Ohio Department of Health (ODH). Verify that the lab you refer to is CLIA certified and approved by the ODH, and that the lab reports all blood lead test results to the ODH. Contact the Surveillance Coordinator at the ODH Childhood Lead Poisoning Prevention Program, 614-728-6816, for information on CLIA certified labs.
- B. If possible, draw the blood specimen in your office. If the specimen must be drawn at a lab, **stress the importance** of the test to the child's parent/guardian, **and encourage** that the test be done right after the visit. If the lab requires an appointment, **schedule the appointment before the child leaves your office.**
- C. Ohio law requires that physicians **provide the following information** when ordering blood lead tests, as stated in the Ohio Administrative Code section 3201-32-05, including: patient information (name, Medicaid number, address, birth date, sex, race/national origin, telephone); procedure information (the procedure used to obtain the specimen and when it was drawn); physician's name, address, telephone number.

FOLLOWING-UP on TEST RESULTS

- A. Be sure to check for the return of the lab slip and record the test result in the patient's record. For example, if you do not receive test results within 30 days, someone in your office should contact the patient and the lab to verify if the blood was drawn and if the analysis was completed.
- B. **Follow-up** with appropriate medical management. Environmental investigations are initiated by the state and local health departments for all confirmed blood tests of 10 micrograms/dL and above. Medical intervention guidelines are available from the OHP website at; <http://jfs.ohio.gov/ohp/bhpp/lpplpt/mmchart1.pdf> or from the ODH Childhood Lead Poisoning Prevention Program, at 614-728-9454.

Need more information? Contact Donna Bush at the Office of Ohio Health Plans, 614-466-6420.