

**Agency for Toxic Substances and Disease Registry  
Case Studies in Environmental Medicine (CSEM)  
Lead Toxicity**

Course: **WB 1105**

Original Date: **August 20, 2007**

Expiration Date: **August 20, 2010**

**Table of Contents**

How to Use This Course .....	3
Initial Check.....	5
What is Lead?.....	9
Where Is Lead Found? .....	11
How Are People Exposed to Lead? .....	16
Who Is at Risk of Lead Exposure?.....	18
What Are the U.S. Standards for Lead Levels?.....	22
What Is the Biologic Fate of Lead?.....	27
What Are the Physiologic Effects of Lead Exposure? .....	30
How Should Patients Exposed to Lead Be Evaluated?.....	39
What Tests Can Assist with the Diagnosis of Lead Toxicity? .....	45
How Should Patients Exposed to Lead be Treated and Managed? .....	49
What Instructions Should Be Given to Patients?.....	54
Where Can I Find More Information?.....	56
Posttest Instructions.....	58
Literature Cited .....	63
Appendix 1: Key to Acronyms/Abbreviations .....	68
Appendix 2. Patient Information Sheet .....	69
Answers to Progress Check Questions .....	71

---

**Environmental  
Alert**

- Children of all races and ethnic origins are at risk of lead toxicity throughout the U.S.
- Lead may cause irreversible neurological damage as well as renal disease, cardiovascular effects, and reproductive toxicity.
- Blood lead levels once considered safe are now considered hazardous, with no known threshold.
- Lead poisoning is a wholly preventable disease.

---

**About This and  
Other Case Studies  
in Environmental  
Medicine**

This educational case study document is one in a series of self-instructional publications designed to increase the primary care provider's knowledge of hazardous substances in the environment and to promote the adoption of medical practices that aid in the evaluation and care of potentially exposed patients. The complete series of Case Studies in Environmental Medicine is located on the ATSDR Web site at <http://www.atsdr.cdc.gov/csem/>. In addition, the [downloadable PDF](#) version of this educational series and other environmental medicine materials provides content in an electronic, printable format, especially for those who may lack adequate Internet service.

---

---

**How to Apply for and Receive Continuing Education Credit** See Internet address [www2.cdc.gov/atsdrce/](http://www2.cdc.gov/atsdrce/) for more information about continuing medical education credits, continuing nursing education credits, and other continuing education units.

---

**Acknowledgements** We gratefully acknowledge the work that the medical writers, editors, and reviewers have provided to produce this educational resource. Listed below are those who have contributed to development of this version of the Case Study in Environmental Medicine.

**Please Note:** Each content expert for this case study has indicated that there is no conflict of interest to disclose that would bias the case study content.

**ATSDR Authors:** Oscar Tarragó, MD, MPH, CHES

**ATSDR Planners:** Oscar Tarragó, MD, MPH, CHES

**ATSDR Commentators:**

**Contributors:** Raymond Demers, MD, MPH

**Peer Reviewers:** Charles Becker, MD; Jonathan Borak, MD; Joseph Cannella, MD; Bernard Goldstein, MD; Alan Hall, MD; Richard J. Jackson, MD, MPH; Jonathan Rodnick, MD; Robert Wheeler, MS; Brian Wummer, MD

---

**Disclaimer** The state of knowledge regarding the treatment of patients potentially exposed to hazardous substances in the environment is constantly evolving and is often uncertain. In this educational monograph, ATSDR has made diligent effort to ensure the accuracy and currency of the information presented, but makes no claim that the document comprehensively addresses all possible situations related to this substance. This monograph is intended as an educational resource for physicians and other health professionals in assessing the condition and managing the treatment of patients potentially exposed to hazardous substances. It is not, however, a substitute for the professional judgment of a health care provider. The document must be interpreted in light of specific information regarding the patient and in conjunction with other sources of authority.

Use of trade names and commercial sources is for identification only and does not imply endorsement by the Agency for Toxic Substances and Disease Registry or the U.S. Department of Health and Human Services.



U.S. Department of Health and Human Services  
Agency for Toxic Substances and Disease Registry  
Division of Toxicology and Environmental Medicine  
Environmental Medicine and Educational Services Branch

---

How to Use This Course

<b>Introduction</b>	The goal of <i>Case Studies in Environmental Medicine</i> (CSEM) is to increase the primary care provider's knowledge of hazardous substances in the environment and to help in evaluation and treating of potentially exposed patients. This CSEM focuses on lead toxicity.
<b>Available Versions</b>	<p>Two versions of the Lead Toxicity CSEM are available.</p> <ul style="list-style-type: none"> <li>• the HTML version <a href="http://www.atsdr.cdc.gov/csem/lead/">http://www.atsdr.cdc.gov/csem/lead/</a> provides content through the Internet;</li> <li>• the <a href="#">downloadable PDF</a> version provides content in an electronic, printable format, especially for those who may lack adequate Internet service.</li> </ul> <p>The HTML version offers interactive exercises and prescriptive feedback to the user.</p>
<b>Instructions</b>	<p>The following steps are recommended to make the most effective use of this course.</p> <ul style="list-style-type: none"> <li>• Take the Initial Check to assess your current knowledge about lead toxicity</li> <li>• Read the title, learning objectives, text, and key points in each section</li> <li>• Complete the progress check exercises at the end of each section and check your answers</li> <li>• Complete and submit your assessment and posttest response online if you wish to obtain continuing education credit. Continuing education certificates can be printed immediately upon completion.</li> </ul>
<b>Instructional Format</b>	This course is designed to help you learn efficiently. Topics are clearly labeled so that you can skip sections or quickly scan sections you are already familiar with. This labeling will also allow you to use this training material as a handy reference. To help you identify and absorb important content quickly, each section is structured as follows

<b>Section Element</b>	<b>Purpose</b>
Title	Serves as a "focus question" that you should be able to answer after completing the section
Learning Objectives	Describes specific content addressed in each section and focuses your attention on important points
Text	Provides the information you need to answer the focus question(s) and achieve the learning objectives
Key Points	Highlights important issues and helps you review
Progress Check exercises	Enables you to test yourself to determine whether you have mastered the learning objectives
Progress Check answers	Provide feedback to ensure you understand the content and can locate information in the text

<b>Learning Objectives</b>	Upon completion of the Lead Toxicity CSEM, you will be able to
<b>Content Area</b>	<b>Objectives</b>
What is lead?	Explain what lead is
Where is lead found?	Describe potential sources of lead exposure in the U.S. today
How are people exposed to lead?	Identify the most important routes of exposure to lead
Who is at risk of lead exposure?	Identify the populations most heavily exposed to lead
What are the US standards for lead levels	Identify the CDC's level of concern for lead in children's blood Identify the OSHA blood lead level for first intervention from occupational exposure to lead Describe the types of environmental standards in the U.S.
What is the biologic fate of lead?	Describe how lead is taken up, distributed, and stored throughout the body Identify the half-life of lead in the blood
What are the physiologic effects of lead exposure?	Describe how lead affects adults and children Describe the major physiologic effects of chronic/ low level lead exposure Describe the major physiologic effects of acute high level lead exposure
How should patients exposed to lead be evaluated?	Describe the CDC's recommendations for screening Describe key features of the exposure history Name the symptoms of low dose lead toxicity Describe how exposure dose and symptoms can vary Describe key features of the physical examination
What tests can assist with the diagnosis of lead toxicity?	Name the most useful test for lead toxicity
How should patients exposed to lead be treated and managed?	Identify three steps that should be taken at blood lead levels between 10 and 19 µg/dL Describe additional steps that should be taken for BLL 20-44 µg/dL, 45-69 µg/dL and 70 µg/dL and above
What instructions should be given to patients?	Identify steps patients with domestic exposures can take to reduce lead exposure Identify steps patients with occupational exposures should take to reduce lead exposure

Initial Check

---

**Instructions** This Initial Check will help you assess your current knowledge about lead toxicity. To take the Initial Check, read the case below, and then answer the questions that follow.

---

**Case Study** A father brings his two-year-old boy into a pediatrician's office for a routine well-child visit. From the father, the doctor learns that the boy's parents are divorced and that he generally lives with his mother and her parents. (The mother had to accompany her parents to her aunt's funeral this weekend and therefore could not make the appointment.) The doctor makes a note of this information.

The pediatrician examines the boy and finds no abnormalities. The boy's growth and development indicators are within normal limits for his age.

Three years later, concerned that her child is hyperactive, the mother brings the same child, now five years old, to your office (his previous pediatrician recently retired). At a parent-teacher conference last week, the kindergarten teacher said that the boy seems impulsive and has trouble concentrating, and recommended evaluation by a physician as well as by the school psychologist. The mother states that he has always seemed restless and easily distracted, but that these first six months in kindergarten have been especially trying.

He has also complained recently of frequent intermittent abdominal pains and constipation. The mother gave him acetaminophen for stomach pains with little change, and has been giving him a fiber laxative, which has reduced the frequency and severity of constipation. She wonders if the change to attending kindergarten has a role in his increased complaints.

Family history reveals that the boy lives with his sister, mother, and maternal grandparents in an older suburb of your community. The child visits with his father one weekend a month, which is working out fine. However, he seems to be fighting more with his sister, who has been diagnosed with attention-deficit disorder and is repeating first grade. Since the mother moved in with her parents after her divorce four years ago, she has worked with the grandfather in an automobile radiator repair shop, where her children often come to play after school. She was just laid off, however, and expressed worry about increasing financial dependence on her parents. She also worries that the grandfather, who has gout and complains increasingly of abdominal pain, may become even more irritable when he learns that she is pregnant.

Her third child is due in 6½ months.

On chart review, you see that the previous pediatrician examined the boy for his preschool physical one year ago. A note describes a very active four year old who could dress himself without help but could not correctly name the primary colors. His vision was normal, but hearing acuity was below normal according to a hearing test administered for his preschool physical. The previous doctor noted

---

---

that the boy's speech and language abilities were slightly delayed. Immunizations are up to date.

Further history on last year's visit indicated adequate diet, with no previous pica behavior. Hematocrit was diminished at 30%. Peripheral blood smear showed hypochromia and microcytosis. There was no evidence of blood loss, and stool examination was negative for occult blood. The diagnosis was "mild iron deficiency anemia," and elemental iron 5 mg/kg per 24 hours (three times daily after meals) was prescribed. The family failed to keep several follow-up appointments, but the child did apparently complete the prescribed 3-month course of iron supplements. He receives no medications at this time and has no known allergies.

On physical examination today, you note that the boy is in the 10th percentile for height and weight. The previous year he fell within the 20th percentile. His attention span is very short, making him appear restless, and he has difficulty following simple instructions. Except for slightly delayed language and social skills, the boy has reached most important developmental milestones.

---

**Initial Check  
Questions**

1. Is there any information that the previous physician should have asked about or looked for (or did not note down) when the boy was brought in as a two year old?
    - A. whether either parent smoked
    - B. age and condition of boy's primary residence and occupations of family members
    - C. the child's birth weight
    - D. whether the child takes vitamins
  2. What should be included in this boy's problem list?
    - A. delayed language ability, slightly impaired hearing
    - B. short stature, anemia and abdominal pain
    - C. possible attention deficit disorder
    - D. All of the above
  3. What test would you order to confirm or rule out your diagnosis?
    - A. capillary blood draw (fingerstick)
    - B. abdominal radiograph
    - C. venous blood lead level
    - D. erythrocyte protoporphyrin (EP) / zinc protoporphyrin (ZPP)
  4. Which other family member is at greatest risk for effects of lead exposure at this time?
    - A. the mother
    - B. the older sister
    - C. the unborn baby
    - D. the grandfather
-

---

**Initial Check  
Answers**

1. **Is there any information that the previous physician should have asked about or looked for (or did not note down) when the boy was brought in as a two year old?**

Answer B. Age and condition of boy's primary residence and occupations of family members

Two of the obvious sources of lead suggested in the case study are leaded paint at home (paint flakes, household dust, and soil) and fumes and dust from solder at the radiator repair shop. You can ask questions about the age of the family's house, when it was most recently painted, and the condition of the paint to get a preliminary sense of the potential extent of this exposure pathway. If the house was built before 1978, the child may be exposed to lead paint chips, lead-contaminated soil, or lead in dust in the home.

Additionally, you should determine if the boy ever had pica (a compulsive eating of nonfood items, to be distinguished from normal hand-to-mouth behavior of children). Pica is more common in children aged two to five, so it is unlikely that this is a present behavior. You can also ask about the length, type, and precise location of the boy's play at the radiator shop.

The previous pediatrician would have done a better job if he or she had asked about the condition of the boy's primary residence as well as the occupations of mother and father.

*The information for this answer comes from section "How Should Patients Exposed to Lead be Evaluated?"*

2. **What should be included in this boy's problem list?**

Answer D. All of the above

History suggests delayed language ability, slightly impaired hearing, short stature, possible attention deficit disorder, anemia and abdominal pain. The child is also experiencing passive exposure to his mother's cigarette smoke and family disruption and possible stress related to his parents' divorce or possibly attending kindergarten.

*The information for this answer comes from section "How Should Patients Exposed to Lead be Evaluated?"*

3. **What test(s) would you order to confirm or rule out your diagnosis?**

Answer C. Venous blood lead level

To confirm lead poisoning, the best test is a venous blood lead level. Capillary blood draws (fingersticks) are not considered reliable for

---

---

diagnosis purposes. A venous or a screening capillary BLL, is usually the first test drawn, instead of the EP/ZPP. Erythrocyte protoporphyrin (EP), commonly assayed as zinc protoporphyrin (ZPP) is not sufficiently sensitive at lower BLLs and therefore is not as useful a screening test for lead exposure in children.

If the blood lead level is below 25 µg/dL, then a serum ferritin level and other iron studies can be used to determine if iron deficiency anemia exists.

*The information for this answer comes from section "What Tests Can Assist with Diagnosis of Lead Toxicity?"*

**4. Which other family member is at greatest risk for effects of lead exposure at this time?**

Answer C. The unborn baby

The mother has recently been laid off, ending the potential occupational exposure. The grandfather may be exposed, as he shows irritability and abdominal pain. Therefore, if this source is removed he should recover. You should, however, suggest that he be tested and talk to his physician about it. The older sister might be at risk from exposure in the home or automotive repair shop, although because she is older she probably will ingest less lead through hand to mouth behavior at this time. However, her history also suggests she may have been exposed as a younger child as well.

The unborn baby is at risk from several sources if the mother has current or past exposure, since lead stored in the bones is mobilized during pregnancy and passed to the fetus through the mother's blood. In addition, the baby will be at risk to potential home-based sources when he or she begins to move around and mouth objects. Prenatal exposure and exposure at a very young age to lead can damage development of the brain.

*The information for this answer comes from section "What Are the Physiologic Effects of Lead Exposure?"*

---



## What is Lead?

<b>Learning Objectives</b>	Upon completion of this section, you will be able to <ul style="list-style-type: none"><li>• explain what lead is.</li></ul>
<b>Definition</b>	Lead is a soft, blue-gray metal. Lead occurs naturally, but much of its presence in the environment stems from its historic use in paint and gasoline and from ongoing or historic mining and commercial operations.
<b>Forms of Lead</b>	<p>Lead exists in both organic and inorganic forms.</p> <p><i>Inorganic lead</i></p> <p>The lead found in old paint, soil, and various products described below is inorganic lead. Leaded gasoline exhaust contributed to ambient inorganic lead contamination. For this reason, the focus of this document is on inorganic lead.</p> <p><i>Organic Lead</i></p> <p>Leaded gasoline contained organic lead before it was burned; however, since the elimination of lead from gasoline in the U.S. starting in 1976, exposure to organic lead is generally limited to an occupational context. However, organic lead <b>can be more toxic</b> than inorganic lead because the <b>body more readily absorbs</b> it. Potential exposures to organic lead should be taken <u>very</u> seriously.</p>
<b>Properties</b>	<p>Lead is a very soft, dense, ductile metal. Lead is very stable and resistant to corrosion, although acidic water may leach out of pipes, fittings, and solder. It does not conduct electricity. Lead is an effective shield against radiation.</p> <p>Because of these properties, and because it is relatively easy to mine and work with, lead has been used for many purposes for thousands of years. Ancient Romans used lead for plumbing, among other uses. In modern times, lead was added to paint and gasoline to improve their performance but was eliminated in the 1970's due to health concerns. Current uses of lead are discussed further in the next section.</p> <p>Accumulation is the result of anthropogenic use, which has concentrated lead throughout the environment. Because lead is spread so widely throughout the environment, it can be found in everyone's body today. The levels found today in most people are orders of magnitude greater than that of ancient times (Flegal 1995). These levels are within an order of magnitude of levels that have resulted in adverse health effects (Budd <i>et al.</i> 1998).</p>
<b>Key Points</b>	<ol style="list-style-type: none"><li>A. Lead is a naturally occurring metal.</li><li>B. Lead is still used widely in commercial products.</li><li>C. Lead is very stable and accumulates in the environment.</li><li>D. Most lead encountered in the environment today is inorganic.</li><li>E. The body absorbs organic lead (as was used in leaded gasoline and is used in occupational settings) faster than inorganic lead.</li></ol>

---

**Progress  
Check**

1. Lead is useful commercially, but also accumulates in the environment, because it
  - A. reacts easily with acids, alkalis, and other chemicals
  - B. does not break down over time
  - C. is very soluble in water
  - D. is most commonly found in the inorganic form.

---

*To review relevant content, see "Properties" in this section.*

---

## Where Is Lead Found?

---

### Learning Objectives

Upon completion of this section, you will be able to

- describe potential sources of lead exposure in the U.S. today.

---

### Introduction

The distribution of lead in the environment varies from place to place. Each of the following sources of lead is discussed further below.

- The most widespread source of lead today for U.S. children is in lead paint that remains in older buildings.
- Lead may be found in and around workplaces that involve lead.
- Lead may contaminate water, food, and beverages, but the contaminant cannot be seen, tasted, or smelled.
- Lead may still be found in some commercial products.
- Some imported home remedies and cosmetics contain lead.
- Lead concentrations in soil, air, and water can be especially high near the sites of historic or ongoing mining operations or smelters.
- While blood lead levels over time are consistently declining, it is still a serious health problem for many, particularly children in urban areas.

Landrigan (2002) estimates that the U.S. incurs \$43.4 billion annually in the costs of all pediatric environmental disease, with childhood lead poisoning alone accounting for the vast majority of it. This is a very high cost to our society, which include medical costs, disability, education and parental lost work time.

---

### Homes and Buildings

Lead was banned from consumer use paint in the U.S. in 1977. Even though leaded paint may be covered with non-leaded paint, lead may still be released into the home environment by peeling, chipping, chalking, friction, or impact. Lead may also be released through past or ongoing home renovation. Lead-contaminated household dust is the major course of lead exposure to children in the U.S. (Lanphear *et al.* 2002)

Between 83% and 86% of all homes built before 1978 in the U.S. have lead-based paint in them. (CDC 1997a)

- The older the house, the more likely it is to contain lead-based paint and to have a higher concentration of lead in the paint.
  - The number of existing U.S. housing units built before 1950, when paint had high lead content, decreased from 27.5 million in 1990 to 25.8 million in 2000 (CDC 2003); despite the gradual decline in the number of houses containing lead paint, however, it still poses a risk.
  - Before 1955, a significant amount of white house paint sold and used was 50% lead and 50% linseed oil. In 1955, manufacturers adopted a voluntary house paint lead-content standard of 1%, but house paint with higher levels of lead continued to be manufactured. (Rabin 1989 as cited in AAP 1993)
  - The amount of lead allowable in paint was lowered by federal law to 1% in 1971 and then to 0.06% in 1977.
  - Workers renovating highway overpasses and bridges are frequently exposed to lead paint applied to these structures over many years before current regulations were in place.
-

- 
- In addition to degradation of interior paint, lead may be tracked into homes in significant quantities from exterior soil that was contaminated by historical use of lead in paint, gasoline, or industries.

---

**Drinking Water**

Lead occurs in drinking water through leaching from lead-containing pipes, faucets, and solder, which in turn can be found in plumbing of older buildings.

- Homes built before 1986 are more likely to have lead pipes, fixtures and solder, although newer homes may also be at risk.
- Boiling water will not get rid of lead.
- Other potential sources of lead contamination include brass fixtures, older drinking water coolers, and older coffee urns (Mushak *et al.* 1989 as cited in AAP 1993).

---

**Foods and Beverages Contaminated with Lead**

Even when lead is not intentionally used in a product, it may contaminate items such as food, water, or alcohol. Lead may contaminate food during

- production and processing
- packaging
- storage

*Production*

Production sources may include

- root vegetables uptake from soil
- atmospheric lead deposition into leafy vegetables (Mushak *et al.* 1989 as cited in AAP 1993)
- grinding or cutting equipment during processing

*Packaging*

Lead in packaging may contaminate food.

- Bright red and yellow paints on bread bags and candy may contain lead (ATSDR 2005; Mushak *et al.* 1989 as cited in AAP 1993).
- Although lead was phased out of cans in the U.S. in the 1980's, some imported cans may still contain lead.

*Storage*

Food or beverages may be stored in lead-containing vessels that contaminate the product.

- Even "safe" pottery and ceramic-ware can become harmful if the protective glaze wears off and exposes people to lead-containing pigments.
  - Lead-glazed pottery, particularly if it is imported, is a potential source of exposure that is often overlooked.
-

- 
- Wine and homemade alcohol that was distilled and/or stored in leaded containers.
  - Wine or other alcoholic drinks stored in leaded-crystal glassware may become contaminated.

*Other*

Other sources of food contamination include

- candies, especially chili-based imported from Mexico
- certain "natural" calcium supplements
- some ceramic tableware (especially imported)

---

**Commercial Products**

While lead is prohibited from many products in the U.S., imported or pre-regulation products may still pose a risk. Consumer products are not routinely tested for lead.

Lead is still used in commercial products such as

- automotive batteries
- bridge paint
- computers
- jewelry
- pewter
- some ceramic glazes

---

**Imported Home Remedies and Cosmetics**

Using certain imported home remedies or cosmetics. Several examples are listed below.

The Mexican folk remedies azarcon and greta used to treat the colic-like illness "empacho" contain lead. These remedies are also known as

- alarcon
- coral
- liga
- Maria Luisa
- rueda

Lead-containing remedies used by some Asian communities are

- ba-baw-san
- bali goli
- chuifong
- ghasard
- kandu
- tokuwan

Middle Eastern remedies and cosmetics include

- alkohl
- cebagin
- saott

---

For more information on these products, see the Centers for Disease Control web site, especially Appendix 1 of the document "Managing Elevated Blood Lead Levels Among Young Children" (CDC 2002) at [http://www.cdc.gov/nceh/lead/CaseManagement/caseManage\\_main.htm](http://www.cdc.gov/nceh/lead/CaseManagement/caseManage_main.htm) or Saper *et al.* 2004.

---

**The Natural Environment**

Because of widespread human use of lead, lead is ubiquitous in the environment. These background levels vary depending on historic and ongoing uses in the area.

- Even abandoned industrial lead sites, such as old mines or lead smelters, may continue to pose a potential public health hazard.
- Industrial sources range in size from large mines and hazardous waste sites (*e.g.*, Superfund sites) to small garages working with old car batteries.
- Industries such as mining and lead smelting contribute to high levels of lead in the environment around such facilities.
- Local community members may be exposed to lead from these sources through ingestion (or inhalation) of lead-contaminated dust or soils.
- Old leaded paint may also contaminate soil, especially in areas immediately adjacent to pre-1978 houses.
- People may be exposed to lead in soils directly or by eating foods grown on lead-contaminated soils.
- The past use of lead in gasoline has contaminated soils, especially along roadways. Tetraethyl lead was phased out of gasoline in the U.S. between 1976 and 1996.

---

**Workplaces**

The major exposure pathways for workers are inhalation and ingestion of lead-bearing dust and fumes.

Workers in the lead smelting, refining, and manufacturing industries experience the highest and most prolonged occupational exposures to lead (ATSDR 2005).

Increased risk for occupational lead exposure occurs among

- battery manufacturing plants
  - construction workers especially renovation/rehabilitation
  - rubber products and plastics industries
  - soldering
  - steel welding/cutting operations
  - other manufacturing industries (ATSDR 2005)
  - bridge maintenance and repair workers
  - municipal waste incinerator workers
  - people who work with lead solder
  - radiator repair mechanics
  - pottery/ceramics industry employees
-

<b>Primary Exposure</b>	<p>It is important to note that occupational exposures can also result in secondary exposure for workers' families if workers bring home lead-contaminated dust on their skin, clothes, or shoes.</p> <ul style="list-style-type: none"> <li>• Children may also be exposed to occupational lead sources if parents work in these industries and allow their children to visit them at work.</li> <li>• Many small businesses and cottage industries are actually located in the home.</li> </ul>
<b>Secondary Exposure</b>	<p>Workers showering and/or changing clothing and shoes can prevent secondary exposures before returning home.</p>

**Table 1: Where Is Lead Found?**

Lead Source	Contaminated Media
Lead solder/pipes	Drinking water
Packages or storage containers	Food, beverages
Paint (pre-1978)	Household dust and soil
Production sources	Imported foods, remedies, cosmetics, jewelry
Mining and smelting	Outdoor air and dust
Workplaces involving lead	Outdoor and indoor air and dust
Gasoline (pre-1988)	Soil

<b>Key Points</b>	<ul style="list-style-type: none"> <li>• Prior to the 1970s, lead was widely used in paint and gasoline.</li> <li>• Lead paint is a primary source of environmental exposure to lead. Lead may be released from old paint in home environments if the paint is disturbed (<i>e.g.</i>, renovation), deteriorated (peeling, chipping, and chalking), or subject to friction or impact (doors, windows, porches, etc...).</li> <li>• The past use of lead in gasoline and paint can result in high lead levels in soil.</li> <li>• Some commercial products still contain lead.</li> <li>• Workers in many industries (and secondary exposure to their families) may have occupational exposure to lead.</li> <li>• Contaminated drinking water, food, alcohol, and home remedies are sources of environmental exposure to lead.</li> <li>• Historic or ongoing lead-related industries (including mining and smelting) can result in high lead levels in surrounding soil.</li> </ul>
<b>Progress Check</b>	<p>2. In older urban areas, most of the lead in the environment today comes from</p> <ul style="list-style-type: none"> <li>A. contaminated drinking water</li> <li>B. lead-contaminated dust, soil, and deteriorated lead-based paint</li> <li>C. imported food, home remedies, and cosmetics</li> <li>D. commercial products containing lead.</li> </ul> <p><i>To review relevant content, see "Homes and Buildings" in this section.</i></p>

## How Are People Exposed to Lead?

---

<b>Learning Objectives</b>	Upon completion of this section, you will be able to <ul style="list-style-type: none"><li>• identify the most important routes of exposure to lead.</li></ul>
<b>Introduction</b>	Today almost everyone is exposed to environmental lead. Exposure to lead and lead chemicals can occur through <b>inhalation</b> , <b>ingestion</b> and <b>dermal contact</b> . <ul style="list-style-type: none"><li>• <b>Most</b> human exposure to lead occurs through <b>ingestion</b> or <b>inhalation</b>.</li><li>• In the U.S. the public is not likely to encounter lead that readily enters the human body through the skin (dermal exposure), as leaded gasoline additives are no longer used.</li><li>• Lead exposure is a global issue. Lead mining and lead smelting are common in many countries, where children and adults can receive substantial lead exposure from sources uncommon today in the U.S. (Kaul <i>et al.</i> 1999; Rothenberg <i>et al.</i> 1994; Litvak <i>et al.</i> 1999; López-Carrillo <i>et al.</i> 1996; Wasserman <i>et al.</i> 1997). Most countries will have phased out use of leaded gasoline by 2007.</li></ul>
<b>Ingestion</b>	Lead exposure in the general population (including children) occurs <b>primarily through ingestion</b> , although inhalation also contributes to lead body burden and may be the major contributor for workers in lead-related occupations. <ul style="list-style-type: none"><li>• Lead paint is the major source of lead exposure for children. (AAP 1993; ATSDR 2005) As lead paint deteriorates, peels, chips, or is removed (<i>e.g.</i>, by renovation), or pulverizes due to friction (<i>e.g.</i>, in windowsills, steps and doors), house dust and surrounding soil may become contaminated. Lead then enters the body through normal hand-to-mouth activity. (Sayre <i>et al.</i> 1974 as cited in AAP 1993)</li><li>• Ingestion of contaminated food, water or alcohol may be significant for some populations. In addition, ingesting certain home remedy medicines may expose people to lead or lead compounds. (See <i>Where Is Lead Found?</i>).</li></ul>
<b>Inhalation</b>	Inhalation is the <b>second major pathway</b> of exposure. Almost all inhaled lead is absorbed into the body, whereas from 20% to 70% of ingested lead is absorbed (with children generally absorbing a higher percentage than adults do) (ATSDR 2005). (See <i>What are the physiologic effects of lead exposure?</i> ). <ul style="list-style-type: none"><li>• Since leaded gasoline additives were phased out beginning in the 1970s, and control measures were implemented in industries, which have reduced air emissions, inhalation is no longer the major exposure pathway for the general population in the U.S.</li><li>• In some foreign countries, however, leaded gasoline is still used, and the resulting emissions pose a major public health threat.</li><li>• Inhalation may be the primary route of exposure to some workers in industries that involve lead.</li><li>• Inhalation may be the primary route of exposure for adults involved in home renovation activities.</li></ul>

---



---

**Dermal**

Dermal exposure plays a role for exposure to organic lead among workers, but is not considered a significant pathway for the general population.

- Organic lead may be absorbed directly through the skin.
- Organic lead (tetramethyllead) is more likely to be absorbed through the skin than inorganic lead.
- Dermal exposure is most likely among people who work with lead.

---

**Endogenous Exposure**

Endogenous exposure to lead may contribute significantly to an individual's current blood lead level, and of particular risk to the developing fetus (see *What are the physiologic effects of lead?*).

- Once absorbed into the body, lead may be stored for long periods in mineralizing tissue (*i.e.*, teeth and bones).
- The stored lead may be released again into the bloodstream, especially in times of calcium stress (*e.g.*, pregnancy, lactation, osteoporosis), or calcium deficiency.

---

**Key Points**

- Ingestion is the most common route of exposure to lead for children, and the route that most commonly leads to illness.
- Inhalation can be a significant exposure pathway, particularly for workers exposed to lead or do-it-yourself home renovators.

---

**Progress Check**

3. The most important route(s) of exposure to lead for children is/are

- A. ingestion and inhalation
- B. inhalation
- C. dermal contact
- D. endogenous sources

*To review relevant content, see "Ingestion" in this section.*

---

## Who Is at Risk of Lead Exposure?

---

<b>Learning Objectives</b>	Upon completion of this section, you will be able to <ul style="list-style-type: none"><li>• identify the populations most heavily exposed to lead.</li></ul>
<b>Introduction</b>	Both children and adults are susceptible to health effects from lead exposure, although the typical exposure pathways and effects are somewhat different. <ul style="list-style-type: none"><li>• Children who reside in pre-1978 housing facilities (and especially those in inner cities or those built before 1950) are at greatest risk for exposure, because the houses may contain lead-based paint.</li><li>• Adults who work in jobs involving lead may be occupationally exposed.</li><li>• Developing fetus are also at risk for adverse health outcomes (less than 1% have levels greater than or equal to 10 µg/dL), as levels that present risk to the fetus do not present risk to the mother.</li></ul>
<b>Children</b>	While children's lead levels have steadily declined in recent decades, some populations of <b>children are still at significant risk</b> of lead poisoning. <ul style="list-style-type: none"><li>• In particular, children who live in older housing are more likely to have elevated BLLs than the population of U.S. children as a whole.</li><li>• It is important to note, however, that no economic or racial/ethnic subgroup of children is free from the risk of having BLLs high enough to cause adverse health effects.</li><li>• Of the children reported with confirmed elevated BLLs between 1997 and 2001, approximately 17% were non-Hispanic whites, 60% were non-Hispanic blacks, 16% were Hispanic, and 7% were of other races or ethnicities. (CDC, 2003)</li><li>• The children affected are more likely to be poor and from racial/ethnic minority groups that cannot afford appropriate housing.</li></ul> <p>Because of their behavior and physiology, <b>children are more affected</b> by exposure to lead than are adults.</p> <ul style="list-style-type: none"><li>• Children absorb more ingested lead than do adults.</li><li>• Children generally ingest lead-contaminated soil and house dust at higher rates than adults because of mouthing and hand-to-mouth behaviors.</li><li>• Children who exhibit pica, a compulsive hand-to-mouth behavior and repeated eating of nonfood items, are at greatest risk.</li><li>• Children have a higher breathing rate than adults, breathing in a greater volume of air per pound.</li><li>• Being shorter than adults are, children are more likely to breathe lead-contaminated dust and soil as well as fumes close to the ground.</li><li>• In addition, the percent of lead absorbed in the gut, especially in an empty stomach, is estimated to be as much as five to 10 times greater in infants and young children than in adults. (Alexander <i>et al.</i> 1974; Chamberlain <i>et al.</i> 1978; James <i>et al.</i> 1985; Ziegler <i>et al.</i> 1978 as cited in ATSDR 1999)</li></ul>

---

- 
- Gastrointestinal absorption of lead in children is increased by iron, calcium, zinc, and ascorbate deficiency. (Mahaffey *et al.* 1990 as cited in AAP 1993)

**Children are more sensitive** than adults are to elevated BLLs. Children's **developing brains** and **nervous system** (and other organ systems) are very sensitive to lead.

- **Childhood lead exposure** has been associated with
  - higher absenteeism in high school
  - lower class rank
  - poorer vocabulary and grammatical reasoning scores
  - longer reaction time
  - poorer hand-eye coordination (AAP, 1993)
- The incomplete development of the blood-brain barrier in fetuses and in very young children (up to 36 months of age) increases the risk of lead's entry into the developing nervous system, which can result in prolonged or permanent neurobehavioral disorders.
- Children's renal, endocrine, and hematological systems may also be adversely affected by lead exposure.

There is **no known threshold exposure level** (as indicated by BLLs) for many of these effects. No blood lead threshold for adverse health effects has been identified in children.

---

## Adults

Although children are at greater risk from lead exposure, adult exposures can also result in harmful health effects.

- **Most adult exposures are occupational** and occur in lead-related industries such as lead smelting, refining, and manufacturing industries.
  - One frequent source of lead exposure to adults is **home renovation** that involves scraping, remodeling, or otherwise disturbing lead-based paint. Renovation involving lead based paint should only be undertaken after proper training, or with the use of certified personnel.
  - Adults can also be exposed during **certain hobbies** and **activities** where lead is used. Some of the more common examples include
    - artistic painting
    - car repair
    - electronics soldering
    - glass or metal soldering
    - glazed pottery making
    - molding of bullets, slugs, or fishing sinkers.
    - stained-glass making
  - target shooting
  - Workers may inhale lead dust and lead oxide fumes, as well as eat, drink, and smoke in or near contaminated areas, thereby increasing their probability of lead ingestion.
  - Between 0.5 and 1.5 million workers are exposed to lead in the workplace (ATSDR, 1999).
  - If showers and changes of clothing are not provided, workers can bring lead dust home on their skin, shoes, and clothing, thus
-

---

inadvertently exposing family members.

- People using paints, pigments, facial makeup, or hair coloring with lead or lead acetate also increase their lead exposure risk. Cosmetics containing lead include surma sindhoor and kohl, popular in certain Asian countries.
- Other than the developmental effects unique to young children, the health effects experienced by adults from adult exposures are similar to those experienced by children, although the thresholds are generally higher.

**Table 2. Populations at Risk of Exposure to Lead in the Workplace**

- Auto repairers
- Battery manufacturers
- Bridge reconstruction workers
- Construction workers
- Firing range instructor
- Gas station attendants (past exposures)
- Glass manufacturers
- Lead manufacturing industry employees
- Lead mining workers
- Lead refining workers
- Lead smelter workers
- Plastic manufacturers
- Plumbers, pipe fitters
- Police officers
- Printers
- Rubber product manufacturers
- Shipbuilders
- Steel welders or cutters

---

**Pregnant Women and Developing Fetuses**

The mother's blood lead level is an important indication of risk to the fetus and neurological problems in newborns. In addition, mothers who had exposure to lead in the past may store lead in their bones. Lead may be released from bones during times of calcium stress such as pregnancy and lactation. Pregnant women with elevated BLLs may have an increased chance of

- preterm labor
- miscarriage
- spontaneous abortion or stillbirth
- low birth weight

See *What are the physiologic effects of lead?* for more information.

---

---

<b>Key Points</b>	<ol style="list-style-type: none"><li>1. Today, the population at greatest risk for lead poisoning is children who live in pre-1978 older housing.</li><li>2. Adults who work with lead or have hobbies involving lead may also be significantly exposed.</li><li>3. Developing fetuses are also at risk for adverse health outcomes.</li></ol>
-------------------	---

---

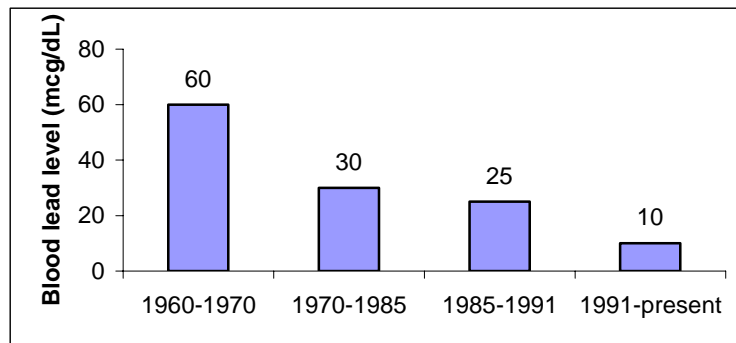
<b>Progress Check</b>	<ol style="list-style-type: none"><li>4. All of the following occupations entail significant exposure to lead except<ol style="list-style-type: none"><li>A. automobile mechanic</li><li>B. construction workers</li><li>C. plumbers</li><li>D. electrician</li></ol></li></ol>
-----------------------	---

*To review relevant content, see "Adults" in this section.*

---

What Are the U.S. Standards for Lead Levels?

<b>Learning Objectives</b>	<p>Upon completion of this section, you will be able to</p> <ul style="list-style-type: none"> <li>• identify the CDC’s level of concern for lead in children’s blood identify the OSHA blood lead level for first intervention from occupational exposure to lead</li> <li>• describe the types of environmental standards in the U.S.</li> </ul>
<b>Introduction</b>	<p>Because of lead’s importance as a cause of public health problems, a number of federal agencies have issued advisory standards or enforceable regulations that set lead levels in different media. The table below summarizes these standards and regulations for 2006; see subsequent sections for further explanation.</p>
<b>Biologic Guidelines</b>	<p>As new information has emerged about the neurological, reproductive, and possible hypertensive toxicity of lead, and as parameters that are more sensitive are developed, the BLLs of concern for lead exposure have been progressively lowered by CDC. (See <b>Figure 1</b> below).</p>



**Figure 1.** Lowering of CDC-recommended action level for blood lead in children over time

**Ten µg/dL (micrograms /deciliter) was adopted by CDC in 1991 as an action level for children, an advisory level for environmental and educational intervention.**

- CDC case management guidelines are designed to keep children’s BLLs below 10 µg/dL (CDC, 2002).
- There are also requirements that children receiving Medicaid be screened.
- Studies have found neurobehavioral impairment in children with BLLs below 10 µg/dL. (Canfield, 2003; Lanphear *et al.* 2000)
- No blood lead threshold has been identified in children.

The Biological Exposure Index (BEI) is a guidance value for assessing biological monitoring results.

The BEI for blood lead is 30 µg/dL. (ACGIH 2005)

The BEI indicates exposure at the Threshold Limit Value (TLV) (See “Workplace Air” below).

<b>Physician</b>	Most states ask or require primary care physicians and persons in charge
------------------	--

---

**Reporting Requirements** of screening programs to report both presumptive and confirmed cases of lead toxicity to the appropriate health agency. This is to ensure

- abatement of the lead source
- education of the patient
- remediation steps are undertaken

In some states, the clinical laboratories performing blood lead testing are required to report cases of lead toxicity.

Even if not required, a physician should strongly consider consulting a health agency in the case of lead toxicity, as health agencies are important sources of resources and information.

In some states, laboratories performing BLL or EP (ZPP) tests are also required to report abnormal results to the appropriate health agency.

---

**Workplace Air** The OSHA Lead Standard specifies the permissible exposure limit (PEL) of lead in the workplace, the frequency and extent of medical monitoring, and other responsibilities of the employer.

OSHA has set a PEL (enforceable) of lead in workplace air at 50  $\mu\text{g}/\text{m}^3$  averaged over an 8-hour workday for workers in general industry.

- For those exposed to air concentrations at or above the action level of 30  $\mu\text{g}/\text{m}^3$  for more than 30 days per year, OSHA mandates periodic determination of BLLs.
- If a BLL is found to be greater than 40  $\mu\text{g}/\text{dL}$ , the worker must be notified in writing and provided with a medical examination.
- If a worker's one-time BLL reaches 60  $\mu\text{g}/\text{dL}$  (or averages 50  $\mu\text{g}/\text{dL}$  or more on three or more tests), the employer is obligated to remove the employee from excessive exposure, with maintenance of seniority and pay, until the employee's BLL falls below 40  $\mu\text{g}/\text{dL}$ .

A copy of the lead standard can be obtained by calling your regional office of OSHA or from the CFR website .

NIOSH at CDC has set a Recommended Exposure Limit (REL) of 50  $\mu\text{g}/\text{m}^3$  to be maintained so that worker blood lead remains < 60  $\mu\text{g}/\text{dL}$  of whole blood. <http://www.cdc.gov/niosh/npg/npgd0368.html>.

The ACGIH has set a threshold limit value for a time-weighted average (TLV/TWA) of 50  $\mu\text{g}/\text{m}^3$  for lead in workplace air (except for lead arsenate). .

---

**Soil** Lead contaminated soil can pose a risk through direct ingestion, uptake in vegetable gardens, or tracking into homes.

- Uncontaminated soil contains lead concentrations less than 50 ppm but soil lead levels in many urban areas exceed 200 ppm. (AAP 1993)
  - The EPA's standard for lead in bare soil in play areas is 400 ppm by weight and 1200 ppm for non-play areas. This regulation applies to
-

---

cleanup projects using federal funds.

The [soil screening level](#) (SSL) for lead represents a conservative estimate for a level that would be protective of public health in residential soils based on an analysis of the direct ingestion pathway for children. This value is for guidance only and is not enforceable.

---

**Drinking Water**

EPA has set drinking water standards with two levels of protection.

- The maximum contaminant level goal (MCLG) is zero. This is the levels determined to be safe by toxicological and biomedical considerations, independent of feasibility.
- EPA's final rule establishes an action level is set at 15 µg/L.

For further information, call the EPA Safe Drinking Water Hotline toll-free at 1-800-426-4791. <http://www.epa.gov/safewater/>

The use of lead solder and other lead-containing materials in connecting household plumbing to public water supplies was banned by EPA as of June 1988.

- Many older structures, however, still have lead pipe or lead-soldered plumbing internally, which may substantially increase the lead content of water at the tap.
- Regulations controlling the lead content of drinking-water coolers in schools went into effect in 1989.
- Residents can buy inexpensive drinking water lead screening kits (see [www.afhh.org](http://www.afhh.org)) or hire professionals to test their water.

---

**Food**

FDA has set a number of action levels (enforceable) and levels of concern for lead in various food items. These levels are based on FDA calculations of the amount of lead a person can consume without ill affect.

- For example, FDA has set an action level of 0.5 µg/mL for lead in products intended for use by infants and children and has banned the use of lead-soldered food cans. (FDA 1994 and FDA 1995 as cited in ATSDR 1999)

---

**Paint**

White house paint contained up to 50% lead before 1955. Federal law lowered the amount of lead allowable in paint to 1% in 1971. The CPSC has limited since 1977 the lead in most paints to 0.06% (600 ppm by dry weight). Paint for bridges and marine use may contain greater amounts of lead.

---



**Table 3: Standards and Regulations for Lead**

Agency	Media	Level	Comments
CDC	Blood	10 µg/dL	Advisory; level for individual management
OSHA	Blood	40 µg/dL	Regulation; cause for written notification and medical exam
		60 µg/dL	Regulation; cause for medical removal from exposure
ACGIH	Blood	30 µg/dL	Advisory; indicates exposure at the threshold limit value (TLV)
OSHA	Air (workplace)	50 µg/m <sup>3</sup>	Regulation; PEL (8-hr average.) (general industry)
		30 µg/m <sup>3</sup>	Action level
CDC/NIOSH	Air (workplace)	100 µg/m <sup>3</sup>	REL (non-enforceable)
ACGIH	Air (workplace)	150 µg/m <sup>3</sup>	TLV/TWA guideline for lead arsenate
		50 µg/m <sup>3</sup>	TLV/TWA guideline for other forms of lead
EPA	Air (ambient)	0.15 µg/m <sup>3</sup>	Regulation; NAAQS; 3-month average
EPA	Soil (residential)	400 ppm (play areas)	Soil screening guidance level; requirement for federally funded projects only (40 CFR Part 745, 2001)
		1200 ppm (non play areas)	
EPA	Water (drinking)	15 µg/L	Action level for public supplies
		0 µg/L	Non-enforceable goal; MCLG
FDA	Food	Various	Action levels for various foods; example: lead-soldered food cans now banned
CPSC	Paint	600 ppm (0.06%)	Regulation; by dry weight. There is a new standard for lead in children's jewelry.

---

**Key Points**

- CDC lowered the recommended blood lead action level for lead exposure in children to 10 µg/dL in 1991.
- States may have their own levels of concern for adults and children.
- Most states have reporting systems for lead poisoning.
- OSHA has set required standards for the amount of lead allowed in workroom air at 50 µg/m<sup>3</sup> averaged over an 8-hour workday.
- EPA has set a standard for lead in the ambient air of 0.15 µg/m<sup>3</sup> averaged over a calendar quarter.
- EPA has established 400 ppm for lead in bare soils in play areas and 1200 ppm for non-play areas for federally funded projects. This may be used as a guidance level elsewhere.
- EPA's action level for lead in water delivered to users of public drinking water systems is 15 µg/L. Its goal for lead is zero.
- FDA has set various action levels regarding lead in food items. Use of lead-soldered food cans is now banned.
- Today, paint intended for residential use is limited to 0.06% lead content.

---

**Progress  
Check**

5. The CDC's action level of 10 µg/dL for children's blood is
- A. the blood lead level below which no effects have been found
  - B. also used by OSHA as a level of concern in workers
  - C. an advisory level for environmental and educational intervention
  - D. a regulatory level at which children must be removed immediately removed from any pre-1978 residences.

---

*To review relevant content, see "Biologic Guidelines" in this section.*

## What Is the Biologic Fate of Lead?

---

<b>Learning Objectives</b>	Upon completion of this section, you will be able to <ul style="list-style-type: none"><li>• describe how lead is taken up, distributed, and stored throughout the body</li><li>• identify the half-life of lead in the blood.</li></ul>
<b>Introduction</b>	<p>The absorption and biologic fate of lead once it enters the human body depend on a variety of factors including nutritional status, health, and age.</p> <ul style="list-style-type: none"><li>• Adults typically absorb up to 20% of ingested lead.</li><li>• Most inhaled lead in the lower respiratory tract is absorbed.</li><li>• Most of the lead that enters the body is excreted in urine or through biliary clearance (ultimately, in the feces).</li></ul> <p>The chemical form of lead, or lead compounds, entering the body is also a factor for the absorption and biologic fate of lead.</p> <ul style="list-style-type: none"><li>• Inorganic lead, the most common form of lead, is not metabolized in the liver.</li><li>• Nearly all organic lead that is ingested is absorbed.</li><li>• Organic lead compounds (far rarer today after EPA's ban on gasoline additives containing lead) are metabolized in the liver.</li></ul> <p>Absorbed lead that is not excreted is exchanged primarily among <u>three compartments</u></p> <ul style="list-style-type: none"><li>• Blood</li><li>• Mineralizing tissues (bones and teeth), which typically contain the vast majority of the lead body burden</li><li>• Soft tissue (liver, kidneys, lungs, brain, spleen, muscles, and heart)</li></ul> <p>These compartments, and the dynamics of the exchange between them, are discussed below.</p>
<b>Lead in the Blood</b>	<p>Although the blood generally carries only a small fraction of total lead body burden, it does serve as the initial receptacle of absorbed lead and distributes lead throughout the body, making it available to other tissues (or for excretion).</p> <ul style="list-style-type: none"><li>• The half-life of lead in adult human blood has been estimated to be from 28 days (Griffin <i>et al.</i> 1975 as cited in ATSDR 2005) to 36 days. (Rabinowitz <i>et al.</i> 1976 as cited in ATSDR 2005)</li><li>• Approximately 99% of the lead in blood is associated with red blood cells; the remaining 1% resides in blood plasma. (DeSilva 1981; EPA, 1986a; Everson and Patterson, 1980, as cited in ATSDR, 1999)</li><li>• In addition, the higher the lead concentration in the blood, the higher the percentage partitioned to plasma. This relationship is curvilinear – as blood lead levels (BLLs) increase as the high-end plasma level increases more.</li></ul>

---

---

Blood lead is also important because the BLL is the most widely used measure of lead exposure.

- These tests, however, do not measure total body burden—they are more reflective of recent or ongoing exposures (see “*Laboratory Evaluation*” section).

---

**Lead in Mineralizing Tissues (Bones and Teeth)**

The bones and teeth of adults contain about 94% of their total lead body burden; in children, the figure is approximately 73%. (Barry 1975 as cited in ATSDR 2005) Lead in mineralizing tissues is not uniformly distributed. It tends to accumulate in bone regions undergoing the most active calcification at the time of exposure.

- Known calcification rates of bones in childhood and adulthood suggest that lead accumulation will occur predominately in trabecular bone during childhood, and in both cortical and trabecular bone in adulthood (Auf der Heide and Wittmets 1992; as cited in ATSDR 1999).
- A new test to measure lead in bone (K-XRF, or K X-ray fluorescence) usually measures lead levels in trabecular bone at the patella or calcaneus and cortical bone at the tibia. However, this test is mostly used for research now.

Two physiological compartments appear to exist for lead in cortical and trabecular bone (ATSDR, 2005; ATSDR, 2000).

- the inert component stores lead for decades
- the labile component readily exchanges bone lead with the blood.

Under certain circumstances, however, this apparently inert lead will leave the bones and reenter the blood and soft tissue organs.

- Bone-to-blood lead mobilization increases during periods of pregnancy, lactation, menopause, physiologic stress, chronic disease, hyperthyroidism, kidney disease, broken bones, and advanced age, all which are exacerbated by calcium deficiency.
- Consequently, the normally inert pool poses a special risk because it is a potential endogenous source of lead that can maintain BLLs long after exposure has ended.

---

**Implications of Biologic Fate**

Because lead from past exposures can accumulate in the bones (endogenous source), symptoms or health effects can also appear in the absence of significant current exposure.

- In most cases, toxic BLLs reflect a mixture of current exposure to lead and endogenous contribution from previous exposure.
  - An acute high exposure to lead can lead to high short-term BLLs and cause symptoms of lead poisoning.
  - It is important that primary care physicians evaluate a patient with potential lead poisoning, examine potential current *and* past lead exposures and look for other factors that affect the biokinetics of lead (such as pregnancy or poor nutrition).
-

---

**Key Points**

- Once in the bloodstream, lead is primarily distributed among three compartments—blood, mineralizing tissue, and soft tissues. The bones and teeth of adults contain more than 95% of total lead in the body.
- In times of stress (particularly pregnancy and lactation), the body can mobilize lead stores, thereby increasing the level of lead in the blood.
- The body accumulates lead over a lifetime and normally releases it very slowly.
- *Both past and current* elevated exposures to lead increase patient risks for lead effects.

---

**Progress  
Check**

6. What is the approximate half-life of lead in the blood?
- A. seven days
  - B. thirty days
  - C. three to six months
  - D. one year

*To review relevant content, see "Lead in the Blood" in this section.*

---

## What Are the Physiologic Effects of Lead Exposure?

---

<b>Learning Objectives</b>	Upon completion of this section, you will be able to <ul style="list-style-type: none"><li>• describe how lead affects adults and children</li><li>• describe the major physiologic effects of chronic low- level lead exposure</li><li>• describe the major physiologic effects of acute high-level lead exposure.</li></ul>
<b>Introduction</b>	<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"><li>• Lead toxicity can affect every organ system.</li><li>• 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).</li></ul> <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"><li>• The practicing health care provider can distinguish overt clinical symptoms and health effects that come with high exposure levels on an individual basis.</li><li>• However, lack of overt symptoms does not mean “no lead poisoning.”</li><li>• Lower levels of exposure have been shown to have many subtle health effects.</li><li>• 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).</li><li>• It is important to prevent all lead exposures.</li></ul> <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>
<b>Neurological Effects</b>	<p>The nervous system is the most sensitive target of lead exposure.</p> <ul style="list-style-type: none"><li>• There may be no lower threshold for some of the adverse neurological effects of lead in children.</li><li>• Neurological effects of lead in children have been documented at exposure levels once thought to cause no harmful effects (&lt;10 µg/dL). (Canfield 2003; CDC 1997a)</li><li>• 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.</li></ul>

---

---

**Children**

In children, acute exposure to very high levels of lead may produce encephalopathy and other accompanying signs of

- ataxia
- coma
- convulsions
- death
- hyperirritability
- stupor

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)

- Even without encephalopathy symptoms, these levels are associated with increased incidences of lasting neurological and behavioral damage. (ATSDR 2005)

Children suffer neurological effects at much lower exposure levels.

- 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 *et al.*, 1981; Schroeder *et al.*, 1985; Fulton *et al.*, 1987; Landsdown *et al.* 1986; Hawk *et al.* 1986; Winneke *et al.* 1990 as cited in AAP 1993)
- 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.

---

**Adults**

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.

- 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.

Lead encephalopathy may occur at extremely high BLLs, *e.g.*, 460 µg/dL. (Kehoe 1961 as cited in ATSDR 2005)

- Precursors of encephalopathy, such as dullness, irritability, poor
-

---

attention span, muscular tremor, and loss of memory may occur at lower BLLs.

Less severe *neurological and behavioral effects* have been documented in lead-exposed workers with BLLs ranging from 40 to 120 µg/dL. (ATSDR 2005) These effects include

- decreased libido
- depression/mood changes, headache
- diminished cognitive performance
- diminished hand dexterity
- diminished reaction time
- diminished visual motor performance
- dizziness
- fatigue
- forgetfulness
- impaired concentration
- impotence
- increased nervousness
- irritability
- lethargy
- malaise
- paresthesia
- reduced IQ scores
- weakness

There is also some evidence that lead exposure may affect adults' postural balance and peripheral nerve function. (ATSDR 1997a, b; Arnvig *et al.* 1980; Haenninen *et al.* 1978; Hogstedt *et al.* 1983; Mantere *et al.* 1982; Valciukas *et al.* 1978 as cited in ATSDR 1999)

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

---

### Renal Effects

Many studies show a strong association between lead exposure and renal effects. (ATSDR 1999)

- 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.e.*, chronic interstitial nephritis).

The lowest level at which lead has an adverse effect on the kidney remains unknown.

- 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).
  - 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 uncommon occurrence in non lead-associated gout. (Goyer 1985, as cited in ATSDR 2000)
- A study by Batuman *et al.* (1981) suggests that renal disease is more frequent and more severe in saturnine gout than in primary gout.

---

**Hematological Effects**

Lead inhibits the body's ability to make hemoglobin by interfering with several enzymatic steps in the heme pathway.

- Specifically, lead decreases heme biosynthesis by inhibiting *d*-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 d-aminolevulinic acid (ALA) and free erythrocyte protoporphyrins (FEP) (EPA 1986a as cited in ATSDR 1999).

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 (*e.g.*, 25 µg/dL) for children. (EPA 1986b as cited in ATSDR 1999; ATSDR 1999)

- 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 “*Laboratory Evaluation*” section for further discussion of EP testing.).

Lead can induce two types of anemia, often accompanied by basophilic stippling of the erythrocytes. (ATSDR 1999)

- 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.
- 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.

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.

- There is a concern about the meaning of and possible sequelae of these biochemical and enzyme changes at lower levels of lead.

---

**Endocrine Effects**

Studies of children with high lead exposure have found that a strong inverse correlation exists between BLLs and vitamin D levels.

- 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
-

	<p>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)</p> <ul style="list-style-type: none"> <li>• However, Rosen et al (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.</li> <li>• Lead appears to have a minimal, if any, effect on thyroid function.</li> </ul>
<b>Gastro-intestinal Effects</b>	<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>
<b>Cardiovascular Hypertension Effects</b>	<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"> <li>• Lead exposure is one factor of many that may contribute to the onset and development of hypertension.</li> <li>• Although low to moderate lead level exposures (BLL &lt; 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.</li> <li>• 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)</li> <li>• 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.</li> </ul>
<b>Reproductive Effects</b>	<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><i>Male Reproductive Effects</i></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"> <li>• Effects may begin at BLLs of 40 µg/dL. (ATSDR 2005)</li> <li>• 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</li> </ul>

---

2000)

- It is unclear how long these effects may last in humans after lead exposure ceases.

#### *Fertility*

It is not currently possible to predict fertility outcomes based on current BLLs or past lead exposure levels. (ATSDR 2000)

#### *Pregnancy Outcomes*

The effect of low-level lead exposures on pregnancy outcomes is not clear. Thus it appears that at higher (*e.g.*, 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.

- Some studies of women living near smelters versus those living some distance away did show increased frequency of spontaneous abortions (Nordstrom *et al.* 1979) and miscarriages and stillbirths. (Baghurst *et al.* 1987; McMichael *et al.* 1986)
- In contrast, Murphy *et al.* (1990) evaluated past pregnancy outcomes among women living in the vicinity of a lead smelter and did not 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, *et al.* 1999).

---

#### **Developmental Effects**

Developmental effects examined in the literature include pregnancy outcomes (*e.g.*, premature births and low birth weights), congenital abnormalities, and post birth effects on growth or neurological development.

- 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 *et al.* (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 *et al.* 1985, 1986; McMichael *et al.* 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).

---

**Other  
Potential  
Effects**

Lead has been linked to problems with the development and health of bones. At high levels, lead can result in slowed growth in children.

- 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.

Current available data are not sufficient to determine the carcinogenicity of lead in humans.

- 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 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).

---

**Key Points**

- Effects in children generally occur at lower BLLs than in adults.
  - The developing nervous system of a child can be affected adversely at BLLs of less than 10 µg/dL. It is often impossible to determine these effects upon clinical examination.
  - There is a wide range of neurological effects associated with lead exposure, some of which may likely be irreversible.
  - Lead exposure can lead to renal effects such as Fanconi-like syndromes, chronic nephropathy, and gout.
-

- 
- Most lead-associated renal effects or disease are a result of ongoing chronic or present high acute exposure or can be a latent effect of chronic past lead exposure.
  - Lead inhibits several enzymes critical to the synthesis of heme, causing a decrease in blood hemoglobin.
  - Today, lead exposure in children only rarely results in frank anemia.
  - Lead's impairment of heme synthesis can affect other heme-dependent processes in the body outside of the hematopoietic system.
  - Lead interferes with a hormonal form of vitamin D, which affects multiple processes in the body, including cell maturation and skeletal growth.
  - Lead exposure may lead to increased risk for hypertension and its sequelae.
  - Evidence suggests an association between lead exposure and certain reproductive and developmental outcomes.
  - Maternal blood lead, from exogenous and endogenous sources, can cross the placenta and put the fetus at risk.
  - Other potential health effects of lead are currently being studied.

---

**Progress  
Check**

7. How do lead's effects differ in children and adults?
- A. Effects in children are more likely to be reversible.
  - B. Adults suffer more neurological damage.
  - C. Children are less likely to become anemic.
  - D. Effects in adults tend to begin at higher exposure levels than in children.

*To review relevant content, see "[Adults](#)" in this section.*

8. Lead toxicity can affect
- A. the kidneys and brain
  - B. IQ and neurological development in children
  - C. sperm count
  - D. all of the above.

*To review relevant content, see "[Neurological Effects, Children, Renal Effects, and Reproductive Effects](#)" in this section.*

---

## How Should Patients Exposed to Lead Be Evaluated?

---

### Learning Objectives

Upon completion of this section, you will be able to

- describe the CDC's recommendations for blood screening
- describe key features of the exposure history
- name the symptoms of low dose lead toxicity
- describe how exposure dose and symptoms can vary
- describe key features of the physical examination.

---

### Introduction

Because children may be exposed to levels of lead which could adversely affect their health without exhibiting clinical symptoms, it is vital that primary care providers adopt a preventive approach to determine which of their patients may be at risk.

Primary care providers can adopt a preventive approach by asking questions to assess a patient's potential for exposure to lead and/or by following statewide protocols for screening. Where the potential for exposure exists, a patient's blood lead levels (BLL) should be tested.

This section focuses on preventive screening, physical examination, and signs and symptoms. Recommended tests are discussed in the next section.

---

### Preventive Assessment and Screening

A primary care provider may identify individuals who may be exposed to potentially dangerous levels of lead before symptoms of lead poisoning manifest themselves. It is often possible and many times crucial for the primary care provider to screen appropriately, manage patients, and facilitate appropriate environmental and nutritional intervention.

Recognition of a lead exposure often depends on the initial reporting of high BLLs by primary care providers.

- In the case of children, CDC recommends that states develop statewide plans for BLL screening (CDC 1997a).
  - These plans and practices vary from state to state (NCHH 2001) and may advocate universal screening of children from high-risk areas at ages one or two and of all children up to age seven who have not previously been screened. Alternatively, they may call for targeted screening based on responses to several questions intended to determine risk more selectively (*e.g.*, type and age of house and whether or not patient's family is a Medicaid recipient).
  - Some local health departments, such as the City of Chicago, recommend testing every six months beginning at six or nine months of age.
  - Contact your state or local health department to see if your state has a lead screening plan.
  - If your pediatric patient falls into a category such as Medicaid where screening is required or recommended, it is important to follow the guidelines and screen the patient. It is equally important to report a positive test to the appropriate agency(s).
  - For occupationally exposed adults, OSHA is responsible for issuing standards and regulations that pertain to workplace exposures
-

---

(<http://osha.gov/SLTC/lead/index.html>). However, the primary care provider should be aware if a patient fits into an occupational group exposed to lead and whether the BLL is being monitored, when evaluating the patient.

---

**Lead Exposure  
Risk Questions**

The first step in identifying individuals with potential lead exposure is to determine through appropriate questioning whether any of the typical lead exposure pathways are cause for heightened concern. (In the case study, the fact that the previous pediatrician apparently did not pursue this line of questioning constitutes a missed opportunity for preventive action.)

- Many health departments can provide physicians with personal risk questionnaires and/or localized risk information to help in this process (see the "*Sources of Information*" section).
- Another useful resource is the CDC publication, "[Managing elevated blood lead levels among young children](#)" (CDC 2002).

Here are some of the issues a physician might discuss with the patient and/or family (see also [Case Studies in Environmental Medicine: Taking an Exposure History](#)" [ATSDR 1992]).

- condition of household pets
- drinking water source and type of pipes
- family history, including possibility of maternal/family exposure and potential use of unusual medicines or home remedies
- frequency of visits to houses or facilities built before 1950
- hobbies of all family members
- home remodeling activities
- location, age, physical condition of current residence, school, and day-care center, etc. (to identify potential for lead paint as well as proximity to industrial facilities, hazardous waste sites, and other potential lead sources)
- nutritional status
- occupational history of all home occupants
- past living conditions (international background is important)
- siblings or playmates who have been diagnosed with lead poisoning
- use of imported or glazed ceramics.

Lead is most harmful to children *under six years of age*.

- Every child who has a developmental delay, behavioral disorder, or speech impairment, or who may have been exposed to lead, should be screened with a blood lead test.
- Equally important, siblings, housemates, and playmates of children with suspected lead toxicity probably have similar exposures to lead and should be screened.

Individuals with potentially high lead exposure should be screened with a blood lead test.



- 
- They (and/or their parents) should also receive lead education, including:
    - behavioral interventions
    - guidance on appropriate nutrition
    - environmental interventions (see "*Treatment and Management*" section).
  - Physicians may want to consider giving parents anticipatory guidance prenatally and before a child reaches one year of age.
  - Physicians should take advantage of the programs and printed materials available through state and/or local health departments in providing this guidance.
- 

**Physical Examination**

In addition to the environmental/family history assessment and BLL screening described above, physicians should conduct a complete physical examination of patients with potential exposure to lead.

It is important to keep in mind, however, that even a complete physical examination may not identify subtle neurological effects that may be associated with low-level lead exposure in children.

The physical examination should include special attention to these systems

- neurological
- hematological
- cardiovascular
- gastrointestinal
- renal

Areas of special concern for health care providers.

- Carefully evaluate the nervous system, including behavioral changes.
- Check blood pressure to evaluate whether the patient is hypertensive and pay special attention to the renal system in those who are positive for hypertension.
- Check for a purplish line on the gums (lead line). This is rarely seen today, but if present, usually indicates severe and prolonged lead poisoning.

Areas of special concern for children by health care providers.

- Hearing, speech, and other developmental milestones should be carefully evaluated and documented.
  - Since iron and calcium deficiencies are known to enhance the absorption of lead and to aggravate pica, it is especially important to assess the nutritional status of young children.
  - The opening case study illustrates a second missed opportunity: despite the delayed growth (20<sup>th</sup> percentile) and speech indicators discovered during the preschool physical (at age four), no BLL test was ordered at that time.
  - When the neurological exam, milestones, or behavior suggest it, further neurobehavioral testing, or evaluation for ADHD, may be indicated.
-

---

**Signs and Symptoms**

Most patients who suffer from lead poisoning are asymptomatic, hence the importance of exposure assessment and screening. There is a continuum of signs and symptoms depending on level and duration of lead exposure (see **Table 4.**)

- At the low exposure levels found today, most children will be asymptomatic, but these levels may still impair the health of children and adults. With increasing exposure dose, the severity of symptoms can be expected to increase.
- Because of differences in individual susceptibility, symptoms of lead exposure and their onset may vary.
- The impaired abilities may occur at BLLs ranging from 10 to 25 µg/dL, whereas in symptomatic lead intoxication, BLLs generally range from 35 to 50 µg/dL in children and 40 to 60 µg/dL in adults.
- Severe toxicity (high exposure dose) is frequently found in association with BLLs of 70 µg/dL or more in children and 100 µg/dL or more in adults.

The impaired abilities that may be associated with lead exposure in an apparently asymptomatic patient are listed in **Table 4.** Also shown are overt symptoms of lead toxicity associated with ongoing exposure. In interpreting this table, it is important to remember that

- Some of the hematological abnormalities of lead poisoning are similar to those of other diseases or conditions. In the differential diagnosis of microcytic anemia, lead poisoning can usually be ruled out by obtaining a venous blood lead concentration; if the BLL is less than 25 µg/dL, the anemia usually reflects iron deficiency or hemoglobinopathy. Two rare diseases, acute intermittent porphyria and coproporphyrinuria, result in heme abnormalities similar to those of lead poisoning, too.
- Patients exhibiting neurological signs due to lead exposure have been treated only for peripheral neuropathy or carpal tunnel syndrome, delaying treatment for lead intoxication.
- Failure to diagnose correctly lead-induced gastrointestinal distress has led to inappropriate abdominal surgery.
- Current health effects (*e.g.*, neurological/developmental) resulting from past exposure, even without current exposure, may also need intervention, if, for example, special education is needed, or if the danger of exposure is still present and/or to prevent exposure in others.

Keep in mind that dividing the signs and symptoms by exposure dose from lowest to high is somewhat artificial — the signs and symptoms generally increase with increasing BLL but in some individuals may appear at variance with these designations. The importance for the clinician is to recognize ongoing lead exposure, interrupt that exposure, and treat the patient as appropriate.

---

**Table 4: Continuum of signs and symptoms of ongoing lead exposure**

**Lowest Exposure Dose Signs and Symptoms:  
Impaired Abilities (patient may appear  
asymptomatic)**

- Decreased learning and memory
- Lowered IQ
- Decreased verbal ability
- Impaired speech and hearing functions
- Early signs of hyperactivity or ADHD

**Low Exposure Dose Signs and Symptoms**

- Myalgia or paresthesia
- Mild fatigue
- Irritability
- Lethargy
- Occasional abdominal discomfort

**Moderate Exposure Dose Signs and Symptoms**

- Arthralgia
- General fatigue
- Difficulty concentrating/Muscular exhaustibility
- Tremor
- Headache
- Diffuse abdominal pain
- Vomiting
- Weight loss
- Constipation

**High Exposure Dose Signs and Symptoms**

- Paresis or paralysis
- Encephalopathy—may abruptly lead to seizures, changes in consciousness, coma, and death
- Lead line (blue-black) on gingival tissue
- Colic (intermittent, severe abdominal cramps)

---

**Key Points**

- Because children may be exposed to potentially adverse levels of lead without exhibiting clinical symptoms, it is vital that primary care providers adopt a preventive approach to determine which of their patients may be at risk.
- While important for monitoring the effects of lead exposure and, in some cases, for identifying the symptoms of lead poisoning, the physical examination alone will not always reveal when a patient is at risk from elevated lead exposure.
- The first signs of lead poisoning in children are often subtle neurobehavioral problems that adversely affect classroom behavior and social interaction.
- Developmental, speech, and hearing impairments are not uncommon in lead-exposed children (ATSDR 2005).
- Most persons with lead toxicity are not overtly symptomatic.
- Some of the health effects of lead exposure on the various organ systems (see "*Physiological Effects*" section) are permanent or latent and may appear after exposure has ceased.

---

**Progress Check**

9. As part of the exposure history, you should explore
- A. possible lead exposure at work or home remodeling
  - B. hobbies that might involve lead
  - C. use of imported home remedies and cosmetics
  - D. all of the above.

*To review relevant content, see "Lead Exposure Risk Questions" in this section.*

10. How should individuals and children with potentially high lead exposures be screened?
- A. a long bone X-ray
  - B. ultrasound
  - C. Blood Lead Levels (BLL) test
  - D. all of the above.

*To review relevant content, see "Lead Exposure Risk Questions" in this section.*

---

### What Tests Can Assist with the Diagnosis of Lead Toxicity?

---

<b>Learning Objectives</b>	Upon completion of this section, you will be able to <ul style="list-style-type: none"><li>• name the most useful test for lead toxicity.</li></ul>
<b>Introduction</b>	Venous Blood Lead Level (BLL) testing is the most useful screening and diagnostic test for recent or ongoing lead exposure as opposed to past exposures.
<b>Blood Lead Levels</b>	<p>Different tests have been used in the past to evaluate lead exposure and/or to gauge the effects of lead exposure.</p> <ul style="list-style-type: none"><li>• Venous BLL testing is the most useful screening and diagnostic test for recent or ongoing lead exposure as opposed to past exposures.</li><li>• Given the greater risk of contamination using the finger-stick method, an elevated BLL obtained through finger sticking should always be confirmed through venipuncture. (AAP 1993 and CDC, 1997a)</li><li>• BLLs respond relatively rapidly to abrupt or intermittent changes in lead intake (for example, ingestion of lead paint chips by children) and, for relatively short exposure periods, bear a linear relationship to those intake levels.</li><li>• For individuals with high or chronic past exposure, however, BLLs often under-represent the total body burden because most lead is stored in the bone and may have “normal” levels in the blood.</li><li>• One exception is patients with a high body burden under physiological stressful circumstances whose BLLs may be elevated from the release of lead stored in bones.</li><li>• Erythrocyte protoporphyrin (EP), commonly assayed as zinc protoporphyrin (ZPP), was previously considered the best test for screening for asymptomatic children, however, is not sufficiently sensitive at lower BLLs and therefore is not as useful a screening test for lead exposure as previously thought.</li></ul> <p>Over the past 30 years, there has been a dramatic decline, nationwide, in blood lead levels (BLLs). Findings from the most recent National Health and Nutrition Examination Survey (NHANES), 1999 – 2002, indicate that BLLs are continuing to decrease across all age and racial/ethnic groups in the U.S. Although BLLs remained higher for young non-Hispanic black children, this group also experienced the greatest decline (72%) in elevated BLLs since 1991-1994. (CDC 2005)</p> <ul style="list-style-type: none"><li>• The overall prevalence of elevated BLLs (<math>\geq 10 \mu\text{g/dL}</math>) for the U.S. population was 0.7%. (CDC 2005)</li><li>• The average BLL for children 1-5 years of age was <math>1.9 \mu\text{g/dL}</math> in 2002, down from <math>15.0 \mu\text{g/dL}</math> in 1976-1980 (before leaded gasoline was banned; CDC 2005).</li><li>• The average BLL for adults 18-74 years of age was <math>14.2 \mu\text{g/dL}</math> from 1976-70; in 1988-1991, the average BLL for adults was <math>3.0 \mu\text{g/dL}</math> (CDC 1997b).</li></ul>

---

- 
- An attempt should be made to identify and minimize lead exposures when BLLs indicate that they are occurring at any blood lead level above background population levels.
  - If an adult has a BLL of 20 µg/dL, *e.g.*, an unusual exposure is likely occurring and should be interrupted, if possible. This is especially important for fertile and pregnant females.

---

**EP and ZPP Levels**

Recent data indicate that the EP/ZPP assay at lower BLLs is not sufficiently sensitivity, and therefore is not as useful a screening test for lead exposure as previously thought. EP/ZPP assays continue to be used at times as a complement to venous BLL testing and are required by OSHA for some workplace testing.

Normal values of ZPP are usually below 35 µg/dL.

EP is also elevated in

- iron deficiency anemia
- jaundice
- sickle cell
- other hemolytic anemias

In erythropoietic protoporphyria, an extremely rare disease, EP is markedly elevated (usually above 300 µg/dL).

---

**Other Evaluation Methods**

There are different evaluation methods used to study patients with elevated BLL.

- Complete blood count (CBC) may be useful for patients with extensive exposure. In lead-exposed patients, the hemocrit and hemoglobin values may be slightly to moderately low in the CBC, and the peripheral smear may be either normochromic and normocytic or hypochromic and microcytic.
    - There may be **basophilic stippling** in patients who have been significantly poisoned for a prolonged period.
    - However, because these results are not specific to lead exposure, the CBC test is not as valuable for detecting lead exposure as the BLL and EP assays.
    - A hypochromic, microcytic anemia should be appropriately differentiated from other causes, especially iron-deficiency anemia by the use of testing for iron, iron binding capacity, and ferritin.
  - Abdominal radiographs may show the presence of radio-dense lead foreign bodies in the gastrointestinal tract. These are helpful only in cases of acute ingestion (*e.g.*, of lead sinkers, curtain weights, jewelry, or paint chips) or unusual persistence of high blood lead values. Longbone radiographs can show "Lead Lines". These are lines of increased density on the metaphysis growth plate of the bone, showing radiological growth retardation. This is not a routine procedure to identify lead poisoning, but a radiological finding of chronic exposure. See **Figures 2 & 3** - Longbone Radiographs.
  - Because hair and fingernails are subject to external environmental
-

---

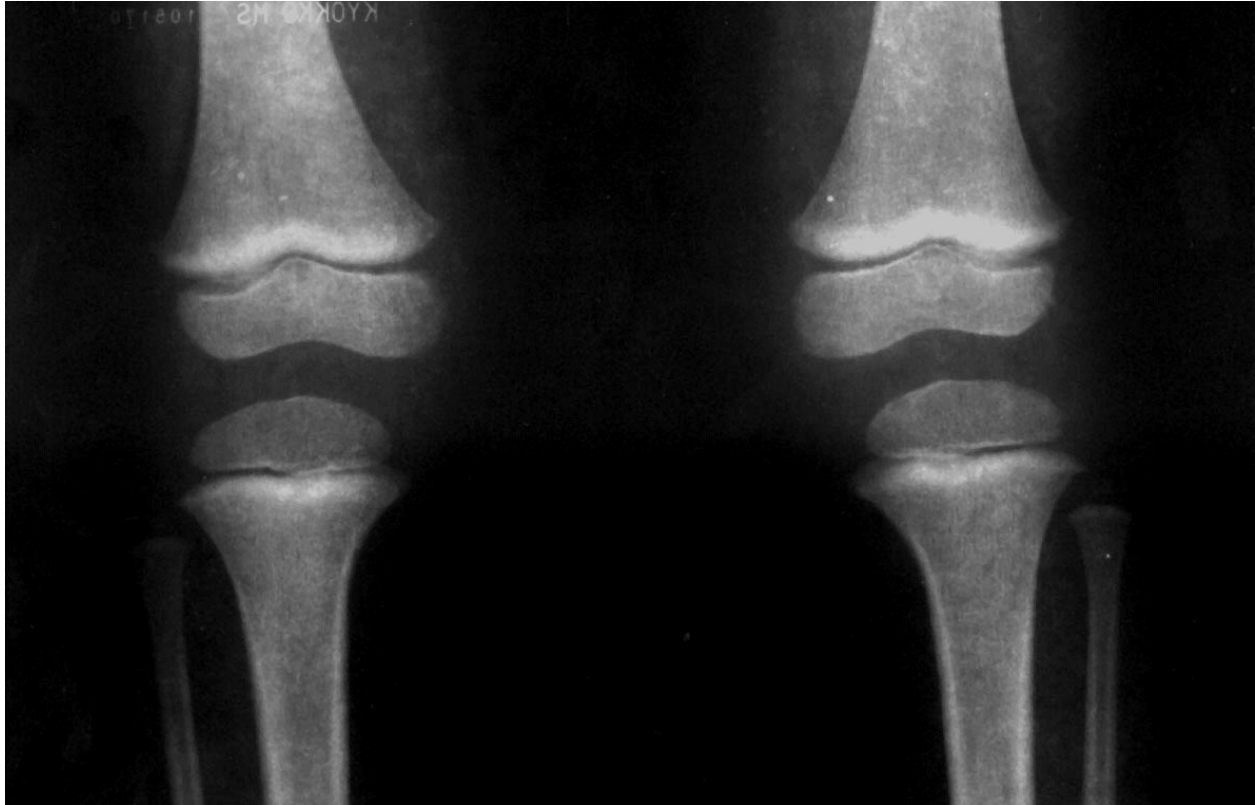
contamination, assaying their lead content is an uncertain estimate of body burden and is not recommended (AAP 1993; CDC 2002).

- Longbone radiographs are not recommended for diagnosing lead exposure (CDC 2002), however, they are useful to determine reduced growth retardation.
  - Second tier tests (such as neurobehavioral/psychological evaluation for children with indicative findings on exam) should be considered, as appropriate.
  - Evaluation may also appropriately include tests for the health effects of lead.
- 



**Figure 2. Longbone radiographs of hands**

"Lead lines" in five year old male with radiological growth retardation and blood lead level of 37.7 $\mu$ g/dL. (Photo courtesy of Dr. Celsa López, Clinical Epidemiologic Research Unit, IMSS, Torreón, México)



**Figure 3. Longbone radiographs of knees.** “Lead lines” in three-year-two-month-old girl with Blood lead level of 10.6 µg/dL. Notice the increased density on the metaphysis growth plate of the knee. (Photo courtesy of Dr. Celsa López, Clinical Epidemiologic Research Unit, IMSS, Torreón, México)

**Key Points**

- The best screening and diagnostic tool for evaluating lead exposure is the venous BLL test.
- Using an EP or ZPP assay to screen children for lead exposure is not as useful as once thought, and not recommended.
- Other tests may be appropriate for specific situations, such as abdominal radiographs to detect swallowed objects.
- Secondary testing for other health effects may also be useful.

**Progress Check**

11. What test is best to confirm lead poisoning in a child?
- A. EP/ZPP
  - B. capillary blood lead level (fingerstick)
  - C. venous blood lead level (BLL)
  - D. abdominal radiograph

*To review relevant content, see “Blood Lead Levels” in this section.*



**How Should Patients Exposed to Lead be Treated and Managed?**

---

<b>Learning Objectives</b>	Upon completion of this section, you will be able to <ul style="list-style-type: none"><li>• identify three steps that should be taken at blood lead levels between 10 and 19 µg/dL</li><li>• describe additional steps that should be taken for BLL 20-44 µg/dL, 45-69 µg/dL and 70 µg/dL and above.</li></ul>
<b>Introduction</b>	In general, the most important management tool for lead-associated diseases is to remove the source of lead exposure. In addition, lead may cause a variety of other diseases and conditions (see “What Are the Physiologic Effects of Lead section) that should be managed appropriately. Since none of these effects is specific to lead poisoning, treatment of these conditions is not discussed here.
<b>Clinical Management</b>	Table 5 provides treatment guidance for children according to BLL based on CDC recommendations. (CDC 2005) <a href="http://www.cdc.gov/lead/guidelines.htm">http://www.cdc.gov/lead/guidelines.htm</a> Most of the treatment actions listed in the table are described in the bullets below: <ul style="list-style-type: none"><li>• It is important to determine the sources of lead exposure.</li><li>• Health departments often provide environmental investigations for children with elevated blood lead levels. These may include looking for lead hazards in their homes and other places where they spend time. If this is not offered, refer patients to private risk assessors or information on home lead hazards available from health departments or agencies such as the U.S. Department of Housing and Urban Development. Practices vary from state to state. (NCHH 2001)</li><li>• Health departments can also help patients by looking for other sources of lead exposure, such as ceramic with leaded glazes, home remedies or imported foods containing lead, and family members’ occupations or hobbies.</li></ul> <p>Lead education and referrals</p> <ul style="list-style-type: none"><li>• Patients with elevated BLLs and their families should receive education about the potential health effects of lead exposure, important environmental and behavioral interventions to reduce potential for lead exposure, and the importance of good nutrition in reducing the absorption and effects of lead.</li><li>• Health departments can often furnish educational materials to health-care providers, and many times have an established program for education and coordination of care (case management). In some cases, physicians may want to refer patients to appropriate social services providers (<i>e.g.</i>, for learning assistance if the child is falling behind in school) and even, in more extreme cases, to physicians with experience in treating lead poisoning.</li></ul> <p>Appropriate clinical referrals can and should also be made for lead's health outcomes based on</p> <ul style="list-style-type: none"><li>• a positive clinical exam.</li><li>• and/or positive tests (such as 2<sup>nd</sup> tier neurobehavioral tests, which</li></ul>

---

---

may also require a referral for diagnosis) if specialty consultation is needed

Diagnostic testing refers to collecting and analyzing a venous blood sample to confirm a capillary blood-screening test, before acting on the result.

- A follow-up test is a venous BLL to monitor the status of a child with an elevated diagnostic BLL, to assure that the elevated BLL is not continuing or rising.
- It may be helpful to compare the patient's blood lead level over time to analyses of expected rate of declines to help confirm removal from the lead source (Roberts *et al.* 2002).

#### Clinical evaluation and management

- Clinical management means that the care should be provided by a health care provider and includes the
    - evaluation
    - family lead education and referrals
    - chelation therapy as appropriate (see below)
    - follow-up testing at appropriate intervals
  - The evaluation should include
    - a medical history (focusing on developmental progress in the case of children)
    - environmental history
    - nutritional history
    - evaluation of child's iron status
    - physical examination, to include complications of lead poisoning
  - Aggressive environmental intervention.
    - Aggressive environmental intervention refers to investigating potential lead exposure pathways and taking immediate steps to control the actual lead hazards identified.
    - If exposure is severe enough, immediate separation from the source is indicated (such as relocation from housing with lead-based paint).
    - For less severe exposure, for example, if lead paint is a major exposure pathway, immediate interim steps such as damp mopping and covering old paint can be taken before long-term measures (*e.g.*, moving or taking all the lead out of the house) are implemented.
    - Environmental intervention should be coordinated through the state or local health department, which is likely to have the best resources and expertise for coordination or support. It is especially important to connect patients and their families with health departments or housing agencies, which can provide guidance on how to find and fix lead hazards safely. Unsafe repairs can easily make lead hazards worse. These agencies may have resources for funding lead hazard reduction. (see: *What Instructions Should Be Given to Patients? section.*)
-

- Chelation therapy.
  - Chelating agents are drugs that bind with heavy metals in the bloodstream, causing them to be more rapidly discharged from the body in urine and bile.
  - Chelation therapy can be effective at reducing the total lead body burden (and acute toxicity effects) in individuals with high current BLLs, but it is generally not indicated for individuals with  $BLLs < 45 \mu\text{g/dL}$ .
  - Chelation therapy is not recommended for those persons with high past exposures to lead and low BLLs who wish to remove lead from their bodies, due to the risk of potential harmful effects of the chelating agents and the remobilized lead.
  - Instead, a calcium-rich diet or supplements might be recommended, to prevent calcium deficiency and subsequent release of lead from the bones.
  - Chelation therapy should always be accompanied by aggressive environmental intervention, and the patient should not be returned to the same environmental exposure situation without a correction (*e.g.*, interdiction, remediation) having taken place.
  - The four chelating agents commonly used in treating patients with high BLLs or signs of encephalopathy are shown in the table below.
  - $\text{Na}_2\text{EDTA}$  (disodium ethylenediaminetetraacetic acid or edetate disodium) should not be used to chelate children because it can cause fatal hypocalcemia (Brown *et al.* 2006)

**Potential Medical Error:**

There are several commercial drugs with the active ingredient EDTA. Only  $\text{CaNa}_2\text{EDTA}$  (Calcium disodium versenate) is appropriate for chelation.  **$\text{Na}_2\text{EDTA}$  (disodium ethylenediaminetetraacetic acid) is not.**

Please write your script carefully and legibly, should you be choosing this particular chelating agent (Please see Table 6 for generic and chemical names).

*Because there are potential side effects associated with each drug, and because treatment protocol differ for each, it is vital that physicians with experience in chelation therapy be consulted before any chelation therapy is begun (AAP 1995).*

An accredited regional poison control center, a university medical center, or a state or local health department can help identify an experienced physician. Note also that the  $\text{CaNa}_2\text{EDTA}$  (edetate disodium calcium) mobilization (challenge) test is no longer recommended because of its difficulty, expense, and potential for increasing lead toxicity (AAP 1995).

Table 5: Guidance for Treatment Actions According to BLL.

BLL (µg/dL)	Treatment Actions
10-19	<ul style="list-style-type: none"> <li>• Provide lead education and referrals</li> <li>• Provide diagnostic testing within 3 months and follow-up testing within two to three months</li> <li>• Proceed according to guidelines in 20-44 range if BLLs persist in 15-19 range</li> <li>• (The presence of a large proportion of children in the 10-14 µg/dL range should trigger community-wide lead poisoning prevention.)</li> </ul>
20-44	<ul style="list-style-type: none"> <li>• Provide lead education and referrals</li> <li>• <i>Provide coordination of care (case management)</i></li> <li>• <i>Perform clinical evaluation and management</i></li> <li>• Provide diagnostic testing (<i>from within one month to within one week</i>) and follow-up testing (<i>every one to two months</i>)</li> <li>• <i>Perform aggressive environmental intervention</i></li> </ul>
45-69	<ul style="list-style-type: none"> <li>• Provide lead education and referrals</li> <li>• Provide coordination of care (case management) <i>within 48 hrs</i></li> <li>• Perform clinical evaluation and management <i>within 48 hrs</i></li> <li>• Provide diagnostic testing <i>within 24-48 hours and follow-up testing (in accordance with chelation therapy, at least once a month)</i></li> <li>• Perform aggressive environmental intervention</li> <li>• <i>Provide appropriate chelation therapy</i></li> </ul>
≥70 (or in case of encephalopathy)	<ul style="list-style-type: none"> <li>• <i>This is a medical emergency.</i></li> <li>• <i>Perform diagnostic testing immediately as an emergency lab test</i></li> <li>• <i>Hospitalize and begin immediate chelation therapy</i></li> <li>• Begin other activities as above</li> </ul>

Table 6. Common Chelating Agents Used in Treating Children With High BLLs

Product Name	Generic Name	Chemical Name	Abbreviation
Calcium disodium versenate	Edetate disodium calcium	Calcium disodium ethylenediaminetetracetate	CaNa <sub>2</sub> EDTA
BAL in oil(British antilewisite)	Dimercaprol	2,3-dimercapto-propanol	BAL
Cuprimine	D-penicillamine	3-mercapto-D-valine	D-penicillamine
Chemet	Succimer	Meso-2,3-dimercaptosuccinic acid	DMSA

---

**Key Points**

- There is a continuum of options—including education, aggressive environmental intervention, and, for more extreme cases, chelation therapy—available to treat patients with elevated BLLs ( $\geq 10$   $\mu\text{g}/\text{dL}$ ). Selection of treatment options depends largely on a patient's BLL and physical exam.
- For the majority of lead-exposed patients, some combination of lead education, aggressive environmental intervention, clinical management, and continued monitoring is indicated. Chelation therapy is only indicated in patients with extremely high or high and persistent BLLs.
- All elevated BLL tests should be reported to the local or state health department as required in the particular state and the HCP should coordinate with the health department in case management as well.

---

**Progress Check**

12. All but which of the following steps should *not* be taken when a child has a venous blood lead level of 10-19  $\mu\text{g}/\text{dL}$ ?
- A. Report the level to the health department.
  - B. Advise the family to find and address possible sources of the child's lead exposure.
  - C. Consult with an experienced clinic or hospital about possible chelation.
  - D. Arrange for follow-up testing within three months.

*To review relevant content, see "[Clinical Management](#)" in this section.*

---

## What Instructions Should Be Given to Patients?

---

<b>Learning Objectives</b>	<p>Upon completion of this section, you will be able to</p> <ul style="list-style-type: none"><li>• identify steps patients with domestic exposures can take to reduce lead exposure</li><li>• identify steps patients with occupational exposures should take to reduce lead exposure.</li></ul>
<b>Introduction</b>	<p>The main instruction to the patient should be to eliminate the source of lead exposure. These recommendations will depend on the type of sources and setting (i.e., home or workplace).</p>
<b>Domestic Environmental Exposures to Lead</b>	<p>People may be exposed to lead through a variety of sources including deteriorated paint, contaminated soil, water, or other products. For children (and adults) with domestic exposures, there may be multiple sources. Therefore, it is important to encourage patients to address all potential sources of lead and to continue blood lead monitoring to help confirm that the source(s) has been effectively eliminated. In all cases, patients should be advised to</p> <ul style="list-style-type: none"><li>• eliminate source(s) of lead exposure</li><li>• Flushing the standing water from the lines and faucet for a few minutes before use and using cold water for drinking may reduce exposure.</li><li>• maintain a diet high in calcium and iron</li><li>• continue to monitor blood lead levels.</li></ul> <p>It can be difficult for low-income patients to permanently address all lead hazards in their homes due to costs and/or landlord relationships (if they are renters). There are federal disclosure laws for potential renters/buyers that require landlords and sellers to disclose any known lead hazard. However, in many communities grants are available to help with lead hazard control. Additionally, there are many low-cost ways to temporarily reduce lead hazards in homes. The patient instruction sheets provide several helpful references.</p> <p>Covering bare soil contaminated with lead in the yard with grass or other type of covering can reduce exposures. Guidelines are available from governmental and nongovernmental organizations to help health care providers instruct their patients in reducing home lead hazards (CEHN 1999). It is also important that residents wet-clean regularly, as children's blood lead levels have been found to be directly correlated with levels of lead in dust in their homes (Lanphear <i>et al.</i> 1998).</p> <p>Patient-friendly, lead resources on the web.</p> <ul style="list-style-type: none"><li>• ATSDR ToxFAQs and ToxFAQs-Chemical Agent Briefing Sheet (ToxFAQs-CABS) answer the most frequently asked health questions about lead (<a href="http://www.atsdr.cdc.gov/tfacts13.html">http://www.atsdr.cdc.gov/tfacts13.html</a> and <a href="http://www.atsdr.cdc.gov/cabs/lead/lead_cabs.pdf">http://www.atsdr.cdc.gov/cabs/lead/lead_cabs.pdf</a>)</li><li>• EPA provides information about lead in paint, dust and soil and how to protect children from lead poisoning at <a href="http://www.epa.gov/lead">www.epa.gov/lead</a></li></ul>

---

- 
- EPA's "Lead in Your Home; A Parent's Reference Guide" [www.epa.gov/lead/pubs/leadrev.pdf](http://www.epa.gov/lead/pubs/leadrev.pdf)
  - The National Lead Information Center (NLIC), funded by EPA, the Centers for Disease Control and Prevention (CDC) and the Department of Housing and Urban Development (HUD), provides the general public and professionals with information about lead hazards and their prevention [www.epa.gov/lead/pubs/nlic.htm](http://www.epa.gov/lead/pubs/nlic.htm).
  - Health Departments and other agencies can provide copies of other materials that will be useful to patients with environmental lead exposures.

---

**Occupational Exposures to Lead**

OSHA requires that the patient speaks to employer about removal from exposure that can help avoid further exposure. These include

- administrative controls
- personal protective equipment
- engineering controls

---

**Clinical Follow-up**

Patients should be reminded of the importance of scheduling follow-up medical surveillance (blood lead testing)

Patients also need to understand when and why they should call their physician for further medical attention. In particular, they should know to have their blood lead monitored on an ongoing basis to confirm removal from the source of exposure.

The ATSDR's patient education sheet on lead toxicity, included in this case study, provides a more detailed checklist that can be used to indicate which types of follow-up are relevant for a given patient.

---

**Key Points**

- Patients exposed to lead in their homes should take steps to reduce environment sources of lead.
- Patients exposed to lead at work should talk to their employers about removal from the source of lead, OSHA regulations for workplace safety, and medical surveillance.
- Patients should also avoid other potential sources of lead.
- A patient education sheet and prescribed follow up check list on lead toxicity is available at .

---

**Progress Check**

13. Patients who have been exposed to lead in their pre-1978 home should

- A. make sure all paint is in good condition and wet-clean regularly
- B. follow lead safe work practices
- C. cover bare soil in the yard
- D. A & C.

*To review relevant content, see "Domestic Environmental Exposures to Lead" in this section.*

---

### Where Can I Find More Information?

---

**For More  
Information**

Please refer to the following Web resources and toll-free telephone numbers for more information on the adverse effects of lead, the treatment of lead-associated diseases, and management of persons exposed to lead. You may also contact ATSDR (see URLs provided below), your state and local health departments, and university medical centers.

CDC Childhood Lead Poisoning Prevention Program

<http://www.cdc.gov/nceh/lead/>

U.S. Environmental Protection Agency, Lead Awareness Program

<http://www.epa.gov/lead/>

ATSDR Information Center Contact Information

<http://www.atsdr.cdc.gov/contacts.html>

Association of Occupational and Environmental Clinics

<http://www.aoec.org>

Agency for Toxic Substances and Disease Registry (ATSDR)

<http://www.atsdr.cdc.gov>

Pediatric Environmental Health Specialty Units (PEHSUs)

[www.aoec.org/PEHSU.htm](http://www.aoec.org/PEHSU.htm)

American College of Occupational and Environmental Medicine

<http://www.acoem.org>

American College of Medical Toxicologists

<http://www.acmt.net>

American College of Preventive Medicine

<http://www.acpm.org>

Centers for Disease Control and Prevention

<http://www.cdc.gov/nceh/lead>

---



---

Available information on toll-free telephone numbers

Centers for Disease Control and Prevention (CDC) Info at 1(888) 422-8737.

[The National Lead Information Center \(NLIC\)](#) at **1-800-424-LEAD (5323)**.

Information is available in Spanish with the use of a translator.

National Lead Information Center Clearinghouse

Phone: 800-424-LEAD (1-800-424-5323)

**Other CSEMs**

Case Studies in Environmental Medicine: Lead Toxicity is one monograph in a series. To view the Taking an Exposure History CSEM and other publications in this series, please go to: <http://www.atsdr.cdc.gov/csem/>

---

Posttest Instructions

<b>Introduction</b>	<p>ATSDR seeks feedback on this course so we can assess its usefulness and effectiveness. We ask you to complete the assessment questionnaire online for this purpose.</p> <p>In addition, if you complete the assessment and posttest online, you can receive continuing education credits as follows.</p>
---------------------	---

Accrediting Organization	Credits Offered
<a href="#">Accreditation Council for Continuing Medical Education (ACCME)</a>	The Centers for Disease Control and Prevention (CDC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. CDC designates this educational activity for a maximum of <b>2.0 AMA PRA Category 1 Credit(s)</b> <sup>™</sup> . Physicians should only claim credit commensurate with the extent of their participation in the activity.
<a href="#">American Nurses Credentialing Center (ANCC), Commission on Accreditation</a>	This activity for <b>2.0</b> contact hours is provided by the Centers for Disease Control and Prevention, which is accredited as a provider of continuing education in nursing by the American Nurses Credentialing Center's Commission on Accreditation.
<a href="#">National Commission for Health Education Credentialing, Inc. (NCHEC)</a>	CDC is a designated provider of continuing education contact hours (CECH) in health education by the National Commission for Health Education Credentialing, Inc. The Centers for Disease Control and Prevention is a designated provider of continuing education contact hours (CECH) in health education by the National Commission for Health Education Credentialing, Inc. This program is a designated event for the Certified Health Education Specialist (CHES) to receive <b>2.0</b> Category I contact hours in health education, CDC provider number GA0082.
<a href="#">International Association for Continuing Education and Training (IACET)</a>	The Centers for Disease Control and Prevention (CDC) has been reviewed and approved as an Authorized Provider by the International Association for Continuing Education and Training (IACET), Suite 800, McLean, VA 22102. CDC will award <b>0.15</b> of CEU's to participants who successfully complete this program.

<b>Disclaimer</b>	<p>In compliance with continuing education requirements, all presenters must disclose any financial or other relationships with the manufacturers of commercial products, suppliers of commercial services, or commercial supporters as well as any use of unlabeled product(s) or product(s) under investigational use.</p> <p>CDC/ATSDR, our planners, and the presenters for this seminar do not have financial or other relationships with the manufacturers of commercial products, suppliers of commercial services or commercial supporters. This presentation does not involve the unlabeled use of a product or product under investigational use.</p>
-------------------	---

<b>Instructions</b>	<p>To complete the assessment and posttest, go to <a href="http://www2.cdc.gov/atsdrce/">www2.cdc.gov/atsdrce/</a> and follow the instructions on that page.</p> <p>You can immediately print your continuing education certificate from your personal transcript online. No fees are charged.</p>
---------------------	--

---

**Posttest**

*Please select the best correct answer.*

1. Lead is a
    - A. Soft, heavy, blue-gray metal.
    - B. Naturally occurring substance.
    - C. Commercially used substance.
    - D. All of the above.
  
  2. Which statement is true of organic lead?
    - A. It is more commonly found in home environments today than is inorganic lead.
    - B. It was the most available source of exposure through natural processes.
    - C. It was a common source of lead exposure in the U.S. when leaded gasoline was used.
    - D. It cannot enter the body through dermal exposure.
  
  3. Of the following, the U.S. population most at risk from exposure to lead today is
    - A. People who work in lead mining and smelting.
    - B. Household contacts of workers engaged in the manufacture of lead-containing products.
    - C. Children living in pre-1978 buildings with deteriorated paint.
    - D. Construction workers.
  
  4. In older urban areas, most of the lead in the environment today comes from
    - A. Contaminated drinking water.
    - B. Lead-contaminated dust, soil, and deteriorated lead-based paint.
    - C. Imported food, home remedies, and cosmetics.
    - D. Commercial products containing lead.
  
  5. Which of the following is *not* considered a potential source of lead exposure?
    - A. Jewelry.
    - B. Treated lumber.
    - C. Imported cosmetics and home remedies.
    - D. Glazed ceramics.
  
  6. What is the Center for Disease Control's blood lead action level for children?
    - A. 5 µg/dL.
    - B. 10 µg/dL.
    - C. 25 µg/dL.
    - D. 40 µg/dL.
-

- 
7. The most important route of exposure to lead by children is
    - A. Ingestion.
    - B. Inhalation.
    - C. Dermal contact.
    - D. All are equally important.
  
  8. Which of the following signs or symptoms is *not* consistent with childhood lead poisoning?
    - A. Recurrent headaches.
    - B. Attention Deficit Hyperactivity Disorder.
    - C. Decreased hearing and speech abilities.
    - D. Difficulty learning.
  
  9. In caring for an adult patient with a blood lead level of 40  $\mu\text{g}/\text{dL}$ , it is most important to
    - A. Continue to monitor with monthly capillary blood tests.
    - B. Take steps to avoid further exposure to lead.
    - C. Immediately start chelation therapy.
    - D. Encourage a diet high in calcium.
  
  10. As part of the exposure history, you should explore
    - A. Possible lead exposure at work or during hobbies.
    - B. Hobbies that might involve lead.
    - C. Use of imported home remedies and cosmetics.
    - D. All of the above.
  
  11. OSHA requires written notification and a medical examination for workers with blood lead levels of
    - A. 10  $\mu\text{g}/\text{dL}$ .
    - B. 25  $\mu\text{g}/\text{dL}$ .
    - C. 40  $\mu\text{g}/\text{dL}$ .
    - D. 70  $\mu\text{g}/\text{dL}$ .
  
  12. At a 24-month well-child check up, under what circumstances should you order a venous blood lead level?
    - A. If the 12-month blood test showed a prior elevation over 10  $\mu\text{g}/\text{dL}$  or no prior blood lead level is available.
    - B. If child is living or spending significant time in pre-1978 housing.
    - C. If a household member works in a job involving lead.
    - D. All of the above.
-

- 
13. Why would a patient's BLL drop only gradually, even with complete removal from the source of exposure?
- A. Lead's half-life in the blood is almost one year.
  - B. Everyone is exposed to high background levels of lead.
  - C. Lead stored in the bones and soft tissues may be released over time.
  - D. None of the above.
14. Chronic lead exposure is not believed to contribute to which of the following conditions
- A. Hypertension.
  - B. Kidney disease.
  - C. Diabetes.
  - D. Low sperm count.
15. You should tell patients who are concerned about lead in their drinking water that
- A. As long as they do not have well water, their water is safe.
  - B. Until they can get their water tested, boil their drinking water.
  - C. Drinking water is non-acidic and will not leach lead out of old pipes, fixtures, or solder.
  - D. Until they can get their water tested, run cold water for one to two minutes before use.
16. Which of the following statements about sources of lead in the environment is true?
- A. Lead dust can raise children's blood lead levels above the level of concern.
  - B. Lead is heavy, so it does not travel far in the air from smelters or industries.
  - C. Lead is only a problem in urban areas with pre-1978 housing.
  - D. Children who eat paint chips make up the majority of those with blood lead levels above 10 µg/dL.
-

**Relevant Content** To review content relevant to the posttest questions, see:

Question	Location of Relevant Content
1.	What is lead?
2.	What is lead?
3.	Who is at risk of lead exposure?
4.	Where is lead found?
5.	How are people exposed to lead?
6.	What are U.S. standards for lead levels?
7.	How are patients exposed to lead?
8.	How should patients exposed to lead be evaluated?
9.	What are U.S. standards for lead levels?
10.	How should patients exposed to lead be evaluated?
11.	What are U.S. standards for lead levels?
12.	What is the biologic fate of lead?
13.	What are the physiologic effects of lead?
14.	What instructions should be given to patients?
15.	Where is lead found?
16.	Where is lead found?

Literature Cited

1. Agency for Toxic Substances and Disease Registry. 1999. Toxicological profile for lead. Atlanta: US Department of Health and Human Services, Public Health Service.
2. Agency for Toxic Substances and Disease Registry. 2005. Toxicological profile for lead. Atlanta: US Department of Health and Human Services, Public Health Service.
3. Alexander H, Checkoway H, van Netten C, *et al.* 1996. Semen quality of men employed at a lead smelter. *Occup Environ Med* 53:411-416.
4. Alexander FW, Clayton BE, Delves HT. 1974. Mineral and trace-metal balances in children receiving normal and synthetic diets. *QJ Med* 43:89-111.
5. American Academy of Pediatrics. 1993. Lead poisoning: from screening to primary prevention. *Pediatrics* 92(1): 176-183.
6. American Academy of Pediatrics. 1995. Treatment guidelines for lead exposure in children. *Pediatrics* 96(1): 155-1601.
7. American Conference of Governmental Industrial Hygienists (ACGIH) 2005. TLVs and BEIs. 2005. Signature publications. Cincinnati, OH.
8. Aufderheide AC, Wittmers LE Jr. 1992. Selected aspects of the spatial distribution of lead in bone. *Neurotoxicol* 13:809-820.
9. Baghurst PA, Robertson EF, McMichael AJ, *et al.* 1987. The Port Pirie cohort study: lead effects on pregnancy outcome and early childhood development. *Neurotoxicology* 8:395-401.
10. Barry PSI. 1981. Concentrations of lead in the tissues of children. *Br J Ind Med* 38:61-71.
11. Batuman V, Maesaka JK, Haddad B, Medicaid *et al.* 1981. The role of lead in gout nephropathy. *New England Journal of Medicine* 304:520-3.
12. Bennett WM. 1985. Lead nephropathy. *Kidney Int* 28:212-20.
13. Borja-Aburto, *et al.* 1999. Blood Lead Levels Measured Prospectively and Risk of Spontaneous Abortion. *Am J Epidemiol* 1999; 150:590-7
14. Bowen WH. 2001. Exposure to metal ions and susceptibility to dental caries. *Journal of Dental Education*. 65(10): 1046-1053.
15. Buchanan LH, Counter SA, Ortega F, Laurell G. 1999. Distortion product oto-acoustic emissions in Andean children and adults with chronic lead intoxication. *Acta Otolaryngol.* 1999; 119(6):652-8.
16. Budd P, Montgomery J, Cox A, Krause P, Barreiro B, Thomas RG. 1998. The distribution of lead within ancient and modern human teeth: implications for long-term and historical exposure monitoring, *Sci Total Environ (Sept)* 18;220(2-3):121-36.
17. Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Juski TA, Lanphear BP. 2003. Intellectual impairment in children with blood lead concentrations below 10 µg per Deciliter. *New England Journal of Medicine.* 348(16): 1517-1526.
18. Centers for Disease Control and Prevention. 1997a. Screening young children for lead poisoning: guidance for state and local public health officials. Atlanta: US Department of Health and Human Services, Public Health Service, CDC Childhood Lead Poisoning Prevention Program. November 1997.
19. Centers for Disease Control and Prevention. 1997b. Update: blood lead levels. *MMWR* 46(7)141-146.
20. Centers for Disease Control and Prevention. 2002. Managing elevated blood lead levels among young children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention. Atlanta: US Department of Health and Human Services.
21. Centers for Disease Control and Prevention. 2003. Surveillance for Elevated Blood Lead Levels Among Children --- United States, 1997—2001. Atlanta: US Department of Health and Human Services. September 12, 2003 / 52(SS10);1-21. Available from URL: <http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5210a1.htm>.

22. Centers for Disease Control and Prevention 2005. Blood lead levels- Unites States 1999-2002. MMWR 54(20)513-516.
23. Chamberlain A, Heard C, Little MJ, *et al.* 1978. Investigations into lead from motor vehicles. Harwell, United Kingdom: United Kingdom Atomic Energy Authority. Report no. AERE-9198. 1979. The dispersion of lead from motor exhausts. Philos Trans R Soc Lond A 290:557-589.
24. Children's Environmental Health Network (CEHN). 1999. Training manual on pediatric environmental health: Putting it into practice. Available from URL: [www.cehn.org/cehn/trainingmanual/manual-front.html](http://www.cehn.org/cehn/trainingmanual/manual-front.html).
25. Cooper WC. 1976. Cancer mortality patterns in the lead industry. Ann NY Acad Sci 271:250-259.
26. DeSilva PE. 1981. Determination of lead in plasma and studies on its relationship to lead in erythrocytes. Br J Ind Med 38:209-217.
27. EPA. 2008. National Air Quality Standards for Lead. EPA 40 CFR Parts 50, 51, 53 and 58. [http://www.epa.gov/air/lead/pdfs/20081015\\_pb\\_naaqs\\_final.pdf](http://www.epa.gov/air/lead/pdfs/20081015_pb_naaqs_final.pdf) in <http://www.epa.gov/air/lead/actions.html> viewed on Oct. 17, 2008.
28. EPA. 1986a. Air quality criteria for lead. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment. Environmental Criteria and Assessment Office. EPA 600/8-83-028F.
29. EPA. 1986b. Determination of reportable quantities for hazardous substances. U.S. Environmental Protection Agency. Code of Federal Regulations. 40 CFR 117.
30. Ernhart CB, Wolf AW, Kennard MJ, *et al.* 1985. Intrauterine lead exposure and the status of the neonate. In: Lekkas TD, ed. International Conference on Heavy Metals in the Environment, Athens, Greece. September, Vol. 1. Edinburgh, United Kingdom: CEP Consultants, Ltd. 35-37.
31. Everson J, Patterson CC. 1980. "Ultra-clean" isotope dilution/mass spectrometric analyses for lead in human blood plasma indicate that most reported values are artificially high. Clin Chem 26:1603-1607.
32. FDA. 1994. Action Levels for Poisonous or Deleterious Substances in Human Food and Animal Feed. Department of Health and Human Services. Public Health Service. Food and Drug Administration.
33. FDA. 1995. Substances prohibited from use in human food. Substances prohibited from indirect addition to human food through food-contact surfaces. U.S. Food and Drug Administration. Code of Federal Regulations. 21 CFR 189.240.
34. Flegal AR and Smith DR. 1995. Measurements of environmental lead contamination and human exposure. Rev Environ Contam Toxicol 143:1-45.
35. Fulton M, Raab G, Thomson G, Laxen D, Hunter R, Hepburn W. 1987. Influence of blood lead on the ability and attainment of children in Edinburgh. Lancet 1: 1221-1226.
36. Gennart J-P, Buchet J-P, Roels H, *et al.* 1992. Fertility of male workers exposed to cadmium, lead or manganese. Am J Epidemiol 135: 1208-1219.
37. Goyer RA. 1985. Renal changes associated with lead exposure. In: Mahaffey KR, rd. Dietary and environmental lead: Human health effects. Amsterdam, The Netherlands: Elsevier Science Publishers B.V.
38. Griffin TB, Couiston F, Wills H. 1975. Biological and clinical effects of continuous exposure to airborne particulate lead. Arh Hig Toksikol 26:191-208. (Yugoslavian)
39. Hawk BA, Schroeder SR, Robinson G, *et al.* 1986. Relation of lead and social factors to IQ of low SES children: a partial replication: Am J Ment Defic 91:178-183.
40. Hu H. 1991. Knowledge of diagnosis and reproductive history among survivors of childhood plumbism. Am J Public Health 81:1070-1072.
41. Hu H, Aro A, Payton M, *et al.* 1996. The relationship of bone and blood lead to hypertension. The normative aging study. JAMA 275:1171-6.



42. IARC. 1987. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans: Overall evaluations of carcinogenicity. Suppl 7: An updating of the IARC monographs volumes 1 to 42. Lyon, France: World Health Organization, International Agency for Research for Research on Cancer, 230-232.
43. James HM, Milburn ME, Blair JA. 1985. Effects of meals and meal times on uptake of lead from the gastrointestinal tract of humans. *Human Toxicol* 4: 401-407.
44. Kaul B, Sandhu RS, Depratt C, and Reyes F. 1999. Follow-Up Screening of Lead-Poisoned Children Near an Auto Battery Recycling Plant, Haina, Dominican Republic. *Environ Health Perspect* 107:917-920.
45. Kehoe RA. 1961. The metabolism of lead in man in health and disease: Present hygienic problems relating to the absorption of lead: The Harben lectures, 1960. *J R Inst Public Health Hyg* 24: 177-203.
46. Kim R, Rotnitzky A, Sparrow D, *et al.* 1996. A longitudinal study of low-level lead exposure and impairment of renal function. The normative aging study. *JAMA* 275: 1177-81.
47. Koo WWR, Succop PA, Bornschcin RL, *et al.* 1991. Serum vitamin D metabolites and bone mineralization in young children with chronic low to moderate lead exposure. *Pediatrics* 87: 680-687.
48. Korrick SA, Hunter DJ, Rotnitzky A, *et al.* 1999. Lead and hypertension in a sample of middle-aged women. *Am J Public Health.* 89(3): 330-5.
49. Landrigan PJ, Schechter CB, Lipton JM, Fahs MC, and Schwartz J. 2002. Environmental pollutants and disease in American children: Estimates of morbidity, mortality, and costs for lead poisoning, asthma, cancer, and developmental disabilities. *Environmental Health Perspectives.* 110(7): 721-728.
50. Landsdown R, Yule W, Urbanowicz MA, Hunter J. 1986. The relationship between blood-lead concentrations, intelligence, attainment and behavior in a school population: the second London study. *Int Arch Occup Environ Health* 57: 225-235.
51. Lanphear BP, Matte TD, Rogers J, Clickner RP, Dietz B, Bornschein RL, Succop P,
52. Mahaffey KR, Dixon S, Galke W, Rabinowitz R, Farfel M, Rohde C, Schwartz J, Ashley P, and Jacobs DE. 1998. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels: A pooled analysis of 12 epidemiological studies. *Environmental research* 79: 51-68.
53. Lanphear BP, Dietrich K, Auinger P, Cox. C 2000. Cognitive deficits associated with BLLs <10 µg/dL in US children and adolescents. *Public Health Rep.* 115: 521-529.
54. Lanphear BP, Hornung R, Ho M, Howard CR, Eberley S, and Knauf K. 2002. Environmental lead exposure during early childhood. *Journal of Pediatrics* 140: 40-47.
55. Lerda D. 1992. Study of sperm characteristics in persons occupationally exposed to lead. *Am J Ind Med* 22: 567-571.
56. Lin S, Hwang S, Marshall EG, *et al.* 1996. Fertility rates among lead workers and professional bus drivers: A comparative study. *Ann Epidemiol* 6: 201-208.
57. Litvak PF, Wasserman G, Kline JK, Jgraziano. 1999. The Yugoslavia Prospective Study of Environmental Lead Exposure. *Environ Health Perspect* 107: 9-15.
58. López-Carrillo L, Torres-Sánchez L, Garrido F, Papaqui-Hernández J, Palazuelos-Rendón E, López-Cervantes M. 1996. Prevalence and determinants of lead intoxication in Mexican children of low socioeconomic status. *Environ Health Perspect* 104: 1208-1211.
59. Mahaffey KR. 1990. Environmental lead toxicity: nutrition as a component of intervention. *Environ Health Perspect* 89: 75-78.
60. McMichael AJ, Vimpani GV, Robertson EF, *et al.* 1986. The Port Pirie cohort study: Maternal blood lead and pregnancy outcome. *J Epidemiol Community* 40: 18-25.
61. Murphy MJ, Graziano JH, Popovac D, *et al.* 1990. Past pregnancy outcomes among women living in the vicinity of a lead smelter in Kosovo, Yugoslavia. *Am J Public Health* 80: 33-5.

62. Mushak P, Davis JM, Crocetti AF, Grant LD. 1989. Prenatal and postnatal effects of low-level lead exposure: integrated summary of a report to the US Congress on childhood lead poisoning. *Environ Res* 50:11-36
63. National Center for Healthy Housing (NCHH). 2001. Another link in the chain: State policies and practices for case management and environmental investigation for lead poisoned children: Update. Columbia, MD.
64. National Toxicology Program (NTP) 2004. Lead (CAS No. 7439-92-1) and lead compounds. Report on carcinogens, eleventh edition.
65. Needleman HL, Rabinowitz M, Leviton A, *et al.* 1984. The relationship between prenatal exposure to lead and congenital anomalies. *JAMA* 251:2956-2959.
66. Needleman H.L. 2002. Bone lead levels in adjudicated delinquents: A case control study. *Neurotoxicology and Teratology* 24: 711-717.
67. Nevin R. 2000. How lead exposure relates to temporal changes in IQ, violent crime, and unwed pregnancy.
68. Nordstrom S, Beckman L, Nordenson I. 1979. Occupational and environmental risks in and around a smelter in northern Sweden. V. Spontaneous abortion among female employees and decreased birth weight in their offspring. *Hereditas* 90:291-6.
69. Payton M, Hu H, Sparrow D, *et al.* 1994. Low-level lead exposure and renal function in the normative aging study. *Am J Epidemiol* 140(9):821-9.
70. Puzas JE, Sickel MJ, Felter ME. 1992. Osteoblasts and chondrocytes are important target cells for the toxic effects of lead. *Neurotoxicology* 13:800-806.
71. Rabin R. Warnings unheeded: a history of child lead poisoning. 1989. *Am J Publ Health*. 79:1668-1674.
72. Rabinowitz MB, Wetherill GW, Kopple JD. 1976. Kinetic analysis of lead metabolism in healthy humans. *J Clin Invest* 58:260-270.
73. Rothenberg SJ, Karchmer S, Schnaas L, Perroni E, Zea F, and Alba JF. 1994. Changes in serial blood lead levels during pregnancy. *Environ Health Perspect* 102: 876-880.
74. Saper RB, Kales SN, Paquin J, Burns MJ, Eisenberg DM, Davis RB, Phillips RS. 2004. Heavy metal content of Ayurvedic herbal medicine products. *JAMA*. 292(23): 2868-2873.
75. Sayre JW, Charney E, Vostal J, Pless BI. 1974. House and hand dust as potential source of childhood lead exposure. *Am J Dis Child*. 127:167-170.
76. Schwartz J. 1995. Lead, blood pressure, and cardiovascular disease in men. *Arch Environ Health* 50:31-37.
77. Schroeder SR, Hawk B, Otto DA, Mushak P, Hicks RE. 1985. Separating the effects of lead and social factors on IQ. *Environ Res* 38:144-154.
78. Staessen JA, Lauwerys RR, Buchet JP, *et al.* 1992. Impairment of renal function with increasing blood lead concentrations in the general population. *N Engl J M* 327(3): 151-6.
79. Telisman S, Cvitkovic P, Jurasovic J, Pizent A, Gavella M, Rocic B. 2000. Semen quality and reproductive endocrine function in relation to biomarkers of lead, cadmium, zinc, and copper in men. *Environ Health Perspect* 108:45-53.
80. Victory W, Throler HA, Volpe R, *et al.* 1988. Summary of discussion sessions: Symposium on lead blood pressure relationships. *Environ Health Perspect* 78:139-155.
81. Wasserman GA, Liu X, Lolocono NJ, Factor-Litvak P, Kline JK, Popovac D, Morina N, Musabegovic A, Vrenezi N, Capuni-Paracka S, Lekic V, Preteni-Redjepi E, Hadzialjevic S, Slavkovich V, Graziano JH. 1997. Lead exposure and intelligence in 7-year-old children: the Yugoslavia prospective study. *Environ Health Perspect* 105:956-962.
82. Watson GE, Davis BA, Raubertas RF, Pearson SK, Bowen WH. 1997. Influence of maternal lead ingestion on caries in rat pups, *Nat Med* 3(9): 1024-1025.
83. Weeden RP, D'Haese P, Van de Vyver FL, *et al.* 1986. Lead nephropathy. *Am J Kidney Dis* 3(5): 380-3.

84. Winneke G, Brockhaus A, Ewers U, Kramer U, Neuf M. 1990. Results from the European multicenter study on lead neurotoxicity in children: implications for risk assessment. *Neurotoxicol Teratol* 12:553-559.
85. Yule W, Lansdown R, Millar IB, Urbanowicz MA. 1981. The relationship between blood lead concentrations, intelligence and attainment in a school population: a pilot study. *Dev Med Child Neurol* 23:567-576
86. Ziegler EE, Edwards BB, Jensen RL, *et al.* 1978. Absorption and retention of lead by infants. *Pediatr Res* 12:29-34.

**Appendix 1: Key to Acronyms/Abbreviations**

<b>CDC</b>	Centers for Disease Control and Prevention
<b>OSHA</b>	Occupational Safety and Health Administration
<b>NIOSH</b>	National Institute for Occupational Safety and Health
<b>ACGIH</b>	American Conference of Governmental Industrial Hygienists
<b>EPA</b>	Environmental Protection Agency
<b>FDA</b>	Food and Drug Administration
<b>CPSC</b>	Consumer Product Safety Commission
<b>PEL</b>	Permissible Exposure Limit
<b>REL</b>	Recommended Exposure Limit
<b>TLV/TWA</b>	Threshold Limit Value/Time-Weighted Average
<b>NAAQS</b>	National Ambient Air Quality Standard
<b>MCLG</b>	Maximum Contaminant Level Goal

Appendix 2. Patient Information Sheet

---

<b>What is lead?</b>	<ul style="list-style-type: none"><li>• Lead is a soft, blue-gray metal that is mined from the earth's crust.</li><li>• Lead has been used for many industrial purposes for centuries.</li><li>• Lead was widely used in paint and gasoline in the U.S. until the 1970's.</li><li>• Lead does not break down over time.</li><li>• Lead is present in all parts of the environment, including inside homes.</li></ul>
<b>How are people exposed to lead?</b>	<ul style="list-style-type: none"><li>• Most people, especially children, who suffer from lead poisoning are exposed through lead-contaminated household dust or soil that gets into their mouths.</li><li>• Homes that were built before 1978 are likely to have paint that contains lead. If this paint is disturbed, rubbed, peels or chips, people living in the house may come in contact with lead.</li><li>• Some people may be exposed to lead through working with or near lead.</li><li>• Other routes of exposure include:<ul style="list-style-type: none"><li>◦ Eating or drinking water, food, or alcohol that contains lead.</li><li>◦ Practicing religious and cultural rituals that include lead.</li><li>◦ Mouthing or swallowing other lead-containing products, including some imported jewelry.</li></ul></li></ul>
<b>What are the health effects of lead?</b>	<ul style="list-style-type: none"><li>• More commonly, lower levels of lead in children over time may lead to reduced IQ, slow learning, Attention Deficit Hyperactivity Disorder (ADHD), or behavioral issues.</li><li>• Lead also affects other parts of the body including the kidneys, heart, and reproductive system,</li><li>• <u>Pregnant women</u> should know that the developing fetus is very sensitive to the effects of lead exposure.</li><li>• The effects of lead may be seen right away or may not be noticed for many years.</li></ul>
<b>How can I prevent exposure to lead?</b>	<ul style="list-style-type: none"><li>• Make sure that your home is lead safe. If your house was built before 1978 and you cannot afford to have all the lead-based paint eliminated from it, please follow these tips:<ul style="list-style-type: none"><li>◦ Make sure that the paint is not chipping or peeling. Pay special attention to the paint around windows, porches, and doors.</li><li>◦ Use lead-safe work practices when doing any remodeling work that causes paint to chip, peel or become dust. (for more information, see: <a href="http://www.epa.gov/lead/pubs/epahudrrmodel.htm">http://www.epa.gov/lead/pubs/epahudrrmodel.htm</a> (Joint EPA/HUD Renovation Training Curriculum; modules and resources.)</li><li>◦ Wet mop floors and window sills <u>at least weekly</u> to control dust.</li><li>◦ Keep children out of areas in the yard with bare soil.</li><li>◦ Wash children's hands and toys with soap and water <u>often</u>.</li></ul></li></ul>

---

---

	<ul style="list-style-type: none"><li>○ Run cold water for <u>one to two minutes</u> before drinking or cooking with it.</li><li>○ Do not use glazed ceramics, home remedies, cosmetics, or leaded-crystal glassware unless you know that they are lead safe.</li><li>○ If you live near an industry, mine, or waste site that may have contaminated the area with lead, be especially careful to <u>avoid exposure to soil</u>.</li></ul>
<b>Is there a medical test for lead exposure?</b>	<ul style="list-style-type: none"><li>● Blood samples can be tested for exposure to lead.</li><li>● Children should have blood tests at ages one and two.</li><li>● Children should also be tested between ages three and six if they are at risk of lead poisoning (see: ).</li></ul>
<b>Who can I call to get more information about lead?</b>	<ul style="list-style-type: none"><li>● <b>The CDC Information Center:</b> 1-888-422-8737)</li><li>● You can also obtain more information about lead from your regional poison control center and your state, county, or local health department.</li></ul>
<b>Where can I go for more in-depth information?</b>	<ul style="list-style-type: none"><li>● Centers for Disease Control and Prevention: <a href="http://www.cdc.gov/lead">www.cdc.gov/lead</a></li><li>● National Lead Information Center: 1-800-424-LEAD or <a href="http://www.epa.gov/lead/pubs/nlic.htm">http://www.epa.gov/lead/pubs/nlic.htm</a></li><li>● National Lead Information Center Clearinghouse Phone: 800-424-LEAD (1-800-424-5323)</li><li>● U.S. Environmental Protection Agency Lead Awareness Program <a href="http://www.epa.gov/lead">http://www.epa.gov/lead</a></li><li>● EPA publication "<a href="http://www.epa.gov/lead/pubs/leadrev.pdf">Lead in Your Home; A Parent's Reference Guide</a>": <a href="http://www.epa.gov/lead/pubs/leadrev.pdf">http://www.epa.gov/lead/pubs/leadrev.pdf</a></li><li>● National Institute for Occupational Safety and Health (NIOSH) 1-800-311-3435 or <a href="http://www.cdc.gov/niosh/">www.cdc.gov/niosh/</a></li><li>● Occupational Safety and Health Administration (OSHA): 1-800-321-6742 or <a href="http://www.osha.gov">www.osha.gov</a>.</li></ul>

---

**Answers to Progress Check Questions**

1. The correct answer is B. Lead does not break down over time.
2. The correct answer is B. Lead-contaminated dust, soil, and deteriorated lead-based paint.
3. The correct answer is A. Ingestion and inhalation.
4. The correct answer is D. electrician.
5. The correct answer is C. an advisory level for environmental and educational intervention.
6. The correct answer is B. 30 days.
7. The correct answer is D. Effects in adults tend to begin at higher exposure levels than in children.
8. The correct answer is D. all of the above.
9. The correct answer is D. all of the above.
10. The correct answer is C. blood lead levels (BLL) test
11. The correct answer is C. venous blood lead level (BLL).
12. The correct answer is C. consult with an experienced clinic or hospital about possible chelation.
13. The correct answer is D. A & C.