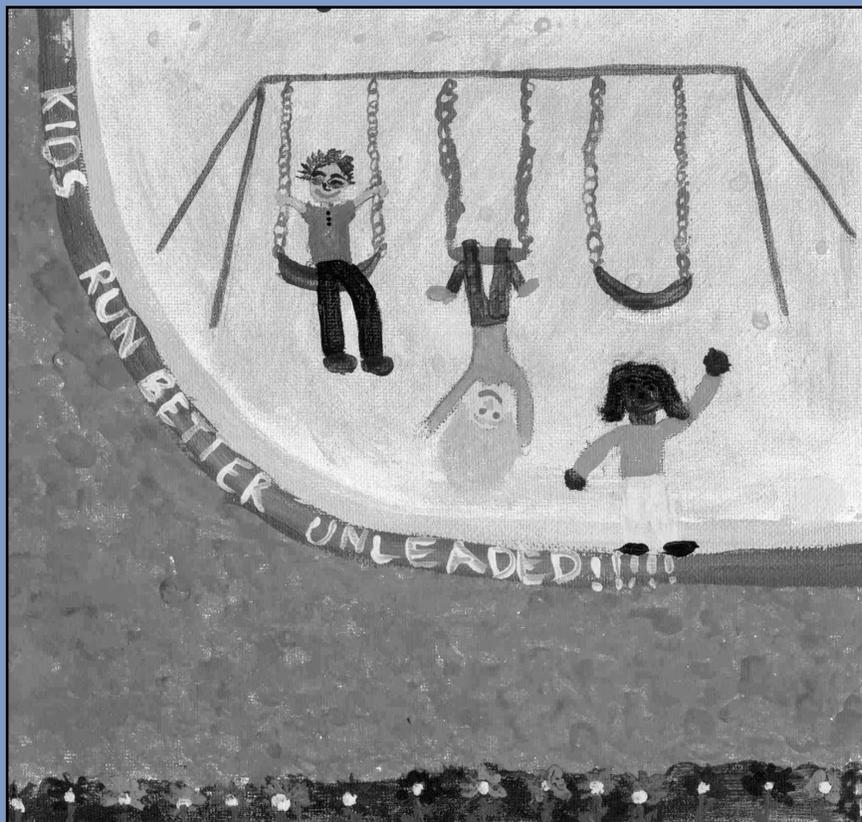


CDC uses a [blood lead reference value](#) of 3.5 micrograms per deciliter ( $\mu\text{g}/\text{dL}$ ) to identify children with higher levels of lead in their blood compared to most children. This level is based on the 97.5th percentile of the blood lead values among U.S. children ages 1-5 years from the 2015-2016 and 2017-2018 National Health and Nutrition Examination Survey (NHANES) cycles. Children with blood lead levels at or above the BLRV represent those at the top 2.5% with the highest blood lead levels.

This document refers to a blood lead level of  $10 \mu\text{g}/\text{dL}$  as the CDC level of concern for adverse health outcomes in children. This terminology has changed, and readers are referred to the [ACCLPP recommendations of 2012](#).

# Managing Elevated Blood Lead Levels Among Young Children:

Recommendations from the  
Advisory Committee on Childhood  
Lead Poisoning Prevention



March 2002



Cover illustration courtesy of the Los Angeles County  
Childhood Lead Poisoning Prevention Program

# **Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention**

Edited by  
Birt Harvey, MD

**Centers for Disease Control  
and Prevention**

**Jeffrey P. Koplan, MD, MPH, Director**

*National Center for Environmental Health*  
**Richard J. Jackson, MD, MPH, Director**

*Division of Environmental Hazards and Health Effects*  
**Michael A. McGeehin, PhD, MSPH, Director**

*Lead Poisoning Prevention Branch*  
**Gary P. Noonan, MPA, Acting Chief**

**U.S. Department of Health  
and Human Services, Public Health Service  
March 2002**

Suggested reference:

Centers for Disease Control and Prevention. Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention. Atlanta: CDC; 2002.



# Table of Contents

List of Tables .....	iii
List of Figures .....	v
Foreword .....	vii
Preface .....	ix
Members of the Advisory Committee on Childhood Lead Poisoning Prevention .....	xi
Authors .....	xv
Acknowledgments .....	xvii
Glossary .....	xix
Chapter 1. Introduction .....	1
Development of the Case Management Recommendations .....	3
Overview of Comprehensive Case Management .....	4
General Considerations .....	9
Chapter 2. Assessment and Remediation of Residential Lead Exposure .....	13
Summary of Recommendations .....	15
Introduction .....	16
Sources and pathways of residential lead exposure .....	16
Effectiveness and Safety of Lead Hazard Control Measures .....	19
Recommendations for Assessment and Remediation .....	22
Enforcement of Laws and Regulations .....	25
Financial Resources for Lead Hazard Control .....	26
Recommendations for Future Research .....	27
Chapter 3. Medical Assessment and Interventions .....	39
Summary of Recommendations .....	41
Introduction .....	42
General Principles of Medical Case Management .....	42
Medical History .....	43
Physical Examination .....	48
Laboratory and Imaging Evaluation .....	48
Chelation Therapy .....	49

Monitoring Blood Lead Levels .....	50
Monitoring the Child .....	52
Recommendations for Future Research .....	53
 Chapter 4. Nutritional Assessment and Interventions .....	 59
Summary of Recommendations .....	61
Introduction .....	62
Nutritional Interventions: Summary of the Evidence .....	62
General Recommendations .....	68
Recommendations for Future Research .....	70
 Chapter 5. Developmental Assessment and Interventions .....	 77
Summary of Recommendations .....	79
Introduction .....	80
Detailed Bases for Recommendations .....	80
General Recommendations .....	85
Recommendations for Future Research .....	88
 Chapter 6. Educational Interventions for Caregivers .....	 97
Summary of Recommendations .....	99
Introduction .....	100
Sources and Pathways of Residential Lead Exposure .....	100
General Principles .....	100
Studies of Various Interventions .....	101
General Recommendations .....	105
Recommendations for Future Research .....	110
 Appendixes .....	 115
Appendix I. Published Reports of Less Common Causes of Elevated Blood Lead Levels (EBLLs) in Children. ....	117
Appendix II. Sources of Information on Lead Abatement .....	128

## List of Tables

Table 1.1. Possible Elements of a Case Management Plan Based on Individualized Child Assessment .....	11
Table 2.1. Summary of Recommendations for Assessment and Remediation of Residential Lead Exposure .....	15
Table 2.2. Time Frames for Environmental Investigation and Other Case Management Activities According to a Child's Blood Lead Level .....	36
Table 2.3. Common Sources of Lead Exposure to Consider in an Environmental Investigation	37
Table 3.1. Summary of Recommendations for Children with Confirmed (Venous) Elevated Blood Lead Levels .....	41
Table 3.2. Guidelines for Questions to Ask Regarding a Child's Environmental History .....	45
Table 3.3. Recommended Schedule for Obtaining a Confirmatory Venous Sample .....	51
Table 3.4. Schedule for Follow-Up Blood Lead Testing .....	51
Table 4.1. Summary of Recommendations for Nutritional Assessment and Interventions .....	61
Table 5.1. Summary of Recommendations for Developmental Assessment and Interventions	79
Table 6.1. Summary of Recommendations for Educational Interventions for Caregivers .....	99



## List of Figures

Figure 2.1. Pathways of Lead Exposure in the Residential Environment . . . . .	33
Figure 2.2. Relationship of Housing Age and Condition to Dust Lead Levels . . . . .	34
Figure 2.3. Relationship of Dust Lead Levels to Blood Lead Levels in Children . . . . .	35
Figure 3.1. Lowest Reported Effect Levels of Inorganic Lead in Children . . . . .	58
Figure 4.1. Median Calcium Intake by Race from NHANES III . . . . .	76



## Foreword

The overall reduction in childhood lead levels over the last three decades has been one of the great environmental health success stories in this country. However, our goal has not yet been reached. There are still far too many lower-income children living in older housing who are being hurt by elevated blood lead levels. The public health, housing, and environmental communities must continue to work together to eliminate the threat of lead poisoning for our future generations.

An important factor in the battle against lead poisoning is the proper management of children who have been identified as having elevated blood lead levels. In this publication, the Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) and other public health practitioners have developed guidelines for assessment and interventions in the areas of medicine, nutrition, environmental exposure, childhood development, and education. Implementation of these “Best Practices” will greatly assist case managers, medical care providers, and others in delivering the most effective services to the lead poisoned child and the child’s caregiver.

I congratulate the ACCLPP and all the authors of these guidelines and thank them for their efforts. This report is a critical piece in the nation’s effort to eliminate childhood lead poisoning in America by the year 2010.

Richard Joseph Jackson, MD, MPH  
Director, National Center for Environmental Health



## Preface

Because case management of children with elevated blood lead levels varies markedly among states, cities, and other jurisdictions, the Advisory Committee on Childhood Lead Poisoning Prevention developed these nationally applicable recommendations. Based on recently published studies and augmented with opinions of experts, this report defines the elements of case management and offers assessment and management guidelines for health departments, case managers, primary care physicians, and other professionals. Not all recommendations are appropriate for any individual child because of variations in age, blood lead level, housing status, and—most important—the ability of caregivers to respond to recommendations without being overwhelmed.

The report contains five chapters in addition to the introduction: home environment investigation and interventions, medical evaluation and treatment, nutritional assessment and dietary modification, developmental surveillance and interventions, and education for caregivers. At the beginning of each chapter is a summary table of specific management recommendations. (The remainder of the tables, the figures, and the references are at the end of each chapter.) The text of the chapters provides the detailed information and references upon which most recommendations are based. Each chapter concludes with suggestions for further research.

This report, in addition to addressing the case management of individual children, also discusses the importance of state laws, regulations, and financing related to lead abatement efforts and the provision of appropriate services for affected children. Finally, the authors of this report recognize that case management is involved with the secondary prevention of elevated blood lead levels and that primary prevention by the removal of ongoing lead exposure sources should be promoted as the ideal and most effective means of preventing elevated blood lead levels.



# Members of the Advisory Committee on Childhood Lead Poisoning Prevention

March 2002

## **ACTING CHAIR**

Carla C. Campbell, MD, MS  
Children's Hospital of Philadelphia  
Philadelphia, Pennsylvania

## **EXECUTIVE SECRETARY**

Gary P. Noonan, MPA  
Acting Chief, Lead Poisoning Prevention Branch  
National Center for Environmental Health, CDC  
Atlanta, Georgia

## **MEMBERS**

Cushing N. Dolbeare  
Housing and Public Policy Consultant  
Washington, D.C.

Anne M. Guthrie, MPH  
Alliance to End Childhood Lead Poisoning  
Charlottesville, Virginia

Birt Harvey, MD  
Pediatrician  
Palo Alto, California

Richard E. Hoffman, MD, MPH  
Physician  
Denver, Colorado

Amy A. Murphy, MPH  
City of Milwaukee Health Department  
Milwaukee, Wisconsin

Estelle B. Richman, M.A.  
Philadelphia Department of Public Health  
Philadelphia, Pennsylvania

Joel D. Schwartz, PhD  
Harvard School of Public Health  
Boston, Massachusetts

Michael W. Shannon, MD, MPH  
Children's Hospital Boston  
Boston, Massachusetts

Michael L. Weitzman, MD  
University of Rochester  
Rochester, New York

**EX OFFICIO MEMBERS**

Agency for Toxic Substances and Disease Registry  
Olivia Harris, MA

Centers for Medicare and Medicaid Services  
Jerry Zelinger, MD

Health Resources and Services Administration  
Byron P. Bailey, MPH

National Institute for Occupational Safety and Health, CDC  
Robert J. Roscoe, MS

National Institute of Environmental Health Sciences  
Walter Rogan, MD

U.S. Agency for International Development  
John Borrazzo, PhD

U.S. Consumer Product Safety Commission  
Lori Saltzman

U.S. Department of Housing and Urban Development  
David Jacobs, PhD

U.S. Environmental Protection Agency  
William H. Sanders, III, DrPH

U.S. Food and Drug Administration  
Michael P. Bolger, PhD

## **LIAISON REPRESENTATIVES**

American Academy of Pediatrics

J. Routt Reigart, II, MD

American Association of Poison Control Centers

George C. Rodgers, Jr., MD, PhD

American Industrial Hygiene Association

Steve M. Hays

American Public Health Association

Rebecca Parkin, PhD, MPH

Association of Public Health Laboratories

Henry Bradford, Jr., PhD

Association of State and Territorial Health Officials

Karen Pearson

Council of State and Territorial Epidemiologists

Ezatollah Keyvan-Larijani, MD, DrPH

National Center for Healthy Housing

Pat McLaine, MPH



## Authors

### *Assessment and Remediation of Residential Lead Exposure*

Prepared by:

Thomas D. Matte, MD, MPH

Dennis Kim, MD, MPH

Division of Environmental Hazards and Health Effects

National Center for Environmental Health

Mark R. Farfel, PhD

Kennedy Krieger Institute

### *Developmental Assessment and Interventions*

Prepared by:

David Bellinger, PhD, MSc

Harvard Medical School

Leonard Rappaport, MD

Harvard Medical School

### *Educational Interventions for Caregivers*

Prepared by:

James R. Campbell, MD, MPH

University of Rochester School of Medicine and Dentistry

Michael L. Weitzman, MD

University of Rochester School of Medicine and Dentistry

### *Medical Assessment and Interventions*

Prepared by:

James R. Roberts, MD, MPH

Medical University of South Carolina

J. Rountt Reigart, MD

Medical University of South Carolina

*Nutritional Assessment and Interventions*

Prepared by:

James D. Sargent, MD  
Dartmouth Medical School

## Acknowledgments

I am indebted to many people for their input into this document. The Working Group decided on the format and appropriate individuals to develop each chapter, reviewed with those authorities the initial drafts, and suggested modifications that the chapter authors invariably felt benefitted their work. The Working Group members were Carla Campbell, Isabella Clemente, Susan Cummins, Patricia McLaine, Tom Matte, Joel Schwartz, and Michael Weitzman.

For each chapter, its authors, CDC experts in the subject, and outside experts attended a Working Group meeting. They provided background information, additional citations, and suggestions for modifications. Among these experts were Susan Adubato, Carol Ballew, Andrea Carlson, Julian Chisholm, Mary Cogswell, Peter Dallman, Mark Farfel, Warren Galke, Scott Grosse, Randy Louchart, Kathryn Mahaffey, Morri Markowitz, Tim Morta, George Rhoads and Walter Rogan.

The document was reviewed at meetings attended by ACCLPP members, ex officio members, liaison representatives, and chapter authors. Changes requested by attendees were incorporated in the final document.

Special thanks should go to CDC staff members Alan Bloch and Jerry Hershovitz, who assisted in the initial stages of reviewing and commenting on the document; Becky Wright, who organized meetings and typed revisions; Philip Jacobs and Nikki Kilpatrick, who assisted with final formatting; Pamela Meyer, who shepherded the document to its present form; and Joey Johnson and Connie Woodall, who provided graphics support.

Sheila Jurik, Kevin Moran, and Pam Gillis Watson provided editorial support.

Last, my personal thanks to Susan Cummins, chair of the ACCLPP at the time this project was initiated, without whose efforts this document would never have been developed or published.

Birt Harvey, Editor  
Chair, Working Group



## Glossary

ACCLPP—Advisory Committee for Childhood Lead Poisoning Prevention.

Acidosis—a condition resulting from the accumulation of acid or depletion of bicarbonate content in the blood and tissues.

Aminoaciduria—an excess of amino acids in the urine.

Asymptomatic—without signs or symptoms.

Ataxia—failure of muscular coordination; irregularity of muscular action.

Bioavailable—readily absorbed and used by the body.

BLL—blood lead level, usually measured in micrograms per deciliter (Fg/dL).

Caregiver—parent, guardian, or other person involved in a child’s daily care.

CDC—Centers for Disease Control and Prevention; part of the U.S. Department of Health and Human Services, Public Health Service.

Chelation therapy—the use of chelating agents (chemical compounds that bind to metals) to remove toxic metals such as lead from the body.

Clearance standards—maximum allowable lead levels on surfaces (e.g., floors, windowsills, and window wells) after a residence has undergone lead abatement.

Drip line—the area under the edges of a roof.

EBLL—elevated blood lead level, defined as any blood lead level  $\geq 10$ Fg/dL.

Encephalopathy—extensive swelling of the brain.

Environmental investigation—an investigation by trained personnel at a child’s residence (or any secondary addresses where the child spends significant amounts of time) to identify lead hazards.

Gingival lead lines—darkening of the gums just distal to the insertion of the tooth.

Glucosuria—the presence of glucose in urine.

HUD—U.S. Department of Housing and Urban Development.

Hypophosphatemia—an abnormally low blood phosphate level.

Fg/dL—micrograms per deciliter, the usual unit of measure for blood lead levels.

Fg/ft<sup>2</sup>—micrograms per square foot, a unit of measure for measuring dust lead loading.

Papilledema—excessive fluid in the optic disk; also called choked disk.

PCP—primary care provider, the health professional who oversees a child’s care, usually a physician, nurse practitioner, or physician’s assistant.

Phosphaturia—an abnormally high urine phosphate level.

Pica—compulsive eating of nonnutritive substances such as dirt or flaking paint.

ppb—parts per billion.

ppm—parts per million.

Primary prevention—preventing a problem before it occurs. Primary prevention of lead poisoning would eliminate lead sources, thus preventing exposure.

Proteinuria—excess protein in the urine.

Radiograph—a film record of internal structures produced by passing x-rays or gamma rays through the body; frequently referred to as an “x-ray.”

Renal—having to do with the kidneys.

Secondary prevention—responding to a problem after it has been detected. Secondary prevention of lead poisoning involves identifying children with EBLLs and eliminating or reducing their lead exposure.

WIC—Special Supplemental Nutrition Program for Women, Infants, and Children.

## **Chapter 1. Introduction**



## Development of the Case Management Recommendations

This report from the Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) is intended to facilitate the management of children with elevated blood lead levels (EBLLs) by providing case managers with information and guidance. Some of the assessments and interventions recommended herein will be the primary responsibility not of case managers, but of other individuals or groups—primary care providers (PCPs), public health agencies, nutritionists, managed care organizations, and environmental inspectors—for whom this document should be considered as only a supplementary, not primary, source of information. Through this document, however, case managers can become familiar with the activities and responsibilities of others, and thus be better prepared to offer them guidance, assistance, and support.

Many studies published since the 1991 Centers for Disease Control and Prevention (CDC) report *Preventing Lead Poisoning in Young Children* (1) have provided updated or new information that can assist case managers of affected children and their families. In response, some states and localities have implemented a variety of changes in case management procedures. The plethora of new information and the marked variation in assessment and management policies among various jurisdictions were the main stimuli for the development of these guidelines.

This report is divided into five chapters other than this introduction: four that present assessment and intervention guidelines from environmental, medical, nutritional, and developmental viewpoints plus one that presents caregiver education guidelines. Experts in each subject area were asked to summarize recommended case management actions; to provide a detailed, referenced basis for their recommendations; and to suggest the most important areas for future research to support, modify, or eliminate poorly justified or empirically based recommendations.

Recommendations in each chapter are based on the results of evidence-based studies wherever possible. The most convincing basis for a specific recommendation is data from prospective, randomized, controlled trials. Unfortunately, such data are scarce; therefore, experts who developed each chapter had to rely primarily on softer data from cross-sectional studies, cohort or case controlled studies, uncontrolled studies, epidemiologic data, and—if appropriate—case reports or animal studies. They were also asked to note whether studies of interventions used to support their recommendations were efficacy studies (studies performed under ideal conditions) or effectiveness studies (studies performed in ordinary settings).

In the absence of sufficient study data, the opinions of respected authorities were considered in the formulation of these recommendations. Recommendations, particularly those not based on controlled studies, were often modified by the ACCLPP working group and subsequently by the full committee. Thus, in their final form, the recommendations in this report represent the consensus of the ACCLPP rather than individual opinions of the authors of each chapter.

This report is written primarily for those who will provide case management for children with EBLLs and for health department personnel who oversee case management follow-up. Because there is unavoidable overlap among chapters, interested professionals may gain insight from chapters covering areas outside their own expertise. For example, a nutritionist or a PCP will find iron stores and anemia discussed in both the medical and the nutritional sections.

Although the primary cause of EBLLs in children is exposure to deteriorated paint in housing built before 1950, other sources of lead are found in some states and localities. Consequently, users of these guidelines may need to modify them to meet the needs unique to specific communities. Further, because the prevalence of EBLLs among children will vary markedly among and within states, the number of children managed will show corresponding variation.

Because there is no apparent threshold below which adverse effects of lead do not occur, “EBLL” must be defined arbitrarily. This report uses the definition given in the 1997 CDC report *Screening Young Children for Lead Poisoning* (2), which defined child blood lead levels (BLLs)  $\geq 10$  Fg/dL as elevated. Although the BLL at which particular elements of case management will be initiated is variable, education and follow-up BLL monitoring should be available for any child who has a confirmed BLL  $\geq 10$  Fg/dL. More intense management, including home visiting and environmental investigation, should be available to any child with a BLL  $\geq 20$  Fg/dL, or persistent levels in the 15 to 19 Fg/dL range.

Another variable, the duration of management, will depend on the effects of lead on the child being treated. As noted in Chapter 5, “Developmental Assessment and Interventions,” the effect of lead on a child may not be demonstrable until the child is well into the elementary school years, meaning that some children will need continued tracking by PCPs or others long after their case management ends.

The interventions recommended in this report are for the secondary prevention of EBLLs—which is to prevent further lead exposure and to reduce BLLs in children who have been identified as having EBLLs—and involve a number of scientific, technical, and implementation issues. The ultimate goal, primary prevention—the removal of harmful lead exposure sources (especially older, deteriorated housing) and the elimination of lead from products with which children may come in direct or indirect contact—involves other, sometimes overlapping, issues. The importance of primary prevention should not be overlooked, since the behavioral and cognitive effects of EBLLs in young children are apparently irreversible.

### **Overview of Comprehensive Case Management**

#### *What Is Case Management?*

Case management of children with EBLLs involves coordinating, providing, and overseeing the services required to reduce their BLLs below the level of concern (i.e., 10 Fg/dL). It is based

on the efforts of an organized team that includes the child's caregivers. A hallmark of effective case management is ongoing communication with the caregivers and other service providers, and a cooperative approach to solving any problems that may arise during efforts to decrease the child's BLL and eliminate lead hazards in the child's environment. Case management is not simply referring a child to other service providers, contacting caregivers by telephone, or other minimal activities.

The current model of case management has eight components: client identification and outreach; individual assessment and diagnosis; service planning and resource identification; the linking of clients to needed services; service implementation and coordination; the monitoring of service delivery; advocacy; and evaluation (3). Once an eligible child is identified, the case manager should do the following:

- Visit the child's residence (and other sites where the child spends significant amounts of time) a minimum of two times.
- Assess factors that may impact the child's BLL (including sources of lead, nutrition, access to services, family interaction, and caregiver understanding).
- Oversee the activities of the case management team.
- Develop a written plan for intervention.
- Coordinate the implementation of the plan.
- Evaluate compliance with the plan and the success of the plan.

An environmental inspector should also visit the child's residence, with the case manager if possible, to conduct a thorough investigation of the site and identify sources of environmental lead exposure. The case management team can then use the results of this investigation to develop a plan to protect the child and correct hazardous conditions. Although environmental services may be provided by the case manager, the environmental inspector, or other program staff, the case manager is responsible for ensuring that a child receives services in a timely fashion.

### *Funding*

Nationally, an estimated 83% of children with BLLs  $\geq 20$  Fg/dL are eligible for Medicaid (4). Both the case management of eligible children and the environmental investigation of their surroundings are reimbursable according to federal Medicaid policy, with each state responsible for setting reimbursement rates for eligible services.

Despite this, funding for services remains a critical resource issue for most states. Fewer than half of all states provide Medicaid reimbursement for lead follow-up services, with the level of reimbursement varying widely. In addition, most state programs do not know how many children with BLLs  $\geq 20$  Fg/dL also receive Medicaid. As of 2000, only 10 state lead programs

were able to successfully identify Medicaid children by linking their Medicaid and lead screening data bases (5).

### *Who Provides Case Management?*

Ninety percent of programs use professionals (nurses or social workers) to deliver case management services (6). The case manager is usually a member of the local health department staff, although nearly half of all states also use other providers to deliver case management services.

In most cases, a management team can best meet the needs of an individual child. The team may include the case manager, the child's caregiver, the child's PCP, an environmental inspector, a health educator, a nutritionist, and the local public health agency.

### *Time Frames for Initiating Case Management Services*

A case manager should schedule an appointment with the child's caregiver as soon as possible after being assigned to the case. Where feasible, public health agencies providing case management services should give priority to children with the highest BLLs and those less than 2 years of age. If the caregiver does not have a telephone, the case manager should visit the child's home and leave information at the door if no one is there. For children with BLLs  $\geq 45$  Fg/dL, the case manager should contact the child's PCP immediately to determine whether the child is being chelated at home or in the hospital. If the child is hospitalized, the initial visit may take place at the hospital. However, it is critical that team members conduct a hazard assessment in the child's home as quickly as possible. (See Chapter 2, "Assessment and Remediation of Residential Lead Exposure," and Chapter 3, "Medical Assessment and Interventions.")

<u>Blood lead level (Fg/dL)</u>	<u>Time frame for initial home visit</u>
15-19 (persistent*)	within 2 weeks of referral
20-44	within 1 week of referral
45-70	within 48 hours of referral
$\geq 70$	within 24 hours of referral

\*two venous BLL measurements at this level more than 3 months apart

### *The Case Management Plan*

The case manager is responsible for developing and implementing a written management plan based on a needs assessment done at visits to the child's home and other sites where the child spends significant amounts of time. Although all cases require a

minimum of two home visits, additional visits are often necessary. The caregivers also should be involved in developing the plan to ensure that it is realistic and meets their perceived needs. Areas the plan should cover are detailed in Table 1.1 and in specific sections of this report.

### *Coordination of Care by the Case Manager*

The case manager is responsible for coordinating care and ensuring that all team members, including the caregiver, stay in communication and work together. Such communication includes verbal consultations with and written summaries of progress for team members. Case managers need not directly provide all follow-up care, but they are responsible for seeing that needed care is provided, including medical follow-up. In most jurisdictions, the environmental inspector or program issues and enforces lead hazard remediation orders. The case manager must be sufficiently knowledgeable about environmental investigation and follow-up, however, to ensure that inspection and remediation take place in a timely fashion and that short-term efforts are made to decrease an affected child's exposure to lead hazards. Similarly, the case manager is responsible for ensuring that someone follows up on referrals for other problems identified during case management.

### *Case Closure*

It often takes an extended period of time to complete all the elements in a case management plan. When the environmental lead hazards have been eliminated, the child's BLL has declined to below 15 Fg/dL for at least 6 months, and other objectives of the plan have been achieved, the case should be closed. However, the case manager should discuss with the PCP and caregiver provisions for appropriate long-term developmental follow-up. (See Chapter 5, "Developmental Assessment and Interventions".) Case closure criteria should also include provisions for administrative closeout if at least three documented attempts to locate or gain access to the child and caregiver have failed.

### *Public Health Agency Role*

Although the recommended public health agency activities are not part of case management *per se*, they are necessary to achieve optimum results. With their focus on the core public health functions of assessment, policy development, and quality assurance, public health agencies play a broad role in coordinating care at the state and local level. They also are responsible for initiating and implementing laws and regulations that will help to eliminate childhood lead poisoning. Local jurisdictions must have the political will

to take enforcement actions, where needed, to protect the health of children. The identification of affected children and exposure sources will have little impact unless lead hazards are eliminated in a timely manner. Public health agencies should take the following steps to coordinate care in six areas:

- *Screening and surveillance:* Ensure that screening of at-risk children is conducted in accordance with the state or local plan. Develop and maintain communication and good working relationships with PCPs and public and private health delivery organizations (including Medicaid).
- *Laboratory testing and reporting:* Require that EBLL test results (and, ideally, all blood lead test results) be reported in a timely and accurate manner, and provide oversight to ensure such reporting. Implement quality control measures for both environmental and blood specimens to ensure the validity and reliability of results.
- *Case management:* Set standards for follow-up of children with BLLs  $\geq 10$  Fg/dL; ensure that these standards are met. Establish procedures for identifying new cases, assigning cases to case managers, providing oversight of case management activities and case managers, and providing oversight for environmental inspection and remediation. Secure Medicaid reimbursement for case management and environmental services. Identify service gaps and take appropriate action.
- *Care coordination:* Work with public and private organizations including health care providers, managed care organizations, Medicaid agencies, housing organizations, mortgage lenders, property owners, and community groups. Provide consultation, education, and technical assistance to these groups, and prepare and distribute educational materials to them. Develop program policy supporting the effective management of children with EBLBs (7).
- *Environmental interventions:* Ensure that laws and regulations related to lead hazard remediation are sufficient to address identified hazards. Enforce safety standards for lead in housing, food, and water. Oversee appropriate exposure reduction for each child. (See Chapter 2, “Assessment and Remediation of Residential Lead Exposure.”)
- *Evaluation:* Evaluate and report on the outcomes of the follow-up care provided to children with EBLBs. Promote necessary changes in programs and policies. Develop an annual report that includes evaluations of screening, reporting, and case management efforts.

## General Considerations

There are several guiding principles to consider when making recommendations for children with EBLs. First, interventions should be directed at children whose risk for lead exposure is high. Second, where possible, interventions should be targeted at children less than 2 years old because neurotoxicity is greater and lead exposure is more likely to result in a rapid increase in BLLs in very young children. Finally, when intervention recommendations are based on tenuous data or on expert opinion, as are some in this document, case managers and other involved professionals should more than ever remember *primum non nocere* (first, do no harm). Most children with EBLs come from economically disadvantaged families who may have difficulty meeting the daily challenges of life and who may be overwhelmed if presented with a long list of interventions. Further, as has been found in many studies of interventions to combat other childhood problems (injury prevention, dietary counseling), behavioral change recommendations usually have only a modest effect at best. Thus, better results may be achieved by focusing on the most important recommendations (usually those designed to eliminate environmental lead hazards) and assisting caregivers in implementing them. Encouraging and supporting families without making them feel guilty for their child's EBL or making unrealistic demands on them may offer the greatest benefit to the child.

## References

1. CDC. Preventing lead poisoning in young children. Atlanta, Georgia: US Department of Health and Human Services, CDC; 1991.
2. CDC. Screening young children for lead poisoning: guidance for state and local public health officials. Atlanta, Georgia: US Department of Health and Human Services, CDC; 1997.
3. Weil M, Karls JM. Case management in human service practice. San Francisco, CA: Jossey Bass Publishers; 1985.
4. US General Accounting Office (GAO). Medicaid: elevated blood lead levels in children. GAO/HEHS-98-78. Washington, DC: GAO; 1998.
5. National Center for Lead-Safe Housing. Another link in the chain update: state policies and practices for case management and environmental investigation for lead-poisoned children. Washington, DC: Alliance to End Childhood Lead Poisoning and the National Center for Healthy Housing; 2001.
6. Alliance To End Childhood Lead Poisoning, the National Center for Lead-Safe Housing. Another link in the chain: state policies and practices for case management and environmental investigation for lead-poisoned children. Washington, DC: Alliance to End Childhood Lead Poisoning and the National Center for Lead-Safe Housing; 1999: p. 33.
7. Epstein SG, Taylor AB, Brown MJ. Coordinating care from clinic to community. Boston, MA: New England SERVE, Rhode Island Department of Health; 1998.

**Table 1.1. Possible Elements of a Case Management Plan Based on Individualized Child Assessment**

<b>Activities</b>	<b>Partnerships needed</b>	<b>Health department resources</b>	<b>Refer to:</b>
<b>Reduction/elimination of environmental hazards</b>			
Assessment of all possible exposure sources	CM, EI, caregiver, PCP	Environmental investigation service or referral list of investigators, laboratory services	<i>Chapter 2, Chapter 3, Appendix I</i>
Temporary/short-term hazard reduction Short-term reduction of residential hazards	CM, caregiver, CHW, caregiver, lead educator, EI	Case-management services, educational materials, HEPA vacs, cleaning materials, cleaning service, temporary coverings	<i>Chapter 2, Chapter 6</i>
Temporary relocation to lead-safe housing	CM, caregiver, SW, PO, community- and faith-based organizations	Case-management services, referrals, linkages with PO, funds for relocation assistance	
Long-term hazard elimination Remediation/lead hazard control	CM, caregiver, SW, PO, EI, HA, community- and faith-based organizations	Case-management services, lead hazard control program	<i>Chapter 2</i>
Permanent relocation to lead-safe housing	CM, caregiver, SW, PO, EI, HA, utility company, community- and faith-based organizations	Case-management services, funds for relocation assistance, lead-safe housing registry	
Identification and removal of non-residential exposures (e.g., remedies, leaded objects, take-home exposures from parent's occupation)	CM, CHW, caregiver, PCP, EI, occupational health specialist	Educational materials on non-paint exposure sources, laboratory services	<i>Chapter 2, Chapter 3, Appendix I</i>

Activities	Partnerships needed	Health department resources	Refer to:
<b>Improvement of nutrition</b>			
Caregiver counseling	CM, nutritionist, PCP, CHW	Case-management services, referrals, linkages, and educational materials	<i>Chapter 3, Chapter 4, Chapter 6</i>
Referrals to WIC or other community food resources	CM, nutritionist, PCP, CHW	Case-management services, WIC program, referrals	
<b>Caregiver lead education</b>			
Counseling re: lead and lead-exposure risks, decreasing identified risks, cleaning practices, importance of follow-up blood lead tests	CM, CHW, caregiver, PCP, EI	Case-management services, educational materials, cleaning materials	<i>Chapter 6</i>
<b>Medical follow-up care</b>			
Child with EBLL Siblings or other at-risk children living in home	CM, PCP, caregiver	Case-management services, transportation services	<i>Chapter 3</i>
<b>Follow-up of other identified problems</b>			
Counseling/referral for: medical services, early intervention and developmental assessment, housing services, social services, Head Start, parent support	CM, PCP, caregiver, community- and faith-based organizations	Case-management services, referrals, linkages	<i>Chapter 3, Chapter 4</i>

**Abbreviations:**

CHW—Community health worker

CM—Case manager

EI—Environmental inspector

HA—Housing authority

HEPA—high-efficiency particulate air (filter)

PCP—Primary care provider (health professional)

PO—Property owner

SW—Social worker

WIC—Special Supplemental Nutrition Program for Women, Infants, and Children

## **Chapter 2. Assessment and Remediation of Residential Lead Exposure**

Prepared by Thomas D. Matte, MD, MPH, Dennis Kim, MD, MPH,  
and Mark R. Farfel, PhD



**Table 2.1. Summary of Recommendations for Assessment and Remediation of Residential Lead Exposure**

- Make prompt and effective environmental management for children with EBLLs the highest priority of all childhood lead poisoning prevention programs.
1. Conduct an environmental investigation for all children with blood lead levels  $\geq 20$  Fg/dL, or persistently  $\geq 15$  Fg/dL. This investigation should include:
    - a. An inspection of the child's home and other sites where the child spends significant amounts of time.
    - b. A history of the child's exposure.
    - c. Measurements of environmental lead levels, including at a minimum
      - i. House dust;
      - ii. Paint that is not intact or is subject to friction;
      - iii. Exposed soil, especially in play areas;
      - iv. Other media as appropriate;
  2. Ensure that interventions to reduce ongoing exposure:
    - a. Focus on control of current lead hazards.
    - b. Include prompt interim measures (e.g., house dust control by professional cleaners) where appropriate, to rapidly reduce lead exposure.
    - c. Be performed in accordance with safe practices by trained workers to avoid increasing lead exposure to occupants and workers.
    - d. Keep to a minimum on-site removal of intact leaded paint.
    - e. Replace or enclose building components when elimination of intact leaded paint is performed.
    - f. Include clearance testing following lead hazard reduction work to ensure that lead levels are safe prior to a structure being re-occupied.
    - g. Include temporary occupant relocation or other measures to protect occupants from exposure to leaded dust produced by lead hazard control activities.
    - h. Relocate children permanently to lead-safe housing if necessary to reduce their lead exposure in a timely manner.
  3. Encourage state and local governments to assess the effectiveness of their laws, ordinances, housing codes, and enforcement structures in dealing with identified lead hazards and to identify changes required to ensure that children are protected.
  4. Promote the expansion of existing federal, state, and local subsidies to help finance lead hazard control in economically distressed communities, and the creation of new subsidies, if necessary.

### Introduction

Recent research concerning lead exposure from leaded paint in the residential environment has shown that some of the recommendations on managing lead hazards in the child's environment made in the 1991 Centers for Disease Control and Prevention (CDC) guidance, *Preventing Lead Poisoning in Young Children*, need updating (1). In addition, a regulation to control lead exposure from public drinking water (2), implemented during the 1990s, makes possible a more focused approach to assessing that source than was previously recommended. This chapter summarizes current knowledge concerning children's lead exposure in the residential environment, recommends interventions directed at reducing or eliminating lead exposure, and provides information to guide state and local officials in developing and updating policies and procedures for identifying and managing lead hazards in the residential environment of children with elevated blood lead levels (EBLLs).

Detailed technical protocols for assessing and correcting lead hazards in a variety of situations can be found in guidance developed by the Department of Housing and Urban Development (HUD) for property owners, private contractors, and government housing agencies (3). These are cited where appropriate.

### Sources and pathways of residential lead exposure

Lead can be found in high concentrations in three media to which children may be directly or indirectly exposed: paint, interior dust, and exterior soil or dust. This section discusses the distribution of lead in these media and their relationships to one another and to blood lead levels (BLLs) in children (Figure 2.1). Lead in tap water, generally a lower dose source of exposure, is also addressed.

#### *Paint*

Although the addition of lead to residential paint and similar surface-coating materials, such as varnishes and stains, was banned in 1978 (4), 74% of dwellings constructed prior to 1980 contain some leaded paint.\* The amount of lead in paint is much greater in homes built before 1950 than in homes built later but prior to the ban on leaded house paint. For example, 90% of

---

\*Throughout this document, the term "paint" will be used to refer to paint and, where appropriate, similar surface-coating materials such as varnishes and stains. Paints and coatings manufactured since 1978 must contain < 0.06% lead by weight. For testing of lead content in existing structures, the regulatory threshold for defining "lead-based paint" is \$ 1 milligram of lead per square centimeter of paint film or \$ 0.5 % lead by weight. These standards, however, were based on the limitations of measurement techniques available when they were formulated rather than on health considerations.

dwellings built before 1940 have paint containing more than 1 mg/cm<sup>2</sup> of lead, compared with 62% of dwellings built from 1960 through 1979. The relative contrast is much greater for paint containing more than 2 mg/cm<sup>2</sup>: 75% versus 18%, respectively (5). Direct and indirect exposure of children to leaded paint that has deteriorated because of deferred maintenance is likely the major factor in the increased risk for EBLL associated with poverty and living in older housing. Data from the Third National Health and Nutrition Examination Survey (NHANES III) indicate that the prevalence of EBLs among children living in homes built before 1946 is five times higher than that among children living in homes built after 1973 (most of which do not have leaded paint) (6). Furthermore, for low-income children living in pre-1946 dwellings, the prevalence of EBLs is 16%, compared with 4% for middle-income children living in such dwellings (6).

Although children may be exposed to lead from paint directly by ingesting paint chips (7), they are more commonly exposed by ingesting house dust or soil contaminated by leaded paint (8, 9). Federal law defines a leaded paint hazard as a condition in which exposure to lead from lead-contaminated dust, lead-contaminated soil, or deteriorated leaded paint would have an adverse effect on human health (10).

Lead contamination of dust or soil occurs when leaded paint deteriorates or is subject to friction or abrasion (as on window sashes). In addition, lead can be dispersed when paint is disturbed during demolition, remodeling, paint removal, or preparation of painted surfaces for repainting. In a population-based study in Wisconsin, about two-thirds of children who had a blood lead test lived in a home that had undergone some type of renovation, repair, or remodeling work in the prior year. These children were at 1.3 times greater risk of having an EBL than were children not exposed to such activities (11). The risk was even higher among children living in homes where certain practices, such as the removal of paint with heat guns, had been used.

### *Interior dust*

Interior house dust can become contaminated with lead as the result of the deterioration or disturbance of leaded paint, the tracking or blowing in of contaminated soil, and the fallout of airborne lead particulate from industrial or vehicular sources. A simple visual inspection of older homes can identify those in poor condition. The condition of leaded paint more accurately predicts lead exposure than the lead content of paint by itself (12, 13). Older homes in poor condition have much higher dust lead levels than older homes in good condition (Figure 2.2) (14). The amount of lead in house dust, in turn, has a strong correlation with the BLLs of young children (12, 13, 15, 16) and is more predictive of BLLs in children than is the amount of lead in house paint (13). Lead levels in house dust can be measured either as a mass concentration (mass of lead/mass of dust) or as surface loading (mass of lead per surface area sampled). The most widely used sampling technique, in which a wipe sample is collected with commercially available baby wipes (3), can determine only lead surface loading. However, this measure

predicts BLLs as well as or better than mass concentration (17). Lead loadings vary considerably among the types of surfaces commonly tested, with levels on interior window sills and window “wells” (the part of the window that receives the lower sash when closed) often being, respectively, 1 and 2 orders of magnitude higher than those found on floors. Higher levels on window components may reflect a combination of lead dust derived from friction and the deterioration of leaded paint on the windows themselves and from the settling of airborne dust from outside of the dwelling. Dust lead loading on all three surfaces (floors, windowsills, and window wells) correlates with BLLs in children (12).

A recent statistical analysis of data from 12 studies relating lead in dust to BLLs in children between 6 and 36 months of age found a strong direct association between dust lead loading and the risk of having an EBLL (13). The association extended well below the 40 Fg/ft<sup>2</sup> threshold for a lead hazard in dust samples collected from floors as defined by HUD (18) and the Environmental Protection Agency (EPA) (19). For example, the estimated probability of a child having an EBLL increases from 7% to 18% with an increase in floor dust lead loading from 10 to 40 Fg/ft<sup>2</sup> (Figure 2.3) (13). For dust samples collected from window sills, lead levels  $\geq$  250 Fg/ft<sup>2</sup> are defined as hazardous (18, 19).

### *Soil and Exterior Dust*

Contamination of soil and exterior dust has been linked to point source emissions, such as lead smelters, fall-out from past use of leaded gasoline, and weathering of exterior leaded paint (20). Soil located next to dwellings typically has higher lead content than that sampled from other locations in a yard.

Potentially hazardous levels of lead in soil are not uncommon. Results of a national survey in which soil samples were collected from both bare and covered soil showed that residences with intact exterior leaded paint are more than three times as likely to have soil lead levels exceeding 500 ppm than are dwellings without lead in exterior paint (21% vs. 6%). Results also showed that soil contamination is eight times more common at residences with non-intact leaded exterior paint than at residences without exterior leaded paint (48% vs. 6%) (5). In urban neighborhoods, high levels of lead have also been found in exterior dust collected from paved surfaces, such as sidewalks (21).

Soil lead content is an important predictor of children’s risk for an EBLL, though less important than the lead content of interior floor dust (13). Soil samples taken from play areas in a yard have a stronger relationship to children’s BLLs than samples from other locations. The EPA defines a soil lead hazard as bare soil that contains 400 ppm of lead in a play area or 1200 ppm in other parts of a yard (19).

### *Tap water*

Lead found in tap water usually is from the corrosion of lead-containing materials found in water distribution systems and household plumbing (22). Exposure to lead in tap water has been reduced by measures taken during the last two decades under the requirements of the 1986 and 1996 amendments to the Safe Drinking Water Act and a subsequent EPA regulation (the Lead and Copper Rule) (2). The latter regulation, which only applies to public water systems, requires those systems to monitor tap water for lead and to implement public education and other measures to reduce lead levels in drinking water if they exceed 15 Fg/L in more than 10% of household samples (2). Lead levels are reduced by treating the supplied water to make it less corrosive and, in some cases, by replacing lead water-service lines. These regulations do not apply to the more than 40 million households supplied by private well water that can have elevated levels of lead if the water is corrosive and lead is present in the well pump or household plumbing system (23). In most jurisdictions, there is no monitoring for lead in the drinking water supplied by private wells.

A number of studies, mostly of adults, have attempted to characterize the relationship between lead levels in drinking water and BLLs (24-26). Data from these studies indicate that exposure to water with a lead content close to the EPA action level would not, by itself, be expected to produce an EBLL. However, the individual risk will vary depending upon the circumstances and amount of water consumed. For example, infants consuming formula prepared with lead-contaminated water may be at particular risk because of the large amount of water they consume relative to their body size (27).

### **Effectiveness and Safety of Lead Hazard Control Measures**

Interventions to reduce exposure to lead in the residential environment include measures focused on immediate hazards to current occupants, such as removing or covering nonintact leaded paint, repairing or replacing windows to prevent abrasion of leaded paint on moving surfaces, sealing floors to create smooth and cleanable surfaces, using professional cleaners to control household dust, and covering bare, contaminated soil. Additional interventions may be carried out to prevent lead hazards from developing in the future, such as replacing building components that have leaded paint (whether intact or not) and removing (stripping) leaded paint from components left in the dwelling.

Most studies evaluating the effectiveness of lead hazard control measures for reducing EBLLs have lacked controls. In addition, many studies evaluated interventions prior to the institution of stringent procedures for limiting the contamination of residences with leaded dust. In general, these earlier studies showed that among children with baseline BLLs greater than about 25 Fg/dL, measures to remove or repair nonintact leaded paint were followed by declines

in BLLs of 20% to 30% over the following year (28). In one controlled study, the decline in BLLs for children in treated dwellings was about twice that of children in untreated dwellings (29).

In homes of children with EBLLs, extensive removal of leaded paint without measures to prevent the children's exposure to abatement dust and debris has also been associated with increases in the children's BLLs (30-32). These increases were apparently the result of corresponding increases in house dust lead levels. Consequently, regulations in many jurisdictions now prohibit certain hazardous paint removal methods, such as uncontained power sanding, and require safe work practices, cleaning, and dust lead testing to protect occupants from lead exposure associated with the disturbance of leaded paint. Most jurisdictions require that post-intervention dust lead levels be below clearance standards—the maximum allowable levels of lead. If the dust lead levels in a particular dwelling exceed the clearance standard, that dwelling cannot be reoccupied until additional cleaning or other measures reduce dust lead contamination to less than the clearance threshold. Clearance standards for public and federally assisted housing are 40 Fg/ft<sup>2</sup> for floors, 250 Fg/ft<sup>2</sup> for windowsills, and 400 Fg/ft<sup>2</sup> for window wells. Some state and local jurisdictions have established other clearance standards (20).

Recent longitudinal studies have evaluated leaded paint abatement programs that combined multiple lead hazard control methods (33-35). Interventions used in these programs included measures to prevent the generation of leaded paint chips and dust (treatments to eliminate nonintact leaded paint and windows containing leaded paint subject to friction), leaded dust removal (specialized cleaning), and measures to make floors smooth and cleanable (by sealing or using durable floor coverings). The elimination of leaded paint hazards in the programs relied primarily on component replacement, enclosure, and paint stabilization, with limited on-site paint removal. Although these studies did not include randomly assigned control homes that received no treatment, their results strongly suggest that these treatments resulted in substantial, sustained reductions in interior dust lead loading and little if any risk of children having substantial short-term increases in BLLs. While average BLLs in children occupying treated dwellings fell by approximately 20% to 25% over the following year (from baseline averages in the 5-15 Fg/dL range) (35), no data on children in untreated dwellings are available to directly estimate the proportion of decline attributable to the hazard-reduction treatments. In one of these studies, greater initial and sustained reductions in interior dust lead loadings were achieved with more intensive treatments, including window replacement (rather than repair) and the use of durable floor coverings (rather than paints and sealants) (34). However, among children living in the more intensively treated dwellings, average BLL declines following the intervention were not significantly greater than those among children whose dwellings had more limited interventions.

These studies generally involved interventions that left some intact leaded paint in place. The only certain way to prevent future exposure to lead from paint in a dwelling is to remove all leaded paint from the dwelling. However, no studies are available that compare changes in children's BLLs following the total "deleading" of their dwelling with changes following

interventions that leave some leaded paint intact. If many components in a dwelling contain leaded paint, complete deleading may be impractical unless performed as part of a substantial or “gut” renovation.

One study of children with baseline BLLs of 10 to 24 Fg/dL found that leaded paint hazard-control measures, including extensive on-site paint removal, resulted in increases in children’s BLLs after abatement (36). These increases occurred despite a protocol for safe work practices, cleaning, and clearance testing. However, the clearance standard used for floors was 200 Fg/ft<sup>2</sup>, which may have been too high to prevent continued or increased exposure to leaded dust when compared with pre-intervention levels. The previously cited impact of relatively “low” levels of lead in house dust on children’s BLLs could explain the increases.

Interventions focused on reducing exposure to leaded dust have been evaluated in several studies (37-39). Household dust control performed repeatedly by professional cleaners was associated with decreases in children’s mean BLL with the greatest benefits seen among children whose dwellings were cleaned at least 20 times during a 1-year follow-up period (38). To be effective, dust control should be conducted every 2-3 weeks. However, simply educating parents about the need to perform dust control as a preventive measure has not proven effective in preventing increases in children’s mean BLLs (39). See Chapter 6, “Educational Interventions for Caregivers,” for a detailed discussion of the effects of such education.

In a controlled study, soil removal and replacement with uncontaminated soil was associated with a 15% reduction in BLLs among children whose average baseline BLL was from 10 to 24 Fg/dL and who were exposed to high levels of lead in soil (40). Two other studies of the lead abatement of soil with lower baseline contamination showed no reduction in children’s BLLs following such abatement (21, 41).

In the studies noted above and reviewed in detail in Chapter 6, the benefits of environmental interventions have generally been modest—BLL reductions in the range of 10% to 30%. A number of factors might explain the limited effectiveness of these interventions. One such factor is that the interventions were limited in scope: lead hazard control often involved the interior but not the exterior of homes. Another factor is that most interventions were performed in scattered rather than contiguous blocks of homes. Thus, children’s continued exposure to lead from sources in the neighborhood might limit the effectiveness of the interventions. In the Baltimore repair and maintenance study, for example, one comparison group consisted of modern urban homes located in contiguous blocks of such dwellings that were built where older row homes with leaded paint once stood. The geometric mean level of lead contamination in the floor dust of the modern urban homes was less than one-tenth that of older homes that had previously undergone complete lead paint abatement but which were still surrounded by other homes with leaded paint. The geometric mean BLL for children living in the modern homes was one-fourth that of the children living in the older homes (34). A final factor is that the release of lead from bone might also reduce the impact of environmental interventions. By one estimate, an

intervention reducing total lead exposure by half for a 5-year-old child would, because of mobilized bone lead stores, cause the child's BLL to decline by only 25% after 1 year (42).

### Recommendations for Assessment and Remediation

#### *General Recommendations*

*Conduct prompt and effective environmental management.* The identification and control of ongoing sources of lead exposure for children with EBLLs should be the highest priority. In addition, identifying children with EBLLs may help officials identify and control potential sources of lead exposure for other children. Because the main objective of environmental management is to reduce lead exposure quickly, investigations should be initiated as soon as possible after a case is identified.

Priority should be placed on responding to children with the highest BLLs and to infants and children less than 2 years of age with any EBLL, because their BLLs are more likely to increase and they are more sensitive to lead's neurotoxic effects. Table 2.2 shows the recommended maximum time frames for initiating environmental investigations and interventions according to a child's BLL.

*Obtain an exposure history.* Investigations to identify sources of a child's lead exposure should begin with an interview with the child's caregiver. Whenever possible, the interview should take place at the child's residence. The interviewer should question the caregiver concerning a range of possible exposure sources. (See Table 2.3 and Appendix I.) It is also important to collect information concerning locations outside the home, such as childcare sites, where the child spends significant amounts of time. The interview should be guided by a checklist tailored to sources of lead exposure found in a given jurisdiction. Checklists facilitate data collection and ensure that potential sources are not overlooked. A sample checklist is provided in Chapter 3, "Medical Assessment and Interventions," and in the 1995 HUD guidelines (3).

*Visually inspect the residential environment.* A visual inspection can quickly identify areas where deteriorating paint may be contributing to lead exposure and should include windows, porches, bare soil, and common areas in multifamily dwellings, as well as any other locations where the child spends time.

*Measure lead in environmental media.* Selection of the media to be tested should be guided by the visual inspection and the child's exposure history. Depending on the inspector's training, the equipment available, and the media to be tested, environmental analysis may be done either on-site with portable instruments or at an environmental laboratory. Personnel performing environmental sampling and on-site testing should be appropriately trained and be certified as risk assessors (43) or have equivalent qualifications.

*Communicate results.* Results of investigations, including recommended actions to protect the child from further exposure, should be communicated promptly to caregivers, to primary care providers (PCPs), and, where relevant, to property owners and housing code enforcement authorities. Environmental management activities should be coordinated with other health professionals, including those providing clinical care, case management, and social services.

### *Specific Recommendations*

Since leaded paint and associated lead in house dust and soil are the most common sources of exposure, they should be the focus of environmental investigations and control efforts. State and local health officials should review current policies concerning childhood lead poisoning prevention and revise them as needed to be consistent with the following recommendations.

*Measure lead levels in house dust, paint, and bare soil.* Investigations of the residential environment of children with EBLs should focus on immediate lead hazards. At a minimum, testing should include house dust, paints, and similar surface coatings that are not intact or that are located on surfaces subject to friction, and bare soil, especially in play areas. Detailed protocols for sampling and measuring lead in these media can be found in the 1995 HUD guidelines (3).

There is no evidence that complete testing of all building components for leaded paint, regardless of the condition or location of the paint, is helpful in identifying ongoing exposure. Such testing may serve other purposes, however, such as educating occupants about the health hazards of leaded paints, planning the abatement of potential future leaded paint hazards, planning renovation work that may involve disturbance of intact paint, or complying with state and local regulations.

*Test for lead in tap water.* For homes served by public water systems, data on lead in drinking water should be obtained from the water supplier. Many public water systems post data on the Internet on the quality of drinking water, including results of lead testing. Links to such data can be found at the following EPA Web site:

<http://yosemite.epa.gov/ogwdw/ccr.nsf/America?OpenView>. If prior testing of a public water system shows that lead contamination is not a problem in homes served by that system, no additional testing is necessary, unless no other source of a child's EBL can be found. For all other children with EBLs, including children living in homes served by private wells, water that the child may consume should be tested. If necessary, measures should be implemented to prevent the child's further exposure to lead (e.g., the use of bottled water or appropriate water filters). If bottled water is used, fluoride supplementation should be discussed with the PCP and the caregiver. More information on lead in drinking water can be found at <http://www.epa.gov/ogwdw/dwh/o-ioc/lead.html> or by contacting the Safe Drinking Water hotline at (800) 426-4791 or [hotline-sdwa@epamail.epa.gov](mailto:hotline-sdwa@epamail.epa.gov). Additional sources of information

about lead in drinking water can be found in Chapter 6, “Educational Interventions for Caregivers.”

*Control immediate hazards.* Interventions to reduce ongoing exposure should include:

1. Replacing or stabilizing the paint in building components containing nonintact leaded paint.
2. Replacing or repairing windows and other building systems to eliminate the abrasion of leaded paint.
3. Covering or replacing bare lead-contaminated soil.
4. Conducting specialized cleaning to reduce lead loading in house dust.
5. Sealing or covering floors to make them smooth and cleanable.

Lead hazard control work must be performed in accordance with safe practices by trained workers to avoid exposing workers to unsafe lead levels or increasing the level of lead exposure to occupants. Detailed guidelines for residential lead hazard control work have been published by HUD (3).

On-site removal of intact leaded paint should be kept to a minimum, and safer alternatives, such as component replacement, enclosure, encapsulation, off-site paint removal, and paint-film stabilization should be used when possible. Replacing building components that have intact leaded paint reduces the potential for future lead exposure as the leaded paint deteriorates or is disturbed during renovation. However, such work can generate leaded dust, and workers should follow the precautions described in HUD guidelines.

As discussed previously, there is no evidence that environmental interventions that include complete removal of all leaded paint are more effective at reducing residents’ BLLs than interventions focused on current lead hazards. Furthermore, some evidence suggests that extensive on-site paint removal increases the potential for lead exposure, at least in the short run. The amount of lead in 1 ft<sup>2</sup> of paint containing 1 mg/cm<sup>2</sup> of lead (approximately 1 g or 1 million Fg) is very large relative to the amount of lead in dust associated with an increased risk for EBLs (approximately 10 Fg/ft<sup>2</sup>). Thus, performing extensive on-site removal of leaded paint in a dwelling without increasing the occupants’ lead exposure requires a degree of caution that may be difficult to achieve and monitor in the routine, large-scale implementation of health codes.

Long-term control of residential hazards from leaded paint may involve considerable time and expense. Obtaining the compliance of property owners may cause additional delays in reducing residents’ lead exposure. Therefore, interim measures to rapidly reduce lead exposure, including specialized cleaning to reduce exposure to leaded dust, are often required.

*Perform clearance testing.* Following lead hazard reduction work, repeat testing for lead in house dust is essential to see whether the work has resulted in levels of lead low enough for safe re-occupancy. Post-intervention tests showing increased or persistently high dust lead levels indicate the need for further cleaning or other additional work. Available evidence indicates that current and proposed guidelines for levels of lead in dust on floors may not adequately protect young children and that levels well below these guidelines are achievable and are often present even before intervention. Therefore, the goal should be to attain post-intervention dust lead levels that are as low as is feasible, which is generally less than 10 Fg/ft<sup>2</sup> on floors (44), and that are at or below baseline levels. Where leaded paint is left in place, periodic monitoring with visual inspection and dust testing should be performed.

*Relocate occupants.* Temporary occupant relocation is generally required to safely conduct lead hazard control activities that may increase dust lead levels. In some cases, it may be feasible to protect occupants during lead control activities by creating barriers, monitoring the work site daily, and, where appropriate, obtaining serial dust lead measurements. In other cases, permanently relocating occupants to lead-safe housing may be the best way of quickly reducing their lead exposure. Examples of situations that might require relocation include a child living in a dwelling that is structurally unsound or a child living in a dwelling where temporary measures to reduce exposure cannot be taken or are ineffective. Case managers and social workers with experience in assisting families with housing difficulties can play a vital role in assessing the needs and desires of the family and arranging such relocation. A registry of lead-safe housing units in a community can also be helpful. When families permanently relocate from a dwelling where lead hazards are identified, measures should be taken to ensure that the hazards are corrected before any other families with young children occupy the dwelling.

### **Enforcement of Laws and Regulations**

Although enforcing laws and regulations pertaining to lead hazards is not part of case management *per se*, it is essential to realizing the long-range goal of reducing those hazards. Individual states should provide health and housing officials with the necessary legal authority to require that timely and effective actions are taken to eliminate lead hazards at properties where children with EBLLs have been identified. Health and housing officials should take all steps necessary to prevent additional or repeated cases of children with EBLLs at one property. In a recent national survey, only 18 states indicated that they have legal authority to order remediation at properties where children with EBLLs reside, with only 14 states reporting that their authority was based on lead-specific state laws or regulations (45). State and local governments should examine their laws, ordinances, and housing codes and their enforcement structure to determine whether they are effective in dealing with identified lead hazards and make changes to ensure that children are protected. At a minimum, legislation or ordinances should include the action

level at which the law applies, procedures for investigation and re-inspection, standards for lead-safe housing, requirements for completing lead hazard control work (including permits, time frames, permissible methods, waste disposal methods, and clearance standards) and enforcement provisions for noncompliance. In addition, states and localities should be encouraged to develop lead-safe housing standards to protect children from exposure and to ensure that older rental housing is safe for children with EBLs. Finally, state and local governments should also ensure that they have the ability and necessary resources to take emergency actions (including cleaning the rental units, stabilizing the paint in them, and relocating the occupants) to protect children from identified lead hazards.

### **Financial Resources for Lead Hazard Control**

Many of the homes in which children with EBLs live are poorly maintained, deteriorated, low-income rental properties. For some economically distressed housing, subsidies and other financial assistance for lead hazard control are required to enable owners to make timely corrections of residential lead hazards. Because resources for addressing lead hazards, particularly in low-income housing, are inadequate in most areas of the country, an increase in resources at the federal, state, and local level should be strongly supported.

In addition, state and local health agencies should develop strong partnerships with local housing and community development organizations, investigate currently available resources for improving low-income housing, and establish mechanisms to apply such resources to lead hazard control in homes of children with EBLs. A detailed discussion and recommendations concerning financing of lead hazard control work can be found in a HUD publication (46). Some examples of current programs providing resources for this purpose are provided in the following paragraphs.

HUD's Lead Hazard Control Grant Program (47) enables state or local agencies to provide grants or loans to property owners for conducting lead hazard control measures in low-income housing. Federal regulations require the timely identification and remediation of lead hazards in federally assisted housing, including rental property, whose owners receive tenant-based assistance (Section 8 housing) (19). This program should create a growing pool of lead-safe housing in the future. Decisions on specific priorities for tenant selection under Section 8 and for public housing have been devolved to state and local public-housing agencies. This local flexibility gives health departments in jurisdictions where lead exposure is a major problem an opportunity to urge that priority for assistance be given to families of children with the highest BLLs who are unable to find or afford lead-safe housing.

State and local governments can use HUD's Community Development Block Grant (CDBG) and HOME Investment Partnership block grant funds to make housing lead-safe. The resources available for state and local block grants under these programs (\$6.4 billion in FY 2000) dwarf the \$60 million available under the Lead Hazard Control Grant Program. Both the CDBG and

HOME programs provide a high degree of flexibility in the use of funds. Indeed, CDBG funds are used by some jurisdictions to support emergency programs dealing with problems such as the breakdown of plumbing or heating systems. A similar approach would be desirable for controlling lead hazards.

State and local governments receiving these block grants must submit a consolidated plan (ConPlan) containing a 5-year strategic plan and a 1-year action plan for their use of these and other available funds. The strategic plan must include actions to evaluate and reduce leaded paint hazards and describe how hazard reduction will be integrated into other housing activities. Evaluating and reducing leaded paint hazards is also a required component of the annual action plan. HUD regulations require that eligible jurisdictions consult with state or local health and child welfare agencies as well as health and social service providers as part of the planning process. State and local health departments with identified lead problems should involve themselves in this planning process to ensure that lead hazard control is a priority for federal CDBG and HOME funding.

### **Recommendations for Future Research**

Technical knowledge concerning the identification and control of lead hazards in homes has advanced greatly over the past several years, resulting in more efficient, safe, and effective environmental management for children with EBLs. Still, prevention efforts could be improved with further work in several areas.

Additional studies are needed to assess the long-term impact of current lead hazard control methods on children's EBLs, especially on levels from 10 to 20 Fg/dL. Available data indicate that these methods are safe and effective (i.e., they do not increase children's BLLs in the short run and they decrease children's exposure to leaded dust). Because BLL changes over time may be influenced by a child's age, the season, and secular trends, as well as by regression to the mean, controlled studies are needed to determine how much of the observed decline in BLLs among children living in these dwellings can be attributed to the interventions. Future research should also evaluate the cost effectiveness of interventions.

Until recently, most residential lead hazard control work and studies have involved children who already had EBLs and presumably relatively high body stores of lead from chronic exposure. The effectiveness of residential lead hazard control in preventing future increases in BLLs among infants and toddlers needs further study.

The level of neighborhood lead exposures appears to make an important contribution to the risk for EBLs among children. Research is needed to examine how community-level lead sources, such as lead from building demolitions, contribute to children's exposure. Finally, the effectiveness of community-level interventions to reduce children's exposure to lead in dwellings and in exterior dust and soil should be further studied.

## References

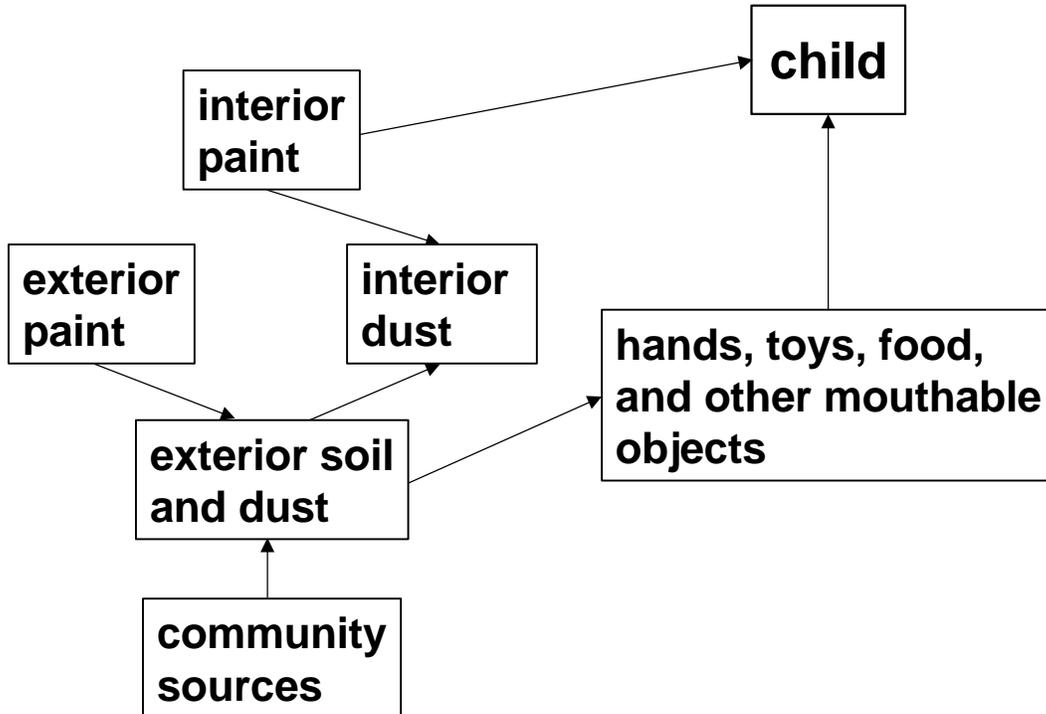
1. CDC. Preventing lead poisoning in young children. Atlanta, Georgia: US Department of Health and Human Services, CDC; 1991.
2. U.S. Environmental Protection Agency. 40 CFR Ch.1 (7-1-00 Edition). Subpart I—Control of lead and copper. Available at <http://www.epa.gov/safewater/regs/cfr141.pdf>. Pages 438-70. Accessed 12/19/01.
3. Department of Housing and Urban Development (HUD). Guidelines for the evaluation and control of lead-based paint hazards in housing. Washington, DC: HUD; 1995. Available at <http://www.hud.gov/lea/learules.html>. Accessed 12/19/01.
4. Consumer Product Safety Commission. Ban of lead-containing paint and certain consumer products bearing lead-containing paint. 42 CFR § 1303.1-5, 1977.
5. Clickner RP, Albright VA, Weitz S. The prevalence of lead-based paint in housing: findings from a national survey. In: Breen JJ, Stroup CR (eds). Lead Poisoning: Exposure, Abatement, Regulation. Boca Raton, FL: CRC Press; 1995:3-12.
6. Pirkle JL, Kaufmann RB, Brody DJ, et al. Exposure of the U.S. population to lead, 1991-1994. *Environ Health Perspect* 1998;106:745-50.
7. McElvaine MD, DeUngria EG, Matte TD, et al. Prevalence of radiographic evidence of paint chip ingestion among children with moderate to severe lead poisoning, St. Louis, Missouri, 1989 through 1990. *Pediatrics* 1992;89:740-2.
8. Bornschein RL, Succop P, Kraft KM, et al. Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In: Hemphill DD (ed). Trace substances in environmental health, XX. Proceedings of University of Missouri's 20th Annual Conference, June 1986. Columbia, MO: University of Missouri; 1987.
9. Lanphear BP, Roghmann KJ. Pathways of lead exposure in urban children. *Environ Res* 1997;74:67-73.
10. Public Law 102-550. Residential Lead-Based Paint Hazard Reduction Act of 1992.

11. U.S. Environmental Protection Agency (EPA). Lead exposure associated with renovation and remodeling activities: Phase III. Wisconsin Childhood Blood Lead Study. Washington, DC: EPA; 1999. EPA 747-R-99-002.
12. Lanphear BP, Weitzman M, Winter NL, et al. Lead-contaminated house dust and urban children's blood lead levels. *Am J Public Health* 1996;86:1416-21.
13. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. *Environ Res* 1998;79:51-68.
14. Clark CS, Bornschein RL, Succop P, et al. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. *Environ Res* 1985;38:46-53.
15. Charney E, Sayre J, Coulter M. Increased lead absorption in inner city children: where does the lead come from? *Pediatrics* 1980;65:226-31.
16. Charney E. Lead poisoning in children: the case against household lead dust. In: Chisolm JJ, O'Hara DM (eds). *Lead absorption in children—management, clinical, and environmental aspects*. Baltimore, MD: Urban & Schwarzenberg; 1982.
17. Lanphear BP, Emond M, Jacobs DE, et al. A side-by-side comparison of dust collection methods for sampling lead-contaminated house dust. *Environ Res* 1995;68:114-23.
18. Department of Housing and Urban Development. Requirements for notification, evaluation and reduction of lead-based paint hazards in federally owned residential property and housing receiving federal assistance; final rule. 24 CFR Part 35. *Federal Register* 1999;64, Number 178: 50139-231.
19. U.S. Environmental Protection Agency. Lead: identification of dangerous levels of lead; final rule. 40 CFR Part 745. *Federal Register* 2001;66(4). Available at <http://www.epa.gov/fedrgstr/EPA-TOX/2001/January/Day-05/t84.htm>. Accessed 12/19/01.
20. U.S. Environmental Protection Agency. Sources of lead in soil: a literature review. Washington, DC: EPA; 1998. EPA 747-R-98-001a.
21. U.S. Environmental Protection Agency. Urban soil lead abatement demonstration project. Volume IV: Cincinnati Report. Washington, DC: US EPA;1993. EPA 600/AP93/001d.

22. U.S. Environmental Protection Agency. National Primary Drinking Water Regulations: Technical Factsheet on Lead. Available at <http://www.epa.gov/safewater/dwh/t-ioc/lead.html>. Accessed 3/28/02.
23. Estimated use of water in the United States in 1995. U.S. Geological Survey. Available at <http://water.usgs.gov/watuse/pdf1995/html/>. Accessed 12/19/01.
24. U.S. Environmental Protection Agency. Air quality criteria document for lead. Research Triangle Park, NC: EPA; 1986. EPA-600/8-83/028aF-dF.
25. Watt GCM, Gilmour WH, Moore MR, et al. Is lead in tap water still a public health problem? An observational study in Glasgow. *BMJ* 1996; 313:979-81.
26. Gulson BL, Giblin AM, Sheehan A, et al. Maintenance of elevated lead levels in drinking water from occasional use and potential impact on blood leads in children. *Sci Total Environ* 1997;205:271-5.
27. Shannon MW, Graef JW. Lead intoxication in infancy. *Pediatrics* 1992;89:87-90.
28. U.S. Environmental Protection Agency (EPA). Review of Studies Addressing Lead Abatement Effectiveness: Updated Edition. Washington DC: EPA; 1998. EPA 747-B-98-001.
29. Staes C, Matte T, Copley CG, et al. Retrospective study of the impact of lead-based paint hazard remediation on children's blood lead levels, St. Louis. *Am J Epidemiol* 1994;139:1016-26.
30. Farfel MR, Chisolm JJ. Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. *Am J Public Health* 1990;80:1240-5.
31. Amitai Y, Graef JW, Brown MJ, et al. Hazards of 'deleading' homes of children with lead poisoning. *Am J Dis Child* 1987;141:758-60.
32. Swindell SL, Charney E, Brown MJ, et al. Home abatement and blood lead changes in children with class III lead poisoning. *Clin Pediatr* 1994;33:536-41.
33. Farfel MR, Chisolm JJ, Rohde CA. The longer-term effectiveness of residential lead paint abatement. *Environ Res* 1994;66:217-21.

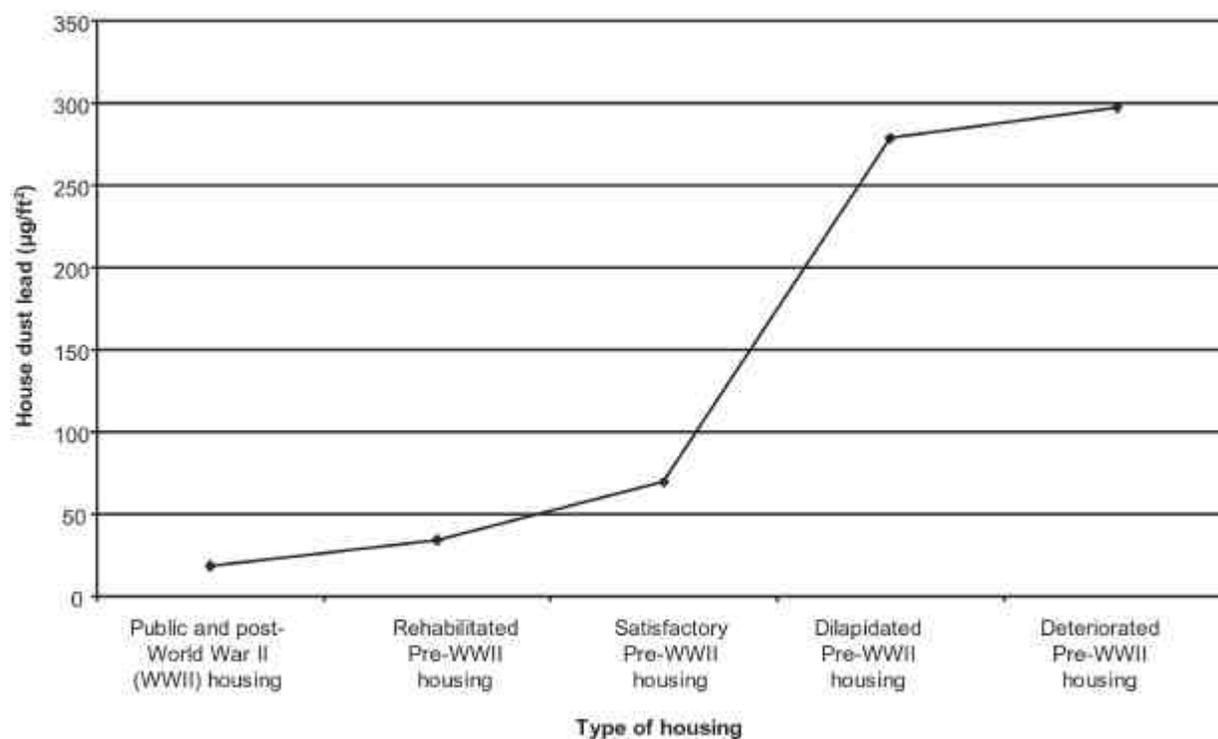
34. U.S. Environmental Protection Agency (EPA). Lead-based paint abatement and repair and maintenance study in Baltimore: Findings based on two years of follow-up. Washington, DC: EPA; 1997. EPA 747-R-97-005.
35. National Center for Lead-Safe Housing. Evaluation of the HUD Lead-Based Paint Hazard Control Grant Program. Fifth interim report. Columbia, MD: National Center for Lead-Safe Housing; 1998.
36. Ashengrau A, Beiser A, Bellinger D, et al. Residential lead-based-paint hazard remediation and soil lead abatement: their impact among children with mildly elevated blood lead levels. *Am J Public Health* 1997;87:1698-702.
37. Rhoads GG, Ettinger AS, Weisel CP, et al. The effect of dust lead control on blood lead in toddlers: a randomized trial. *Pediatrics* 1999;103(3):551-5.
38. Ashengrau A, Hardy S, Mackey P, et al. The impact of low technology lead hazard reduction activities among children with mildly elevated blood lead levels. *Environ Res* 1998;79:41-50.
39. Lanphear BP, Howard C, Eberly S, et al. Primary prevention of childhood lead exposure: a randomized trial of dust control. *Pediatrics* 1999;103:772-7.
40. Weitzman M, Ashengrau A, Bellinger D, et al. Lead-contaminated soil abatement and urban children's blood lead. *JAMA* 1993;269:1647-54.
41. Farrell KP, Brophy MC, Chisholm JJ Jr, et al. Soil lead abatement and children's blood lead levels in an urban setting. *Am J Public Health* 1998;88:1837-9.
42. Rust SW, Kumar P, Burgoon DA, et al. Influence of bone-lead stores on the observed effectiveness of lead hazard intervention. *Environ Res* 1999;81:175-84.
43. U.S. Environmental Protection Agency. Lead; requirements for lead-based paint activities in target housing and child-occupied facilities; final rule. 40 CFR Part 745. *Federal Register* 1996;61:45777-830.
44. Galke W, Clark S, Wilson J, Jacobs D, Succop P, Dixon S, Bornschein B, McLaine P, Chem M. Evaluation of the HUD Lead Hazard Control Grant Program: Early Overall Findings. *Environ Res*. 2001; 86:149-156..

45. Alliance To End Childhood Lead Poisoning, National Center for Lead-Safe Housing. Another Link in the Chain: state policies and practices for case management and environmental investigation for lead-poisoned children. Available for purchase at <<http://www.aeclp.org>>. Accessed 12/27/00.
46. Lead-Based Paint Hazard Reduction and Financing Task Force. Putting the pieces together: controlling lead hazards in the nation's housing. Washington DC: US Department of Housing and Urban Development; July 1995. HUD-1547-LBP.
47. U.S. Department of Housing and Urban Development. Lead-Based Paint Hazard Control Grant Program. Available at <http://www.hud.gov/offices/lead/leagrant.cfm>. Accessed 12/19/01.
48. Roscoe RJ, Gittleman JL, Deddams JA, et al. Blood lead levels among children of lead-exposed workers: a meta-analysis. *Am J Ind Med* 1999;36:475-81.



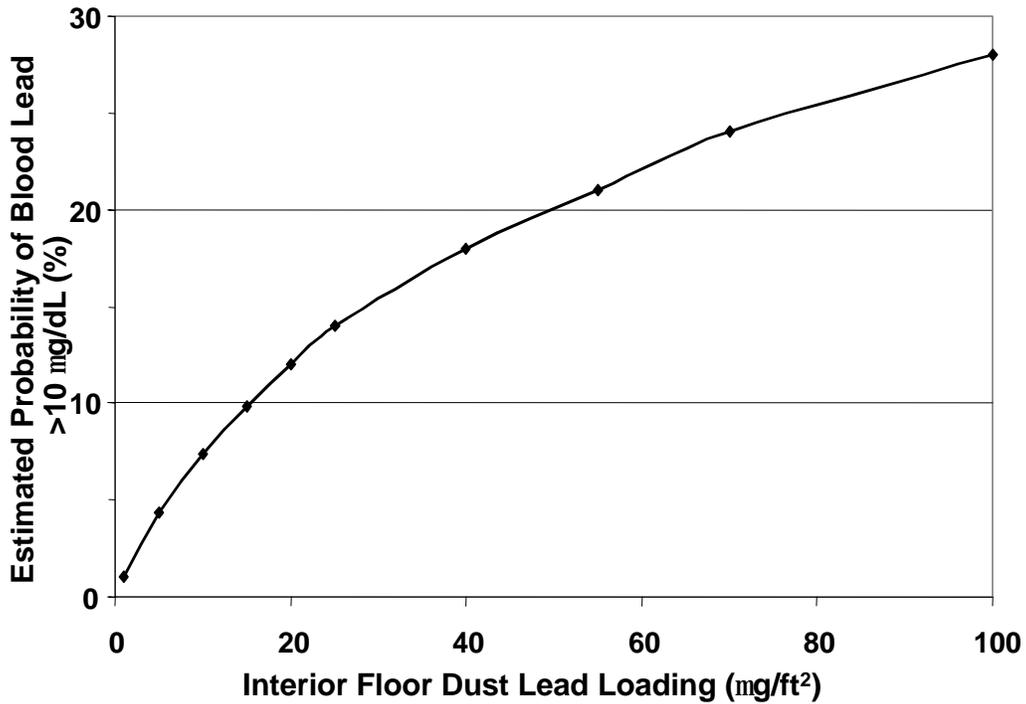
**Figure 2.1. Pathways of Lead Exposure in the Residential Environment**

Sources: Bornschein et al., 1987 (reference 8). Lanphear et al., 1997 (reference 9)



**Figure 2.2. Relationship of Housing Age and Condition to Dust Lead Levels**

Source: Clark et al., 1985 (reference 14)



**Figure 2.3. Relationship of Dust Lead Levels to Blood Lead Levels in Children**

Source: Lanphear et al., 1998 (reference 12). Assumes children are exposed to a soil lead concentration equal to the national average level (72 ppm).

**Table 2.2. Time Frames for Environmental Investigation and Other Case Management Activities According to a Child’s Blood Lead Level<sup>a</sup>**

Blood lead level (Fg/dL) <sup>b</sup>	Actions	Time frame for beginning intervention
10-14	Provide caregiver lead education. Provide follow-up testing. Refer the child for social services if necessary.	Within 30 days
15-19	Above actions, plus: If BLLs persist (i.e., 2 venous BLLs in this range at least 3 months apart) or increase, proceed according to actions for BLLs 20-44.	Within 2 weeks
20-44	Above actions, plus: Provide coordination of care (case management). Provide clinical evaluation and care. <sup>c</sup> Provide environmental investigation and control current lead hazards.	Within 1 week
45-70	Above actions.	Within 48 hours
70 or higher	Above actions, plus hospitalize child for chelation therapy immediately.	Within 24 hours

<sup>a</sup>The ACCLPP encourages programs to develop methods to deliver environmental assessment services to caregivers for children living in high-risk dwellings regardless of the children’s blood lead levels.

<sup>b</sup>Micrograms per deciliter of whole blood measured in a venous sample collected following an elevated screening measurement.

<sup>c</sup>The recommended clinical evaluation is described in Chapter 3, “Medical Assessment and Interventions.”

**Table 2.3. Common Sources of Lead Exposure to Consider in an Environmental Investigation**

(less common sources should be considered where appropriate—see Appendix I)

Source	Standards <sup>a</sup> /Comments	References
Paint	<i>New paint: 600 ppm in dried paint film.</i> <i>Existing paint in structures built prior to 1978: 1 mg/cm<sup>2</sup> or 0.5%</i>  Hazard is increased if leaded paint is deteriorated; present on surfaces subject to friction (e.g., window sashes); or disturbed during maintenance, repair, and renovation, especially during surface preparation for repainting.	3, 4
Interior dust	<i>Floors: 40 micrograms per square foot (µg/ft<sup>2</sup>)<sup>b</sup></i> <i>Interior window sills: 250 µg/ft<sup>2</sup></i> <i>Window troughs: 400 µg/ft<sup>2</sup></i>  Research shows children to be at increased risk for EBLLs at floor lead loading substantially below standard.	18, 19
Residential soil	<i>Bare play area soil: 400 ppm</i> <i>All other soil: 1200 ppm</i>  Dust on paved surfaces in urban areas often contains elevated lead concentrations.	19
Drinking water	<i>First draw from tap (stagnant sample): 15 ppb</i>  Probability of contamination depends on the chemistry of the water. For communities served by public water systems, available data may indicate whether testing is likely to be helpful.	2
Occupations, hobbies	House dust may be contaminated with lead (see above) indirectly via contaminated work clothes, shoes, or hair. Direct contamination can occur from hobbies that generate lead fumes (from heating) or dust.	47

<sup>a</sup>Note: Most standards for lead in environmental media are established on the basis of measurement feasibility or for primary prevention purposes. These standards cannot be used to determine the cause of an EBLL, which requires that environmental measurements be interpreted in the context of a careful exposure history.

<sup>b</sup>EPA has established standards of 40 and 250 µg/ft<sup>2</sup> for hazardous levels of lead in dust on floors and sills, respectively. HUD has established interim standards, 25 and 125 µg/ft<sup>2</sup>, that apply if a more limited assessment known as a "lead hazard screen" is performed. The EPA standard for window troughs is intended only for clearance testing after lead hazard reduction activities.



## **Chapter 3. Medical Assessment and Interventions**

Prepared by James R. Roberts, MD, MPH, and J. Routt Reigart, MD



**Table 3.1. Summary of Recommendations for Children with Confirmed (Venous) Elevated Blood Lead Levels**

Blood Lead Level (µg/dL)				
10-14	15-19	20-44	45-69	≥70
<ul style="list-style-type: none"> <li>• Lead education                             <ul style="list-style-type: none"> <li>S Dietary</li> <li>S Environmental</li> </ul> </li> <li>• Follow-up blood lead monitoring</li> </ul>	<ul style="list-style-type: none"> <li>• Lead education                             <ul style="list-style-type: none"> <li>S Dietary</li> <li>S Environmental</li> </ul> </li> <li>• Follow-up blood lead monitoring</li> <li>• Proceed according to actions for 20-44 µg/dL if:                             <ul style="list-style-type: none"> <li>S A follow-up BLL is in this range at least 3 months after initial venous test</li> </ul> </li> <li style="text-align: center;"><b>or</b></li> <li>S BLLs increase</li> </ul>	<ul style="list-style-type: none"> <li>• Lead education                             <ul style="list-style-type: none"> <li>S Dietary</li> <li>S Environmental</li> </ul> </li> <li>• Follow-up blood lead monitoring</li> <li>• Complete history and physical exam</li> <li>• Lab work:                             <ul style="list-style-type: none"> <li>S Hemoglobin or hematocrit</li> <li>S Iron status</li> </ul> </li> <li>• Environmental investigation</li> <li>• Lead hazard reduction</li> <li>• Neurodevelopmental monitoring</li> <li>• Abdominal X-ray (if particulate lead ingestion is suspected) with bowel decontamination if indicated</li> </ul>	<ul style="list-style-type: none"> <li>• Lead education                             <ul style="list-style-type: none"> <li>S Dietary</li> <li>S Environmental</li> </ul> </li> <li>• Follow-up blood lead monitoring</li> <li>• Complete history and physical exam</li> <li>• Complete neurological exam</li> <li>• Lab work:                             <ul style="list-style-type: none"> <li>Hemoglobin or hematocrit</li> <li>S Iron status</li> <li>S FEP or ZPP</li> </ul> </li> <li>• Environmental investigation</li> <li>• Lead hazard reduction</li> <li>• Neurodevelopmental monitoring</li> <li>• Abdominal X-ray with bowel decontamination if indicated</li> <li>• Chelation therapy</li> </ul>	<ul style="list-style-type: none"> <li>• Hospitalize and commence chelation therapy</li> <li>• Proceed according to actions for 45-69 µg/dL</li> </ul>
<p><b>The following actions are NOT recommended at any blood lead level:</b></p> <ul style="list-style-type: none"> <li style="width: 50%;">• Searching for gingival lead lines</li> <li style="width: 50%;">• Testing of hair, teeth, or fingernails for lead</li> <li style="width: 50%;">• Testing of neurophysiologic function</li> <li style="width: 50%;">• Radiographic imaging of long bones</li> <li style="width: 50%;">• Evaluation of renal function (except during chelation with EDTA)</li> <li style="width: 50%;">• X-ray fluorescence of long bones</li> </ul>				

## Introduction

Case management of children with elevated blood lead levels (EBLLs) requires a different approach from that used in the past. Prior to the development of programs aimed at screening children for EBLLs, lead exposure was generally not detected until a child presented with symptoms of lead toxicity. Neurological findings associated with acute encephalopathy (lethargy, ataxia, seizures, papilledema, and coma) were often the first signs of an EBLL, and children with these symptoms required immediate hospitalization and treatment. Encephalopathy could result from a blood lead level (BLL)  $\geq 70$  Fg/dL and could develop without prior symptoms. Among children with BLLs exceeding 150 Fg/dL, laboratory abnormalities often included phosphaturia, proteinuria, aminoaciduria, glucosuria, and hypophosphatemia (1-3).

Today such presentations are rare. Children with EBLLs usually have BLLs below 30 Fg/dL, and few BLLs exceed 50 Fg/dL. Most children with EBLLs have no symptoms. Case management now focuses on reducing children's exposure to lead and decreasing their BLLs, whether they have symptoms of lead toxicity or not. What follows is a guide to the basic standards and principles of medical case management. It is not intended for use as a complete protocol but rather as a tool for adapting management to local needs and conditions.

## General Principles of Medical Case Management

### *Coordinating Care*

Coordination of care is critical to successful case management. For each child, an individualized plan of follow-up must be devised and implemented. Members of the case management team need to maintain open lines of communication and work together. Case managers and primary care providers (PCPs), in particular, must work collaboratively to ensure proper medical management and follow-up.

### *Conducting Medical Case Management*

Medical case management for children with EBLLs is largely predicated on a secondary prevention model (i.e., intervention after an EBLL has been detected, usually prior to the onset of symptoms). By interrupting the process of lead poisoning through early detection and intervention, case managers working with PCPs can prevent children from dying or suffering severe permanent sequelae of lead toxicity such as persistent seizures and mental retardation (4, 5).

The detrimental effects of EBLLs in the range of 10 to 45 Fg/dL are usually subclinical and may include neurodevelopmental impairment often apparent only at a later age. (See Chapter 4, "Developmental Assessment and Intervention.") Figure 3.1 illustrates the lowest reported BLLs

for some of the effects associated with EBLs. If a child presents without symptoms, the child's PCP and case manager may have trouble convincing the child's caregiver of the importance of suggested interventions. Case managers should manage each child individually, taking into consideration the child's BLL and the ability of caregivers to cooperate and implement interventions.

### *Identifying Children with EBLs*

Screening programs are the main vehicle for identifying children with EBLs. Those found in this manner typically have BLLs from 10 to 30 Fg/dL and present with no abnormalities on routine medical history, physical examination, or laboratory tests (other than their EBL). It is critical that case managers as well as PCPs not equate the absence of clinical symptoms, physical abnormalities, or abnormal laboratory results with an absence of toxicity.

### *Identifying Sources of Lead Exposure*

When evaluating a child with an EBL, case managers must identify the sources of a child's lead exposure. The most common source of lead in children with EBLs is leaded paint. Housing built before 1950 has been shown to be routinely contaminated with lead and to represent a risk for children (6, 7). Contamination of dust or soil occurs when leaded paint chinks or chips, or is subject to friction. (See Chapter 2, "Assessment and Remediation of Residential Lead Exposure.") Other less common sources include lead in water, lead in substances used in caregiver hobbies or occupations, lead in culturally specific substances such as folk remedies, and lead in imported cookware or cosmetics (Appendix 1).

## **Medical History**

### *General Considerations*

Although abdominal pain, vomiting, constipation, change in appetite, and irritability have been described in association with EBLs, they are seldom caused by BLLs less than 40 Fg/dL, and other causes for such symptoms should be sought. Case managers, caregivers, and PCPs may note increased activity among children with BLLs < 45 Fg/dL. However, they should not assume that increased activity is related to the EBL (5, 8).

### *History Taking to Determine Lead Sources*

A child's environmental history can provide information about the child's possible exposure to residential and other sources of lead. It should:

### Chapter 3. Medical Assessment and Interventions

- Include elements specific to the child: ethnic group, caregiver hobbies and occupations, and local lead hazards.
- Be taken from a person who regularly observes the child's activity and behavior.
- Identify all sites where the child spends significant amounts of time.
- Be obtained independently in both office and home settings by the PCP, case manager, or others involved in the child's care.
- Be accompanied by a full environmental investigation for all children with BLLs  $\geq 20$  Fg/dL or two venous BLLs  $\geq 15$  Fg/dL at least 3 months apart.

Taking a history in the child's home allows for direct observation and further in-depth questioning. If a child's BLL remains elevated despite lead hazard reduction, less common sources should be considered. Because a child may be exposed to lead from multiple sources, identifying one source may not be sufficient to eliminate all lead exposure. Repeated history taking by different members of the management team is often required.

Because knowledge about the lead sources in a community and the prevalence of EBLs in specific geographic areas of the community can be useful in determining sources of exposure, the interpretation of a child's environmental history may require consultation with lead experts. Case managers play a crucial role in treating children's EBLs by fostering a multi-disciplinary approach to the environmental evaluation and by coordinating communication among public health officials, PCPs, and caregivers. Table 3.2 outlines suggested questions to ask in determining a child's environmental history. This is only intended to be a guide, and case managers and PCPs are encouraged to tailor this list to local needs.

**Table 3.2. Guidelines for Questions to Ask Regarding a Child's Environmental History**

**Paint and soil exposure**

What is the age and general condition of the residence?  
Is there evidence of chewed or peeling paint on woodwork, furniture, or toys?  
How long has the family lived at that residence?  
Have there been recent renovations or repairs in the house?  
Are there other sites where the child spends significant amounts of time?  
What is the character of indoor play areas?  
Do outdoor play areas contain bare soil that may be contaminated?  
How does the family attempt to control dust/dirt?

**Relevant behavioral characteristics of the child**

To what degree does the child exhibit hand-to-mouth activity?  
Does the child exhibit pica?  
Are the child's hands washed before meals and snacks?

**Exposures to and behaviors of household members**

What are the occupations of adult household members?  
What are the hobbies of household members? (Fishing, working with ceramics or stained glass, and hunting are examples of hobbies that involve risk for lead exposure.)  
Are painted materials or unusual materials burned in household fireplaces?

**Miscellaneous questions**

Does the home contain vinyl mini-blinds made overseas and purchased before 1997?  
Does the child receive or have access to imported food, cosmetics, or folk remedies?  
Is food prepared or stored in imported pottery or metal vessels?

### *Paint and soil exposure\**

Case managers should consider the following elements in assessing a child's exposure to lead in paint or soil:

- *Age and condition of housing:* Pre-1950 housing that is in poor condition poses the greatest risk for children (6,7). Housing built from 1950 through 1978 may also contain leaded paint, although the concentration of lead in paint was lower during this period than previously. The condition of the home is important: deterioration of lead-painted surfaces markedly increases the risk to children (9-12); water damage from roofing and plumbing can increase peeling and chipping of paint; open windows with debris in the window well provide an additional source of exposure.
- *Duration of a child's habitation at that site and whether the child has moved recently:* Because leaded dust generally causes EBLs only after a significant duration of exposure, children with EBLs who recently moved into their current residence may have been exposed to lead at their prior residence. Conversely, those who have moved from a lead-free or lead-safe environment to a more risky one are more likely to need ongoing active surveillance.
- *Whether the residence has been renovated:* Any disturbance of leaded paint, including repairs, renovation, or improper lead abatement can result in generation of leaded dust.
- *Other possible exposure locations:* It is important to document locations where a child with an EBL spends considerable periods of time, such as relatives' or babysitters' houses. Such locations may be a source of exposure and therefore should be subjected to the same scrutiny as the primary habitation.
- *Character of indoor play areas:* Do play areas have lead hazards? Are there window wells, windowsills, or other painted edges near indoor play areas? These potential sources of leaded dust or paint may be subject to chewing or mouthing behaviors typical of young children.
- *Soil exposure:* In outdoor areas, the most heavily contaminated soil is adjacent to the house, particularly in the drip line. Soil contamination may also occur from industrial sites and past automobile emissions, particularly along heavily traveled thoroughfares.
- *Dust and dirt control:* Children's BLLs declined when a team of professionals thoroughly and regularly cleaned the children's homes (13). At present, there is no evidence that routine house cleaning by family members or frequent and thorough hand washing decreases BLLs. However, because leaded dust is a primary contributor to EBLs (14-17), strict attention to

---

\*See Chapter 2, "Assessment and Remediation of Residential Lead Exposure," for a detailed discussion of paint and soil exposure.

house cleaning and preventing dust accumulation may be protective. (See Chapter 6, “Educational Interventions for Caregivers.”)

#### *Relevant child behaviors*

While pica has long been a known risk factor for EBLs, typical hand-to-mouth activity during the toddler years is a more frequent cause of lead ingestion (18, 19). Although no supporting data are available, frequent hand washing may help lower EBLs of young children.

#### *Caregiver exposures and at-risk behaviors*

Household members who work in lead-contaminated environments or participate in certain hobbies can bring lead into the home on their clothing or shoes (20). It is important to ask such household members whether they regularly shower and change clothes and shoes after these activities. Caregivers may also introduce airborne lead into the home by burning lead-contaminated materials in an indoor fireplace. (See Appendix I for a detailed discussion of caregiver exposures.)

#### *Miscellaneous*

*Water:* When no other source of lead is found, the water supply should be considered. (See Chapter 2, “Assessment and Remediation of Residential Lead Exposure.”) While public water systems must monitor, and, if necessary, treat tap water for elevated lead levels, regulations governing lead in drinking water do not apply to households supplied by private wells. Municipal water companies and private industrial laboratories can advise case managers and caregivers on how to collect and process water. In areas where there are no known hazardous water-supply lines, lead contamination of the water may occur within the home from lead solder in plumbing fixtures or from fixtures made of lead-containing alloys. Contamination is increased when the water is relatively acidic (pH < 6.5). Moore found that water lead levels and the subsequent BLLs of children drinking such water substantially decreased when the pH of drinking water was raised to > 8.5. He also found that low mineral content increased lead contamination in water, but not as greatly as acidity (21). Water that is hot or has been stationary in pipes overnight contains more lead than freely running cold water. Lead contamination decreases with the aging of home plumbing, particularly if the water supply is alkaline or contains significant calcium or other mineral deposits (22, 23).

*Mini-blinds:* Imported vinyl mini-blinds may contain lead and result in exposure. (See Appendix I.)

*Cultural practices:* Specific ethnic groups may use imported folk remedies, cosmetics, food, or cookware contaminated with lead. Practices resulting in lead exposure from these sources are

often localized, and determining children's risk from such practices requires knowledge about a specific group's cultural habits. Caregivers may be reluctant to admit using some of these items, and it is important not to put them on the defensive when taking the history. (See Appendix I.)

*Newly identified sources:* In the past, various items have emerged as lead hazards, often first presented through the news media. Case managers and PCPs should be cognizant of recent media and Consumer Product Safety Commission reports that may be relevant to children with whom they have contact (see <http://www.cpsc.gov>).

### Physical Examination

Children evaluated as a consequence of an EBLL found by screening most often have no physical findings specific for lead toxicity. Gingival lead lines, although often stressed during medical training, are rarely seen in clinical practice and are of no use in the diagnosis and management of children with EBLs. Pallor, papilledema, and other neurologic findings suggestive of acute encephalopathy would not be expected.

A thorough evaluation of all children with BLLs  $\geq 20$  Fg/dL is recommended for three reasons. First, it will allow PCPs to ascertain whether children with such EBLs have any findings suggestive of encephalopathy. The BLL threshold for encephalopathic findings is believed to be 70 Fg/dL, although encephalopathy is usually associated with much higher BLLs (1, 3). Second, it will allow PCPs to assess whether children with EBLs are engaging in at-risk behaviors such as pica and hand-to-mouth activity. Finally, it will allow PCPs to identify behavioral and neurodevelopmental disorders, such as distractibility, aggression, or speech delay. If a child has any of these findings, regardless of their etiology, the case manager or PCP should, when appropriate, refer the child for a further evaluation. (See Chapter 5, "Developmental Assessment and Interventions.")

### Laboratory and Imaging Evaluation

Among asymptomatic children, most clinical laboratory results other than BLLs will be normal and therefore will not be of assistance in case management. However, all children should have a hemoglobin or hematocrit test performed, as anemia is associated with EBLs. That iron deficiency rather than lead is the cause of such anemia does not diminish the need for follow-up (24, 25). PCPs may wish to assess children's iron stores by one or more of a variety of laboratory tests. Iron deficiency may delay children's neurodevelopment independently of the effects of lead (26). Because basophilic stippling is not specific for lead toxicity, a peripheral blood smear is of no use in the hematologic evaluation.

The inhibition of heme synthesis leads to the accumulation of excess porphyrins, particularly protoporphyrin IX in red cells. The fluorescence of these porphyrins has led to the development of methods to detect extracted porphyrins—free erythrocyte protoporphyrin (FEP) or erythrocyte

porphyrin (EP) testing—and to the observation of zinc protoporphyrin (ZPP) in red cells. Because the relationship between the results of these tests and BLLs is log-linear, these tests can be used to evaluate and follow children with very high BLLs. However, the results are confounded by concomitant iron deficiency and show poor correlation with BLLs  $\leq 25$  Fg/dL (27). Therefore, EP tests should be used infrequently except in evaluating children with BLLs well above 25 Fg/dL whose BLLs do not show a steady decline in response to medical and environmental interventions. In such situations, these measures may assist PCPs in differentiating BLL rebound after treatment from the effects of re-exposure.

While very high BLLs have been associated with serious to severe renal tubular dysfunction (2, 28), there is no evidence to support routinely evaluating the renal status of children with presymptomatic BLLs. However, if potentially nephrotoxic chelating agents such as EDTA are to be used in treatment, renal function testing is appropriate prior to and during therapy.

Abdominal radiographs may be useful in determining whether children are currently ingesting lead-contaminated non-food items, including paint chips. They are particularly useful when children have an unexpected acute rise in BLL or are not responding to case management as expected. Long-bone films for the presence of growth arrest lines (“lead lines”) may be of interest but rarely provide information useful for a child’s case management (29). Lead lines are not present unless BLLs exceed 50 Fg/dL and are indicative of chronic exposure (30). Also of no documented utility in the management of children with EBLLs are hair (31), fingernail, and tooth (dentin) lead measurements. Hair and fingernails are subject to external contamination, which makes the results of lead tests on them uninterpretable.

A few studies have demonstrated alteration of neurophysiologic function (e.g., postural sway, auditory evoked potentials, nerve conduction) with BLLs observed today (32-35). However, further research is needed to define normative standards and determine inter-individual variation and clinical significance. Until then, such measures are of little use in the diagnosis or management of an individual child.

X-ray fluorescence of long bones uses a radioactive source to provide noninvasive estimation of lead in bone (36). At the present time, it should be considered a research tool to be used only to characterize groups of children in epidemiological studies. As with the neurophysiologic methods discussed previously, it is insufficiently standardized, and results show significant inter-laboratory variation.

### **Chelation Therapy**

While chelation therapy is considered a mainstay in the medical management of children with BLLs  $> 45$  Fg/dL, it should be used with caution. Primary care providers should consult with an expert in the management of lead chemotherapy prior to using chelation agents. If unaware of a center with such expertise, PCPs should contact their local or state lead poisoning prevention program, local poison control center, or the Lead Poisoning Prevention Branch at

CDC (404-498-1420) for the names of accessible experts. A child with an EBLL and signs or symptoms consistent with encephalopathy should be chelated in a center capable of providing appropriate intensive care services!

Controversy exists as to the appropriate level at which to initiate chelation therapy, and which drugs are most appropriate. Succimer treatment of young children with BLLs < 45 Fg/dL lowered their BLLs but failed to improve their neurodevelopmental test scores (37). (See Chapter 5, “Developmental Assessment and Interventions.”) Chelation therapy with succimer is addressed in a document on pharmaceutical agents in the treatment of lead poisoning (38).

If oral outpatient chelation therapy is undertaken, the case manager should ensure that caregivers adhere to the prescribed dosing schedule and should serve as the liaison between the medical community and the child’s caregiver. Treatment should occur in a lead-safe environment.

### Monitoring Blood Lead Levels

Measurement of BLLs is the main method of determining whether significant absorption of lead has occurred, how urgently intervention is needed, and how successful case management has been. When a child’s BLL does not fall within a reasonable amount of time, it is the responsibility of the case manager and other team members to determine the cause of failure. The rate of BLL decrease can depend on both the amount of lead in the child’s body and the duration of the BLL elevation. A course of chelation therapy with succimer results in a rapid fall in BLL after 1 week of treatment. However, BLLs of those treated rebound after treatment ends, and by approximately 7 weeks after an initial course of therapy, BLLs of treated patients may reach almost 75% of prechelation levels (39). CDC recommends rechecking children’s BLLs 7 to 21 days after completion of chelation therapy (40). A continuing increase in children’s BLLs above the rebound level during the follow-up period may indicate continuing or possibly increased exposure to lead and definitely indicates a need for further environmental investigation. Common causes of rising BLLs include failure to address hazards in the child’s environment, improper environmental lead abatement techniques, and continued use of imported pottery, cosmetics, or folk medicines that are contaminated with lead. However, medical conditions resulting in bed rest or similar immobilization (41), or in acidosis (42), can cause children’s BLLs to rise unexpectedly, or fail to fall.

#### *Confirmation of BLL by Venous Sample*

Any screening BLL above 10 Fg/dL must be confirmed with a venous sample. The time frame for confirmation depends upon the initial BLL (Table 3.3). In general, the higher the screening BLL, the sooner the confirmatory test. However, if a child is less than 12 months old,

or if there is reason to believe that the BLL is rising rapidly, an earlier diagnostic confirmation may be indicated.

**Table 3.3. Recommended Schedule for Obtaining a Confirmatory Venous Sample**

Screening test result (µg/dL)	Perform a confirmation test within:
10-19	3 months
20-44	1 week-1 month <sup>a</sup>
45-59	48 hours
60-69	24 hours
> 70	Immediately as an emergency lab test

<sup>a</sup>The higher the BLL on the screening test, the more urgent the need for confirmatory testing.

Table adapted from: *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials*. Atlanta: CDC; 1997.

*Follow-Up Venous Blood Lead Testing*

Medical management includes follow-up blood lead testing. Table 3.4 presents the suggested frequency of follow-up tests. This table is to be used as guidance. Case managers and PCPs should consider individual patient characteristics and caregiver capabilities and adjust the frequency of follow-up tests accordingly.

**Table 3.4. Schedule for Follow-Up Blood Lead Testing<sup>a</sup>**

Venous blood lead level (µg/dL)	Early follow-up (first 2-4 tests after identification)	Late follow-up (after BLL begins to decline)
10-14	3 months <sup>b</sup>	6-9 months
15-19	1-3 months <sup>b</sup>	3-6 months
20-24	1-3 months <sup>b</sup>	1-3 months
25-44	2 weeks-1 month	1 month
> 45	As soon as possible	Chelation with subsequent follow-up

<sup>a</sup>Seasonal variation of BLLs exists and may be more apparent in colder climate areas. Greater exposure in the summer months may necessitate more frequent follow ups.

<sup>b</sup>Some case managers or PCPs may choose to repeat blood lead tests on all new patients within a month to ensure that their BLL level is not rising more quickly than anticipated.

### Monitoring the Child

#### *Managing a Child's Nutrition*

Although the effectiveness of nutritional interventions has not been established, the following recommendations are common sense and are appropriate advice for all children, including those with EBLs:

- Consume adequate amounts of bioavailable calcium and iron.
- Consume at least two servings daily of foods high in vitamin C, such as fruits, vegetables, and juices.
- Eat in areas that pose a low risk for lead exposure; for example, at a table rather than on the floor.
- Participate in the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) if the family is eligible.

Dietary information is discussed in detail in Chapter 4, “Nutritional Assessment and Interventions.”

#### *Educating Caregivers*

Educating caregivers is an important part of case management. Caregivers need to understand EBLs and the risks that an EBL poses to their child, what they can do to eliminate their child's exposure to lead, and the importance of follow-up. It is important to not overburden caregivers and to provide them with understandable information and manageable interventions. (See Chapter 6, “Educational Interventions for Caregivers,” for a detailed discussion.)

#### *Monitoring a Child's Developmental Progress*

Follow-up also requires attention to the behavioral sequelae of EBLs. Neurodevelopmental monitoring should continue long after a case meets BLL closure criteria, as many deficits will not manifest themselves until after a child starts school. Because developmental history and testing at the time of an EBL usually will not identify lead-caused problems, a child's EBL history should be part of his or her permanent medical record. A referral for testing of intellectual and behavioral performance, whether or not related to EBLs, should be made if indicated. (See Chapter 5, “Developmental Assessment and Interventions,” for details.) The PCP and case manager should be intimately involved with any educational and behavioral interventions, in consultation with developmental and behavioral experts.

### *Monitoring Caregiver Compliance with Follow-Up Measures*

For many reasons, caregivers may have trouble adhering to follow-up measures. Case managers, PCPs, and other members of the case management team must be careful not to blame the caregivers but should continue to make them aware that follow-up is for the benefit of the child. Caregivers may have trouble appreciating the importance of follow-up for asymptomatic children. Many caregivers have problems with basic needs such as transportation, food, or paying monthly bills. Therefore, it is important to limit interventions to those most likely to benefit the child while being within the capabilities of the caregiver. Punitive interventions, such as referring children to protective services, should be done as a last resort, when all more constructive approaches have been exhausted. Members of the case management team should always remember that virtually all caregivers are doing the best they can for their children and should be assisted in their efforts.

### **Recommendations for Future Research**

- Develop improved screening methods through the use of geographic information systems.
- Compare succimer with edetate calcium disodium in the treatment of children with EBLLs.
- Assess the benefits of hand washing and other low-intensity educational interventions.
- Identify primary prevention interventions that are effective in reducing lead exposure.
- Establish an effective approach to referral and intervention for children with EBLLs who are suspected of having developmental or behavioral problems.
- Evaluate the effectiveness of dietary interventions.
- Develop and refine additional screening questions for an effective “environmental checklist.”
- Determine an appropriate endpoint for completion of chelation therapy.

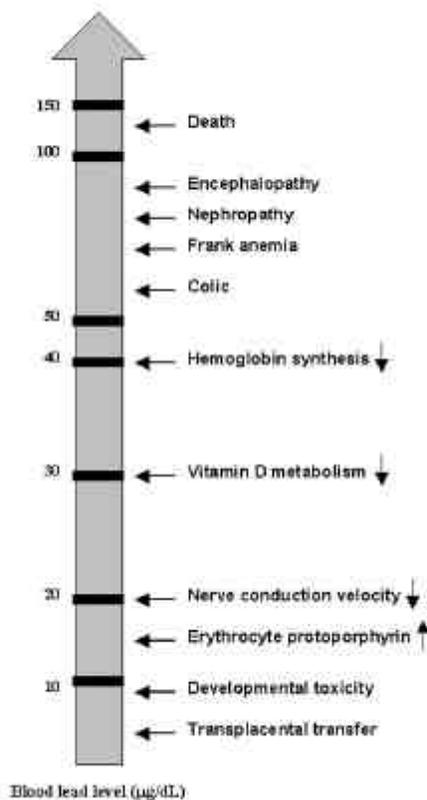
## References

1. Chisolm JJ, Harrison HE. The treatment of acute lead encephalopathy in children. *Pediatrics* 1957;19:2-20.
2. Chisolm JJ. Aminoaciduria as a manifestation of renal tubular injury in lead intoxication and a comparison with patterns of aminoaciduria seen in other diseases. *J Pediatr* 1962;60:1-17.
3. Chisolm JJ. The use of chelation agents in the treatment of acute and chronic lead intoxication in childhood. *J Pediatr* 1968;73:1-38.
4. Byers RK, Lord EE. Late effects of lead poisoning on mental development. *Am J Dis Child* 1943;66:471-94.
5. Perlstein MA, Attala R. Neurologic sequelae of plumbism in children. *Clin Pediatr* 1966;5:292-98.
6. Sargent JD, Bailey A, Simon P, et al. Census tract analysis of lead exposure in Rhode Island children. *Environ Res* 1997;74:159-68.
7. CDC. Update: blood lead levels—United States, 1991-1994. *MMWR* 1997;46:141-6.
8. Committee on Measuring Lead in Critical Populations. *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations*. National Research Council Commission on Life Sciences, Washington, DC: National Academy Press, 1993. Available at <http://books.nap.edu/books/030904927X/html/R1.html#>. Accessed 12/19/01.
9. Chisolm JJ, Mellits ED, Quaskey SA. The relationship between the level of lead absorption in children and the age, type, and condition of housing. *Environ Res* 1985;38:31-45.
10. Clark CS, Bornschein RL, Succop S, et al. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. *Environ Res* 1985;38:46-53.
11. Sargent JD, Brown MJ, Freeman JL, et al. Childhood lead poisoning in Massachusetts communities: its association with sociodemographic and housing characteristics. *Am J Public Health* 1995; 85:528-34.

12. Bronson MA, Tilden RL, Renier CM. Community-based screening for childhood lead poisoning. Identification of risk factors and susceptible populations in Duluth. *Minn Med* 1999;82:25-9.
13. Rhoads GG, Ettinger AS, Weisel CP, et al. The effect of dust lead control on blood lead in toddlers: a randomized trial. *Pediatrics* 1999;103(3):551-5.
14. Charney E, Sayre J, Coulter M. Increased lead absorption in inner city children: where does the lead come from? *Pediatrics* 1980;65:226-31.
15. Bornschein RL, Succop P, Kraft KM, et al. Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In Hemphill DD (ed). *Trace Substances in Environmental Health, XX. Proceedings of University of Missouri's 20th Annual Conference, June 1986. University of Missouri, Columbia, Missouri, 1987.*
16. Lanphear BP, Burgoon DA, Rust SW, et al. Environmental exposures to lead and urban children's blood lead levels. *Environ Res* 1998;76:120-30.
17. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. *Environ Res* 1998a;79:51-68.
18. Freeman NC, Ettinger A, Berry M, et al. Hygiene- and food-related behaviors associated with blood lead levels of young children in lead contaminated homes. *J Expo Anal Environ Epidemiol* 1997;7:103-18.
19. Duggan MJ. Contribution of lead in dust to children's blood lead. *Environ Health Perspect* 1983;50:370-81.
20. Roscoe RJ, Gittleman J, Deddens JA, et al. Blood lead levels among children of lead-exposed workers: a meta-analysis. *Am J Indust Med* 1999;36:475-481.
21. Moore MR. Influence of acid rain upon water plumbosolvency. *Environ Health Perspect* 1985;63:121-6.
22. Agency for Toxic Substances and Disease Registry. *The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress. Atlanta, Georgia: US Department of Health and Human Services, Public Health Service; 1988.*

23. Environmental Protection Agency. Reducing lead in drinking water: A Benefit Analysis. Washington, DC: US Environmental Protection Agency, Office of Policy Planning and Evaluation; 1986. EPA-230-09-86-019.
24. Serwint JR, Damokoab A, Berger OG, et al. No difference in iron status between children with low and moderate lead exposure. *J Pediatr* 1999;135:108-10.
25. Carvalho FM, Barreto ML, Silvany-Neto AM, et al. Multiple causes of anaemia amongst children living near a lead smelter in Brazil. *Sci Total Environ* 1984;35:71-84.
26. Lozoff B, Jimenez E, Wolf AW. Long-term developmental outcome of infants with iron deficiency. *N Engl J Med* 1991;325:687-94.
27. McElvaine MD, Orbach HG, Binder S, et al. Evaluation of the erythrocyte protoporphyrin test as a screen for elevated blood lead levels. *J Pediatr* 1991;119:548-50.
28. Goyer RA. The renal tubule in lead poisoning. Mitochondrial swelling and aminoaciduria. *Lab Invest* 1968;19:71-7.
29. Sachs HK. The evolution of the radiologic lead line. *Radiology* 1981;139:81-5.
30. Blickman JG, Wilkinson RH, Graef JW. The radiologic "lead band" revisited. *Am J Roentgenology* 1986;146:245-7.
31. Esteban E, Rubin CH, Jones RL, et al. Hair and blood as substrates for screening children for lead poisoning. *Arch Environ Health* 1999;54:436-40.
32. Bhattacharya A, Shukla R, Dietrich K, et al. Effect of early lead exposure on children's postural balance. *Develop Med Child Neurol* 1995;37:861-78.
33. Otto D, Robinson G, Baumann S, et al. Five-year follow-up study of children with low-to-moderate lead absorption: electrophysiological evaluation. *Environ Res* 1985;38:168-86.
34. Osman K, Pawlas K, Schutz A, et al. Lead exposure and hearing effects in children in Katowice, Poland. *Environ Res* 1999;80:1-8.
35. Landrigan PJ, Baker EL, Feldman RG, et al. Increased lead absorption with anemia and slowed nerve conduction in children near a lead smelter. *J Pediatr* 1976;89:904-10.

36. Hu H, Milder FL, Burger DE. X-ray fluorescence: issues surrounding the application of a new tool for measuring burden of lead. *Environ Res* 1989;49:295-317.
37. Rogan WJ, Dietrich KN, Ware JH, et al. Succimer chelation and neuropsychological development in lead-exposed children. *N Engl J Med* 2001;344:1421-6.
38. American Academy of Pediatrics Committee on Drugs. Treatment guidelines for lead exposure in children. *Pediatrics* 1995;96:155-60.
39. Treatment of Lead-Exposed Children (TLC) Trial Group. Safety and efficacy of succimer in toddlers with blood lead levels of 20-44 Fg/dL. *Pediatr Res* 2000;48:593-9.
40. CDC. Preventing lead poisoning in young children. Atlanta, Georgia: US Department of Health and Human Services, CDC; 1991.
41. Markowitz ME, Weinberger HL. Immobilization-related lead toxicity in previously lead-poisoned children. *Pediatrics* 1990;86:455-7.
42. Aub JC, Fairhall LT, Minot AS, et al. Lead poisoning. *Medicine* 1925;4:1-250.



**Figure 3.1. Lowest Reported Effect Levels of Inorganic Lead in Children**

Source: Preventing Lead Poisoning in Young Children, Centers for Disease Control and Prevention; 1991.

## **Chapter 4. Nutritional Assessment and Interventions**

Prepared by James Sargent, MD



**Table 4.1. Summary of Recommendations for Nutritional Assessment and Interventions**

Nutritional measures have not yet been proven to have a clinically important impact on elevated blood lead levels (EBLLs) in children. However, children with EBLLs are often at risk for poor nutrition, and their caregivers should receive nutritional counseling to help these children obtain a well-balanced and age-appropriate diet.

**Assessment**

- Test children at risk for anemia (e.g., those from low income, migrant, or recently arrived refugee families, or those qualifying for the Special Supplemental Nutrition Program for Women, Infants, and Children [WIC]).
  - Between ages 9 and 12 months
  - 6 months later
  - Annually from ages 2 to 5 years
- Evaluate the diet of children at risk for anemia, paying particular attention to dietary iron, vitamin C, and calcium.

**Interventions**

- Evaluate the WIC eligibility of children with EBLLs and ensure their access to this program if eligible.
- Advise caregivers to provide children with an adequate intake of iron-containing foods. Recommend that they:
  - Introduce pureed meats as soon as the child is developmentally ready.
  - Provide one serving of lean red meat per day to older children.
  - Provide supplements only under the supervision of a physician or nutritionist and only when anemia or iron deficiency is documented.
- Encourage caregivers to provide children with adequate intake of vitamin C-containing foods. Recommend that they:
  - Provide two servings of fruit juices or fruits per day.
  - Provide supplements only under the supervision of a physician or nutritionist.
- Encourage caregivers to provide children with adequate intake of calcium (500 mg/day @ 1-3 years; 800 mg/day @ 4-8 years). Recommend that they:
  - Provide two servings per day of dairy products or other calcium-rich foods.
  - Provide supplements only under the supervision of a physician or nutritionist.

**Always keep recommended interventions within the ability of the caregiver to implement them.**

### Introduction

While the assessment and remediation of lead sources should be the top priority for the management of children with EBLs, nutritional interventions may also be beneficial (1-4). This chapter evaluates the evidence supporting commonly used nutritional interventions, makes recommendations, and suggests an agenda for future clinical research. In evaluation studies on the effects of various nutritional interventions on EBLs, we considered both the design of the studies and the effectiveness of the interventions. Because of a lack of randomized, controlled clinical trials of nutritional interventions among children with EBLs, most recommendations are based on generally accepted nutritional principles, as well as on the results of adult human, animal, or cross-sectional studies, with greater weight being given to those studies with designs that are less subject to bias and inferential error.

### Nutritional Interventions: Summary of the Evidence

#### *Iron*

*Are children at higher risk for EBLs also at higher risk for iron deficiency?*

Despite declines in the prevalence of iron deficiency over the past 30 years with the routine supplementation of infant foods with iron, iron deficiency remains the most common nutritional deficiency in infants and young children (5). Data from the Third National Health and Nutrition Examination Survey (NHANES III) indicate that in 1988-94, 9% of toddlers aged 1 to 2 years were iron deficient (6). As with EBLs, young age, poor nutrition, and low socioeconomic status are associated with iron deficiency. In addition, some reports suggest that iron deficiency in young children is associated with pica, a risk factor for lead ingestion (7-10). In short, many nutritional and behavioral factors associated with iron deficiency may also be found in children with EBLs.

*Is iron deficiency associated with EBLs?*

Because animal studies and other evidence suggest that iron deficiency and EBLs are associated, the Centers for Disease Control and Prevention (CDC) in the past has recommended providing an iron-rich diet for all children with EBLs, evaluating children with blood lead levels (BLLs)  $\geq 20$  Fg/dL for iron deficiency, and treating iron deficiency if present (11). However, the association between EBLs and iron deficiency in children is not well defined. It is unknown whether this relationship is causal and operating through a nutritional or physiological mechanism or whether it is merely the result of shared risk factors. Prospective studies of children with and without iron deficiency living in lead-contaminated environments are difficult to conduct since treatment is indicated for both iron deficiency and EBLs. Therefore, most studies that address this question are case series, case-control studies, or cross-sectional surveys. Though the results of most early studies suggested that iron deficiency is more common among

children with EBLLs, these studies can be criticized for one or more of the following reasons: 1) they lacked an appropriate comparison group; 2) they screened for EBLLs with erythrocyte protoporphyrin, an indicator of both lead and iron status; or 3) they failed to adjust results for factors associated with both EBLLs and iron deficiency, including age and socioeconomic status.

Of the four studies we found that avoided these methodological problems, two reported a positive association between iron deficiency and BLLs in children and two suggested no association. Each study used different definitions of iron status and EBLL. Of the studies finding a positive association, one suggested iron deficiency in children was associated with a 60% increased risk for a BLL  $\geq 10$  Fg/dL after adjustments for children's age, hemoglobin level, and insurance status (12). The second, a study on dietary iron, found that children in the highest quartile for iron intake were at a significantly lower risk of having a BLL  $\geq 15$  Fg/dL, after adjustments for maternal education, children's lead exposure, age, and total caloric intake (odds ratio 0.4, 95% confidence interval, 0.2-0.9) (13). Of the studies that indicated no association, one was conducted among black children 11 to 33 months of age who resided in urban areas, and the results may not be applicable to other groups (14). In that study, the prevalence of iron deficiency was 7% among children with BLLs 20 to 44 Fg/dL and 5% among children with BLLs  $\leq 10$  Fg/dL. The other study, using NHANES III data and published only in abstract form, reported no association between iron deficiency (with or without anemia) and BLLs  $\geq 10$  Fg/dL after adjusting for age of housing; education of household head; and children's age, race, and poverty status, and intake of fat, calcium, and vitamin C (15).

During the 1980s, some prospective studies of children's BLLs and development gathered data on the children's iron status as well; most of the data from these studies are unpublished. Bornshein (personal communication, University of Cincinnati Medical Center, November 1988) found that Cincinnati children who became more iron deficient (as evidenced by increased total iron-binding capacity) had greater increases in BLLs, but McMichael et al. (16) and Bellinger (personal communication, Harvard Medical School, March 1989) found no association between BLLs or changes in BLLs and initially low serum ferritin levels. Neither study, however, adjusted for children's use of iron supplements or for other factors. If children with initially low serum ferritin levels received iron supplements, this could have affected the association between initial low serum ferritin levels and changes in BLLs.

#### *Does iron deficiency increase absorption of lead?*

Some animal studies suggest mechanisms by which iron levels could affect lead retention. For example, one study of rats indicates that iron and lead absorption may be mediated by common carriers and that ingested iron decreases the absorption of lead in a dose-related manner, presumably by competitive inhibition of the carrier protein (17). Moreover, iron-deficient animals have significantly higher rates of lead absorption than iron-replete ones (18). However, the effect of iron levels and iron supplementation on radiolabeled lead retention in humans is controversial, with at least one study finding an effect (19) and at least one not (20). In their latest

study, Watson and colleagues (19) found a correlation between lead and iron absorption; however, the mean lead-absorption value for iron-deficient subjects was not significantly different from the value for those who were not iron deficient. No data are available for children.

### *Does iron deficiency enhance the adverse effect of lead on development?*

Although iron deficiency may not modify children's risk for lead exposure or retention, iron deficiency and EBLs have similar toxicity profiles. Both result in a lower production in heme; this is manifested clinically by higher erythrocyte protoporphyrin levels in children with EBLs and iron deficiency than in those children with either condition alone (21). More importantly, both iron deficiency and EBLs have a deleterious effect on cognitive development. This raises the possibility that the neurodevelopmental effects of lead may be more severe when iron deficiency is also present. However, there is no evidence to suggest that iron deficiency modifies the neurodevelopmental effect of EBLs. Instead, in one study comparing the cognitive development of children living near a lead smelter with that of those in a nearby town in Yugoslavia, researchers found the neurodevelopmental effects of iron deficiency and EBLs to be independent of one another (22).

### *Does iron supplementation have an effect on BLLs?*

There is evidence to suggest that iron-sufficient children excrete more urinary lead when chelated with EDTA, although the increase is small and probably not clinically significant. In addition, after iron administration, chelation-induced lead excretion increased among patients with iron deficiency. The study in which this occurred, however, did not address the effect of iron deficiency on BLLs and lead excretion in the absence of chelation (23). In a study conducted by Ruff and colleagues (24), children with EBLs and iron deficiency were given iron supplements, whereas children with EBLs but no iron deficiency were not. The children who were iron deficient and received supplements had only half the reduction in BLLs of the children who were iron sufficient and did not. However, it is not clear whether this was due to the effect of iron supplementation on hemoglobin concentration or to another factor affecting lead biokinetics. The problem with using BLL as an indicator of body burden of lead in iron studies is that, because 99% of lead in blood is intraerythrocytic, any intervention that causes a significant increase in the hemoglobin concentration will similarly affect the BLL.

### *Summary*

Although iron may help prevent lead absorption in animals, studies of the association between iron deficiency and BLLs in children have produced inconsistent results. There is little evidence that iron promotes a clinically important increase in lead excretion. However, the use of iron supplements among children with EBLs and iron deficiency has been shown to improve their developmental scores, suggesting that the effects of iron deficiency on cognition can be partially reversed among children with EBLs (24). This finding is consistent with a wealth of

data indicating that neurodevelopmental impairment among children with iron-deficiency anemia can be partially resolved by treatment with iron supplements (25-28). However, treatment with succimer (dimercaptosuccinic acid) to lower EBLLs (20 to 44 Fg/dL) in toddlers has not been shown to improve their cognition (29). Since the effects of iron deficiency on children's development appear to be independent of the effects of lead, there is no compelling reason to screen and treat children with lead exposure differently from children of similar age on the basis of their risk for iron deficiency, assessed independently of their lead exposure. Detailed recommendations for the prevention of iron deficiency can be found in a recent CDC report (30). Several of these recommendations are summarized later in this chapter.

### *Vitamin C*

#### *Could increasing children's vitamin C intake decrease their BLLs?*

Decreased lead retention has been shown in rats fed vitamin C and exposed to lead (31-33). Clinical studies in humans actually predate these and other animal studies, as case reports of lead-poisoned workers' response to ascorbate began to appear in the literature as early as 1939 (34). Later, clinical trials were conducted among workers and other adults. An uncontrolled experiment involving 39 workers showed that their BLLs had declined 24 weeks after they began treatment with vitamin C (35). Results of a single-blind clinical trial of vitamin C (1 g daily) among lead smelter workers with BLLs of 28 to 76 Fg/dL did not show vitamin C to affect their urinary excretion of lead (36). In a double-blind randomized clinical trial, however, adult male smokers given a daily dose of vitamin C (1 g) experienced a statistically significant 80% decline in BLLs (from 36 to 20 Fg/dL) after 1 week of treatment that persisted through the 4-week period of the study (37).

Much less is known about the effect of vitamin C on BLLs in children. One correlative cross-sectional study using NHANES III data showed high levels of serum vitamin C to be associated with a low prevalence of EBLLs for both children and adults. Results of this study also showed an association between serum vitamin C levels and log BLLs among adults but not among children (38). This study, however, did not control for environmental lead risk or include children below the age of 4 years who are the usual subjects for case management (39). In summary, although there is fairly strong evidence to support giving vitamin C to adults with EBLLs, there is insufficient evidence to recommend for or against vitamin C supplementation for children with EBLLs. It is important to note that CDC recommends giving all children 6 months and older at least two servings of foods rich in vitamin C per day for the prevention of iron deficiency (30).

### *Calcium*

#### *Are children at higher risk for EBLLs also at higher risk for inadequate calcium intake?*

Recommended daily allowance values for calcium intake have been replaced by “adequate intake”(AI) levels (40). AIs for young children are age-specific: 0-6 months, 210 mg/day; 7-12 months, 270 mg/day; 1-3 years, 500 mg/day; and 4-8 years, 800 mg/day. Actual mean calcium intake levels may be estimated from NHANES III data. Figure 4.1 depicts the median, 75<sup>th</sup> percentile, and 25<sup>th</sup> percentile levels for daily calcium intake among children aged 1 to 4 years by race. Calcium intake is below the AI level for more than 25% of Mexican American and non-Hispanic black 1- to 3-year-olds, and approaches 25% for non-Hispanic white 1- to 3-year-olds. Similarly, there is little variation in calcium intake across income groups (results not shown). Although these data do not specifically reflect the calcium intake of children with EBLLs, groups that typically have higher risk for EBLLs in this nationally representative sample of children have only a slightly higher risk for calcium intake below AI levels. One research group assessed the calcium intake of 314 mostly African-American children using a food frequency questionnaire (41). The results for children aged 1 to 3 years were similar to those of NHANES III, with about 30% of children having calcium intakes below the AI level of 500 mg per day. Because calcium intakes were not much higher for 4- to 8- year-old children in this sample, a substantially higher proportion of them (almost 60%) had calcium intakes below the AI level of 800 mg for their age group.

#### *Does inadequate calcium intake confer a higher risk for EBLLs?*

Animal studies have shown higher lead retention in animals fed low-calcium diets, raising the possibility that low-calcium diets could affect the BLLs of humans (42-45). Furthermore, studies of radiolabeled lead absorption in human adults show lower absorption of lead when lead is co-administered with calcium (46, 47). In 89 metabolic balance studies of 12 infants, dietary calcium intake was found to be inversely associated with lead retention (48). As the authors noted, however, dietary calcium intake closely paralleled the intake of phosphorus and other unmeasured components of milk and formula, so it is difficult to attribute this effect solely to calcium.

In NHANES II (1976 - 1980), calcium intake was inversely associated with BLLs in a nationally representative sample of children aged 3 to 11 years (49). The analysis included good controls for children’s socioeconomic status, region of the country, and urban vs. rural residence. Results of this analysis showed that children’s calcium intake had a small, inverse correlation with their BLL, with children’s BLLs declining by only about 0.2% for each 100 mg increase in dietary calcium. The study was subject to the following limitations. First, it included no direct controls for environmental lead exposure. Second, because the backward selection procedure used for the regression analysis removed confounding nutritional variables from the final model, and the p-value for the calcium effect was close to 0.05, statistical significance would probably

be lost with the inclusion of only one nutritional confounder. Results of other smaller published cross-sectional studies generally support an inverse association between children's calcium intake and BLLs, but these studies also did not control for confounding (50, 51).

#### *Calcium supplementation above the AI level*

Meredith et al. showed that increases in dietary calcium of up to 5 mmol decreased lead retention in rats with no pre-existing calcium deficiency; however, they found no further decrease with oral doses of calcium above 5 mmol (100-fold molar excess of calcium) (52). This finding is consistent with those from the studies of radiolabeled lead absorption in human adults mentioned above (46, 47). In an unpublished balance study of the effect of calcium gluconate syrup supplementation (50 mg calcium/kg/day) on lead retention in six children, neither lead absorption nor lead retention was found to be affected by calcium supplementation (personal communication, Ekhard E. Ziegler, University of Iowa College of Medicine, May 14, 1990). Similarly, no effect of calcium supplementation was found in a randomized clinical trial of calcium glycerophosphate supplementation of infant formula involving 105 infants (53). In this study, infants in the treatment group received, on average, 1600 mg of calcium per day. Change in BLL over time was small for all of the infants in the prevention trial (only 1 Fg/dL), limiting the power of the study to examine a treatment effect.

#### *Summary*

There is little evidence that a child typically considered at high risk for lead exposure is at greater risk for low calcium intake than children without EBLs. However, because of the frequency of inadequate calcium intake among all children, it is important to verify that a child with an EBL is receiving enough calcium. The results of both animal studies and human laboratory studies provide good evidence that dietary calcium competitively inhibits lead absorption. The results of one cross-sectional study of older children with controls for socioeconomic status show an inverse association between dietary calcium intake and BLLs. There are few data on young children in the high-risk age range, and no clinical trials have evaluated the efficacy of supplementation among children with low calcium intakes who are at risk for lead exposure. The results of studies among older children and adults, animal studies, and cross-sectional studies all reinforce the importance of adequate calcium intake (i.e., two servings per day of dairy products or other calcium-rich foods). However, there is no clinical evidence that supplementation of calcium beyond the recommended AI level in children with EBLs has a clinical effect on the BLLs; therefore, we do not recommend giving calcium supplements to children with EBLs.

### *Total fat intake*

The link between fat intake and BLLs comes primarily from animal experiments (54). In one cross-sectional experimental study, researchers found a direct association between dietary fat and BLLs (55); however, no such relationship between dietary fat and BLL was found in NHANES II (47). Thus, no strong case can be made for decreasing children's total fat intake. In addition, dietary fat is an important constituent in the diets of children under 2 years of age because calories from fat support high calorie requirements for growth during this period. Thus, we do not recommend low-fat diets for the treatment of younger children with EBLs.

### *Zinc supplementation*

Some evidence from animal studies suggests that high levels of dietary zinc inhibit the absorption and retention of lead in animals (56). However, in one small clinical study in which zinc was given with and without vitamin C to lead-exposed workers, the zinc had no demonstrable effect on their BLLs (36). As with calcium, we do not recommend adding zinc supplements to the diet of children with EBLs.

### *Other factors*

Many other factors have been evaluated as mediators of lead absorption and excretion in adults or animals. These factors include vitamins (thiamin, pyridoxine, vitamin D), minerals (phosphorus), dietary chelators (phytate acid, alginates, oral EDTA), and frequency of meals. These were not included in this review because of a lack of evidence to determine their efficacy in children.

## **General Recommendations**

### *WIC referral*

- Because children with EBLs are at risk for poor diet, refer children with EBLs to supplemental food programs that provide nutritional counseling and access to healthy foods.
- Determine whether children with EBLs are eligible for WIC and ensure their access to this program if they are eligible. An EBL is a condition that should qualify age-eligible older children who might otherwise not be candidates for participation in the program.

### *Iron deficiency*

- Low-income or minority children with EBLs are usually at high risk for iron-deficiency anemia. Detailed recommendations for the prevention of iron deficiency can be found in a recent CDC report (30). Several of these recommendations are included below.

- Test those at risk for anemia (e.g., those from low-income, migrant, or recently arrived refugee families or those qualifying for WIC). These children should be tested at the following ages:
  - S Initially between ages 9 and 12 months
  - S Six months later
  - S Annually from ages 2 to 5 years

#### *Vitamin C intake*

- Advise caregivers to provide children with an adequate intake of vitamin C. For children approximately age 6 months and older, encourage caregivers to provide two feedings per day of foods rich in vitamin C (e.g., fruits, vegetables, or juice), preferably with meals, as a way of improving their iron absorption.

#### *Iron intake*

- Encourage caregivers to provide children with an adequate intake of iron by:
  - S Introducing them to iron-fortified cereals and pureed meats at their appropriate developmental stages.
  - S Providing one serving of lean red meat per day to older children.
  - S Do not recommend giving children iron supplements except under the supervision of a physician or nutritionist and only when iron deficiency or anemia is documented.

#### *Calcium intake*

- Encourage caregivers to see that children with EBLs receive an adequate amount of calcium (500 mg/day @ 1 to 3 years; 800 mg/day @ 4 to 8 years), by:
  - S Providing them with two servings of dairy products per day, unless they are lactase-deficient.
  - S Providing lactase-deficient children with sufficient dietary calcium from other sources (e.g., broccoli, greens, kidney beans, and calcium-fortified juices).
  - S Do not recommend giving children calcium supplements except under the supervision of a physician or nutritionist.
- Do not recommend supplementation in children with EBLs beyond the recommended AI levels.

#### *Fat intake*

- Do not encourage a low-fat diet as a means of lowering children's EBLs. Not only is there no clinical evidence to support the implementation of such a diet, but dietary fat is an important constituent in the diets of children, especially those under 2 years of age.

### *Zinc supplementation*

- Do not recommend zinc supplementation.

### **Recommendations for Future Research**

Evidence suggests that some population-based nutritional measures may reduce lead absorption in children. However, we do not yet know enough about the relationship between children's EBLLs and many specific nutrients to make recommendations. The literature is replete with animal studies that have not been adequately followed up in the human population. When they are, the epidemiologic work consists mostly of correlative cross-sectional studies without adequate controls for environmental lead exposure. Because of the sparse evidence for the efficacy of various nutritional interventions for children with EBLLs, it is premature to call for the implementation of population-based nutritional interventions. Instead, promising interventions should be evaluated in randomized clinical trials.

Such clinical trials are especially important for several reasons. First, correlative nutrition studies are hampered by our limited ability to measure dietary intake. In addition, many nutrient intakes correlate with each other, so only large observational studies can separate the effects of, for example, dietary fat from iron. An association between a nutritional factor and BLL, however, does not mean that manipulating the nutritional factor will have a clinically meaningful effect on an EBLL. Some of the randomized trials of dust control among children illustrate this (57, 58). Only clinical trials employing randomized designs can determine whether modifying children's intake of specific nutrients will actually influence their BLLs.

Furthermore, many patient-compliance and side-effects issues need to be resolved before nutritional interventions can be studied clinically. For example: Which vitamin C supplements will children reliably take? Which ones will caregivers reliably give? Does it matter when the dose is given? Should the supplement be given as a medication or through a frequently eaten food item? What is the level of supplementation that could result in side effects?

Finally, many nutritional interventions will involve behavioral change for families. Results of clinical studies of behavioral change (e.g., parental smoking cessation [59, 60] and environmental tobacco exposure reduction [61]), suggest that only modest behavioral changes can be expected from limited-contact counseling interventions. In addition, injury research shows that passive interventions (e.g., using childproof medicine caps) are much more effective in preventing injuries than are active interventions (e.g., asking parents to keep medicines out of reach). Therefore, controlled randomized studies should identify and evaluate actions and interventions that may result in improved compliance with recommendations.

**References**

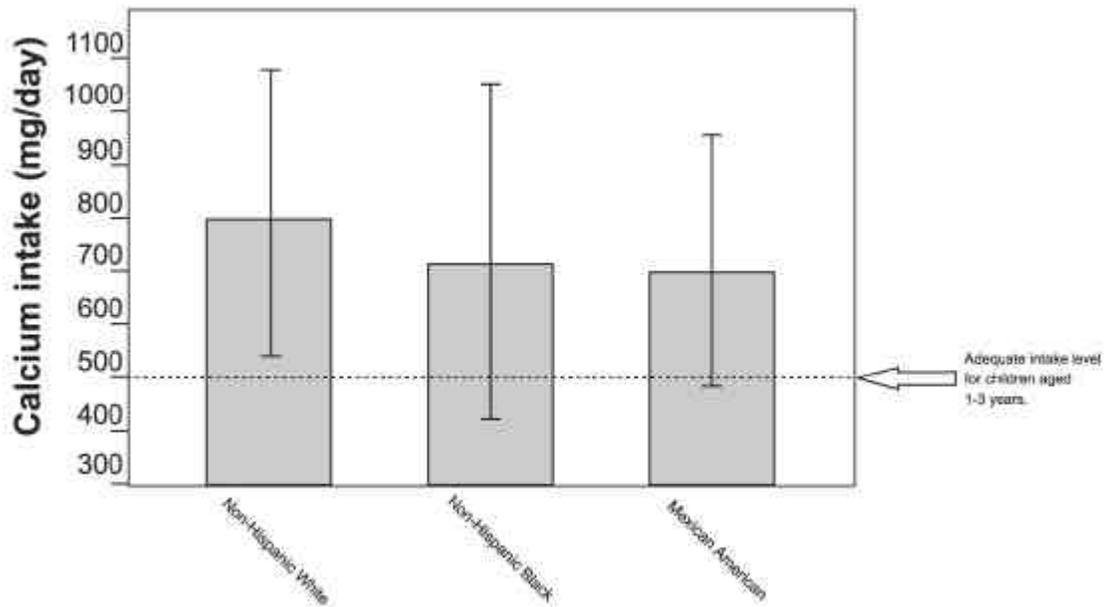
1. Mahaffey KR. Nutrition and lead: strategies for public health. *Environ Health Perspect* 1995;103(Suppl 6):191-6.
2. Bogden, JD, Oleske JM, Louria DB. Lead poisoning--one approach to a problem that won't go away. *Environ Health Perspect* 1997;105:1284-7.
3. Hu H, Kotha S, Brennan T. The role of nutrition in mitigating environmental insults: policy and ethical issues. *Environ Health Perspect* 1995;103 (Suppl 6):185-90.
4. Mushak P, Crocetti AF. Lead and nutrition. *Nutrition Today* 1996; 31:12- 7.
5. Rees JM, Monsen ER, Merrill JE. Iron fortification of infant foods: a decade of change. *Clin Pediatr* 1985;24:707-10.
6. Looker AC, Dallman PR, Carroll MS, et al. Prevalence of iron deficiency in the United States. *JAMA* 1997;277:973-6.
7. Barltrop D. The prevalence of pica. *Am J Dis Child* 1966;112:116-23.
8. Giebel HN, Suleymanova D, Evans GW. Anemia in young children of the Muynak District of Karakalpakistan, Uzbekistan: prevalence, type, and correlates. *Am J Public Health* 1998;88:805-7.
9. Danford DE, Pica and nutrition. *Annu Rev Nutr* 1982;2:303-22.
10. Mooty J, Ferrand CF, Harris P. Relationship of diet to lead poisoning in children. *Pediatrics* 1975;55:636-9.
11. CDC. Preventing lead poisoning in young children. Atlanta, Georgia: US Department of Health and Human Services, CDC; 1991.
12. Wright RO, Shannon MW, Wright RJ, et al. Association between iron deficiency and low-level lead poisoning in an urban primary care clinic. *Am J Public Health* 1999;89:1049-53.
13. Hammad TA, Sexton M, Langenberg P. Relationship between blood lead and dietary iron intake in preschool children, a cross-sectional study. *Ann Epidemiol* 1996;6:30-3.

14. Serwint JR, Damokosh AI, Berger OG, et al. No difference in iron status between children with low and moderate lead exposure. *J Pediatr* 1999;135:108-10.
15. Campbell JR, Auinger P, Weitzman M. Absence of an association between iron status and BLLs in a nationally representative sample (Abstract). *Pediatr Res* 2000;47:179A.
16. McMichael AJ, Baghurst PA, Wigg NR, et al. Environmental exposure to lead and cognitive deficits in children (letter). *N Engl J Med* 1989;320:596.
17. Barton JC, Conrad ME, Nuby S. Effects of iron on the absorption and retention of lead. *J Lab Clin Med* 1978;92:536-47.
18. Mahaffey-Six K, Goyer RA. The influence of iron deficiency on tissue content and toxicity of ingested lead in the rat. *J Lab Clin Med* 1972;79:128-36.
19. Watson WS, Morrison J, Bethel MI. Food iron and lead absorption in humans. *Am J Clin Nutr* 1986;44:248-56.
20. Flanagan PR, Chamberlain MJ, Valberg LS. The relationship between iron and lead absorption in humans. *Am J Clin Nutr* 1982;36:823-9.
21. Mahaffey KR, Annet JL. Association of erythrocyte protoporphyrin with blood lead levels and iron status in the Second National Health and Nutrition Examination Survey, 1976-1980. *Environ Res* 1986;41:327-38.
22. Wasserman G, Graziano JH, Factor-Litvak P, et al. Independent effects of lead exposure and iron deficiency on developmental outcomes at age 2 years. *J Pediatr* 1992;121: 695-703.
23. Markowitz ME, Rosen JF, Bijur PE. Effects of iron deficiency on lead excretion in children with moderate lead intoxication. *J Pediatr* 1990;116:360-4.
24. Ruff HA, Markowitz ME, Bijur PE, et al. Relationships among blood lead levels, iron deficiency, and cognitive development in two-year-old children. *Environ Health Perspect* 1996;104:180-5.
25. Pollitt E. Iron deficiency and cognitive function. *Annu Rev Nutr* 1993;13:521-37.
26. Idjradinata P, Pollitt E. Reversal of developmental delays in iron-deficient anaemic infants treated with iron. *Lancet* 1993;341:1-4.

27. Lozoff B. Behavioral alterations in iron deficiency. *Adv Pediatr* 1988;35:331-60.
28. Lozoff B, Jimenez E, Hagen J, et al. Poorer behavioral and developmental outcome more than 10 years after treatment for iron deficiency in infancy. *Pediatrics* 2000;105:1-11.
29. Rogan WJ, Dietrich KN, Ware JH, et al. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. *N Engl J Med* 2001;344:1421-6.
30. CDC. Recommendations to prevent and control iron deficiency in the United States. *MMWR* 1998;47(RR-3):1-29.
31. Goyer RA, Cherian GM. Ascorbic acid and EDTA treatment of lead toxicity in rats. *Life Sci* 1979;24:433-8.
32. Suzuki T, Yoshida . Effect of dietary supplementation of iron and ascorbic acid on lead toxicity in rats. *J Nutr* 1979;109:982-8.
33. Flora SJS, Tandon SK. Preventive and therapeutic effects of thiamine, ascorbic acid, and their combination on lead intoxication. *Acta Pharmacol Toxicol* 1986;58:374-8.
34. Holms HN, Campbell K, Amberg EJ. Effect of vitamin C on lead poisoning. *J Lab Clin Med* 1939;24:1119-27.
35. Papaioannou RA, Sohler A, Pfeiffer CC. Reduction of blood lead levels in battery workers by zinc and vitamin C. *Orthomolecular Psychiatry* 1978;7:94-106.
36. Lauwerys R, Roels H, Buchet JP, et al. The influence of orally-administered vitamin C on the absorption of and the biological response to lead. *J Occup Med* 1983;25:668-78.
37. Dawson EB, Evans DR, Harris WA, et al. The effect of ascorbic acid supplementation on the blood lead levels of smokers. *J Am Col Nutr* 1999; 18:166-70.
38. Simon JA, Hudes ES. Relationship of ascorbic acid to blood lead levels. *JAMA* 1999;281:2289-93.
39. Matte TD. Reducing blood lead levels: benefits and strategies [editorial; comment]. *JAMA* 1999;281:2340-2.

40. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. Washington, DC: Institute of Medicine, National Academy Press; 1997.
41. Bruening K, Kemp FW, Simone N, et al. Dietary calcium intakes of urban children at risk of lead poisoning. *Environ Health Perspect* 1999;107:431-5.
42. Mahaffey KR, Haseman JD, Goyer RA. Dose-response to lead ingested in rats fed low dietary calcium. *J Lab Clin Med* 1973;82:92-101.
43. Barton JC, Conrad ME, Harrison L, et al. Effects of calcium on the absorption and retention of lead. *J Lab Clin Med* 1978;91:366-76.
44. Lederer LG, Franklin CB. Effect of calcium and phosphorus on retention of lead by a growing organism. *JAMA* 1940;114:2457-61.
45. Six KM, Goyer RA. Experimental enhancement of lead toxicity by low dietary calcium. *J Lab Clin Med* 1970;76:933-42.
46. Blake KC, Mann M. Effect of calcium and phosphorus on the gastrointestinal absorption of <sup>203</sup>Pb in man. *Environ Res* 1983;30:188-94.
47. Heard MJ, Chamberlain AC. Effect of minerals and food on uptake of lead from the gastrointestinal tract in humans. *Hum Toxicol* 1982;1:411-5.
48. Ziegler EE, Edwards BB, Jensen RL, et al. Absorption and retention of lead by infants. *Pediatr Res* 1978;12:29-34.
49. Mahaffey KR, Gartside PS, Glueck CJ. Blood lead levels and dietary calcium intake in 1- to 11-year-old children: the Second National Health and Nutrition Examination Survey, 1976 to 1980. *Pediatrics* 1986;78:257-62.
50. Sorell M, Rosen JF, Roginsky M. Interactions of lead, calcium, vitamin D, and nutrition in lead-burdened children. *Arch Environ Health* 1977;32:160-4.
51. Johnson NE, Tenuta K. Diets and lead blood levels of children who practice pica. *Environ Res* 1979;18:369-76.

52. Meredith PA, Moore MR, Goldberg A. The effect of calcium on lead absorption in rats. *Biochem J* 1977;166:531-7.
53. Sargent JD, Dalton MA, O'Connor GT, et al. Randomized trial of calcium glycerophosphate-supplemented infant formula to prevent lead absorption. *Am J Clin Nutr* 1999;69:1224-30.
54. Barltrop D, Khoo HE. The influence of dietary minerals and fat on the absorption of lead. *Sci Total Environ* 1976;6:265-73.
55. Lucas SR, Sexton M, Langenberg P. Relationship between blood lead levels and nutritional factors in preschool children: a cross-sectional study. *Pediatrics* 1996;97:74-8.
56. Cerklewski FL, Forbes RM. Influence of dietary zinc on lead toxicity in the rat. *J Nutr* 1976;106:689-96.
57. Lanphear BP, Winter NL, Apetz L, et al. A randomized trial of the effect of dust control on children's blood lead levels. *Pediatrics* 1996;98:35-40.
58. Lanphear BP, Howard C, Eberly S, et al. Primary prevention of childhood lead exposure: a randomized trial of dust control. *Pediatrics* 1999;103:772-7.
59. Valanis B, Lichtenstein E, Mullooly JP, et al. Maternal smoking cessation and relapse prevention during health care visits. *Am J Prev Med* 2001;20:1-8.
60. Wall MA, Severson HH, Andrews JA, et al. Pediatric office-based smoking intervention: impact on maternal smoking and relapse. *Pediatrics* 1995;96:622-8.
61. Hovell MF, Zakarian JM, Matt GE, et al. Effect of counseling mothers on their children's exposure to environmental tobacco smoke: randomised controlled trial. *BMJ* 2000;321:337-42.



**Figure 4.1. Median Calcium Intake by Race from NHANES III**  
Error bars represent the interquartiles range.

## **Chapter 5. Developmental Assessment and Interventions**

Prepared by David Bellinger, PhD, MSc., and Leonard Rappaport, MD



**Table 5.1. Summary of Recommendations for Developmental Assessment and Interventions**

- Make long term developmental surveillance a component of the management plan for any child with a blood lead level (BLL)  $\geq$  20 Fg/dL, while recognizing that this will not necessarily result in referral for diagnostic assessment or intervention.
- Also consider developmental surveillance for a child who has a BLL that does not exceed 20 Fg/dL but who has other significant developmental risk factors.
- Do not base decisions regarding developmental assessment or intervention on a child's age at the time the child is found to have an elevated blood lead level (EBLL).
- If you wish to refer a child with an EBLL for intervention services, consider referring that child to early intervention/stimulation programs.
- Include a history of a child's EBLL in the problem list maintained in the child's medical record.
- Do not stop developmental surveillance when a child with an EBLL reaches age 6 or when the child's blood lead level is reduced. A responsible party (e.g., the child's PCP) should provide ongoing developmental surveillance of that child after the EBLL case is closed.
- In the developmental surveillance of children with EBLLs:
  - Watch for emerging difficulties at critical transition points in childhood: first, fourth, and sixth/seventh grades.
  - Watch for behaviors that interfere with learning, such as inattention and distractibility.
- Refer children experiencing neurodevelopmental problems for a thorough diagnostic evaluation.
- Be advocates for the child.

## Introduction

Since *Preventing Lead Poisoning in Young Children* was published in 1991 by the Centers for Disease Control and Prevention (CDC) (1), considerable new data have become available on the developmental and neurobehavioral effects of lead, including late results of a number of prospective longitudinal studies begun around 1980. These new data generally bolster the conclusion, reached in the 1991 statement, that lead adversely affects children's performance on tests of cognition at blood lead levels (BLLs) below 10 Fg/dL (2,3). New insights have been generated as well regarding both the most sensitive functional endpoints and the range of endpoints affected. Specifically, recent data suggest that lead toxicity may contribute to neurobehavioral, as well as cognitive, morbidities of childhood. Because of the consistency of these associations and the relatively high prevalence of BLLs in the range associated with these increased risks, it is important to address the issues involved in the identification and treatment of lead-related cognitive and neurobehavioral effects.

Any recommendations regarding neurodevelopmental assessments and interventions for children with elevated blood lead levels (EBLLs) must rest on a firm empirical foundation. Therefore, this chapter presents an overview of numerous studies of the association between children's BLLs and their neurodevelopment and behavior, as well as the recommendations based on the studies.

## Detailed Bases for Recommendations

### *BLLs and IQ*

Several older case series clearly demonstrate that children presenting with symptoms and findings of severe lead intoxication are at substantially increased risk for serious neurological sequelae (4-6). Asymptomatic children with BLLs in the 30- to 60-Fg/dL range also may suffer a variety of neurologic and neurobehavioral adversities (7-9).

Recent epidemiological studies provide a wealth of data on the nature of the dose-effect relationship for children with BLLs below 35 Fg/dL. The relationship between children's BLL and IQ appears to be linear, even at BLLs below 10 Fg/dL (2, 3). Some data suggest, however, that the slope for the dose-effect relationship is steeper for BLLs below 15 Fg/dL than it is for levels above 15 Fg/dL (3). Meta-analyses of the results of several studies indicate that an increase in average postnatal BLL from 10 to 20 Fg/dL is associated with a decrease of 1 to 3 points in the child's IQ measured at age 5 or older (3, 10, 11). The point estimates for the IQ change associated with a doubling of BLL from 10 to 20 Fg/dL were 2.57 points (standard error 0.41) in Schwartz' analysis of a mixed set of prospective and cross-sectional studies (3) and 2.53 points (standard error 0.41) in the analysis of cross-sectional studies by Pocock et al. (10).

The study cohorts were quite diverse ethnically, culturally, and sociodemographically. Children in some cohorts experienced chronic exposure by virtue of living near a smelter (12, 13), while children in other cohorts were impoverished and living in inner-city areas in old housing with leaded paint in poor repair (14, 15). Yet other cohorts consisted largely of children from relatively well-to-do families (16, 17). The likelihood that these interstudy differences were accompanied by differences in the nature and extent of confounding bias makes the overall consistency in the findings of the different studies even more impressive, and increases the plausibility of the conclusion that lead plays a causal role in a child's neurodevelopment. As in most areas of epidemiological research, however, interstudy variability is apparent in the strength of the association, with some investigators reporting that the association between children's BLLs and IQ scores was not statistically significant (15, 17-19). Nevertheless, the overall weight of evidence clearly supports the existence of an inverse association between children's BLLs and their IQ scores.

#### *Other Neurodevelopmental Deficits Associated with EBLs*

Children presenting with severe symptomatic lead intoxication are known to suffer from neurobehavioral problems such as impulsivity, aggression, and short attention span (4). Results of a number of studies support the hypothesis that the spectrum of low-level lead effects on children includes neurobehavioral problems (20-23). At present, there is no compelling evidence that an EBL increases a child's risk for attention deficit hyperactivity disorder (ADHD) (24). However, because the studies mounted to address this question have been cross-sectional or retrospective, children's lead exposure status at earlier developmental periods may have been misclassified. It is noteworthy that elevated blood or tooth lead levels have been repeatedly linked to the types of behavioral problems pertinent to the diagnosis of attention deficit disorder inattentive subtype, a diagnosis included for the first time in the fourth (and most recent) edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (25). These behaviors include distractibility, poor organization, a lack of persistence, and daydreaming (26-29). Elevated bone lead levels have also been linked to an increased risk of engaging in antisocial behaviors in middle childhood (29).

Efforts to identify a "neurobehavioral signature" for children with EBLs have generally been unsuccessful (30), although several studies have found that among preschool children, EBLs were most strongly associated with deficits in nonverbal functions, particularly visual-motor skills (13, 31-33). However, the nature of the deficits identified when children reach school age are less consistent. It is likely that the manner in which lead toxicity is expressed depends on many factors, including the timing and chronicity of exposure, the child's age when outcomes are assessed, and the context of the assessment (30). Given the absence of specificity in the findings associated with an EBL, a child's specific deficits are of little use in making a diagnosis of past or present EBL.

### *Interchild Variability*

The BLLs at which individual children show signs of clinical lead intoxication vary widely, and despite the consistent inverse association between children's BLL and IQ noted above, children have varying sensitivity to the more subtle functional impairments associated with EBLLs (30). Although, on average, children with higher BLLs tend to score lower on IQ tests than do children with lower BLLs, some children seem to be more affected than others by a given lead dose (34). This suggests that not all children with a given BLL should be considered at equivalent neurodevelopmental risk. In other words, an EBLL should be viewed as a risk factor for neurodevelopmental problems, not a diagnosis.

### *Importance of Age*

Identifying the age at which children are most sensitive to the neurodevelopmental effects of lead is complicated by the relatively high degree of stability in children's BLLs and the frequent confounding of age and peak BLL (32, 35). However, data from cohorts in which these obstacles to inference are less severe indicate that children's IQs may be particularly sensitive to lead-associated effects when the children are about 2 years old (16). Pocock et al. for example, found that EBLLs in children from 1 to 3 years of age appear to be the most predictive of children's later development (10). On the other hand, data from several of the prospective studies suggest that recent or concurrent BLLs are among the strongest predictors of children's neurodevelopmental function at school-age (12, 32). Furthermore, primate studies indicate that the period of greatest susceptibility to EBLLs may depend upon the specific deficit being evaluated (36). There is some limited evidence for this from human studies as well (37).

### *Time Lag Associated with the Effects of EBLLs*

For the most part, the evidence from prospective studies regarding the time course of the association between a child's BLL and neurodevelopment is consistent with a lag effect (16, 31). It is much more common to find a significant association between children's previous BLLs and their current neurodevelopmental status than between their current BLLs and current developmental status. This pattern is less clear under circumstances in which children's BLLs remain elevated for extended periods of time, such as when they live near smelters or in hazardous housing; under such conditions, children's current and past BLLs tend to be strongly correlated. The lag may be the result of a toxicological process in which some period of time is required for past lead exposure to affect the central nervous system function. Another explanation is that lead may primarily affect higher-order neurodevelopmental processes that are best tested at later ages when children's response modalities are more highly differentiated. One implication of this lag is that neurodevelopmental assessments conducted when a child has an

EBLL may produce many false-negative results and fail to identify a child who is at risk for later neurodevelopmental dysfunction. Careful long-term surveillance of behavior and neurodevelopment is thus needed to ensure that such children are identified.

The effects of EBLLs on the skills required for academic success may not be appreciated until a child reaches critical transition points in school: 1) first grade, when children are expected to begin acquiring basic academic skills such as reading words or performing arithmetic operations; 2) fourth grade, where the emphasis begins to shift from acquiring basic skills to using those skills to learn new material (“reading to learn” as opposed to “learning to read”); and 3) sixth or seventh grade, when students are expected to use higher-order planning and organizational skills in order to complete long-term projects. Increased BLLs have been associated with difficulties with all three types of skills (20, 26, 38).

### *Persistence of Neurodevelopmental Effects*

Results from a variety of studies indicate that neurodevelopmental problems associated with elevated postnatal BLLs are persistent (8, 16, 38-43). The natural history of these problems appears to correspond to a “constant decrement” model, with the deficits associated with higher BLLs neither increasing nor decreasing over time (44), although few data are available on the persistence of effects. In contrast, the findings from most of the prospective studies are consistent with the hypothesis that neurodevelopmental effects associated with elevations in biomarkers of prenatal lead exposure attenuate during a child’s early years of life (10).

### *Factors Affecting a Child’s Risk for Neurological Sequelae*

Increased exposure to lead frequently occurs in the context of other factors that also place a child at increased neurodevelopmental risk (e.g., poverty, single-parent household, teen-age mother, child abuse, poor nutrition). From this perspective, lead represents an additional “hit,” adding to a child’s cumulative neurodevelopmental risk (45). In multivariate statistical models, children’s BLLs tend to account for a relatively small amount of the variance in their neurodevelopmental status. The amount varies across outcomes measured and across studies, ranging from 0% (i.e., accounting for no variance in neurodevelopmental measurements) to as much as 11% (46), although usually on the order of 1% to 3%. For instance, in the set of cross-sectional studies included in the meta-analysis of Needleman and Gatsonis (47), children’s BLLs accounted for 2.3% of the variance in their IQ scores (based on a weighted partial correlation of  $-0.152$ ). Other factors, particularly social class and parental intelligence, typically account for much larger percentages of outcome variance.

Some evidence suggests that certain characteristics of children and their families are associated with the children’s increased risk for neurodevelopmental impairments from a given level of lead exposure. Several studies, although not all, have identified family social class as one

such characteristic, with children from lower social strata appearing to express the neurodevelopmental effects of lead at a lower BLL (48-51). A sex difference is also sometimes found, although in some studies it is girls (52, 53) and in others it is boys (10, 50, 54) who are found to be at greater risk. One implication of these findings is that lead's association with children's neurodevelopment cannot be accurately expressed as a single number because the magnitude of the association may vary depending on the characteristics of a particular child and his or her environment. A more promising implication, however, is that the effects of lead on a child might be reduced by modifying critical aspects of the environment. For example, indirect observational evidence indicates that the persistence of the link between an EBLL and reduced function varies with factors such as family social class, maternal IQ, and quality of the home environment (54). Specifically, if two children with the same early BLL achieve the same developmental score at time 1, but one child's environment offers greater cognitive stimulation, that child's developmental status at time 2 is likely to be better than that of the child from the less stimulating environment. Thus certain factors might help children to weather the developmental insult of early lead exposure, either preventing neurodevelopmental effects from being expressed or facilitating subsequent recovery of function. Social class is often found to be such an effect modifier, although higher social class is presumably a surrogate for the more proximal influences that confer this greater resilience (e.g., better nutrition, greater access to academic supports, more varied experiences).

### *Effectiveness of Reducing BLLs*

It has not been shown that lowering a BLL after it has been elevated prevents lead-induced cognitive defects. In one study, children with BLLs between 25 and 55 Fg/dL were chelated if a test dose of EDTA increased their urinary lead excretion. Chelation did not change their neurodevelopmental test scores or BLLs at 6 months of follow-up. However, the children whose BLLs fell the most, whether they had chelation or not, had the greatest improvement in test scores at their 6-month follow-up evaluation (55). It is noteworthy that children's test scores increased one point for each decline of 3 Fg/dL in their BLL, a slope that is consistent with the slope for the dose-effect relationship from several observational studies that did not involve any intervention. However, the only large-scale randomized trial assessing the effects of chelation-induced BLL reductions on neurotoxicity showed that oral chelation with succimer (dimercaptosuccinic acid) lowered children's BLLs but did not improve their scores on a range of cognitive, neuropsychological, and neurobehavioral tests. Conducted among children living in deteriorating housing in four inner-city areas, this study involved 780 children (12 to 23 months of age) with BLLs from 20 to 44 Fg/dL who were randomly assigned to receive either a placebo or up to three courses of succimer. While the mean BLL of the treated group was 4.5 Fg/dL lower than that of the control group 6 months after treatment, there were no significant

differences between them in any of the mean test scores 3 years after treatment began, when the children were, on average, 5 years old (56).

### *Effects of Early Enrichment on Children with EBLLs*

No studies have been published on the effectiveness of non-medical interventions, such as early enrichment programs, in ameliorating the effects of EBLLs on children's neurodevelopment. In the absence of such data, it is reasonable to hypothesize that children with neurodevelopmental problems associated with an EBLL would benefit from the types of interventions shown to be effective in facilitating the neurodevelopment of other groups of children with idiopathic neurodevelopmental problems or those known to be at increased risk for such problems, such as low birth weight infants. Evaluations of interventions to foster the development of preschool children at risk for neurodevelopmental problems because of socioeconomic disadvantage, nonorganic failure to thrive, or low birth weight indicate that such programs can produce IQ increases on the order of 8 points (57). Although the magnitude of these IQ effects might attenuate after children complete the programs, participation in such programs is associated with lower rates of grade retention and need for special education (58). Some evidence suggests that programs in which participation begins prior to age 3 are more effective than those in which participation begins later (59). Programs that include procedures to foster both child development and parenting skills tend to be more effective than programs that are solely child-focused or parent-focused (57). Examples of such programs are the Mother-Infant Transaction Program (60-64) and the Infant Health and Development Program (65-67).

### **General Recommendations**

*Make long-term developmental surveillance a component of the management plan for any child with a BLL  $\geq$  20 Fg/dL.*

The precise BLL that one identifies as a "trigger" for neurodevelopmental surveillance will depend on the type and magnitude of deficit that one considers sufficiently large to warrant concern. Current CDC guidelines recommend that a child whose BLL is 20 Fg/dL or above receive environmental and medical evaluations. It makes both clinical and logistical sense to integrate neurodevelopmental surveillance and possible referral for diagnostic assessment or intervention into the overall management plan of such a child. The PCP and case manager, working in close collaboration, are best positioned to organize and oversee these processes. A BLL that exceeds 20 Fg/dL should not necessarily result in a referral for diagnostic assessment or intervention. This clinical decision should be made on a case-by-case basis, taking into account whether other neurodevelopmental risk factors are present (e.g., teen-age mother, poor parenting skills, inadequate cognitive or emotional stimulation, child abuse, poverty, genetic disorder, poor

nutrition, other medical issues). Under some circumstances, such as persistent BLLs of 15 to 19 Fg/dL or the presence of other significant neurodevelopmental risk factors, it would be appropriate to place a child with a lower BLL under increased neurodevelopmental surveillance. The case manager is in a unique position to assist the PCP in this regard by virtue of his or her knowledge of a child's risk factors gleaned from visits to the home or other contacts. Thus, the case manager can serve as a critical information resource to the PCP regarding contextual factors germane to the PCP's decisions about a child's neurodevelopmental needs. Furthermore, a case manager with training in neurodevelopmental assessment can conduct screening evaluations and bring potential problems to the PCP's attention.

The usual absence of associations between concurrent BLLs and risk for neurobehavioral deficits among children aged 0 to 3 years suggests that neurodevelopmental assessment of children while they have an EBLL might not identify children who will later experience cognitive problems (false-negatives). If a child currently has or has ever had an EBLL, however, the PCP and case manager should take a more aggressive approach in assessing that child's neurodevelopment and referring that child for follow-up. Under ordinary circumstances, the PCP is in the best position to follow up with long-term monitoring of a child with an EBLL. The developmental and behavioral screening that PCPs conduct at well-child visits, including taking a clinical history and administering brief instruments such as the Denver Developmental Screening Test, may be sufficient to identify children who are failing to make age-appropriate progress and transitions and who thus require additional diagnostic evaluation. Kindergarten-readiness evaluations generally are not designed to identify vulnerabilities that may be expressed as serious academic problems once children enter school. Because they produce many false negatives, kindergarten evaluations are not sufficiently sensitive to identify potential lead-associated learning difficulties.

*Do not base decisions regarding developmental assessment or intervention on a child's age at the time of the EBLL.*

Age is an inappropriate criterion for determining which children with EBLLs need referral for developmental evaluation. The neurodevelopmental effects of EBLLs are persistent and may be delayed. Also, there is no way of knowing how long a child may have had an EBLL. A child first identified as having an EBLL at age 4 might well have also had an EBLL at age 2 or 3 and, on the basis of a presumed chronic exposure, could be regarded as being in greater need of developmental assessment than a child with an EBLL at age 2. Because detailed information about children's blood lead history is often not available, a child of any age who is found to have a BLL of 20 Fg/dL or greater should be placed under increased surveillance in order to identify any emerging neurodevelopmental problems as early as possible.

*If you wish to refer a child with an EBLL for intervention services, consider referring that child for early intervention/stimulation programs that are available for children at increased developmental risk.*

Although there is no empirical basis for recommending interventions with specific characteristics for children with neurodevelopmental problems resulting from an EBLL, it is reasonable to hypothesize that such children would benefit from the types of interventions shown to be effective in facilitating the neurodevelopment of other groups of children with idiopathic neurodevelopmental problems. Programs in which participation begins prior to age 3 or those that include procedures to foster both child development and parenting skills may be most effective. Examples of such programs are the Mother-Infant Transaction Program and the Infant Health and Development Program.

*Include a history of a child's EBLL in the problem list maintained in the child's medical record.*

If a child changes his or her PCP, ensure that this information, along with other pertinent aspects of the child's medical history, is transmitted to the next provider. The PCP should work with the case manager to ensure appropriate follow-through.

*For the purposes of developmental surveillance, do not consider a child's case "closed" when the child reaches age 6 or when his or her BLLs are reduced.*

The period of increased risk for the expression of lead-associated neurodevelopmental problems continues after lead exposure has been remediated and BLLs reduced. Closure of a child's case by the case manager does not mean that the need for neurodevelopmental monitoring has ended.

*Be especially vigilant for emerging difficulties at critical transition points in childhood.*

There are three periods when different types of learning difficulties are typically expressed:

1. *First grade:* Children begin acquiring basic academic skills.
2. *Fourth grade:* They use these basic skills to learn new material.
3. *Sixth or seventh grade:* They need higher order planning and organizational skills.

A child with a history of EBLs who experienced difficulties making earlier transitions should be viewed as being at increased risk of experiencing difficulties with later transitions. Even children who made early transitions smoothly should be under increased surveillance at later transition points, as they may have problems when new educational demands are placed on them.

*Be alert for behaviors that might interfere with learning.*

An EBLL in early childhood is associated with an increased risk for behaviors such as inattention, distractibility, and impulsivity that can interfere with learning. These behaviors are characteristic of the recently recognized inattentive subtype of ADHD. Even if the behaviors a child presents are not sufficient to warrant the diagnosis of ADHD, the child may be helped by the types of classroom and work accommodations routinely made for children with an attention disorder.

*If you suspect that a child might be experiencing neurodevelopmental problems, consider arranging a thorough diagnostic (as opposed to screening) evaluation.*

The procedures used for assessment and intervention for a child with a history of EBLL and neurodevelopmental problems should be the same as those for a child with neurodevelopmental problems due to known and unknown causes. Ideally, assessments should be conducted by multidisciplinary teams, which might include developmental-behavioral pediatricians, educators, neuropsychologists, neurologists, speech/language pathologists, and child psychiatrists.

*Be advocates for the child.*

This might involve assisting the family in arranging diagnostic evaluations, interpreting the results, and petitioning third parties to pay for the evaluation on the grounds that the evaluation might reduce special education or specialized therapy costs in future years. In regions where access to specialized neurodevelopmental clinics is limited, diagnosis and treatment planning can also be achieved by means of school-based evaluations or private practitioners. It is important to recognize the complexities of school-system involvement in this process. Some school systems may be unwilling to commit resources to evaluate a child in the absence of a complaint that includes reduced academic progress. Furthermore, expecting schools to conduct such evaluations places them in a position of possible conflict of interest insofar as they would have to pay for remedial services deemed necessary as a result of the evaluations.

### **Recommendations for Future Research**

1. Conduct studies to characterize in greater detail the neurodevelopmental presentation associated with an EBLL, including analyses of the degree to which the presentation varies with factors such as the child's age at exposure and the magnitude and chronicity of the exposure.
2. Conduct studies to characterize the associations between EBLs and learning disabilities.

3. Conduct studies to evaluate the role of EBLLs in causing or exacerbating behaviors associated with ADHD, conduct disorder, and other psychiatric diagnoses.
4. Conduct studies to evaluate the potential psychosocial vulnerabilities of children with EBLLs (e.g., self-esteem, self-concept, social competencies, aggression).
5. Conduct randomized trials to evaluate the efficacy of specific interventions in ameliorating lead-associated neurodevelopmental problems.

## References

1. CDC. Preventing Lead Poisoning in Young Children. Atlanta, Georgia: US Department of Health and Human Services, CDC; 1991.
2. National Research Council. Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations. Washington, DC: National Academy Press; 1993.
3. Schwartz J. Low-level lead exposure and children's IQ: A meta-analysis and search for a threshold. *Environ Res* 1994;65:42-55.
4. Byers R, Lord E. Late effects of lead poisoning on mental development. *Am J Dis Child* 1943;66:471-94.
5. Mellins R, Jenkins D. Epidemiological and psychological study of lead poisoning in children. *JAMA* 1955;158:15-20.
6. Perlstein M, Attala R. Neurologic sequelae of plumbism in children. *Clin Pediatr* 1966;5:292-8.
7. Rummo J, Routh D, Rummo N, et al. Behavioral and neurological effects of symptomatic and asymptomatic lead exposure in children. *Arch Environ Health* 1979;34:120-4.
8. Faust D, Brown J. Moderately elevated blood lead levels: effects on neuropsychologic functioning in children. *Pediatrics* 1987;80:623-9.
9. Benetou-Marantidou A, Nakou S, Micheloyannis J. Neurobehavioral estimation of children with life-long increased lead exposure. *Arch Environ Health* 1988;43:392-5.
10. Pocock S, Smith M, Baghurst P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. *BMJ* 1994;309:1189-97.
11. World Health Organization/International Programme on Chemical Safety. Environmental Health Criteria 165. Inorganic Lead. Geneva: World Health Organization, 1995.
12. Tong SL, Baghurst P, McMichael A, et al. Lifetime exposure to environmental lead and children's intelligence at 11-13 years: the Port Pirie cohort study. *BMJ* 1996;312:1569-75.

13. Wasserman G, Liu X, Lolacono N, et al. Lead exposure and intelligence in 7-year-old children: The Yugoslavia Prospective Study. *Environ Health Perspect* 1997;105:956-62.
14. Dietrich K, Succop P, Berger O, et al. Lead exposure and the central auditory processing abilities and cognitive development of urban children: The Cincinnati Lead Study cohort at age 5 years. *Neurotoxicol Teratol* 1992;14:51-6.
15. Ernhart C, Morrow-Tlucak M, Wolf A, et al. Low-level lead exposure in the prenatal and early preschool periods: intelligence prior to school entry. *Neurotoxicol Teratol* 1989;11:161-70.
16. Bellinger D, Stiles K, Needleman H. Low-level lead exposure, intelligence, and academic achievement: a long-term follow-up study. *Pediatrics* 1992;90:855-91.
17. Cooney G, Bell A, McBride W, et al. Low-level exposures to lead: The Sydney Lead Study. *Dev Med Child Neurol* 1989;31:640-9.
18. Smith M, Delves T, Lansdown R, et al. The effects of lead exposure on urban children: The Institute of Child Health/Southampton Study. *Dev Med Child Neurol (suppl)* 1983;47:1-54.
19. Harvey P, Hamlin M, Kumar R, et al. Blood lead, behavior and intelligence test performance in preschool children. *Sci Total Environ* 1984;40:45-60.
20. Bellinger D, Leviton A, Allred E, et al. Pre- and postnatal lead exposure and behavior problems in school-aged children. *Environ Res* 1994;66:12-30.
21. Mendelsohn A, Dreyer B, Fierman A, et al. Low-level lead exposure and behavior in early childhood. *Pediatrics* 1998;101:E10.
22. Wasserman G, Staghezza-Jaramillo B, Shrout P, et al. The effect of lead exposure on behavior problems in preschool children. *Am J Public Health* 1998;88:481-6.
23. Burns J, Baghurst P, Sawyer M, et al. Lifetime low-level exposure to environmental lead and children's emotional and behavioral development at ages 11-13 years. The Port Pirie Cohort Study. *Am J Epidemiol* 1999;149:740-9.
24. Kahn C, Kelly P, Walker W. Lead screening in children with attention deficit hyperactivity disorder and developmental delay. *Clin Pediatr* 1995;34:498-501.

25. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4<sup>th</sup> Edition (DSM-IV). Washington, DC: American Psychiatric Association; 1994.
26. Needleman H, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med* 1979;300:689-95.
27. Yule W, Urbanowicz MA, Lansdown R, et al. Teachers' ratings of children's behaviour in relation to blood lead levels. *Br J Dev Psychol* 1984;2:295-305.
28. Thomson G, Raab G, Hepburn W, et al. Blood-lead levels and children's behaviour: results from the Edinburgh Lead Study. *J Child Psychol Psychiatry* 1989;30:515-28.
29. Needleman H, Riess J, Tobin M, et al. Bone lead levels and delinquent behavior. *JAMA* 1996;275: 363-9.
30. Bellinger D. Interpreting the literature on lead and child development: the neglected role of the "experimental system." *Neurotoxicol Teratol* 1995;17:201-12.
31. Bellinger D, Sloman J, Leviton A, et al. Low-level lead exposure and children's cognitive function in the preschool years. *Pediatrics* 1991;87:219-27.
32. Dietrich K, Berger O, Succop P. Lead exposure and the motor developmental status of urban six-year-old children in the Cincinnati Prospective Study. *Pediatrics* 1993;91:301-7.
33. Baghurst P, McMichael A, Tong S, et al. Exposure to environmental lead and visual-motor integration at age 7 years: the Port Pirie Cohort Study. *Epidemiology* 1995;6:104-9.
34. Bellinger D. Lead and neuropsychological function in children: progress and problems in establishing brain-behavior relationships. In: Tramontana M, Hooper S (eds.) *Advances in Child Neuropsychology*, vol. 3. New York: Springer-Verlag, 1995:12-47.
35. McMichael A, Baghurst P, Robertson E, et al. The Port Pirie Cohort Study. Blood lead concentrations in early childhood. *Med J Aust* 1985;143:499-503.
36. Rice D. Lead exposure during different developmental periods produces different effects on FI performance in monkeys tested as juveniles and adults. *Neurotoxicology* 1992;13:757-70.
37. Shaheen S. Neuromaturation and behavior development: the case of childhood lead poisoning. *Dev Psychol* 1984;20:542-50.

38. Needleman H, Schell A, Bellinger D, et al. The long-term effects of childhood exposure to low doses of lead: an 11-year follow-up report. *N Engl J Med* 1990;322:83-8.
39. Bellinger D, Hu H, Titlebaum L, et al. Attentional correlates of dentin and bone lead levels in adolescents. *Arch Environ Health* 1994;49:98-105.
40. White R, Diamond R, Proctor S, et al. Residual cognitive deficits 50 years after lead poisoning during childhood. *Br J Industrial Med* 1993;50:613-22.
41. Fergusson D, Horwood J, Lynskey M. Early dentine lead levels and educational outcomes at 18 years. *J Child Psychol Psychiatry* 1997;38:471-8.
42. Tong S, Baghurst P, Sawyer M, et al. Declining blood lead levels and changes in cognitive function during childhood. *JAMA* 1998;280:1915-9.
43. Tong S. Lead exposure and cognitive development: persistence and a dynamic pattern. *J Paediatr Child Health* 1998;34:114-8.
44. Fergusson D, Horwood J. The effects of lead levels on the growth of word recognition in middle childhood. *Int J Epidemiol* 1993;22:891-7.
45. Sameroff A, Seifer R, Baldwin A, et al. Stability of intelligence from preschool to adolescence: the influence of social and family risk factors. *Child Dev* 1993;64:80-97.
46. Winneke G, Beginn U, Ewert T, et al. Comparing the effects of perinatal and later childhood lead exposure on neuropsychological outcome. *Environ Res* 1985;38:155-67.
47. Needleman H, Gatsonis C. Low-level lead exposure and the IQ of children. *JAMA* 1990;263:673-8.
48. Winneke G, Kraemer U. Neuropsychological effects of lead in children: interactions with social background variables. *Neuropsychobiology* 1984;11:195-204.
49. Lansdown R, Yule W, Urbanowicz MA, et al. The relationship between blood-lead concentrations, intelligence, attainment and behavior in a school population: the second London Study. *Int Arch Occup Environ Health* 1986;57:225-35.

50. Dietrich K, Krafft K, Bornschein R, et al. Low-level fetal lead exposure effect on neurobehavioral development in early infancy. *Pediatrics* 1987;80:721-30.
51. Bellinger D, Leviton A, Waternaux C, et al. Low-level lead exposure, social class, and infant development. *Neurotoxicol Teratol* 1988;10:497-503.
52. McMichael A, Baghurst P, Vimpani G, et al. Sociodemographic factors modifying the effect of environmental lead on neuropsychological development in early childhood. *Neurotoxicol Teratol* 1992;14:321-7.
53. Rabinowitz MB, Wang JD, Soong WT. Lead and classroom performance at seven primary schools in Taiwan. *Res Human Capital Development* 1993;7:253-72.
54. Bellinger D, Leviton A, Sloman J. Antecedents and correlates of improved cognitive performance in children exposed in utero to low levels of lead. *Environ Health Perspect* 1990;89:5-11.
55. Ruff H, Bijur P, Markowitz M, et al. Declining blood lead levels and cognitive changes in moderately lead-poisoned children. *JAMA* 1993;269:1641-6.
56. Rogan WJ, Dietrich KN, Ware JH, et al. Succimer chelation and neuropsychological development in lead-exposed children. *N Eng J Med* 2001;344:1421-6.
57. World Health Organization. *A Critical Link: Interventions for Physical Growth and Psychological Development: A Review*. Department of Child and Adolescent Health and Development. Geneva: World Health Organization; 1999.
58. Barnett S. Long-term effects of early childhood programs on cognitive school outcomes. *Future Child* 1995;5:25-50.
59. Ramey C, Ramey S. Early intervention and early experience. *Am Psychol* 1998;53:109-20.
60. Nurcombe B, Rauh V, Howell DC, et al. An intervention program for the mothers of low birthweight infants: The outcome at six and twelve months of the Vermont Infant Studies Project. In: Call JD, Galenson E, Tyson RL (eds). *Frontiers of Infant Psychiatry, vol. II*. New York: Basic Books; 1984.
61. Rauh V, Achenbach T, Nurcombe B, et al. Minimizing adverse effects of low birthweight: four-year results of an intervention program. *Child Dev* 1988;59:544-53.

62. Rauh V, Nurcombe B, Achenbach T, et al. The Mother-Infant Transaction Program: the content and implications of an intervention for the mothers of low-birthweight infants. *Clin Perinatol* 1990;17:31-45.
63. Achenbach T, Phares V, Howell C, et al. Seven-year outcome of the Vermont Intervention Program for Low-Birthweight Infants. *Child Dev* 1990;61:1672-81.
64. Achenbach T, Howell C, Aoki M, et al. Nine-year outcome of the Vermont Intervention Program for Low-Birth Weight Infants. *Pediatrics* 1993;91:45-55.
65. Infant Health and Development Program. Enhancing the outcomes of low-birth-weight, premature infants: a multisite, randomized trial. *JAMA* 1990;263:3035-42.
66. Brooks-Gunn J, McCarton C, Casey P, et al. Early intervention in low-birth-weight infants: results through age 5 years from the Infant Health and Development Program. *JAMA* 1994;272:1257-62.
67. McCarton C, Brooks-Gunn J, Wallace I, et al. Results at age 8 years of early intervention for low-birth-weight premature infants. *JAMA* 1997;277:126-32.



## **Chapter 6. Educational Interventions for Caregivers**

Prepared by James Campbell, MD, MPH, and Michael Weitzman, MD



**Table 6.1. Summary of Recommendations for Educational Interventions for Caregivers**

**General Considerations**

- Tailor educational interventions to each child and caregiver.
- Repeat educational interventions as needed.

**Environmental Interventions**

- Provide information about potential sources of lead identified during environmental investigations.
- Explain that lead abatement should be conducted by certified professionals.
- Discuss and demonstrate the following methods that caregivers can use to reduce their child's lead exposure:
  - Create barriers between living/play areas and lead sources.
  - Regularly wash children's hands and toys.
  - Regularly wet mop floors and wet wipe window components.
  - Vacuum carpeted areas before wet mopping floors; cover carpeted floors with throw rugs.
  - Leave shoes at the door. Use entryway mats.
  - Prevent children from playing in soil. If possible, provide sandboxes.
  - Consider relocation if lead contamination is extensive and not easily remediable.
- Discuss with caregivers potential water hazards only if appropriate.
  - Do not cook with or allow children to drink hot tap water.
  - Run the tap water cold for 1-2 minutes in the morning and then fill a pitcher with the water for drinking, cooking, and formula preparation.
  - Use bottled water if drinking water is contaminated.

**Nutritional Interventions**

- Discuss dietary interventions.
- Encourage caregivers to provide children with foods rich in absorbable iron, vitamin C, and calcium.

**Medical Care**

- Discuss the importance of recommended medical follow-up, including the importance of notifying the case manager if the family moves.
- Review the nature of and risks associated with elevated blood lead levels (EBLLs).

## Introduction

The 1990s witnessed dramatic declines in children’s mean blood lead levels (BLLs) and in the percent of children with elevated blood lead levels (EBLLs) (1). During that decade, we learned a lot about children’s exposure to lead in and around their homes, and about how to reduce that exposure through environmental interventions and caregiver education and counseling. In this chapter, we provide current recommendations for educational interventions, review the quality of evidence that supports these recommendations, and identify research needed to improve the effectiveness of caregiver education. Much of the relevant research is discussed in more detail in Chapter 2, “Assessment and Remediation of Residential Lead Exposure.”

The efficacy of most interventions has not been studied in isolation. Studies usually involved multiple interventions, thus limiting our understanding of the utility of individual recommendations.

## Sources and Pathways of Residential Lead Exposure

Leaded paint is the most common high-concentration source of lead for children and is typically seen in homes built prior to 1950. Poorly maintained older homes with deteriorating paint or those undergoing renovation, whether they are the children’s primary residences or secondary sites where children spend much time, pose the highest risk of lead exposure. The usual sites of deteriorating leaded paint are interior painted surfaces, particularly those subject to abrasion such as window components, and exterior surfaces like siding and porches. The paint chinks or chips off from normal wear-and-tear and deteriorates into dust. Leaded dust can also be created by improperly conducted abatement (2). Soil is another significant source of lead for some children (3). Exterior soil can become very contaminated with lead from deteriorating overlying leaded paint, driplines, or lingering fall-out from previously used leaded gasoline, especially along heavily traveled roads.

Children typically ingest leaded dust as a consequence of age-appropriate hand-to-mouth activity. Studies consistently show an association between the amount of lead on children’s hands and their BLLs (4, 5). Children are also exposed by intentionally ingesting paint chips, dust, or soil. Housing and soil sources are the most common cause of EBLLs in children. Additional significant sources of lead in certain communities include water, industrial contamination, folk medicines, and imported cosmetics or pottery (6-8). In addition, as shown in Appendix I, numerous less common lead sources may be the cause of individual cases of EBLLs.

## General Principles

Educational interventions are directed at helping caregivers reduce the exposure of children to residential and other sources of lead. While most children are exposed through the

deterioration of leaded paint, they may also be exposed to lead from other sources; some of these exposures are a consequence of cultural practices or caregiver occupations or hobbies. Case managers should therefore select the information and interventions that are most appropriate to each child, family, and community and avoid overwhelming caregivers with interventions that may be of little or no benefit.

Although there is no risk-perception or risk-communication research specific to childhood lead poisoning, general principles of these fields can be applied to improve the effectiveness of educational interventions to reduce children's BLLs. Case managers must recognize that caregivers understand the "risk" of EBLs in ways different from the ways that experts in lead poisoning understand them, and case managers should tailor their recommended interventions to caregivers' conceptions of risks. If interventions are not tailored to caregivers' conceptions of risks, then caregivers are less likely to act on the information they receive (9, 10).

In addition to educating caregivers about childhood lead poisoning, case managers may also need to provide detailed instructions on intervention techniques, actually demonstrate the techniques, and then ask caregivers to perform the techniques themselves. Such actions should increase caregivers' understanding of the interventions and consequently increase the chances that the interventions will be successful.

## Studies of Various Interventions

### *Interventions to reduce children's lead exposure from residential deteriorating paint*

Interventions to reduce children's exposure to deteriorating paint in their homes include the safe repair of non-intact leaded paint, the safe repair or replacement of windows or other building components to prevent abrasion of leaded paint, and the safe removal (stripping) of leaded paint from components left in the home. In a review of uncontrolled studies involving children with baseline BLLs greater than 25 Fg/dL, the EPA found that BLLs of children in homes where non-intact leaded paint was safely removed or repaired declined 20% to 30% over the following year (11). In one controlled study, the mean BLL of children in treated dwellings declined twice as much as that of children in untreated dwellings (12).

### *Interventions to reduce children's lead exposure from residential dust*

Four clinical trials assessed the efficacy of household dust control by professional cleaners (13-17). Three trials assessed the effectiveness of household dust control done by the families of children with EBLs: two randomized clinical trials (18-20), and one nonrandomized, retrospective analysis with a comparison group (21, 22).

Among the studies of professional house dust control, two (13, 17) found that children in homes that underwent intensive dust-control (i.e., two trained cleaners wet mopping floors and

wet wiping horizontal surfaces for 2 hours every 2-3 weeks) had a 17% - 18% decrease in their mean BLL 1 year after the initial test. In one of these studies (17), a subgroup of children whose homes were cleaned 20 or more times over the year (a mean of once every 2.6 weeks) had a 34% decrease in their BLLs. Since trained cleaners conducted the interventions, this effect size is probably the optimum that can be achieved. The remaining studies (14-16) failed to show that dust control is associated with a decrease in children's BLL. Two of these studies were of a one-time intervention (14, 15), and the other was of cleaning done every 6 weeks (16). However, one-time or infrequent interventions would most likely not prevent EBLs, because household dust builds up again after a short time. This is suggested by Hilts et al., who found that children's lead loading returned to baseline levels 3 weeks after high-efficiency particulate air (HEPA) vacuuming (16). Similarly, Rhoads et al. found no change in the mean BLL of children whose homes were cleaned fewer than 10 times over the year (at most every 5.6 weeks) (17).

Lanphear et al. conducted two trials in which the cleaning was done by the caregivers (18-20). In the first, 104 children (aged 12 to 31 months; BLLs 1.7 to 30.6 Fg/dL) were randomly assigned to an intervention group (in which caregivers received cleaning supplies, were educated about areas likely to be contaminated with lead, and were instructed to clean monthly) or to a control group (in which caregivers received only a brochure about preventing EBLs) (18). Seven months after enrollment, the median change in children's BLL was -0.05 Fg/dL in the intervention group and -0.60 Fg/dL in the control group ( $p=0.50$ ). However, in this study, the researchers could not ensure that the families adhered to the recommended cleaning regimen.

The second trial involved 275 children with a mean baseline BLL of 2.8 Fg/dL (19, 20). These children were randomly assigned to either an intervention group that received education, cleaning supplies, and up to eight home visits by an advisor, or to a control group that did not receive any of these interventions. Again, researchers found no significant differences in the geometric mean BLLs of children in the two groups at 12, 18, 24 and 48 months of age. But again, they could not ensure that the families adhered to the recommended regimen.

Schultz et al. conducted a retrospective analysis of an in-home educational intervention (21, 22). Health department staff visited the homes of children (mean age 3.4 years) with BLLs 20 to 24 Fg/dL and conducted an educational session for caregivers regarding lead sources, methods to reduce children's exposure to these sources, and appropriate nutrition for children. The children in visited homes made up the study group. A reference group was made up of children (comparable with the study group by age, sex, race, and BLL) who did not receive the educational intervention. Follow-up BLLs were obtained about 6 months after the intervention. The study group children had a significantly greater mean decline in BLL (4.2 Fg/dL) than the reference group children (1.2 Fg/dL,  $P<0.001$ ). The authors concluded that home educational visits may have helped lower children's BLLs (22). However, they also noted that "[t]he validity of this conclusion depends upon whether children who received the visits were comparable to reference group children whose families were often unavailable for outreach visits. Families that were unavailable...may have been more likely to exhibit behavior patterns responsible for the

continued elevation of their children's blood lead levels" (22). Thus, with no randomization of subjects, the reference group may not have been comparable in at least one important way.

In a meta-analysis, the findings of several studies were combined to determine the effect of dust control on children's BLL (23). To be eligible for analysis, the studies had to be randomized controlled trials, cost less than \$2,500, and be conducted in a community without a continual lead emission source, such as a lead smelter. Five studies were eligible (15, 17-20). Results of the meta-analysis showed no significant post-intervention differences in mean BLLs between children in the intervention and control groups. However, the intervention groups contained significantly fewer children with BLLs  $\geq 15$  Fg/dL and  $\geq 20$  Fg/dL than did the control groups. For example, only 1.8% of children in the intervention groups had BLLs  $\geq 20$  Fg/dL, whereas 5.3% of those in the control group did (OR=0.29, CI 0.01 - 0.85,  $p=0.024$ ). This finding persisted even after the single study involving professional dust control (17) was removed from the analysis.

The aforementioned studies largely focused on cleaning dust on uncarpeted floors. Although the dust lead loading on uncarpeted floors has a higher correlation with children's BLLs than the dust lead loading on carpets, dust lead loading on carpets does correlate with children's BLLs (24). However, neither HEPA vacuums nor common household vacuums reduced carpet dust lead levels by clinically relevant amounts (16, 25, 26). Furthermore, in one study, children whose homes were HEPA vacuumed actually had higher levels of lead on their hands after the interventions although their BLLs did not change (16). The authors speculate this may have occurred because families who received the vacuuming "...may have relaxed their hygiene efforts...because of a perceived reduction in exposure risk" (16). A report that HEPA vacuuming increased the lead loading on the surface of the carpet by bringing lead from deep in the carpet to the surface (25) offers an alternative explanation for the increase in hand lead levels.

In summary, studies indicate that household dust control performed by professional cleaners is associated with decreases in children's mean BLL, although it appears that to be effective, such dust control must be conducted at least every 2 to 3 weeks. However, simply educating parents of the need to perform dust control has not proven effective in reducing children's mean BLL.

#### *Interventions to reduce children's lead exposure by improving personal hygiene practices*

We found no controlled studies that examined the effect of personal hygiene on BLLs of children, although studies of the correlation between the level of lead on children's hands and their BLLs have consistently found an association between the two (6, 7, 27, 28). Although the frequency of self-reported hand washing has not been associated with children's BLLs (27, 28), the validity of study results based on such self-reported hygiene measures is clouded by the possible effects of social desirability bias.

While there is no evidence that hand washing is associated with a decrease in children's BLLs, it is a simple intervention that poses no risks.

*Interventions to reduce children's lead exposure from residential soil*

The major study examining whether soil abatement is efficacious at reducing children's BLLs, the Urban Soil Lead Abatement Demonstration Project (29), was conducted in three cities: Boston, Baltimore, and Cincinnati.

In Boston, Weitzman et al. studied the effects of paint, dust, and soil lead abatement on the BLLs of 152 children (mean age: 31.6 months; mean baseline BLL: 12.5 Fg/dL; and median surface soil lead level: 2075 ppm) (14). Eleven months after soil lead abatement, the adjusted mean BLL of children in homes having the abatement dropped to 10.26 Fg/dL, and that of control children dropped to 11.54 Fg/dL ( $p=0.02$ ). However, despite the statistical significance, the authors concluded that these differences were clinically irrelevant. In a follow-up of these children for an additional year, they found that soil lead abatement was associated with a 2.25 to 2.70 Fg/dL decline in the children's BLL, but that children who lived in dwellings with consistently elevated floor levels of leaded dust derived no benefit from the soil abatement (30).

In Baltimore, Farrell et al. randomly assigned 408 children (aged 6 to 72 months) to either an intervention group (whose homes underwent exterior paint stabilization followed by soil abatement) or a control group (whose homes underwent exterior paint stabilization but no soil abatement) (31). The children's mean BLL was about 11 Fg/dL. At baseline, only 54% of properties had soil samples with a lead concentration above 1000 ppm. Ten to 13 months after the intervention, the geometric mean BLL of the treatment group was unchanged, while that of the control group had fallen 0.7 Fg/dL (29). Results of multivariate analysis showed no significant difference in the mean BLL of the groups at follow-up.

In the Cincinnati trial, researchers studied the effects of soil lead abatement on the BLLs of 206 children (aged 9 to 72 months; median BLL 10 Fg/dL) by assessing changes in the children's median BLLs 9 to 10 months after the interventions. Through multivariate analysis, they found no significant difference in the mean BLL of children in households receiving and households not receiving soil lead abatement.

There are a number of possible explanations for why these studies of soil abatement showed no effect. First, most of the interventions were performed in scattered homes rather than contiguous blocks of homes, so continued exposure to lead from nearby properties may have limited the effectiveness of the interventions. Second, the studies enrolled children whose sources of lead exposure were primarily from their homes rather than children whose sources were primarily from soil (i.e., those who avidly played in or ingested soil). Finally, the release of lead from children's bones may have attenuated the impact of the interventions (32).

The EPA concluded that when soil is a significant source of lead for a child, the lead abatement of that soil is associated with a reduction in that child's BLL (29). However, the mean

reductions in children's BLLs do not appear to be clinically relevant. Further, an economic analysis concluded that soil lead abatement was not cost-effective (33). Therefore, we do not recommend residential soil lead abatement in the secondary prevention of children's EBLs. Nevertheless, because some children may experience significant lead exposure from soil either because they play in or ingest soil or because their soil has high levels of lead, we do recommend simple, safe measures such as providing sandboxes with covers or covering open soil with grass or mulch.

### *Nutritional Interventions*

Although the effects of various nutritional interventions on children's BLLs are either limited or have not been studied, certain interventions are of value to the children's general health, because many children with EBLs are at risk for poor nutrition. See Chapter 4, "Nutritional Assessment and Interventions," for a detailed discussion.

## **Recommendations**

### *General Recommendations*

*Tailor educational interventions to each child and caregiver.*

Select the interventions and information that are most appropriate to the child. Devise a written plan with specific recommendations to reduce the child's exposure to identified sources of lead in consultation with the caregivers and give a copy of the plan to them.

*Continue educational efforts beyond a one-time intervention.*

Monitor children's follow-up BLLs. If a child's BLL is not decreasing, discuss the case with the primary care provider (PCP) and, if appropriate, an environmental health specialist, to determine whether lead sources are being overlooked. Case managers may need to make further home visits to assess new lead sources and ensure that caregivers understand and are carrying out recommended interventions.

### *Environmental Recommendations*

Prompt and effective control of the sources of children's lead exposure is the highest priority. Ensure that all sites lived in or regularly visited by a child with an EBL are inspected jointly with the caregiver to identify potential sources of lead exposure.

*Provide information about potential sources of lead.*

If caregivers are informed of lead sources identified during the environmental inspection, as well as other potential sources (Appendix I), they may change their attitudes and behaviors in ways that result in secondary prevention. Therefore, encourage caregivers to examine their yards and homes for chipping paint, especially areas where their child spends a good deal of time, and to alert lead inspectors to areas that may be potential sources of exposure.

*Explain that lead abatement should be conducted by trained workers.*

Improperly conducted lead abatement (e.g., grinding or sanding lead-based paint and thus producing lead dust, or allowing children access to areas of abatement) may actually increase children's lead exposure (34, 35). Therefore, recommend that abatement be conducted by certified professionals. However, if caregivers choose to conduct lead abatement themselves, direct them to resources that will at least give them guidance in how to conduct lead abatement safely (Appendix II).

*Discuss and demonstrate methods that caregivers can implement to reduce their children's lead exposure.*

While verbal instructions and written materials are useful, it is important to demonstrate methods of reducing children's lead exposure whenever possible. Demonstrating these methods at the child's home can help in overcoming language and cultural barriers. Encouraging caregivers to practice the methods demonstrated and provide corrective feedback if necessary should help them better understand and adhere to the recommended interventions. Although many of these interventions have not been studied in isolation or shown to be effective, most are simple interventions that pose no risk and should help reduce children's risk for lead exposure.

- *Create barriers between living/play areas and lead sources.* Leaded paint tastes sweet, which may encourage children to ingest deteriorating paint. Until abatement is completed, caregivers should clean and/or isolate all sources of lead. Advise them to close and lock doors to keep children from deteriorated paint on walls and to use temporary barriers such as contact paper or duct tape to cover holes in walls or to block children's access to other sources of lead.
- *Regularly wash children's hands and toys.* Hands and toys can become contaminated from household dust or exterior soil, both known reservoirs of lead. Washing a child's hands may also enhance caregiver-child interaction and reduce the transmission of infectious diseases. Urge caregivers to buy toys that can easily be washed.
- *Regularly wet mop floors and wet wipe window components.* Because household dust is a major source of lead, advise caregivers to wet mop floors and wet wipe horizontal

surfaces every 2-3 weeks until all of their child's hand-to-mouth behaviors cease. Since windowsills and wells can contain high levels of leaded dust, they should be kept clean and, if feasible, shut to prevent abrasion of painted surfaces. Advise caregivers to use disposable cleaning materials or reusable materials used only for cleaning. The EPA recommends the use of a general-purpose, nonphosphate cleaner (36). In studies that found house dust control to be associated with a decrease in children's mean BLL, professional house cleaners used a powdered detergent rather than bleach or ammonia (13, 17).

- *Vacuum carpets before wet mopping floors; cover carpeted areas with throw rugs.* Vacuuming may increase children's lead exposure by bringing lead-contaminated dust from deep in the carpet to its surface. Therefore, advise caregivers to initially vacuum carpeted floors and subsequently wet clean the carpets. After cleaning the carpets, caregivers should wet mop noncarpeted floors to remove dust aerosolized by vacuuming. Advise caregivers to place throw rugs over carpeted children's play areas and to consider replacing the carpet if it is extremely contaminated with dust.
- *Leave shoes at the door; use entryway mats.* Contaminated exterior soil can be tracked into homes on shoes.
- *Prevent children from playing in soil; if possible, provide them with sandboxes.* Do not recommend residential soil abatement, but do advise caregivers to limit their children's play in bare soil. Also advise them to either plant grass on areas of bare soil or cover the soil with grass seed, mulch, or wood chips if possible. Until the bare soil is covered, advise caregivers to move play areas away from bare soil and away from the perimeter of the house. Sandboxes with covers can provide an alternative place for children to play; when not in use, sandboxes should be covered.
- *Consider relocation.* If lead contamination is extensive and not easily remediable, advise caregivers to consider moving to another home. Case managers should be knowledgeable about lead-safe housing where families can be temporarily or permanently placed.

*Discuss the potential for lead-contaminated water, if appropriate.*

Water sources can become contaminated with lead from household pipes made of lead or harboring leaded solder (37). The local health authority will know if this is a prevalent community-wide problem. See Chapter 2, "Assessment and Remediation of Residential Lead Exposure," for a detailed discussion of contamination in municipal or well water. If household water is a suspected source of lead exposure, advise caregivers to implement the following interventions pending the results of water testing:

- *Do not drink or cook with hot tap water.* Lead is more soluble in warm water.
- *Run the tap water cold for 1-2 minutes in the morning, and then fill a pitcher with the*

*water.* The water is then available that day for drinking, cooking, and formula preparation. Although the benefit of regularly running the tap before consuming water has not been studied in isolation, this is a simple intervention that poses no risk.

- *If drinking water in a child's home is contaminated with lead, advise caregivers to use only bottled water until household water lead levels have been corrected.* However, since most bottled water does not contain fluoride, fluoride supplementation may be necessary. For more information on bottled water, contact the United States Food and Drug Administration (301-443-4166); NSF International, an organization that certifies bottled water and water filters (313-769-5106); or the International Bottled Water Association (703-683-5213).

### *Nutritional Recommendations*

*Discuss dietary interventions.*

- Recommend that caregivers provide children with foods rich in absorbable iron, vitamin C, and calcium. Foods such as red meat and iron-enriched cereals are good sources of absorbable iron. Adding foods to a meal that are rich in vitamin C (e.g., fruit juice) can dramatically increase iron absorption. Two servings per day of dairy products are recommended. Unless the child does not ingest dairy products because of lactase deficiency, do not suggest calcium supplements, as they can be contaminated with lead (38). Both iron deficiency and EBLs are common among children of low-income families (39, 40), so providing iron-rich foods to children with EBLs would contribute to the treatment of iron deficiency. (See Chapter 4, "Nutritional Assessment and Interventions," for details.)
- Recommend that caregivers provide regular meals and snacks. In one study of five adults, a higher proportion of lead was absorbed when it was given to people when they were fasting (41). Therefore, encourage caregivers to provide three meals and two snacks (during midafternoon and at bedtime) a day. Refer eligible families to food supplementation programs such as the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC).

### *Medical Recommendations*

*Discuss the importance of regular medical follow-up.*

Follow-up blood tests are the best way to determine the success of environmental and other interventions. Therefore, remind caregivers to:

- Make and keep follow-up appointments for blood tests. When making appointments, follow the schedule in Table 3.4 of Chapter 3, "Medical Assessment and Interventions."
- Notify the case manager if the child moves to a new residence.

- Inform all current and future health care providers of the child that the child had an EBLL. This is important even when the child's BLL is no longer elevated.

*Review the meaning and risks of EBLs.*

Remind caregivers that:

- Children with EBLs are often asymptomatic.
- Knowing the source of lead is critical to preventing further exposure.
- Neurodevelopmental effects of EBLs are usually not immediately identifiable. Because they may only become apparent after a child is in school, be aware of possible later effects. (See Chapter 5, "Developmental Assessment and Interventions.")

*Recommendations for Caregivers Whose Children's Lead Exposure Is from Nonhousing Sources*

*Describe ways to eliminate work- and hobby-related exposure.*

Household members who are exposed to lead from occupations or hobbies may bring lead into the home on lead-contaminated clothing, shoes, and hair (42, 43). A list of occupations and hobbies associated with home lead exposure can be found in Appendix I.

For work-related lead sources, advise the caregivers to:

- If possible, reduce their lead exposure in the workplace.
- Shower before leaving work.
- Change clothes before going home and leave soiled clothing at work to be laundered by the employer. If this is not possible, change clothes in an area at home that is inaccessible to children.
- Store street clothes in separate areas of the workplace to prevent contamination.
- Leave all lead-containing or lead-contaminated material at the workplace.
- Obtain a referral to an occupational health clinic if the caregiver has an EBL.
- Prevent children from visiting the work area.

For hobby-related lead sources, advise the caregivers to:

- Separate hobby areas from living areas.
- Prevent children from visiting hobby areas.
- Have anyone engaging in "lead hobbies" change clothes either before entering the home or in an area that is inaccessible to children.
- Wash contaminated clothing separately from the rest of the family laundry.
- Properly store and dispose of toxic substances.

*Discuss the hazards of food containers, folk remedies, or cosmetics contaminated with lead.*

Items that may be associated with lead exposure are listed in Appendix I.

- Advise caregivers not to use containers, cookware, or tableware purchased abroad to store or cook foods or liquids unless they are shown to be lead-free.
- Advise caregivers not to use folk remedies and cosmetics purchased abroad unless they are shown to be lead-free.

### Recommendations for Future Research

Although dust control performed by trained cleaners has been shown to reduce children's mean BLLs (13, 17), simply educating families on the need to perform dust control does not attain the same results. Further research on how to motivate families to perform regular and effective cleaning is important.

Reports indicate that, in the absence of interventions to reduce ongoing contamination of dust from disintegrating paint, the effect of dust control on children's BLLs is modest (17, 18, 20, 44). However, randomized trials examining the effects of a multifactor intervention involving dust control, nutritional supplementation, and behavioral modification on children's BLLs would be of value.

In other areas of environmental health, a great deal has been learned about ways in which different people view risks, methods of risk reduction, and barriers to addressing risks. Despite this increased understanding of the scientific basis of risk perception and communication in the past 20 years (45), no studies of risk perception or communication have been conducted among caregivers of children with EBLLs. In order to develop more effective educational and risk-reduction strategies to combat EBLLs in children, health officials need better information about what people think about lead hazards and why they think that way. Methods such as mental modeling (46) and value integration (47) would be very valuable approaches to obtaining such information.

Soil lead abatement is costly and has not been associated with clinically significant reductions in BLLs. However, studies are needed to assess the effectiveness and costs of using barriers such as grass, shrubbery, or cement to protect children with high soil lead exposures. Research is also needed on the efficacy of various barriers, such as wallpaper or paneling, in protecting children from exposure inside their homes.

Since the half-life of lead in the blood can be up to 38 months (48), children must be followed for prolonged periods to determine whether their BLLs are decreasing and whether interventions have been effective. Decreases in dust lead levels or hand lead levels might be used as intermediate, proxy measures of children's lead exposure if we could develop practical, inexpensive, and reliable methods of using such assays in a clinical setting.

**References**

1. Pirkle JL, Kaufmann RB, Brody DJ, et al. Exposure of the U.S. population to lead. *Environ Health Perspect* 1998;106:745-50.
2. Rey-Alvarez S, Menke-Hargrave T. Deleading dilemma: pitfall in the management of childhood lead poisoning. *Pediatrics* 1987;79:214-7.
3. Mielke HW, Reagan PL. Soil is an important pathway of human lead exposure. *Environ Health Perspect* 1998;106(Suppl 1):217-29.
4. Sayre JW, Charney E, Vostal J, et al. House and hand dust as a potential source of childhood lead exposure. *Am J Dis Child* 1974;127:167-70.
5. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. *Environ Res* 1998a;79:51-68.
6. Matte TD, Proops D, Palazuelos E, et al. Acute high-dose lead exposure from beverage contaminated by traditional Mexican pottery. *Lancet* 1994;344:1064-5.
7. Ng R, Martin DJ. Lead poisoning from lead-soldered electric kettles. *CMA* 1977;116:508-12.
8. Jones TF, Moore WL, Craig AS, et al. Hidden threats: lead poisoning from unusual sources. *Pediatrics* 1999;104:1223-5.
9. National Research Council. *Improving Risk Communication*. Washington, DC: National Academy Press; 1989.
10. Bennett R, Calman K (eds). *Risk Communication and Public Health*. Oxford: Oxford University Press; 1999.
11. Environmental Protection Agency (EPA). *Review of Studies Addressing Lead Abatement Effectiveness: Updated Edition*. Washington, DC: EPA; 1998a. EPA 747-B-98-001.
12. Staes C, Matte T, Copley CG, et al. Retrospective study of the impact of lead-based paint hazard remediation on children's blood lead levels, St. Louis. *Am J Epidemiol* 1994;139:1016-26.

## Chapter 6. Educational Interventions for Caregivers

13. Charney E, Kessler B, Farfel M, et al. A controlled trial of the effect of dust-control measures on blood lead levels. *N Engl J Med* 1983;309:1089-93.
14. Weitzman M, Ashengrau A, Bellinger D, et al. Lead-contaminated soil abatement and urban children's blood lead. *JAMA* 1993;269:1647-54.
15. Aschengrau A, Hardy S, Mackey P, et al. The impact of low technology lead hazard reduction activities among children with mildly elevated blood lead levels. *Environ Res* 1998;79:41-50.
16. Hilts SR, Hertzman C, Marion SA. A controlled trial of the effect of HEPA vacuuming on childhood lead exposure. *Can J Public Health* 1995;86:345-50.
17. Rhoads GG, Ettinger AS, Weisel CP, et al. The effect of dust control on blood lead in toddlers: a randomized trial. *Pediatrics* 1999;103:551-5.
18. Lanphear BP, Winter NL, Apetz L, et al. A randomized trial of the effect of dust control on children's blood lead levels. *Pediatrics* 1996a;98:35-40.
19. Lanphear BP, Howard C, Eberly S, et al. Primary prevention of childhood lead exposure: a randomized trial of dust control. *Pediatrics* 1999;103:772-77.
20. Lanphear BP, Eberly S, Howard CR. Long-term effect of dust control on blood lead levels. *Pediatrics* 2000;106. Available at: <http://www.pediatrics.org/cgi/content/full/106/4/e48>. Accessed 12/19/01.
21. Schultz B, Pawel D, Murphy A. A retrospective examination of in-home educational visits to reduce childhood lead levels. *Environ Res* 1999;80:364-8.
22. Environmental Protection Agency (EPA). Effect of In-Home Educational Intervention on Children's Blood Lead Levels in Milwaukee. Washington, DC: EPA; 1996. EPA 747-R-95-009.
23. Haynes E, Lanphear BP, Tohn E, et al. The effect of interior lead hazard controls on children's blood lead concentrations: a systematic evaluation. *Environ Health Perspect* 2002 Jan;110(1):103-7.
24. Lanphear BP, Weitzman M, Winter NL, et al. Lead-contaminated house dust and urban children's blood lead levels. *Am J Public Health* 1996b;86:1416-21.

25. Ewers L, Clark S, Menrath W, et al. Clean-up of lead in household carpet and floor dust. *Am Ind Hyg Assoc J* 1994;55:650-7.
26. Yiin L, Rhoads GG, Rich DQ, et al. Comparison of lead reduction techniques on carpets and upholstery: the New Jersey assessment of cleaning techniques study (NJACT). Abstracts of the American Public Health Association 2000 Annual Meeting. Available at: [http://apha.confex.com/apha/128am/techprogram/paper\\_10129.htm](http://apha.confex.com/apha/128am/techprogram/paper_10129.htm). Accessed 12/19/01.
27. Lanphear BP, Roghmann KJ. Pathways of lead exposure in urban children. *Environ Res* 1997;74:67-73.
28. Sargent JD, Dalton M, Stukel TA, et al. Evaluation of capillary collection methods for blood lead screening in children. *Ambulatory Child Health* 1995;1:112-22.
29. Environmental Protection Agency (EPA). Urban Soil Lead Abatement Demonstration Project. Volume I: EPA Integrated Report. Washington, DC: EPA; 1996. EPA 600/P-93/001aF.
30. Aschengrau A, Beiser A, Bellinger D, et al. The impact of soil lead abatement on urban children's blood lead levels: phase II results from the Boston Lead-in-Soil Demonstration Project. *Environ Res* 1994;67:125- 48.
31. Farrell KP, Brophy MC, Chisholm JJ Jr, et al. Soil lead abatement and children's blood lead levels in an urban setting. *Am J Pub Health* 1998;88:1837-9.
32. Rust SW, Kumar P, Burgoon DA, et al. Influence of bone-lead stores on the observed effectiveness of lead hazard intervention. *Environ Res* 1999;81:175-84.
33. Glotzer DE, Weitzman M, Aschengrau A, et al. Economic evaluation of environmental interventions for low-level childhood lead poisoning. *Ambulatory Child Health* 1997;3:255-67.
34. Amitai Y, Graef JW, Brown MJ, et al. Hazards of deleading homes of children with lead poisoning. *Am J Dis Child* 1987;141:758-60.
35. Aschengrau A, Beiser A, Bellinger D, et al. Residential leaded paint hazard remediation and soil lead abatement: their impact among children with mildly elevated blood lead levels. *Am J Public Health* 1997;87:1698-702.

36. Environmental Protection Agency (EPA). Lead-Cleaning Efficacy Follow-up Study. Washington, DC: EPA; 1998b. EPA 747-R-98-008.
37. CDC. Preventing lead poisoning in young children. Atlanta, GA: US Department of Health and Human Services, CDC; 1991.
38. Scelfo GM, Flegal AR. Lead in calcium supplements. *Environ Health Perspect* 2000;108:309-13.
39. Pirkle JL, Kaufmann RB, Brody DJ, et al. Exposure of the U.S. population to lead, 1991-1994. *Environ Health Perspect* 1998;106:745-50.
40. Looker A, Dallman PR, Carroll MD, et al. Prevalence of iron deficiency in the United States. *JAMA* 1997;277:973-6.
41. Rabinowitz MB, Kopple JD, Wetherill GW. Effect of food intake and fasting on gastrointestinal lead absorption in humans. *Am J Clin Nutr* 1980;33:1784-8.
42. Gerson M, Van den Eeden SK, Gahagan P. Take-home lead poisoning in a child from his father's occupational exposure. *Am J Industr Med* 1996;29:507-8.
43. Roscoe RJ, Gittleman JL, Deddens JA, et al. Blood lead levels among children of lead-exposed workers: a meta-analysis. *Am J Ind Med* 1999;36:475-81.
44. Lanphear BP. The paradox of lead poisoning prevention. *Science* 1998b;281:1617-8.
45. Fischhoff B. Risk perception and communication unplugged: twenty years of progress. *Risk Analysis* 1995;15(2):137-45.
46. Bostrom A, Fischhoff B, Morgan MG. Characterizing mental models of hazardous process: a methodology and an application to radon. *J Social Issues* 1992;48:85-100.
47. Gregory RS. Valuing environmental policy options: a case study comparison of multiattribute and contingent valuation survey methods. *Land Economics* 2000;76:151-73.
48. Manton WI, Angle CR, Stanek KL, et al. Acquisition and retention of lead by young children. *Environ Res* 2000;82:60-80.

## **Appendixes**



**Appendix I. Published Reports of Less Common Causes of Elevated Blood Lead Levels (EBLLs) in Children.**

<b>Exposure Source</b>	<b>Description/Exposure Pathway</b>	<b>Study Type*</b>	<b>Study Description</b>	<b>Ref. #</b>
Occupational Take Home Exposures				
<i>Battery reclamation</i>	Lead carried home by battery workers. (Only a minority of battery workers showered or changed clothes before going home.)	E	Twelve (75%) of 16 children of lead-exposed workers had EBLLs and a higher average BLL than neighborhood controls (22.4 vs. 9.8 µg/dL, <i>p</i> =.049).	28
<i>Ceramics</i>	Ceramic-coated capacitors made with fritted glass containing lead.	E	Case-control study of 51 children under 6 years (20 exposed, 31 controls) showed higher average BLLs in exposed children (13.4 vs. 7.1 µg/dL, <i>p</i> <.001).	31
<i>Furniture refinishing</i>	Lead carried home by workers who restored furniture that had undergone chemical stripping and was thought to be lead-free.	CR	Report of six workers and three of their children aged 4-18 months.	19
<i>Construction</i>	Lead dust on skin and clothes taken home.	E	Case-control study of 50 children under 6 years (31 exposed, 19 controls) showed 25.8% of workers' children had EBLLs compared to 5.3% of control children (OR=6.1).	53
<i>Radiator repair</i>	Lead carried home by workers who did soldering to repair radiator.	E	The mean BLL for 18 children (under 7 years) of lead-exposed workers was 10 µg/dL.	42, 34

Exposure Source	Description/Exposure Pathway	Study Type*	Study Description	Ref. #
Imported Cosmetics				
<i>Kohl (Middle East, India, Pakistan, some parts of Africa)</i>	A gray or black eye cosmetic applied to the conjunctival margins of the eyes. Can contain up to 83% lead. It is believed to strengthen and protect the eyes against disease. Also known as Al Kohl.	E	A study of 538 girls aged 6 to 12 years demonstrated that the application of kohl was associated with higher BLLs ( $p=0.0461$ ).	3, 37
<i>Pakistani eye cosmetics</i>	Eye cosmetics are often applied to the eyes of children.	E	Retrospective chart review of 175 children aged 8 months to 6 years showed an average BLL of 4.3 $\mu\text{g}/\text{dL}$ for Pakistani/Indian children not using eye cosmetics and 12.9 $\mu\text{g}/\text{dL}$ for those using eye cosmetics ( $p=0.03$ ).	50, 52
<i>Surma (India)</i>	A black fine powder applied to the eyes for medicinal and cosmetic reasons.	E	A case-control study of 62 children demonstrated higher BLLs in children using surma ( $p<.001$ ).	2, 15
Contaminated Foods				
<i>Apple cider</i>	Cider was made in a maple syrup evaporator that had lead solder joining the interior seams.	CR	Report of a 7-year-old child.	8
<i>Flour (Middle East)</i>	Lead fillings used in stone mills contaminated flour.	E	Investigation of 43 symptomatic patients aged zero to 80 years and their families and of 563 children aged 10 to 18 years demonstrated that 33 (23%) of 146 community stone mills had lead contamination and that 171 (30.4%) of 563 children had BLLs exceeding 30 $\mu\text{g}/\text{dL}$ .	25

Exposure Source	Description/Exposure Pathway	Study Type*	Study Description	Ref. #
<i>Lozeena</i>	An orange powder used to color rice and meat that contains 7.8%-8.9% lead.	CR	Report of brothers aged 2 and 3 years and their parents. In addition, 9 of 18 extended family members had EBLLs.	15
<i>Infant formula</i>	Infant formula was made with contaminated tap water from copper pipes with lead solder.	CR	Report (with environmental sampling data) of a 13-month-old child.	46, 47
<i>Tamarind candy (Mexico)</i>	Tamarind candy jam products from Mexico. During the manufacturing process, the candied jam is packaged in stoneware or terra cotta ceramic jars that can leach lead.	CR	Report of two children under 6 years old, six older children, and one adult.	14
<b>Food and Beverage Containers</b>				
<i>Bulk-water storage tank</i>	Lead leached from soldered seams and brass fittings in bulk-water storage tanks.	CR	Report of three children aged 6, 12, and 14 months.	11
<i>Ceramic glaze</i>	Lead in ceramic glaze can leach into stored beverages, especially juices since they are acidic. The risk is highest for improperly fired containers.	CR	Multiple reports.	7, 12, 49
<i>Cocktail glass</i>	Lead leached from cocktail glass.	CR	Report of a family with one adult and children aged 4, 5, and 14 years.	22
<i>Iranian urn (samovar)</i>	Lead spot solder from the original manufacturing process leached into water used to make baby formula.	CR	Reports of a 10-week-old child with seizures and of a 4-month-old child.	33, 48

Exposure Source	Description/Exposure Pathway	Study Type*	Study Description	Ref. #
<i>Lead-soldered kettle</i>	Lead leached into infant formulas.	CR	Reports of a 3-month-old child and of a 1-day-old child.	40
<i>Azarcon</i>	Also known as <b>alarcon, coral, luiga, maria luisa</b> , or <b>rueda</b> . Bright orange powder used to treat empacho (an illness believed to be caused by something stuck in the gastrointestinal tract, resulting in diarrhea and vomiting). Azarcon is 95% lead.	E	Report of 15-month-old and 3-year-old siblings who expired with seizures and a subsequent survey of 545 systematically selected households for azarcon and greta usage.	16, 15, 18, 4, 51
<i>Ayurvedic medicine (Tibet)</i>	Unnamed traditional medicine.	CR	Single case.	15
<i>Ba-Baw-San (China)</i>	Herbal medicine used to treat colic pain or to pacify young children.	E	Study of 319 children aged 1 to 7 years demonstrated that consumption was associated with increased BLLs ( $p=.038$ ).	21
<i>Bint Al Zahab (Iran)</i>	Rock ground into a powder and mixed with honey and butter given to newborn babies for colic and early passage of meconium after birth.	CR	Report of six children aged 2 days to 3 months.	44
<i>Bint Dahab (Saudi Arabia; means "daughter of gold")</i>	A yellow lead oxide used by local jewelers and as a home remedy.	CR	Report of 10 children aged 7 days to 13 months, including three who took bint dahab.	35

Exposure Source	Description/Exposure Pathway	Study Type*	Study Description	Ref. #
<i>Bokhoor (Kuwait)</i>	A traditional practice of burning wood and lead sulphide to produce pleasant fumes to calm infants.	CR	Report of four children aged 16 days to 4.5 months.	27
<i>Ghasard</i>	Brown powder used as a tonic to aid in digestion.	CR	Report of a 9-month-old child who died.	17
<i>Greta (Mexico)</i>	Yellow powder used to treat empacho (see <b>azarcon</b> ); can be obtained through pottery suppliers, as it is also used as a glaze for low-fired ceramics. Greta is 97% lead.	E	See azarcon.	4, 16, 18, 51
<i>Jin Bu Huan (China)</i>	An herbal medicine used to relieve pain.	CR	Report of three children aged 13 and 23 months and 2.5 years.	10
<i>Pay-loo-ah (Vietnam)</i>	A red powder given to children to cure fever or rash.	CR	Report of a 6-month-old child.	13, 15
<i>Po Ying Tan (China)</i>	An herbal medicine used to treat minor ailments in children.	CR	Report of a 4-month-old child.	20
<i>Santrinj (Saudi Arabia)</i>	An amorphous red powder containing 98% lead oxide used principally as a primer for paint for metallic surfaces, but also as a home remedy for “gum boils” and “teething.”	CR	Report of 10 children aged 7 days to 13 months, including 7 who took santrinj.	35
<i>Surma (India)</i>	Black powder used as a cosmetic and as teething powder.	E	A case-control study of 62 children demonstrated higher BLLs in children using surma (p<.001).	2, 15
<i>Tibetan herbal vitamin</i>	Used to strengthen the brain.	CR	Report of a 5-year-old child.	38

Exposure Source	Description/Exposure Pathway	Study Type*	Study Description	Ref. #
<i>Traditional Saudi medicine</i>	Orange powder prescribed by a traditional medicine practitioner for teething; also has an antidiarrheal effect.	CR	Report of three children aged 11, 22, and 44 months.	1
Miscellaneous				
<i>Automobile key-chain emblem</i>	Ingestion of lead-containing automobile key-chain emblem.	CR	Report of a 23-month-old child.	5
<i>Clothing accessory</i>	Ingestion of a "simulated watch."	CR	Report of 3-year-old child who required endoscopy.	26
<i>Curtain weights</i>	Ingestion of lead-containing curtain weights.	CR	Report of deaths of a 23-month-old child and a 2-year-old child.	30, 6
<i>Fishing sinkers</i>	Ingestion of a lead-containing fishing sinker.	CR	Report of an 8-year-old.	39
<i>Gasoline sniffing</i>	Lead in gasoline absorbed through gasoline sniffing.	CR	Report of six of seven siblings aged 10 to 17 years.	9, 24
<i>Lead bullet</i>	Lead absorbed from a retained bullet.	CR	Report of one adult and review of 18 other cases including seven children under 2 years old.	23, 32
<i>Lead pellets</i>	Ingestion of lead pellets from pellet gun.	CR	Report of a 6-year-old child.	45
<i>Lead shot and toy (boat keel)</i>	Lead shot used in a toy boat keel that was eaten by a child.	CR	Report of a 4-year-old child.	28
<i>Newsprint fireplace log</i>	Lead inhaled during burning of a log made from old newsprint.	CR	Report of a 6-month-old child.	43

Exposure Source	Description/Exposure Pathway	Study Type*	Study Description	Ref. #
<i>Pool cue chalk</i>	Lead contained in pool cue chalk.	CR	Report of two children aged 28 and 27 months.	36
<i>Vinyl miniblinds</i>	Lead dust from vinyl miniblinds.	E	A study of 92 children aged 6 to 72 months attributed 9% of lead poisoning cases to vinyl miniblind exposure.	41

\*CR = case report, E = epidemiological study

**References**

1. Abu Melha A, Ahmed NA, el Hassan AY. Traditional remedies and lead intoxication. *Trop Geogr Med* 1987;39:100-3.
2. Ali AR, Smales OR, Aslam M. Surma and lead poisoning. *BMJ* 1978;2:915-6.
3. Al-Saleh I, Nester M, DeVol E, et al. Determinants of blood lead levels in Saudi Arabian schoolgirls. *Int J Occup Environ Health* 1999;5:107-14.
4. Baer RD, De Alba JG, Cueto LM, et al. Lead-based remedies for empacho: patterns and consequences. *Soc Sci Med* 1989;29:1373-9.
5. Biehusen FC, Pulaski EJ. Lead poisoning after ingestion of a foreign body retained in the stomach. *N Engl J Med* 1956;254:1179-81.
6. Blank E, Howieson J. Lead poisoning from a curtain weight. *JAMA*. 1983;249:2176-7.
7. Browder AA. Lead poisoning from glazes. *Ann Intern Med* 1972;76:665.
8. Carney JK, Garbarino KM. Childhood lead poisoning from apple cider. *Pediatrics* 1997;100:1048-9.
9. CDC. Epidemiologic notes and reports: Gasoline sniffing and lead toxicity among siblings—Virginia. *MMWR* 1985;34:449-50,455.
10. CDC. Epidemiologic notes and reports - Jin Bu Huan toxicity in children—Colorado, 1993. *MMWR* 1993;42:633-6.
11. CDC. Epidemiologic notes and reports: Lead-contaminated drinking water in bulk-water storage tanks—Arizona and California, 1993. *MMWR* 1994; 43:751, 757-8.
12. CDC. Epidemiologic notes and reports: Lead poisoning following ingestion of homemade beverage stored in a ceramic jug. *MMWR* 1989;38:379-80.
13. CDC. Folk Remedy-associated lead poisoning in Hmong children—Minnesota. *MMWR* 1983;32:555-6.
14. CDC. Lead poisoning associated with imported candy and powdered food coloring—California and Michigan. *MMWR* 1998;47:1041-3.

15. CDC. Lead poisoning associated with use of traditional ethnic remedies—California, 1991-1992. *MMWR* 1993;42:521-4.
16. CDC. Lead poisoning from Mexican folk remedies—California. *MMWR* 1983;32:554-5.
17. CDC. Lead poisoning-associated death from Asian Indian folk remedies—Florida. *MMWR* 1984;33:638,643-5.
18. CDC. Use of lead tetroxide as a folk remedy for gastrointestinal illness. *MMWR* 1981;30:546-7.
19. CDC. Occupational and take-home lead poisoning associated with restoring chemically stripped furniture—California, 1998. *MMWR* 2001;50:246-8.
20. Chan H, Billmeier GJ Jr, Evans WE, et al. Lead poisoning from ingestion of Chinese herbal medicine. *Clin Toxicol* 1977;10:273-81.
21. Cheng TJ, Wong RH, Lin YP, et al. Chinese herbal medicine, sibship, and blood lead in children. *Occup Environ Med* 1998;55:573-6.
22. Dickinson L, Reichert EL, Ho RC, et al. Lead poisoning in a family due to cocktail glasses. *Am J Med* 1972;52:391-4.
23. Dillman RO, Crumb CK, Lidsky MJ. Lead poisoning from a gunshot wound. Report of a case and review of the literature. *Am J Med* 1979;66:509-14.
24. Eastwell HD. Elevated lead levels in petrol “sniffers.” *Med J Aust* 1985;143(9 suppl):S63-S64.
25. Eisenberg A, Avni A, Grauer F, et al. Identification of community flour mills as the source of lead poisoning in West Bank Arabs. *Arch Intern Med* 1985;145:1848-51.
26. Esernio-Jenssen D, Donatelli-Guagenti A, Mofenson HC. Severe lead poisoning from an imported clothing accessory: “watch” out for lead. *J Toxicol Clin Toxicol* 1996;34:329-33.
27. Fernando NP, Healy MA, Aslam M, et al. Lead poisoning and traditional practices: the consequences for world health. A study in Kuwait. *Public Health* 1981;95:250-60.

## Appendixes

28. Gittleman JL, Engelgau MM, Shaw J, et al. Lead poisoning among battery reclamation workers in Alabama. *J Occup Med* 1994;36:526-32.
29. Greensher J, Mofenson HC, Balakrishnan C, et al. Lead poisoning from ingestion of lead shot. *Pediatrics* 1974;54:641-3.
30. Hugelmeyer CD, Moorhead JC, Horenblas L, et al. Fatal lead encephalopathy following foreign body ingestion: case report. *J Emerg Med* 1988;6:397-400.
31. Kaye WE, Novotny TE, Tucker M. New ceramics-related industry implicated in elevated blood lead levels in children. *Arch Environ Health* 1987;42:161-4.
32. Kikano GE, Stange KC. Lead poisoning in a child after a gunshot injury. *J Fam Pract* 1992;34:498-500, 502, 504.
33. Lockitch G, Berry B, Roland E, et al. Seizures in a 10-week-old infant: lead poisoning from an unexpected source. *CMAJ* 1991;145:1465-8.
34. Lussenhop DH, Parker DL, Barklind A, et al. Lead exposure and radiator repair work. *Am J Public Health* 1989;79:1558-60.
35. McNeil JR, Reinhard MC. Lead poisoning from home remedies. *Clin Pediatr (Phila)* 1967;6:150-6.
36. Miller MB, Curry SC, Kunkel DB, et al. Pool cue chalk: a source of environmental lead. *Pediatrics* 1996;97:916-7.
37. Mojdehi GM, Gurtner J. Childhood lead poisoning through kohl. *Am J Public Health* 1996;86:587-8.
38. Moore C, Adler R. Herbal vitamins: lead toxicity and developmental delay. *Pediatrics* 2000;106:600-2.
39. Mowad E, Haddad I, Gemmel DJ. Management of lead poisoning from ingested fishing sinkers. *Arch Pediatr Adolesc Med* 1998;152:485-8.
40. Ng R, Martin DJ. Lead poisoning from lead-soldered electric kettles. *Can Med Assoc J* 1977;116:508-9.

41. Norman EH, Hertz-Picciotto I, Salmen DA, et al. Childhood lead poisoning and vinyl miniblind exposure. *Arch Pediatr Adolesc Med* 1997;151:1033-7.
42. Nunez CM, Klitzman S, Goodman A. Lead exposure among automobile radiator repair workers and their children in New York City. *Am J Ind Med* 1993;23:763-77.
43. Perkins KC, Oski FA. Elevated blood lead in a 6-month-old breast-fed infant: the role of newsprint logs. *Pediatrics* 1976;57:426-7.
44. Rahman H, Al Khayat A, Menon N. Lead poisoning in infancy—unusual causes in the U.A.E. *Ann Trop Paediatr* 1986;6:213-7.
45. Roberts JR, Landers KM, Fargason CA Jr. An unusual source of lead poisoning. *Clin Pediatr (Phila)* 1998;37:377-9.
46. Shannon M, Graef J. Hazard of lead in infant formula (letter). *N Engl J Med* 1992;326:137.
47. Shannon M, Graef JW. Lead intoxication from lead-contaminated water used to reconstitute infant formula. *Clin Pediatr (Phila)* 1989;28:380-2.
48. Shannon M. Lead poisoning from an unexpected source in a 4-month-old infant. *Environ Health Perspect* 1998;106:313-6.
49. Sitarz AL. Letter: Severe lead poisoning in a 6-month-old infant. *J Pediatr* 1975;86:810-1.
50. Sprinkle RV. Leaded eye cosmetics: a cultural cause of elevated lead levels in children. *J Fam Pract* 1995;40:358-62.
51. Trotter RT 2d. Greta and Azarcon: a survey of episodic lead poisoning from a folk remedy. *Hum Organ* 1985;44:64-72.
52. Warley MA, Blackledge P, O’Gorman P. Lead poisoning from eye cosmetic. *BMJ* 1968;1:117.
53. Whelan EA, Piacitelli GM, Gerwel B, et al. Elevated blood lead levels in children of construction workers. *Am J Public Health* 1997;87:1352-5.

## Appendix II. Sources of Information on Lead Abatement

- The Office of Lead Hazard Control of the U.S. Department of Housing and Urban Development (HUD) publishes a manual that explains how renovations and remodeling projects can be safely conducted. Single copies of this publication (*Lead Paint Safety—A Field Guide for Painting, Home Maintenance and Renovation Work*) can be ordered from the National Lead Information Center at 1-800-424-5323 or downloaded from the HUD Office of Lead Hazard Control Web site at [www.hud.gov/lea/leahome.html](http://www.hud.gov/lea/leahome.html).
- The Center for National Lead-Safe Housing provides information about safe repair. The Web site is [www.leadsafehousing.org/html/tech\\_assistance.htm](http://www.leadsafehousing.org/html/tech_assistance.htm).
- The Alliance to End Childhood Lead Poisoning provides information about safe repair. The Web site is [www.aeclp.org/painting/index.html](http://www.aeclp.org/painting/index.html).
- HUD offers a web-based, one-hour training course on how to visually assess the condition of paint films. The Web site is [www.hud.gov/lea/lbptraining.html](http://www.hud.gov/lea/lbptraining.html).