CDC

CENTERS FOR DISEASE CONTROL AND PREVENTION

Preventing Lead Poisoning in Young Children

A STATEMENT BY THE CENTERS FOR DISEASE CONTROL — OCTOBER 1991

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES / Public Health Service

During the process of converting this document into electronic format, blank pages were not converted, therefore, there may be missing numbered pages. Again, these were blank pages that were not converted.

Preventing Lead Poisoning in Young Children

A STATEMENT BY THE CENTERS FOR DISEASE CONTROL — OCTOBER 1991

William L. Roper, M.D., M.P.H., *Director*

National Center for Environmental Health and Injury Control
Vernon N. Houk, M.D., Director

Division of Environmental Hazards and Health Effects
Henry Falk, M.D., Director

Lead Poisoning Prevention Branch
Sue Binder, M.D., Chief

U. S. DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service
Centers for Disease Control and Prevention

Use of trade names is for identification purposes only and does not constitute endorsement by the Public Health Service or by the Department of Health and Human Services.

Contents

Preface iii

Advisory Committee Members and Consultants v

Chapter 1. Introduction 1

Chapter 2. Background 7

Chapter 3. Sources and Pathways of Lead Exposure 17

Chapter 4. The Role of the Pediatric Health-Care Provider 27

Chapter 5. The Role of State and Local Public Agencies 35

Chapter 6. Screening 39

Chapter 7. Diagnostic Evaluation and Medical Management of Children with Blood Lead Levels ≥20 μg/dL 51

Chapter 8. Management of Lead Hazards in the Environment of the Individual Child 65

Chapter 9. Management of Lead Hazards in the Community 75

Appendix I. Capillary Sampling Protocol 81

Appendix II. Summary for the Pediatric Health-Care Provider 87

Preface

This is the fourth revision of the statement on *Preventing Lead Poisoning in Young Children* by the Centers for Disease Control (CDC). The recommendations continued herein are based mainly on the scientific data showing adverse effects of lead in young children at increasingly lower blood lead levels. They are tempered, however, by practical considerations, for example, of the numbers of children who would require followup and the resources required to prevent this disease. It is possible that further scientific data and development of infrastructure and technology will result in a lowering of the blood lead level at which interventions are recommended at a future time.

This statement is a departure from previous ones in several ways. Perhaps most important is the emphasis on primary prevention and the need for coordination between pediatric health-care providers and public agencies. This statement reflects the vision expressed in the Department of Health and Human Services' *Strategic Plan for the Elimination of Childhood Lead Poisoning*, which calls for a concerted, coordinated societywide effort to eliminate this disease.

In writing this statement, we identified several areas where better data are needed in order to provide scientifically sound guidance. These range from evaluating the efficacy of chelation therapy at lower blood lead levels in terms of preventing the adverse effects of lead to developing science-based criteria for determining when an abated unit is cleaned up enough for rehabitation. We hope that the appropriate research to answer such questions will be conducted in a timely manner, and we will continue to update the statement to reflect current understanding.

We are aware of concerns about the impact the changes in the statement will have on childhood lead poisoning prevention programs, laboratories, and pediatric health-care providers. In this new statement, we recognize the need for a transition period until we are able to implement fully the new recommendations; it will take time and a concerted effort to implement this new guidance.

CDC is conducting several activities which bear directly on the implementation of the statement. First, as noted above, the *Strategic Plan for the Elimination of Childhood Lead Poisoning* was released by Dr. Louis W. Sullivan, Secretary of the Department of Health and Human Services, on February 21, 1991. In addition to laying out the actions needed to eliminate childhood lead poisoning, this plan describes the need for infrastructure and technology development, including for the evaluation of blood and environmental lead levels. Second, CDC is aggressively pursuing research and development efforts in collaboration with several instrument manufacturers to develop a field-rugged, relatively inexpensive, and simple-to-operate blood lead instrument, which would markedly enhance blood lead screening efforts. Initial results are encouraging, but the effort is still in the developmental stage. If all goes well, new instrumentation could be ready in 2 to 3 years. Third, we are continuing our efforts to help laboratories improve the quality of their blood lead measurements through our proficiency testing program and through our Blood Lead Laboratory Reference System. Finally, CDC also has a grant program in childhood lead poisoning prevention, through which state and local health agencies receive Federal money to screen children for lead poisoning, ensure environmental and medical followup for poisoned children, and provide education about lead poisoning. By the end of FY 1991, we will be funding 13 state and 2 city childhood lead poisoning prevention programs, and the President's budget for 1992 includes almost a doubling of the FY 1991 budget. We continue to encourage CDC-funded programs to address infrastructure issues.

Other Federal agencies, like the Environmental Protection Agency and the Department of Housing and Urban Development, have also released plans that deal with aspects of the childhood lead poisoning problem. These agencies are also working to build the needed infrastructure for and expand the scientific knowledge on reducing exposure to lead in the environment.

I wish to thank the members of the Committee and consultants, as well as the numerous other people who assisted in the development and review of this document. I believe this document will be a major landmark in the effort to eliminate childhood lead poisoning from the United States.

Vernon N. Houk, M.D.
Director
National Center for Environmental Health and Injury Control

Advisory Committee on Childhood Lead Poisoning Prevention Members and Consultants

CHAIR
John F. Rosen, M.D.
Attending Pediatrician
Montefiore Hospital and
Medical Center
111 East 20th Street
Bronx, New York 10467

EXECUTIVE SECRETARY
Henry Falk, M.D.
Director
Division of Environmental Hazards
and Health Effects
National Center for Environmental
Health and Injury Control
Centers for Disease Control
Atlanta, Georgia 30333

MEMBERS
Evelyn S. Bouden, M.D.
Director
Division of Maternal and Child Health
Pennsylvania Department of Health
725 H & W Building
Harrisburg, Pennsylvania 17108

Beverly Coleman-Miller, M.D.
Special Assistant for Medical Affairs
Commission of Public Health
1660 L Street, N.W., Suite 1204
Washington, D.C. 20036

Ronald L. Fletcher, M.D.
Co-Director
Oncology Department
Greene Memorial Hospital
1141 N. Monroe Drive
Xenia, Ohio 44385

Lynn R. Goldman, M.D.
Chief
Environmental Epidemiology and
Toxicology Section
Department of Health Services
5900 Hollis Street, Suite E
Emeryville, California 94608

Dwala S. Griffin
Administrator
Division of Preventive Medicine
Louisville-Jefferson County Board of Health
400 East Gray Street
Louisville, Kentucky 40202

Richard J. Jackson, M.D.
Chief
Office of Environmental Health Hazards
Assessment
California Department of Health Services
2151 Berkeley Way, Room 619
Berkeley, California 94704-1071

Rudolph E. Jackson, M.D.
Professor and Acting Chairman
Department of Pediatrics
Morehouse School of Medicine
720 Westview Drive, S.W.
Atlanta, Georgia 30310

James C. Keck
President
Leadtec Services, Inc.
522 Beck Avenue
Baltimore, Maryland 21221

Herbert L. Needleman, M.D.
Professor of Psychiatry
Western Psychiatric Institute and Clinic
University of Pittsburgh School of Medicine
Iroquois Building, Suite 305
3600 Forbes Avenue
Pittsburgh, Pennsylvania 15213-2593

Sergio Piomelli, M.D.
Director
Babies Hospital
3959 Broadway
New York, New York 10032

Stephanie L. Pollack, J.D.
Attorney
Conservation Law Foundation of New England
3 Joy Street
Boston, Massachusetts 02108-1497

Knut Ringen, Dr.P.H.
Director
Laborers' National Health and Safety Fund
905 16th Street, N.W.
Washington, D.C. 20006

Noel V. Stanton
Lead Chemist
Toxicology Section
State Laboratory of Hygiene
465 Henry Mall
Madison, Wisconsin 53706

CONSULTANTS

David Bellinger, Ph.D.
Neuroepidemiology Unit
Children's Hospital
300 Longwood Avenue
Boston, Massachusetts 02115

James J. Chisolm, Jr., M.D.
Associate Professor
Francis Scott Key Medical Center
Building G, Room 224
4940 Eastern Avenue
Baltimore, Maryland 21224

Charles G. Copley
Deputy Health Commissioner
City of Saint Louis
Department of Health and Hospitals
Office of the Commissioner
634 N. Grand, P. O. Box 147202
Saint Louis, Missouri 63178

Anita S. Curran, M.D.
Assistant Dean for Clinical Affairs
Robert Wood Johnson Medical Center
University of Medicine and Dentistry of New Jersey
One Robert Wood Johnson Place
New Brunswick, New Jersey 08903

John W. Graef, M.D.
Director
Lead/Toxicology Clinic
The Children's Hospital
300 Longwood Avenue
Boston, Massachusetts 02115

Phillip J. Landrigan, M.D.
Director
Division of Environmental and Occupational Medicine
Mount Sinai Medical Center
One Gustave L. Levy Place
New York, New York 10029

John R. Reigart, M.D.
Project Pediatrician
S. C. Childhood Lead Poisoning Project
Department of Pediatrics, Children's Hospital
171 Ashley Avenue, 6th Floor
Charleston, South Carolina 29425

Chapter 1. Introduction

Summary

New data indicate significant adverse effects of lead exposure in children at blood lead levels previously believed to be safe. Some adverse health effects have been documented at blood lead levels at least as low as 10 micrograms per deciliter of whole blood (μg/dL).

The 1985 intervention level of 25 μg/dL is, therefore, being revised downwards to 10 μg/dL.

A multitier approach to followup has been adopted.

Primary prevention efforts (that is, elimination of lead hazards before children are poisoned) must receive more emphasis as the blood lead levels of concern are lowered.

The goal of all lead poisoning prevention activities should be to reduce children's blood lead levels below 10 μg/dL. If many children in the community have blood lead levels ≥10 ug/dL, communitywide interventions (primary prevention activities) should be considered by appropriate agencies. Interventions for individual children should begin at blood lead levels of 15 μg/dL.

Childhood lead poisoning is one of the most common pediatric health problems in the United States today, and it is entirely preventable. Enough is now known about the sources and pathways of lead exposure and about ways of preventing this exposure to begin the efforts to eradicate permanently this disease. The persistence of lead poisoning in the United States, in light of all that is known, presents a singular and direct challenge to public health authorities, clinicians, regulatory agencies, and society.

> Lead poisoning is one of the most common and preventable pediatric health problems today.

Lead is ubiquitous in the human environment as a result of industrialization. It has no known physiologic value. Children are particularly susceptible to lead's toxic effects. Lead poisoning, for the most part, is silent: most poisoned children have no symptoms. The vast majority of cases, therefore, go undiagnosed and untreated. Lead poisoning is widespread. It is not solely a problem of inner city or minority children. No socioeconomic group, geographic area, or racial or ethnic population is spared.

Previous lead statements issued by the Centers for Disease Control (CDC) have acknowledged the adverse effects of lead at lower and lower levels. In the most recent previous CDC lead statement, published in 1985, the threshold for action was set at a blood lead level of 25 μg/dL, although it was acknowledged that adverse effects occur below that level. In the past several years, however, the scientific evidence showing that some adverse effects occur at blood lead levels at least as low as 10 μg/dL in children has become so overwhelming and compelling that it must be a major force in determining how we approach childhood lead exposure.

This document provides guidelines on childhood lead poisoning prevention for diverse groups. Public health programs that screen children for lead poisoning look to this document for guidance on screening regimens and public health actions. Pediatricians and other health-care practitioners look to this document for information on screening and guidance on the medical treatment of poisoned children. Government agencies, elected officials, and private citizens seek guidance about what constitutes a harmful level of lead in blood—what the current definition of lead poisoning is and what blood lead levels should trigger environmental and other interventions.

It is not possible to select a single number to define lead poisoning for the various purposes of all of these groups. Epidemiologic studies have identified harmful effects of lead in children at blood lead levels at least as low as 10 μg/dL. Some studies have suggested harmful effects at even lower levels, but the body of information accumulated so far is not adequate for effects below about 10 μg/dL to be evaluated definitively. As yet, no threshold has been identified for the harmful effects of lead.

Because 10 μg/dL is the lower level of the range at which effects are now identified, primary prevention activities—communitywide environmental interventions and nutritional and educational campaigns—should be directed at reducing children's blood lead levels at least to below 10 μg/dL. Blood lead levels between 10 and 14 μg/dL are in a border zone. While the overall goal is to reduce children's blood lead levels below 10 μg/dL, there are several reasons for not attempting to do interventions directed at individual children to lower blood lead levels of 10-14 μg/dL. First, particularly at low blood lead levels, laboratory measurements may have some inaccuracy and imprecision, so a blood lead level in this range may, in fact, be below 10 μg/dL. Secondly, effective environmental and medical interventions for children with blood lead levels in this range have not yet been identified and evaluated. Finally, the sheer numbers of children in this range would preclude effective case management and would detract from the individualized followup required by children who have higher blood lead levels.

The single, all-purpose definition of childhood lead poisoning has been replaced with a multitier approach, described in Table 1-1. Community prevention activities should be triggered by blood lead levels ≥10 μg/dL. Medical evaluation and environmental investigation and remediation should be done for all children with blood lead levels ≥20 μg/dL. All children with blood lead levels ≥15 μg/dL should receive individual case management, including nutritional and educational interventions and more frequent screening. Furthermore, depending on the availability of resources, environmental investigation (including a home inspection) and remediation should be done for children with blood lead levels of 15-19 μg/dL, if such levels persist. The highest priority should continue to be the children with the highest blood lead levels.

Other differences between the 1985 and 1991 statements are as follows:

Screening test of choice. Because the erythrocyte protoporphyrin level is not sensitive enough to identify children with elevated blood lead levels below about 25 μg/dL, the screening test of choice is now blood lead measurement.

Universal screening. Since virtually all children are at risk for lead poisoning, a phase in of universal screening is recommended, except in communities where large numbers or percentages of children have been screened and found not to have lead poisoning. The full implementation of this will require the ability to measure blood lead levels on capillary samples and the availability of cheaper and easier-to-use methods of blood lead measurement.

Table 1-1. Interpretation of blood lead test results and follow-up activities: class of child based on blood lead concentration

Class	Blood lead concentration (μg/dL)	Comment
I	≤9	A child in Class I is not considered to be lead-poisoned.
IIA	10-14	Many children (or a large proportion of children) with blood lead levels in this range should trigger communitywide childhood lead poisoning prevention activities (Chapter 9). Children in this range may need to be rescreened more frequently.
IIB	15-19	A child in Class IIB should receive nutritional and educational interventions and more frequent screening. If the blood lead level persists in this range, environmental investigation and intervention should be done (Chapter 8).
III	20-44	A child in Class III should receive environmental evaluation and remediation (Chapter 8) and a medical evaluation (Chapter 7). Such a child may need pharmacologic treatment of lead poisoning (Chapter 7).
IV	45-69	A child in Class IV will need both medical and environmental interventions, including chelation therapy (Chapters 7 and 8).
V	≥70	A child with Class V lead poisoning is a medical emergency. Medical and environmental management must begin immediately (Chapters 7 and 8).

Primary prevention. Efforts need to be increasingly focused on preventing lead poisoning before it occurs. This will require communitywide environmental interventions, as well as educational and nutritional campaigns.

Succimer. In January, 1991, the U.S. Food and Drug Administration approved succimer, an oral chelating agent, for chelation of children with blood lead levels over 45 μg/dL.

Childhood lead poisoning prevention programs have had a tremendous impact on reducing the occurrence of lead poisoning in the United States. Because of these programs, deaths from lead poisoning and lead encephalopathy are now rare. These programs have targeted high-risk children for periodic screening; provided education to caretakers about the causes, effects, symptoms, and treatments for lead poisoning; and ensured medical treatment and environmental remediation for poisoned children. Screening and medical treatment of poisoned children will remain critically important until the environmental sources most likely to poison children are eliminated.

Federal regulatory and other actions have resulted in substantial progress in reducing blood lead levels in the entire U.S. population. In the last two decades, the virtual elimination of lead from gasoline has been reflected in reductions in blood lead levels in children and adults. Lead levels in food have also decreased since most manufacturers stopped using leaded solder in cans and since atmospheric deposition of lead on food crops declined as a result of reductions of lead in gasoline. In 1978, the Consumer Product Safety Commission banned the addition of lead to new residential paint.

Nevertheless, important environmental sources and pathways of lead remain. Lead-based paint and lead-contaminated dusts and soils remain the primary sources and pathways of lead exposure for children. In addition, children continue to be exposed to lead through air, water, and food, as well as occupations and hobbies of parents and caretakers. The focus of prevention efforts, therefore, must expand from merely identifying and treating individual children to include primary prevention—preventing exposure to lead before children become poisoned. This will require a shared responsibility among many public and private agencies. Public agencies will have to work with pediatric health-care providers to identify communities with childhood lead-poisoning prevention problems and unusual sources of lead and to ensure environmental followup of poisoned children. Public housing and economic development agencies will have to integrate lead paint abatement into housing rehabilitation policies and programs. Health-care providers will need to phase in virtually universal screening of children. Public and private organizations must continue to develop economical and widely-available blood lead tests to make such screening possible. Public and private housing owners must bear a portion of the financial burden for abatement.

The changes in this statement are not meant to create an enormous burden on primary pediatric health-care providers. These changes will only be useful if public health and other agencies effectively complement health-care providers' activities. Several efforts have begun to increase federal support of childhood lead poisoning prevention programs and of followup activities. Ongoing efforts to develop infrastructure and technology by the public and private sectors include 1) the development of inexpensive, easy-to-use portable methods for measuring blood lead levels; 2) the development of training and certification programs for lead paint inspectors and abatement contractors; and 3) the development and testing of new abatement methods, including encapsulants. The changes in this statement are also not meant to increase the emphasis on screening of children; the long-term goal of this statement is **prevention**. Until primary prevention of childhood lead poisoning can be achieved, however, increased screening and followup of poisoned children is essential.

Elimination of Childhood Lead Poisoning

Will require efforts from both the private and public sectors.

Will require a shift in emphasis to primary prevention.

Will take time and resources.

Should proceed in a rational manner, with the highest risk children being made the highest priority.

Can be achieved.

In February 1991, the U.S. Department of Health and Human Services released a *Strategic Plan for the Elimination of Childhood Lead Poisoning* (HHS, 1991). This plan describes the first 5 years of a 20-year societywide effort to eliminate this disease. It places highest priority on first addressing the children at greatest risk for lead poisoning. The U.S. Department of Housing and Urban Development (HUD, 1990) and the Environmental Protection Agency (EPA, 1991) have both released plans dealing with the elimination of lead hazards. To eliminate this disease will require a tremendous effort from all levels of government as well as the private sector, but we believe that the benefits to society will be well worth it. We look forward to the day when childhood lead poisoning is no longer a public health problem.

References

CDC (Centers for Disease Control). Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta: CDC, 1985; CDC report no. 99-2230.

EPA (Environmental Protection Agency). Strategy for reducing lead exposures: report to Congress. Washington (DC): EPA, 1991.

HHS (U.S. Department of Health and Human Services). Strategic plan for the elimination of childhood lead poisoning. Atlanta: CDC, 1991.

HUD (U.S. Department of Housing and Urban Development). Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing: report to Congress. Washington (DC): HUD, 1990.

Chapter 2. Background

Summary

The blood lead level considered to indicate lead toxicity has progressively shifted downwards.

In general, children are more at risk for lead exposure than adults.

Large numbers of children in the United States continue to have blood lead levels in the toxic range.

This chapter describes the health effects of lead on children and fetuses, the metabolism of lead, and the demographics of lead exposure in the United States. It explains why the definition of childhood lead poisoning is being revised.

EFFECTS OF LEAD ON CHILDREN AND FETUSES

Lead affects virtually every system in the body.
The blood lead level considered to indicate lead poisoning has fallen steadily since the 1970s.
Blood lead levels at least as low as 10 μg/dL are associated with adverse effects.
Although the effects of low-level lead exposure may not seem severe in the individual child, on a population basis they are extremely important.

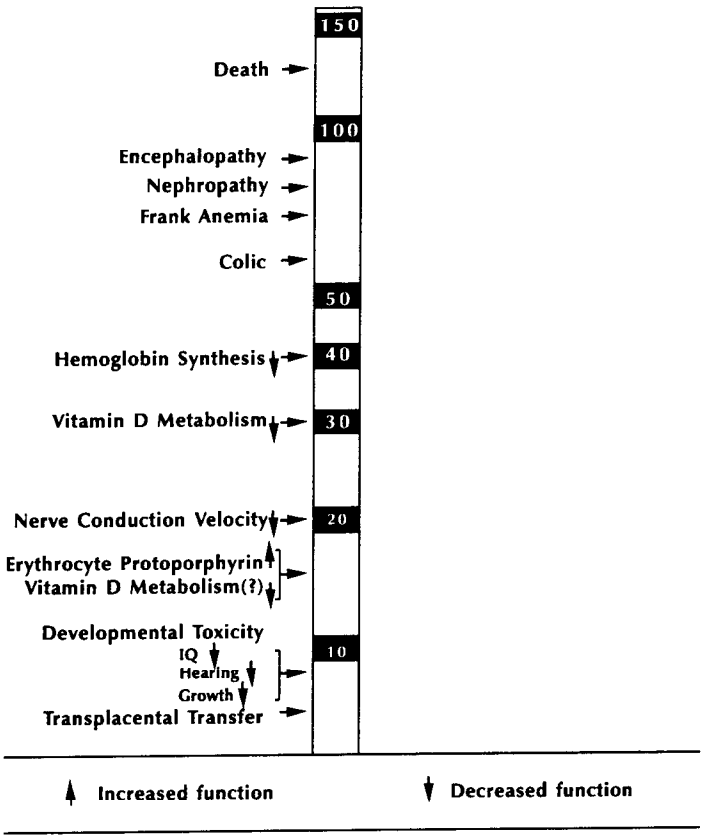
Lead is a poison that affects virtually every system in the body. It is particularly harmful to the developing brain and nervous system of fetuses and young children. The adverse effects of lead on children and adults are summarized in Figure 2-1.

The risks of lead exposure are not based on theoretical calculations. They are well known from studies of children themselves and are not extrapolated from data on laboratory animals or high-dose occupational exposures.

Levels of Concern

Since 1970, our understanding of childhood lead poisoning has changed substantially. As investigators have used more sensitive measures and better study designs, the generally recognized level for lead toxicity has progressively shifted downward. Before the mid-1960s, a level above 60 μg/dL was considered toxic (Chisolm and Harrison, 1956). By 1978, the defined level of toxicity had declined 50% to 30 μg/dL. Figure 2-2 shows how the federal definition of an elevated blood lead level has changed over the years.

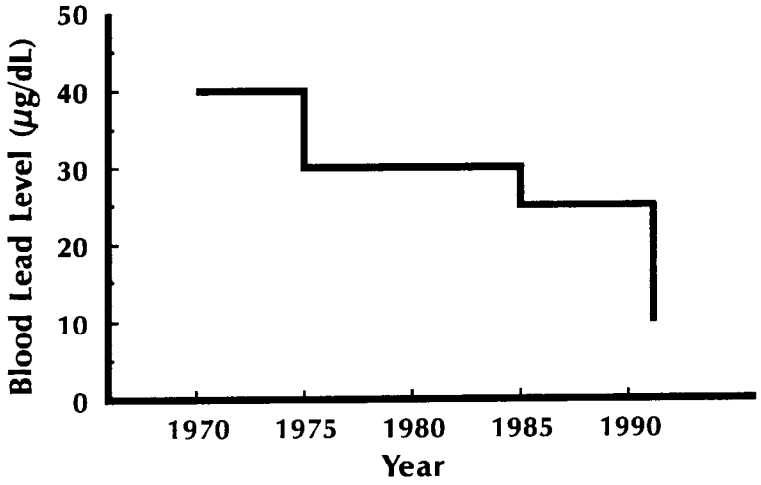
Figure 2-1. Lowest observed effect levels of inorganic lead in children*



*Note: The levels in this diagram do not necessarily indicate the lowest levels at which lead exerts an effect. These are the levels at which studies have adequately demonstrated an effect.

Source: ATSDR, 1990.

Figure 2-2. Blood lead levels considered elevated by the Centers for Disease Control and the Public Health Service



Range of Effects of Lead

Very severe lead exposure in children (blood lead levels ≥80 μg/dL) can cause coma, convulsions, and even death. Lower levels cause adverse effects on the central nervous system, kidney, and hematopoietic system. Blood lead levels as low as 10 μg/dL, which do not cause distinctive symptoms, are associated with decreased intelligence and impaired neurobehavioral development (Davis and Svendsgaard, 1987; Mushak et al., 1989). Many other effects begin at these low blood lead levels, including decreased stature or growth (Schwartz et al., 1986; Bornschein et al., 1986; Shulka et al., 1989), decreased hearing acuity (Schwartz and Otto, 1987), and decreased ability to maintain a steady posture (Bhattacharya et al., 1988). Lead's impairment of the synthesis of the active metabolite 1,25-$(OH)_2$ vitamin D is detectable at blood lead levels of 10-15 μg/dL. Maternal and cord blood lead levels of 10-15 μg/dL appear to be associated with reduced gestational age and reduced weight at birth (ATSDR, 1988). Although researchers have not yet completely defined the impact of blood lead levels <10 μg/dL on central nervous system function, it may be that even these levels are associated with adverse effects that will be clearer with more refined research.

Studies of Low-Level Lead Effects on the Central Nervous System

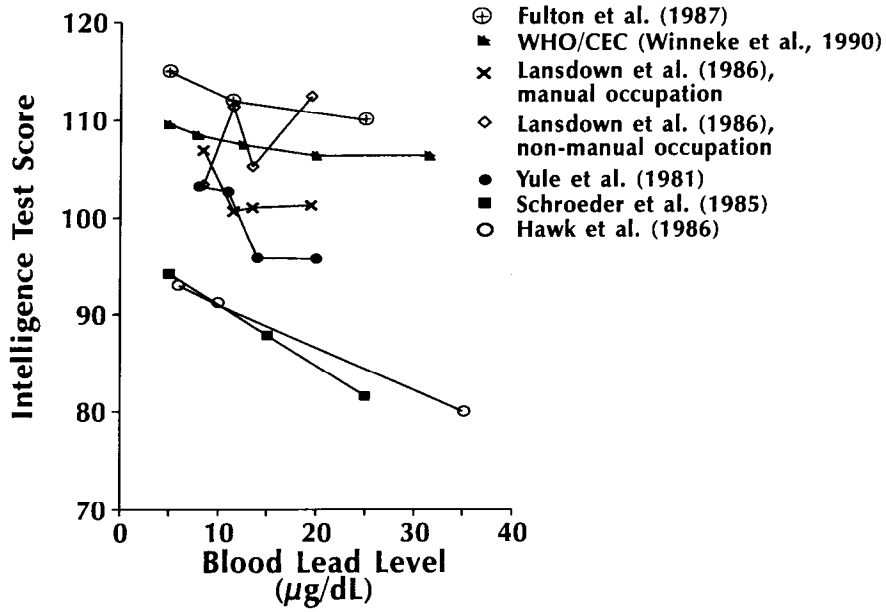
The concern about adverse effects on central nervous system functioning at blood lead levels as low as 10 μg/dL is based on a large number of rigorous epidemiologic and experimental studies. In particular, recent cross-sectional and prospective studies have provided new evidence about the association between low-level lead exposure and child development.

Several well-designed and carefully conducted cross-sectional and retrospective cohort studies in many different countries have been conducted (Lansdown et al., 1986; Fulton et al., 1987; Fergusson et al., 1988; Silva et al., 1988; Bergomi et al., 1989; Hansen et al., 1989; Hatzakis et al., 1989; Winneke et al., 1990; Lyngbye et al., 1990; Needleman et al., 1990; Yule et al., 1981; Lansdown et al., 1986; Hawk et al., 1986; Schroeder et al., 1985). Figure 2-3 shows the mean intelligence quotient (IQ) scores (in most cases adjusted for potential confounding factors) achieved by children with different blood lead levels from several of these studies. Some inconsistencies can be found in the results of these studies, but the weight of the evidence clearly supports the hypothesis that decrements in children's cognition are evident at blood lead levels well below 25 μg/dL. No threshold for the lead-IQ relationship is discernable from these data.

Most investigators report lower IQ scores among the more highly exposed children but these differences have not uniformly reached statistical significance (that is, $p<.05$). One way to synthesize the data from different studies is meta-analysis. Recent evaluation of 24 major cross-sectional studies provides strong support for the hypothesis that children's IQ scores are inversely related to lead burden (Needleman and Gatsonis, 1990).

Although available evidence is not sufficient to conclude that lead-associated deficits are irreversible, a recent follow-up study reported that the educational success of a cohort of young adults was significantly inversely associated with the amount of lead in teeth they shed as first and second graders (Needleman et al., 1990). In this study, dentine lead levels above 20 ppm were associated with a seven-fold risk of not graduating from high school, a six-fold risk of having a reading disability, deficits in vocabulary, problems with attention and fine motor coordination, greater absenteeism, and lower class ranking. Although dentine lead levels did not correspond in any simple way to blood lead levels, the available preschool blood lead levels

Figure 2-3. Blood lead levels and IQ scores of children, from cross-sectional and retrospective cohort studies*



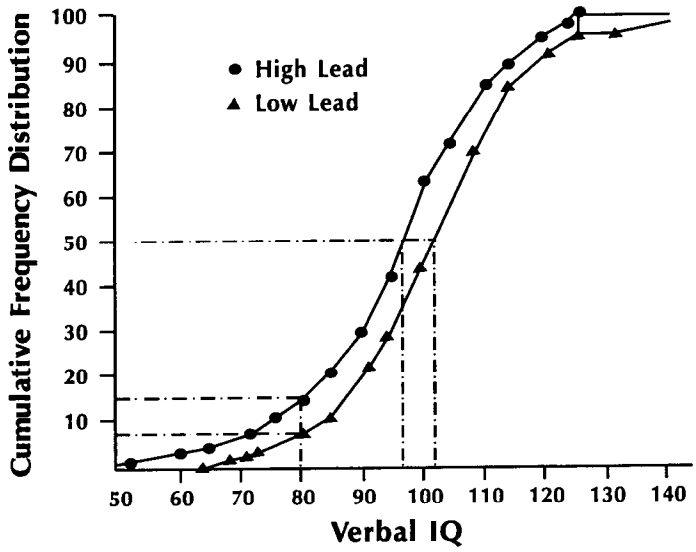
*Data from prospective studies are not included.

of the more highly exposed children averaged 35 μg/dL (Needleman et al., 1979). Increased circumpulpal dentine lead levels (>16 ppm) have been linked to higher rates of learning disabilities in a recent Danish study as well (Lyngbye et al., 1990).

To address methodological limitations of cross-sectional studies of lead and child development, a number of prospective studies were begun during the 1980s. Blood lead measurements were begun during the prenatal period and continued for several years, along with assessment of development. In several but not all cohorts, prenatal exposures have been associated with slower sensory-motor and delayed early cognitive development (Bellinger et al., 1987; Bellinger et al., 1991; Dietrich et al., 1987; Ernhart et al., 1986; Dietrich et al., 1991). With low postnatal exposures and favorable socioeconomic conditions, some of these early associations may attenuate as children grow older (Bellinger et al., 1991). In addition, several studies have noted that children's cognitive performance in the preschool period may be associated with early postnatal lead exposures (McMichael et al., 1988; Bellinger et al., 1991). It will be necessary for these prospective studies to follow their respective cohorts into the school-age years in order for the full implications of these early patterns to become clear.

Questions are frequently raised about the practical significance of the difference frequently observed between the IQ scores of more exposed and less exposed children. For the previously described population of children studied by Needleman et al. (Needleman et al., 1979), a shift in mean IQ score of 4-6 points as a result of lead exposure was associated with a substantial increase in the prevalence of children with severe deficits (that is, IQ scores less than 80) (Figure 2-4). Similarly, in this population the shift was associated with an absence of children who achieved superior function (that is, IQ scores greater than 125).

Figure 2-4. Cummulative frequency distribution of verbal IQ scores in children with high and low tooth lead levels



Source: Needleman et al., 1979.

ABSORPTION OF LEAD

Children are at higher risk for lead exposure because
They have more hand-to-mouth activity than adults.
They absorb more lead than adults.

Many factors can affect the absorption, distribution, and toxicity of lead. Children are more exposed to lead than older groups because their normal hand-to-mouth activities may introduce many nonfood items into their gastrointestinal tract (Lin-Fu, 1973). The efficiency of gastrointestinal absorption of lead in food and beverages in children has been estimated to be around 40% (Ziegler et al., 1978). From experimental studies, gastrointestinal absorption of lead from nonfood sources is decreased in the presence of food (Rabinowitz, 1980). Efficiency of absorption is probably also affected by the particle size and form of lead (Barltrop and Meek, 1979). Deficiencies in iron, calcium, protein, and zinc are related to increased blood lead levels and perhaps increased vulnerability to the adverse effects of lead (Mahaffey, 1981; Mahaffey and Michaelson, 1980).

BLOOD LEAD LEVELS IN THE UNITED STATES

Large numbers of children continue to have blood lead levels high enough to cause adverse effects.

Substantial progress has been made, however, in reducing blood lead levels in the United States.

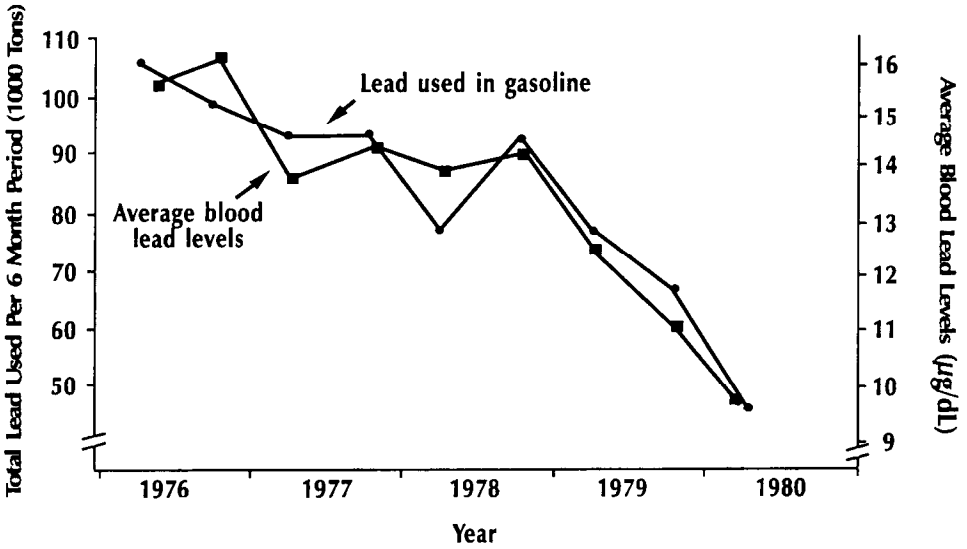
Lead-based paint remains the major source of high-dose lead poisoning in the United States.

The Agency for Toxic Substances and Disease Registry estimated that in 1984, 17% of all American preschool children had blood lead levels that exceed 15 μg/dL (ATSDR, 1988). Although all children are at risk for lead toxicity, poor and minority children are disproportionately affected. Lead exposure is at once a by-product of poverty and a contributor to the cycle that perpetuates and deepens the state of being poor.

Substantial progress has been made in reducing blood lead levels in U.S. children. Perhaps the most important advance has been the virtual elimination of lead from gasoline. Close correlations have been demonstrated between the decline in the use of leaded gasoline and declines in the blood lead levels of children and adults between 1976 and 1980 (Annest, 1983) (Figure 2-5). Levels of lead in food have also declined significantly, as a result both of the decreased use of lead solder in cans and the decreasing air lead levels.

Lead-based paint remains the major source of high-dose lead poisoning in the United States. Although the Consumer Products Safety Commission (CPSC) limited the lead content of new residential paint starting in 1978, millions of houses still contain old leaded paint. The Department of Housing and Urban Development estimates that about 3.8 million homes with young children living in them have either nonintact lead-based paint or high levels of lead in dust (HUD, 1990).

Figure 2-5. Change in blood lead levels in relation to a decline in use of leaded gasoline, 1976–1980



Source: Annest JL, 1983.

References

ATSDR (Agency for Toxic Substances and Disease Registry). Case studies in environmental medicine: lead toxicity. Atlanta: ATSDR, 1990.

ATSDR (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: ATSDR, 1988.

Annest JL. Trends in the blood lead levels of the US population. In: Rutter M, Jones RR, editors. Lead versus health. Chichester and New York: John Wiley and Sons, 1983:33-58.

Barltrop D, Meek F. Effect of particle size on lead absorption from the gut. Arch Environ Health 1979;34:280-5.

Bellinger D, Leviton A, Waternaux C, Needleman H, Rabinowitz M. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. N Engl J Med 1987;316:1037-43.

Bellinger D, Sloman J, Leviton A, Rabinowitz M, Needleman H, Waternaux C. Low-level exposure and children's cognitive function in the preschool years. Pediatrics 1991;87:219-27.

Bergomi M, Borella P, Fantuzzi G, Vivoli G, Sturloni N, Cavazzuti G, Tampieri A, Tartoni PL. Relationship between lead exposure indicators and neuropyschological performance in children. Dev Med Child Neurol 1989;31:181-90.

Bhattacharya A, Shulka R, Bornshein R, Dietrich K, Kopke J. Postural disequilibrium quantification in children with chronic lead exposure: a pilot study. Neurotoxicology 1988;9:327-40.

Bornschein RL, Succop PA, Krafft KM, Clark CS, Peace B, Hammond PB. 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. Columbia (MO): University of Missouri, 1986:322-32.

CDC (Centers for Disease Control). Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta: CDC, 1985; CDC report no. 99-2230.

Chisolm JJ, Harrison HE. The exposure of children to lead. Pediatrics 1956;18:934-55.

Davis JM, Svendsgaard DJ. Lead and child development. Nature 1987;329:297-300.

Dietrich KN, Krafft KM, Bornshein RL, Hammond PB, Berger O, Succop PA, Bier M. Low-level fetal lead exposure effect on neurobehavioral development in early infancy. Pediatrics 1987;80:721-30.

Dietrich KN, Succop PA, Berger O, Hammond P, Bornschin RL. Lead exposure and cognitive developmnent of urban preschool children: the Cincinnati lead study cohort at age 4 years. Neurotoxicology and Teratology 1991;13:203-11.

Ernhart CB, Wolf AW, Kennard MJ, Erhard P, Filipovich HF, Sokol RJ. Intrauterine exposure to low levels of lead: the status of the neonate. Arch Environ Health 1986;41:287-91.

Fergusson DM, Fergussen JE, Horwood LJ, Kinzett NG. A longitudinal study of dentine lead levels, intelligence, school performance, and behavior Part II: dentine lead and cognitive ability. J Child Psych Pyschiat 1988;29:793-809.

Fulton M, Raab G, Thompson G, Laxen D, Hunter R, Hepburn W. Influence of blood lead on the ability and the attainment of children in Edinburgh. Lancet 1987;i:1221-6.

Hansen ON, Trillingsgaard A, Beese I, Lyngbye T, Grandjean P. A neuropsychological study of children with elevated dentine lead level: assessment of the effect of lead in different socio-economic groups. Neurotoxicology and Teratology 1989;11:205-13.

Hatzakis A, Kokkevi A, Maravelias C, Katsouyanni K, Salaminios F, Kalandidi A, Koutselinis A, Stefanis C, Trichopoulos D. Pyschometric intelligence deficits in lead-exposed children. In: Smith M, Grant L, Sors A, editors. Lead exposure and child development: an international assessment. Dordrecht: Kluwer Academic Publishers, 1989:211-23.

Hawk BA, Schroeder SR, Robinson G, Otto D, Mushak P, Kleinbaum D, Dawson G. Relation of lead and social factors to IQ of low-SES children: a partial replication. Am J Ment Def 1986;91:178-83.

HUD (U.S. Department of Housing and Urban Development). Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing: report to Congress. Washington (DC): HUD, 1990.

Lansdown R, Yule W, Urbanowicz MA, Hunter J. The relationship between blood-level concentrations, intelligence, attainment and behaviour in a school population: the second study. Int Arch Occup Environ Health 1986;57:225-35.

Lin-Fu JS. Vulnerability of children to lead exposure and toxicity: Part one. N Engl J Med 1973; 289:1229-33.

Lyngbye T, Hansen ON, Trillingsgaard A, Beese I, Grandjean P. Learning disabilities in children: significance of low-level lead exposure and confounding effects. Acta Pediatr Scand 1990;79:352-60.

Mahaffey KR. Nutritional factors in lead poisoning. Nutr Rev 1981;39:353-62.

Mahaffey KR, Michaelson IA. The interaction between lead and nutrition. In: Needleman HL, editor. Low level lead exposure: the clinical implications of current research. New York (NY): Raven Press, 1980:159-200.

McMichael AJ, Baghurst PA, Wigg NR, Vimpani GV, Robertson EF, Roberts RJ. Port Pirie cohort study: environmental exposure to lead and children's abilities at four years. N Engl J Med 1988;319:468-75.

Mushak P, Davis JM, Crochetti AF, Grant LD. Prenatal and postnatal effects of low level lead exposure: integrated summary of a report to the U.S. Congress on childhood lead poisoning. Environ Res 1989;50:11-36.

Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children. JAMA 1990;263:673-8.

Needleman HL, Gunnoe C, Leviton A, Reed R, Peresie H, Maher C, Barret P. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N Engl J Med 1979;300:689-95.

Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. N Engl J Med 1990;322:83-8.

Rabinowitz MB, Kopple JD, Wetherill GW. Effect of food intake and fasting on gastrointestinal lead absorption in humans. Am J Clin Nutrition 1980;33:1784-8.

Schroeder SR, Hawk B, Otto DA, Mushak P, Hicks RE. Separating the effects of lead and social factors on IQ. Environ Res 1985;38:144-54.

Schwartz J, Angle C, Pitcher H. Relationship between childhood blood lead levels and stature. Pediatrics 1986;77:281-8.

Schwartz J, Otto D. Blood lead, hearing thresholds, and neurobehavioral development in children and youth. Arch Environ Health 1987;42:153-60.

Shukla R, Bornschein RL, Dietrich KN, Buncher CR, Berger OG, Hammond PB, Succop PA. Fetal and infant lead exposure: effects on growth in stature. Pediatrics 1989;84:604-12.

Silva PA, Hughes P, Williams S, Faed JM. Blood lead, intelligence, reading attainment, and behaviour in eleven year old children in Dunedin, New Zealand. J Child Psych Psychiat 1988;29:43-52.

Winneke G, Brockhaus A, Ewers U, Kramer U, Neuf M. Results from the European multicenter study on lead neurotoxicity in children: implications for risk assessment. Neurotoxicity and Teratology 1990; 12:553-9.

Yule W, Lansdown R, Miller I, Urbanowicz M. The relationship between blood lead concentrations, intelligence, and attainment in a school population: a pilot study. Dev Med Child Neurol 1981;23:567-76.

Ziegler EE, Edwards BB, Jensen RL, Mahaffey KR, Fomon SJ. Absorption and retention of lead by infants. Pediatric Research 1978;12:29-34.

Chapter 3. Sources and Pathways of Lead Exposure

Sources and Pathways of Lead Exposure in Children Include:

Lead-based paint.

Soil and dust.

Drinking water.

Parental occupations and hobbies.

Air.

Food.

For some children, other sources and pathways, such as "traditional" medicines, may be critical.

INTRODUCTION

A child's environment is full of lead. Children are exposed to lead from different sources (such as paint, gasoline, and solder) and through different pathways (such as air, food, water, dust, and soil). Although all U.S. children are exposed to some lead from food, air, dust, and soil, some children are exposed to high dose sources of lead. Lead-based paint is the most widespread and dangerous high-dose source of lead exposure for preschool children.

Lead entering the body from different sources and through different pathways presents a combined toxicological threat (ATSDR, 1988). Multiple, low-level inputs of lead can result in significant aggregate exposure. Indeed, for children with lower (but still elevated) blood lead levels (for example, in the range of 10-20 μg/dL) identifying a single, predominant environmental source or pathway is not always possible.

This chapter describes the most important sources and pathways for childhood lead exposure. Information about the levels or concentrations of concern in different pathways is based on information assembled by regulatory agencies and other published data. Nothing in this chapter should be interpreted as suggesting standards for acceptable or unacceptable levels or concentrations of lead in different environmental media.

LEAD-BASED PAINT

- Lead-based paint is the most common high-dose source of lead exposure for children.
- About 74% of privately owned, occupied housing units in the United States built before 1980 contain lead-based paint.
- Children are exposed to lead when they ingest chips of lead-based paint or ingest paint-contaminated dust and soil.
- Many cases of lead poisoning result when homes containing lead-based paint are remodelled or renovated without precautions being taken.
- Removing lead from housing is important both for the treatment of poisoned children and for the primary prevention of childhood lead poisoning.

Lead-based paint remains the most common high-dose source of lead exposure for preschool children. Lead-based paint (containing up to 50% lead) was in widespread use through the 1940s. Although the use and manufacture of interior lead-based paint declined during the 1950s and thereafter, exterior lead-based paint and lesser amounts of interior lead-based paint continued to be available until the mid-1970s (CEH/CAPP, 1987). (Lead-based paint produced after the 1940s tended to have much lower lead concentrations than lead-based paint produced earlier.) In 1978, the Consumer Product Safety Commission banned the manufacture of paint containing more than 0.06% lead by weight on interior and exterior residential surfaces, toys, and furniture. Unfortunately, lead-based paint that is still available for industrial, military, and marine usage occasionally ends up being used in homes.

Nationwide, about 3 million tons of lead remain in an estimated 57 million occupied private housing units built before 1980 (representing 74% of all such housing). Of particular concern are the 14 million housing units believed to contain lead paint in unsound condition and the 3.8 million deteriorated units occupied by young children (HUD, 1990).

Pica, the repeated ingestion of nonfood substances, has been implicated in cases of lead poisoning; however, a child does not have to eat paint chips to become poisoned. More commonly, children ingest dust and soil contaminated with lead from paint which flaked or chalked as it aged or which has been disturbed during home maintenance or renovation. This lead-contaminated house dust, ingested via normal repetitive hand-to-mouth activity, is now recognized as a major contributor to the total body burden of lead in children (Bornschein et al., 1986). Because of the critical role of dust as an exposure pathway, children living in sub-standard housing and in homes undergoing renovation are at particular risk for lead poisoning.

Numerous studies have established that the risk of lead poisoning is related to the presence of lead-based paint and to the condition of such paint (ATSDR, 1988; EPA, 1986). Children who live in rehabilitated lead-free housing or who return to lead-reduced housing after undergoing medical treatment have significantly lower blood levels than children living in similar, nonrehabilitated housing (Bornschein et al., 1986; Chisolm et al., 1985). Data from several urban lead poisoning prevention programs indicate that deleading the home of a poisoned child can reduce blood lead levels substantially (Rosen et al., in press; Amitai et al., in press; G. Copley, unpublished data). Deleading or lead paint abatement can be an effective method of

reducing children's exposure to dangerous levels of lead in paint and house dust if properly done (Farfel and Chisolm, in press), but may actually increase dust lead levels if not done properly (Farfel and Chisolm, 1990).

Lead paint is typically found on kitchen and bathroom walls and throughout pre-1950 homes on doors, windows, and wooden trim. The risks of lead poisoning are greater when lead paint or the underlying surface are in deteriorated condition and when lead paint (even intact paint) is located on surfaces accessible to children (EPA, 1986). Lead paint on interior and exterior window components is particularly of concern because it is abraded into dust by the repeated opening and closing of these windows (Farfel and Chisolm, 1990).

Many cases of childhood lead poisoning that result from renovation or remodelling of homes have been reported (Marino, 1990). Before older homes undergo any renovation that may generate dust, they should be tested for the presence of lead-based paint. If such paint is found, contractors experienced in working with lead-based paint should do the renovations.

There is no uniform standard for safe or allowable amounts of lead in existing painted surfaces. States and the federal government use values ranging from 0.7-1.2 mg/cm^2 of wall when lead is measured using a portable x-ray fluorescence analyzer (XRF) or a standard of 0.5% lead by weight when tests are performed using laboratory analysis. These regulatory limits are based mostly on practical, not health, considerations.

Lead paint also continues to be used on the exterior of painted steel structures, such as bridges and expressways. In addition to the obvious risk to workers, increased lead absorption has been reported in children exposed to chips or dust during the deleading or maintenance of such structures (Landrigan et al., 1982).

Deleading, even when performed in the homes of children who have already been poisoned, is an important method of primary lead poisoning prevention because it reduces or removes the lead hazard from that housing unit for all future occupants. Methods for the safe abatement of residential lead paint are detailed in Chapter 8. The Department of Housing and Urban Development has primary responsibility for issues related to lead-based paint in housing.

SOIL AND DUST

- Soil and dust act as pathways to children for lead deposited from paint, gasoline, and industrial sources.
- The long-term efficacy and cost-effectiveness of different measures to reduce lead levels in soil need to be evaluated.
- Reduction of dust lead is important both as part of deleading and as a means of interim risk reduction.

Soil and dust act as pathways to children for lead deposited by primary lead sources such as lead paint, leaded gasoline, and industrial or occupational sources of lead. Since lead does not dissipate, biodegrade, or decay, the lead deposited into dust and soil becomes a long-term source of lead exposure for children. For example, although lead emissions from gasoline have largely been eliminated, an estimated 4-5 million metric tons of lead used in gasoline remain in dust and soil, and children continue to be exposed to it (ATSDR, 1988).

Because lead is immobilized by the organic component of soil, lead deposited from the air is generally retained in the upper 2-5 centimeters of undisturbed soil (EPA, 1986). Urban soils and other soils that are disturbed or turned under may be contaminated down to far greater depths. Soil lead levels within 25 meters of roadways are typically 30-2,000 parts per million (ppm) higher than natural levels, with some roadside soils having concentrations as high as 10,000 ppm. Soils adjacent to houses painted with exterior lead paints may also have lead levels above 10,000 ppm. Measured lead levels in soil adjacent to smelters range as high as 60,000 ppm (EPA, 1986).

As part of normal play and hand-to-mouth exploratory activities, young children may inhale or ingest lead from soil or dust. Ingestion of dust and soil during meals and playtime activity appears to be a more significant pathway than inhalation for young children (EPA, 1986).

Different investigators have found widely varying relationships between levels of lead in soil and dust and children's blood lead levels. Blood lead levels generally rise 3-7 μg/dL for every 1,000-ppm increase in soil or dust lead concentrations (EPA, 1986; Bornschein et al., 1986; ATSDR, 1988). Particle size and the chemical form of lead may affect the bioavailability of lead in soil and dust; access to soil, behavior patterns, presence of ground cover, and a variety of other factors also influence this relationship (Barltop and Meek, 1979).

Even if ongoing deposition of lead into soil and dust is eventually halted, measures will have to be taken to reduce exposures from lead-contaminated soils and dusts. Until data demonstrating the efficacy and cost-effectiveness of permanent soil and dust abatement measures are available, interim risk reduction steps will be needed in some places. Dust control via wet mopping and frequent hand washing has been shown to reduce the blood lead levels of children with high blood lead levels (Charney et al., 1983), but this is not a permanent solution so long as the source of the lead in the dust remains. For urban and smelter communities, where outdoor soil can be a major source of lead in house dust (Diemel et al., 1981; Yankel et al., 1977), indoor dust abatement may not be effective unless abatement of soil lead is also conducted. Soil abatement may consist of either establishing an effective barrier between children and the soil or the removal and replacement of at least the top few centimeters of soil. Grass cover, if properly maintained, may be an effective means of limiting exposure to dusts originating from lead-contaminated soil (Jenkins et al., 1988).

DRINKING WATER

Contamination of drinking water with lead usually occurs in the distribution system.
Several properties of water and its pattern of use affect how much lead contamination results from a particular water distribution system.
Some practical measures can lower the lead content of drinking water.

Lead levels are typically low in ground and surface water, but may increase once the water enters the water distribution system. Contamination of drinking water can occur at five points in or near the residential, school, public, or office plumbing, including 1) lead connectors (that is, goosenecks or pigtails), 2) lead service lines or pipes, 3) lead-soldered joints in copper

plumbing throughout the building, 4) lead-containing water fountains and coolers, and 5) lead-containing brass faucets and other fixtures. The 1986 Safe Drinking Water Act Amendments banned the use of lead in public drinking water distribution systems and limited the lead content of brass used for plumbing to 8%.

Several properties of water and its patterns of use affect the extent of lead contamination that results from a particular water delivery system. These factors include 1) the corrosiveness of water (that is, pH, alkalinity, and mineral content), 2) age of the lead-soldered joints and other lead components (the newer ones often pose a higher risk), 3) quantity and surface area of lead materials, and 4) standing time and temperature of water in contact with leaded surfaces.

Typically, lead pipes are found in residences built before the 1920s, with the oldest cities having the most frequent use of lead pipes. Pipes made of copper and soldered with lead came into general use in the 1950s. Overall, lead leaching from copper pipes with lead-soldered joints represents the major source of water contamination in homes and public facilities such as schools.

In some areas of the United States (for example, Pennsylvania), cisterns are used to store water, especially rain water that may be acidic. Cisterns also can be roof-collection systems, which are common in some island areas (for example, Hawaii, the Florida Keys). When lead solder is used either in the construction of these cisterns or to repair leaks, or the cistern has a lead liner, the potential for lead contamination of the water is substantial. If the water has a relatively low pH, has low concentrations of cations such as Ca^{++} or Mg^{++} (that is, "soft" water), or has an elevated organic content, the water is probably aggressive in dissolving lead from the cistern. Corrosion control may be effective in reducing water lead levels in the case of corrosive water.

Lead in drinking water is probably absorbed more completely than lead in food. Adults absorb 35%-50% of the lead they drink, and the absorption rate for children may be greater than 50% (ATSDR, 1988).

In general, lead in drinking water is not the predominant source for poisoned children. In some circumstances, however, lead exposures from water are unusually high. Some water cooler-fountains have been found to have lead-soldered or lead-lined tanks. Patterns of intermittent water use from these fountains results in the water standing in the tanks longer than in typical residential situations, which can increase the amount of lead that is leached from the tanks. Several babies have been poisoned when hot tap water, which was then boiled (resulting in concentrating the lead), was used to make baby formula (J. Graef, personal communication).

Practical measures to reduce exposure to lead in drinking water include using fully-flushed water for drinking and cooking and always drawing water for ingestion from the cold water tap. The effectiveness of many point-of-use devices (treatment devices that are installed at the tap) in reducing lead in water varies and may be affected by the location of the device in relation to the lead source and by compliance with manufacturer's use and maintenance instructions. Some, like reverse osmosis and distillation units, may be effective. Carbon, sand, and cartridge filters do not remove lead.

The Environmental Protection Agency regulates the permissible lead content of water.

OCCUPATIONS AND HOBBIES

Children may be exposed to high lead levels when workers take home lead on their clothing or when they bring scrap or waste material home from work.

Hobbyists may also inadvertently expose their families to lead.

The current Occupational Safety and Health Administration standards may not adequately protect the health of workers.

A variety of work and hobby environments expose people to lead and may result in lead exposures for their families. Occupations frequently reported to have resulted in adult lead poisoning are shown in Table 3-1. Many potential hazardous activities, like furniture refinishing and making stained glass, may be either hobbies or occupations. Other activities that may be associated with lead exposure include using indoor firing ranges, doing home repairs and remodeling, and making pottery. "Take-home" exposures may result when workers wear their work clothes home or launder them with the family laundry or when they bring scrap or waste material home from work (Grandjean and Bach, 1986).

Strict compliance by industrial operations with the Occupational Safety and Health Administration (OSHA) General Industry Lead Standard governing lead exposures (29 CFR 1910.1025) would greatly reduce both occupational lead exposure and the associated indirect exposures in the homes of these workers. Unfortunately, not all occupational settings are covered by this regulation. Workers in construction—including lead abatement workers–are excluded from coverage under the General Industry Lead Standard; they are covered under a much weaker construction standard. Numerous workers in these work environments have been

Table 3-1. Industries identified by surveillance for elevated blood lead levels, California and New York, 1991

Industry Description	Standard Industrial Classification Number
Secondary smelting and refining of nonferrous metals	3341
Storage batteries (lead batteries)	3691
Valve and pipe fittings (except plumber's brass goods)	3494
Plumbing fixture fittings and trim (brass goods)	3432
Brass/copper foundry	3362
Glass products, made of purchased glass	3231
Motor vehicle parts and accessories	3714
Firing range workers	7997,9221
Pottery, nec	3269
Chemical and chemical preparations	2899
Bridge, tunnel, and elevated highway construction	1622
Automotive repair shops	7539
Industrial machinery and equipment	5084
Inorganic pigments	2816
Primary batteries, dry and wet	3692

Sources: Baser and Marion, 1990; Maizlish et al., 1990.

excessively exposed to lead, with construction workers particularly having a high risk of very high blood lead levels (Maizlish et al., 1990). Compliance with the OSHA comprehensive lead standard is inadequate (Landrigan, 1990; Maizlish, et al., 1990) even by those industries required to be in compliance. Furthermore, the current OSHA standard may not adequately protect the health of workers (Landrigan, 1990). OSHA plans to revise its standard within the next several years.

AIRBORNE LEAD

Although lead use in gasoline has been markedly reduced, previous use has resulted in widespread contamination of soil and dust.

Except around point sources, airborne lead is only a minor exposure pathway.

Until recently, the combustion of leaded gasoline by motor vehicles was the predominant source of airborne lead in the United States. However, the Environmental Protection Agency (EPA) ordered the reduction of almost all lead in gasoline during the 1970s and 1980s, and 1990 amendments to the Clean Air Act will completely prohibit the use of lead as a gasoline additive beginning as early as January, 1992 and concluding no later than December 31, 1995. As discussed in the previous section, however, soil and dust contaminated by deposition of lead-containing particles can contain high concentrations of lead.

Except around point sources, like smelters and battery manufacturing plants, inhalation of airborne lead is now a minor exposure pathway for individual children. Other industrial activities may also result in localized exposures to lead, including burning solid waste in incinerators and sandblasting or demolishing bridges and other lead-painted metal structures. These localized activities, however, can be important sources of high-dose exposure.

FOOD

The quantity of lead in the U.S. diet has decreased markedly in recent years.

Improperly fired ceramicware, leaded crystal, and lead-soldered cans result in lead leaching into foods.

Some food-handling practices can increase the lead content of foods.

During the 1980s, the quantity of lead in the U.S. diet decreased markedly. "Market basket" data from the U.S. Food and Drug Administration (FDA), used to estimate typical lead intake, show that the average dietary lead intake for a 2-year-old child was about 30 μg/day in 1982, about 13 μg/day by 1985, and about 5 μg/day in the period 1986-1988. This reduction was achieved through substantially restricted use of lead-soldered side-seam cans and the phasing out of lead as an additive in gasoline. In 1980, 47% of domestically produced food and soft drink cans were lead-soldered. By 1989 use of lead-soldered cans declined to 1.4% of domestically produced cans. Counter to this trend is the continued use of lead solder in cans of imported

foods, because cans manufactured outside the United States typically continue to contain lead solder.

Lead in foods comes from several sources in addition to lead solder: soil in which the plant is grown; air and rain; food processing (including lead leaching from some types of metal cans described above); contact with lead solder or ceramic vessels used to store the food; and contact with lead dusts in the home. If lead contamination is unusually severe, the quantity of lead in the diet will be much higher than the "Market Basket" estimates. Examples include imported food from countries that do not restrict the use of lead solder in cans; storage of foods packaged in lead-soldered cans for over a year or so, even if the can is unopened; storage of acidic foods in ceramic containers made with improperly applied leaded glazes; and food processed with lead-contaminated water.

Under some circumstances, food grown in "urban gardens" may have an elevated lead content if the garden soil is high in lead or if there are high lead concentrations in the air or water used for irrigation. Soil conditions (for example, pH, phosphorus content, buffering capacity, and the amount of organic matter) and the type of plant have a great effect on how much lead is transferred to the plant. The amount transferred is difficult to predict because many factors affect lead uptake. It is recommended that the crops grown on contaminated soil be tested to determine their lead uptake. Such tests may be arranged through the Agriculture Extension Service, state or federal departments of agriculture, or private laboratories.

Occasionally, food supplements can be seriously contaminated with lead. Examples have included various dietary supplements from "natural" sources, such as calcium supplements derived from animal bone sources.

In addition, some food-handling practices in the home can increase the lead content of foods and should be avoided. Foods should not be stored in unopened, lead-soldered cans for over a year or so. Foods should not be stored, even under refrigeration, in opened cans even if the can is subsequently covered. Food should be stored only in containers that do not release lead (for example, glass, stainless steel, or plastic containers). If ceramic food containers are ever used to store food, they should be made with lead-free glazes. Leaded crystal should not be used to store food for prolonged periods of time and should not be used to hold baby formula or juices.

Lead solders should never be used to repair food containers or to construct or repair cooking utensils. High lead levels may be present in hot water prepared in lead-soldered tea pots.

OTHER SOURCES

Other Sources and Pathways of Lead Exposure
"Traditional" medicines
Cosmetics
Casting ammunition, fishing weights, or toy soldiers
Making stained glass
Making pottery
Refinishing furniture
Burning lead-painted wood

Published data, as well as anecdotal evidence from clinicians and others who work with lead-poisoned children, have identified a variety of other sources of concern.

Many "non-Western" medicines (for example, greta and azarcon used to treat diarrhea or gastrointestinal upset) and cosmetics (for example, surma or kohl used around the eye for decorative or medicinal purposes) contain substantial quantities of lead and other metals. Rather than occurring as trace ingredients or trace contaminants, various lead compounds are used as major ingredients of traditional medicines in numerous parts of the world. "Traditional healers," using non-Western pharmacopeias, manufacture these products, which are often brought to recent immigrant groups by friends and relatives. Examples of such exposures have been reported from the Arab cultures, from the Indo-Pakistan subcontinent, from China, and from Latin America.

Folk remedies containing lead include:	
Alarcon	Ghasard
Alkohl	Greta
Azarcon	Liga
Bali Goli	Pay-loo-ah
Coral	Rueda

Many hobbies can result in substantial exposures to lead. For example, molten lead can be used in casting ammunition and making fishing weights or toy soldiers; leaded solder is used in making stained glass; leaded glazes and frits are used in making pottery; and artists' paints may contain lead. Furniture refinishing may also result in lead exposure.

In some areas, the burning of lead-painted wood in home stoves and fireplaces is a source of lead exposure. Lead fumes are generated, ashes contaminate the home, and ashes are often disposed of in the back yard, resulting in contamination of the environment.

SOURCES OF LEAD OUTSIDE THE UNITED STATES

Childhood lead poisoning is a problem worldwide.

Childhood lead poisoning is a problem worldwide. In other parts of the world, however, predominant sources of lead are very different than in the United States. For example, leaded gasoline is still widely used in many countries and contributes to elevated blood lead levels, especially in urban children. Poorly glazed pottery leading to high food lead levels can be the most prominent source of lead in some areas, for example, in parts of Latin America. Point industrial sources may dramatically increase air and soil lead levels in parts of the world where environmental controls have not been effectively implemented, for example, in Eastern Europe. Lead contamination from cottage industries that recycle lead, often in backyards, is a problem in Central America and elsewhere. For children moving to or from the United States, an assessment of potential lead hazards requires specific knowledge of the country involved.

References

ATSDR (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: ATSDR, 1988.

Amitai Y, Brown MJ, Graef JW, Cosgrove E. Effects of residential deleading on the blood lead levels of lead poisoned children. Pediatrics (in press).

Barltrop D, Meek F. Effect of particle size on lead absorption from the gut. Arch Environ Health 1979;34:280-5.

Baser ME, D Marion. A statewide case registry for surveillance of occupational heavy metals absorption. Am J Public Health 1990:80:162-4.

Bornschein RL, Succop PA, Krafft KM, Clark CS, Peace B, Hammond PB. Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In: Hemphill D, ed. Trace substances in environmental health. Columbia, (MO): University of Missouri, 1986:322-32.

Charney E, Kessler B, Farfel M, Jackson D. Childhood lead poisoning: a controlled trial of the effect of dust-control measures on blood lead levels. N Engl J Med 1983;309:1089-93.

Chisolm JJ Jr, 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.

CEH/CAPP (Committee on Environmental Hazards/Committee on Accident and Poison Prevention). Statement on childhood lead poisoning. Pediatrics 1987;79:457-65.

Diemel JA, Brunekreef B, Boleij JS, Biersteker K, Veenstra SJ. The Arnheim Lead Study II: indoor pollution and indoor/outdoor relationships. Environ Res 1981;25:449-56.

EPA (Environmental Protection Agency). Air quality criteria for lead. Research Triangle Park (NC): Office of Health and Environmental Assessment, 1986; EPA report no. EPA/600/8-83/028aF.

Farfel MR, Chisolm JJ Jr. Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. Am J Public Health 1990;80:1240-5.

Farfel MR, Chisolm JJ Jr. An evaluation of experimental practices for abatement of residential lead-based paint: report on a pilot project. Environ Res (in press).

Grandjean P, Bach E. Indirect exposures: the significance of bystanders at work and at home. Am Ind Hyg Assoc J 1986;47:819-24.

HUD (Department of Housing and Urban Development). Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing: report to Congress. Washington (DC): HUD.

Jenkins G, Murray C, Thorpe BH. Lead in soil: the Ontario situation. In: Davies BE, Wixson BG, eds. Lead in soil: issues and guidelines. Northwood: Science Reviews Limited, 1988:235-45.

Landrigan PJ. Lead in the modern workplace. Am J Public Health 1990;80:907-8.

Landrigan PJ, Baker EL Jr, Himmelstein JS, Stein GF, Weddig JP, Straub WE. Exposure to lead from the Mystic River Bridge: the dilemma of deleading. N Engl J Med 1982;306:673-6.

Maizlish N, Rudolph L, Sutton P, Jones JR, Kizer KW. Elevated blood lead in California adults, 1987: results of a statewide surveillance program based on laboratory reports. Am J Public Health 1990; 80:931-4.

Marino PE, Landrigan PJ, Graef J, Nussbaum A, Bayan G, Boch K, Boch S. A case report of lead paint poisoning during renovation of a Victorian farmhouse. Am J Public Health 1990;80:1183-5.

Rosen JF, Markowitz ME, Bijur PE, et al. Sequential measurements of bone lead content by L-X-ray fluorescence in $CaNa_2$ EDTA-treated lead-toxic children. Environ Health Perspect (in press).

Yankel AJ, von Lindern IH, Walter SD. The Silver Valley Lead Study: the relationship between childhood blood lead levels and environmental exposure. J Air Pollution Control Organization 1977; 27:763-7.

Chapter 4. The Role of The Pediatric Health-Care Provider

The Pediatric Health-Care Provider Should

Provide anticipatory guidance about childhood lead poisoning and its prevention.

Provide screening for lead poisoning following established screening schedules.

Conduct appropriate diagnostic blood lead testing in children with symptoms or signs consistent with lead poisoning or pica.

Interpret blood lead results.

Educate parents about reducing blood lead levels.

Coordinate with local public health officials.

Ensure that poisoned children receive appropriate medical, environmental, and social service followup.

Pediatric health-care providers, working as part of the public health team, must play a critical role in the prevention and management of childhood lead poisoning. Their roles include 1) educating parents about key causes of childhood lead poisoning; 2) screening children and interpreting blood lead test results; 3) working with appropriate groups in the public and private sectors to make sure that poisoned children receive appropriate medical, environmental, and social service followup; and 4) coordinating with public health officials and others involved in lead-poisoning prevention activities.

ANTICIPATORY GUIDANCE

Anticipatory guidance means

Teaching parents about major sources of lead and how to prevent poisoning.
Tailoring guidance to likely hazards in the community.

Pediatric health-care providers consider education to be an integral part of well-child care. Along with educating parents about nutrition and developmental stages, providers should discuss the potential hazards of lead. They should focus on the major preventable sources of high-dose lead poisoning—lead-based paint and take-home exposures from parents' occupations and hobbies. Parents should be told of the potential dangers of peeling lead-based paint, the potential hazards of renovating older homes, and the need for good work practices if their occupations or hobbies expose them to lead. (These sources and pathways of exposure are

INTERPRETATION OF BLOOD LEAD LEVELS

In interpreting blood lead levels, the provider should

Understand the scientific basis for concern.
Understand the degree of imprecision and inaccuracy in blood lead measurements.
Explain carefully why followup is or is not needed.

The studies which form the basis of our concern about childhood lead poisoning are described in Chapter 2. These studies suggest that adverse effects of lead occur at blood lead levels at least as low as 10 μg/dL. The following paragraphs provide guidance on what might be told to a parent, depending on the blood lead levels of the child.

Blood lead level <10 μg/dL. A blood lead level <10 μg/dL is not considered to be indicative of lead poisoning.

Blood lead level 10-14 μg/dL. Children with blood lead levels in this range are in a border zone. Since the laboratory tests for measuring blood lead levels are not as accurate and precise as we would like them to be at these levels, many of these children's blood lead levels may, in fact, be <10 μg/dL. Although a detailed environmental history should be taken since an obvious remediable source of lead may be found, it is unlikely that there is a single predominant source of lead exposure for most of these children. Thus, a full home inspection is not recommended. It is, however, prudent to try to decrease exposure to lead with some simple interventions (Page 30). (The required education can be done face-to-face or by distributing brochures or other written materials.) In addition, these children should receive followup blood lead testing in about 3 months. The adverse effects of blood lead levels of 10-14 μg/dL are subtle and are not likely to be recognizable or measurable in the individual child. It is important to make sure that these children's blood lead levels do not go up.

Example: Johnny was a 12 month old child without any risk factors for high-dose exposure. A capillary blood lead test was performed, and his blood lead level was 14 μg/dL. His pediatrician told his mother that Johnny's blood lead test was in a kind of "border" zone, but that it was high enough to require careful followup. The pediatrician explained that laboratory test results have some inaccuracy and imprecision, but, nevertheless, suggested some housekeeping and nutritional interventions to reduce Johnny's exposure. Johnny had a venous blood lead measurement three months later, which was 7 μg/dL. Three months after that, when Johnny was 18 months old, his blood lead level was 5 μg/dL. His blood lead level was measured one year later and was 5 μg/dL, and he received no further followup.

Blood lead level 15-19 μg/dL. Children with venous blood lead levels 15-19 μg/dL need more careful followup. The pediatric health-care provider should take a careful history, asking about sources of lead exposure (Chapter 3). Parents should receive guidance about interventions to reduce blood lead levels (Page 30). Children with blood lead levels in this range are at

risk for decreases in IQ of up to several IQ points and other subtle effects. The effects of lead at these levels are significant enough that the health-care provider should emphasize to parents the importance of followup screening to make sure the levels do not increase. The provider should also discuss interventions to reduce the blood lead levels. In addition, these children should receive followup testing (Chapter 6). If their blood lead levels persist at ≥15 μg/dL, environmental investigation and remediation should be completed, if resources permit. In some communities, childhood lead poisoning prevention programs may be able to manage the environmental investigation and remediation.

Blood lead level 20-69 μg/dL. Children with venous blood lead levels in this range should have a full medical evaluation (Chapter 7). This includes a detailed environmental and behavioral history (asking about reading or other learning disabilities, language development, pica, etc.), a physical examination, and tests for iron deficiency. Particularly for children needing urgent medical followup (that is, blood lead level ≥45 μg/dL), pediatric health-care providers with limited experience in treating lead poisoning should consider referring such children to a clinic with experience in managing childhood lead poisoning. These children should also have complete environmental investigations so that lead hazards can be reduced. The local public childhood lead poisoning prevention programs will often work as a team with the pediatric health-care provider and the child's family to ensure appropriate environmental followup.

Blood lead level ≥70 μg/dL. Children with blood lead levels this high constitute a medical emergency that preferably should be managed by someone with experience in treating children who are critically ill with lead poisoning. Medical and environmental management must begin immediately (Chapters 7 and 8).

EDUCATING PARENTS ABOUT REDUCING BLOOD LEAD LEVELS

What can parents do to reduce blood lead levels?

Housekeeping interventions to reduce exposure to dust.
Interventions to reduce exposure to other sources of lead.
Attention to nutrition.

There are many interventions parents can use to help reduce blood lead levels. These interventions are not a substitute for lead hazard abatement.

Housekeeping Interventions

Particularly in older homes, which may have been painted with lead-based paint, interventions to reduce exposure to dust may help reduce blood lead levels. These include:

- Make sure your child does not have access to peeling paint or chewable surfaces painted with lead-based paint. Pay special attention to windows and window sills and wells.

- If the house was built before about 1960 and has hard surface floors, wet mop them at least once a week with a high phosphate solution (for example, 5-8% phosphates). (The phosphate content of automatic dishwashing detergents and other cleaning substances is often listed on the label and may be high enough for this purpose. Otherwise, trisodium phosphate can be purchased in hardware stores.) Other hard surfaces (such as window sills and baseboards) should also be wiped with a similar solution. Do not vacuum hard surface floors or window sills or wells, since this will disperse dust. Vacuum cleaners with agitators remove dust from rugs more effectively than vacuum cleaners with suction only.
- Wash your child's hands and face before he/she eats.
- Wash toys and pacifiers frequently.

Other Interventions To Reduce Exposure To Lead

- If soil around the home is or is likely to be contaminated with lead (for example, if the home was built before 1960 or the house is near a major highway), plant grass or other ground cover. Since the highest concentrations of lead in a yard tend to be near surfaces that were once painted with lead paint, like exterior walls, if exterior lead paint was likely to be used, plant bushes around the outside of your house so your child cannot play there.
- In areas where the lead content of water exceeds the drinking water standard, use only fully-flushed water from the cold-water tap for drinking, cooking, and making formula. In communities where water conservation is a concern, use the first-flush water for other purposes.
- Do not store food in open cans, particularly if the cans are imported.
- Do not use pottery or ceramicware that was inadequately fired or is meant for decorative use for food storage or service.
- Make sure that take-home exposures are not occurring from parental occupations or hobbies (Chapter 3).

Nutrition

- Make sure your child eats regular meals, since more lead is absorbed on an empty stomach.
- Make sure your child's diet contains plenty of iron and calcium.

Examples of Sources of Iron and Calcium

Iron	Calcium
Liver	Milk
Fortified cereal	Yourt
Cooked legumes	Cheese
Spinach	Cooked greens

COORDINATING WITH PUBLIC SECTOR OFFICIALS

Public health officials should tell the pediatric health-care provider about

- The magnitude of the childhood lead poisoning problem in the provider's community.
- Unusual sources of lead exposure in the provider's community.
- Public sector services that can be used to ensure appropriate followup for poisoned children.
- Interventions being conducted through public sector actions for children with lead poisoning.

Pediatric health-care providers should notify public sector officials about

- Poisoned children they identify.
- Unusual sources or pathways of exposure they identify.

The responsibilities of public sector officials are described in Chapter 5. These officials are an important source of information for the pediatric health-care provider. They can alert the provider to the extent of the lead poisoning problem in the provider's catchment area. They can provide information about particular lead sources that may be of concern in a given neighborhood. Often these officials can assist in the management of the lead-poisoned child, doing followup screening, conducting environmental investigations, and ensuring lead hazards are abated. They should keep the provider informed of actions they take on the child's behalf. The pediatric health-care provider is responsible for informing public health officials about lead-poisoned children, reporting any unusual sources or pathways of exposure, and reporting elevated blood lead levels, if this is required.

APPROPRIATE FOLLOWUP

Appropriate followup includes

- Education.
- Followup blood lead testing.
- Medical evaluation, if appropriate.
- Pharmacologic treatment, if appropriate.
- Environmental investigation, if appropriate.
- Referral to infant stimulation or child development programs, if appropriate.
- Referral for social services.

Not all aspects of a poisoned child's followup will be managed by the pediatric health-care provider, although the provider is an important part of the team. Through his or her interactions with the child and family and the responsible public health agency, the provider

should make sure that any appropriate interventions are occurring. The provider should make sure that the family receives education about childhood lead poisoning and ways of preventing it, and he or she should make sure that the child receives the appropriate followup blood lead testing. If the child needs a medical evaluation (for a blood lead level $\geq$20 μg/dL) or pharmacologic treatment (Chapter 7), either the provider should do it or should refer the child to a place that treats large numbers of poisoned children. The provider should make sure that the child receives an appropriate environmental investigation and remediation with the help of the public health agencies. Particularly if the child is developmentally delayed, the provider should refer the child to an appropriate infant stimulation or child development program. In many cases, lead-poisoned children and their families will also benefit from social services followup.

Chapter 5. The Role of State and Local Public Agencies

A variety of local, state and federal agencies play a role in preventing childhood lead poisoning. Pediatric health-care providers and parents should know about what these agencies do so that they can use these resources effectively. In turn, these agencies must coordinate their activities to ensure that all aspects of childhood lead poisoning prevention—health, housing, and environment—are being addressed, and to provide the most comprehensive and cost-effective services to at-risk children, their parents, and their health-care providers.

Government operations vary widely from state to state. In some states, city or local government takes the leading role in providing public health services such as lead poisoning prevention. In other states, county or state agencies take the lead role. Similarly, housing and environmental agencies with responsibility for addressing lead hazards may exist at the local, county, or state level.

PUBLIC HEALTH AGENCIES

The public health agency should

- Ensure that necessary screening services are provided.
- Analyze surveillance and other data to identify exposure patterns and high-risk populations.
- Develop and implement a primary prevention plan that focuses on the highest risk sources and populations.
- Coordinate prevention activities with other pertinent health, housing, and environmental agencies.
- Ensure that medical and environmental followup services for poisoned children are provided.

In most cities and some towns, counties, and states, lead poisoning prevention programs are included within the public health department or agency. Traditionally, these programs have focused on screening for lead poisoning and ensuring medical or environmental followup for children identified as being poisoned. Many also undertake public education activities, but, historically, lack of resources has limited these agencies' ability to focus on primary prevention.

A comprehensive, multifaceted approach to preventing childhood lead poisoning would include screening and surveillance, risk identification, primary prevention activities, interagency coordination, and services for poisoned children.

Screening

As explained in Chapter 6, screening children for lead poisoning is important both to identify poisoned children and to provide data that can be used to target communitywide interventions.

Public health agencies should have the primary responsibility for ensuring that children receive necessary blood lead screening. While it is not realistic for public agencies to actually perform most of the required screening, these agencies must work with individual and institutional pediatric health care providers to ensure that the private sector provides as much screening as possible. Screening by public agencies should focus on children who would not otherwise receive it in the private sector.

Surveillance and Risk Assessment

Before a public health agency can design and implement a primary prevention plan, the agency must assess the sources of lead in the community, exposure patterns, and high-risk populations. The lead public health agency should generally take responsibility for the types of risk assessment activities described in Chapter 9, soliciting cooperation and assistance from housing and environmental agencies when appropriate. As explained in Chapter 9, blood lead screening provides data for assessing the extent and nature of a given community's lead problem. Public health agencies should also take the lead in conducting or coordinating the collection of the environmental, housing, and demographic data needed to undertake a community-based risk assessment.

Primary Prevention

One of the most important themes of this document is the need to identify and remove sources of exposure to lead before children are harmed, that is, the need for primary prevention. Public health agencies must take a leading role in designing and implementing primary prevention programs. One important activity for public health agencies is to use the data collected from screening and surveillance to develop a primary prevention plan designed to target resources to the most pervasive sources and the highest risk populations.

Interagency Coordination

Public health agencies cannot be expected to implement primary prevention activities by themselves. Many steps that must be taken lie within the expertise or jurisdiction of other government agencies, especially those dealing with housing or the environment. To prevent lead poisoning, all public agencies with a connection to this problem need to be enlisted in the effort. The activities of different types of agencies at different levels of government must be coordinated, preferably through formal arrangements under which the different agencies meet and consult. Public health agencies should take the lead in organizing interagency task forces or committees and in ensuring that all involved agencies communicate regularly.

Providing Services to Poisoned Children

Public health agencies have traditionally been responsible for ensuring that lead-poisoned children receive appropriate medical and environmental followup, often through a formal case-management system. Until lead poisoning has successfully been eradicated, public health agencies will have to devote much of their lead-poisoning prevention resources to case management services for poisoned children.

HOUSING AGENCIES

Housing agencies should

Work with public health agencies to provide needed housing and environmental services to poisoned children.

Enforce code requirements regarding lead hazards.

Assist public health agencies in educating property owners, tenants, real estate professionals, and building contractors about lead hazards in housing.

Use regulations and policies to increase the amount of safe and effective abatement performed.

Most states, and some cities and counties, have agencies with the responsibility for regulating housing quality or developing policies to ensure that people are provided with safe and affordable housing. As the focus shifts from severely poisoned children to lead hazards in children's environments, the role of housing agencies will expand. A comprehensive, multifaceted role for housing agencies would include providing services to poisoned children; enforcing housing codes; educating the public; and making regulations and policies to increase the number of safe and effective abatements.

ENVIRONMENTAL AGENCIES

Environmental agencies should

Participate in interagency efforts to prevent lead poisoning.

Adopt a multimedia approach to addressing environmental lead hazards.

Undertake monitoring, regulatory, licensing, and enforcement activities to reduce environmental exposure to lead.

Most states, and some cities and counties, have agencies responsible for regulating exposures to toxic substances in the environment. Traditionally, such agencies have dealt with exposure to lead in the air, in drinking water, and in hazardous and solid waste, but have had little or no role in addressing the hazards associated with lead in paint. Some environmental agencies have begun to address the problems of toxic substances in housing, such as asbestos and radon, and they may also be willing to join an interagency effort to reduce exposure to lead hazards in housing. A comprehensive, multifaceted role for environmental agencies would include participation in interagency efforts, multimedia lead hazard reduction programs, monitoring, and regulation.

Chapter 6. Screening

Summary

Screening is important both to ensure that poisoned children are identified and to generate data to target primary prevention activities.

Virtually all children should be screened for lead poisoning. Screening children with a high probability of exposure to high-dose sources is the highest priority.

Screening should be done using a blood lead test.

Children at greatest risk for high-dose lead exposure should be screened more frequently.

Analytical considerations affect interpretation of blood lead test results, particularly at low levels.

Traditionally, the main purpose of a childhood lead poisoning screening program has been to identify asymptomatic lead-poisoned children and to intervene as quickly as possible to reduce their blood lead levels. An additional benefit of screening programs is that abatement of lead sources for poisoned children results in prevention of lead poisoning for children who would have been exposed to those sources in the future. As the focus in lead poisoning prevention turns more to primary prevention, an additional benefit of screening is that data generated can be used in targeting interventions to places with children at high risk for lead poisoning.

SUGGESTED PRIORITIES FOR SCREENING

Virtually all U.S. children are at risk for lead poisoning.
Children at highest risk should be given the highest priority for screening.

In 1984, the last year for which estimates are available, it is believed that between 3 and 4 million children younger than age 6 years (17% of all U.S. children in this age group) had blood lead levels above 15 μg/dL (ATSDR, 1988). Furthermore, about 74% of occupied, privately owned housing built before 1980 contains lead-based paint (defined as ≥1 milligram per square centimeter (mg/cm^2)) (HUD, 1990). **Because almost all U.S. children are at risk for lead poisoning (although some children are at higher risk than others), our goal is that all children should be screened, unless it can be shown that the community in which these children live does not have a childhood lead poisoning problem.** (Deciding that no problem exists requires that a large number or percentage of children be tested.[1]) The full implementation of this will require the ability to measure blood lead levels on capillary samples and the availability of cheaper and easier-to-use methods of blood lead measurement. Children

[1]The health departments need to take lead role in assessing whether or not a community has a childhood lead poisoning problem.

at highest risk for lead poisoning are the highest priority for screening. Table 6-1 provides guidance on the groups for which repeated screening is most strongly indicated.

Children ages 6 to 72 months who live in or are frequent visitors to deteriorated old buildings, including day care centers, make up the highest priority group. Because the highest concentrations of lead in paint were used in the early 1900s, homes built before about 1960 are of greatest concern. Children whose homes are being renovated are also at extremely high risk. Since siblings, housemates, visitors, and playmates of children with confirmed lead poisoning may have similar exposures to lead, they also should be promptly screened. In communities with a high prevalence of lead poisoning, health departments should consider door-to-door screening, since many children with lead poisoning may be missed by fixed-site screening.

Children with parents whose work or hobbies involve lead may also risk lead exposure (Chapter 3). Also, children living near lead smelters or other industries where lead is processed may be at increased risk for lead poisoning.

In general, screening and assessment for lead poisoning should focus on children younger than 72 months of age, particularly on children younger than 36 months of age. Young children engage in the most hand-to-mouth activity (and therefore are at highest risk for lead exposure) and have the most rapidly developing nervous systems, making them more vulnerable to the effects of lead. Children with developmental delays, who may exhibit pica or have more extensive hand-to-mouth activity than other children, would be expected to be at increased risk for lead poisoning even if they are 72 months of age and older. These children may have to be screened more often during early infancy, and may require screening into their school years.

Children who have unexplained seizures, neurological symptoms, abdominal pain, or other symptoms that are consistent with lead poisoning should also have their blood lead levels measured. In addition, the possibility of lead poisoning should be considered in any child with growth failure, developmental delay, hyperactivity, behavior disorders, hearing loss, anemia, etc.

Table 6-1. Priority groups for screening

- Children, ages 6 to 72 months, who live in or are frequent visitors to deteriorated housing built before 1960.
- Children, ages 6 to 72 months, who live in housing built before 1960 with recent, ongoing, or planned renovation or remodelling.
- Children, ages 6 to 72 months, who are siblings, housemates, or playmates of children with known lead poisoning.
- Children, ages 6 to 72 months, whose parents or other household members participate in a lead-related occupation or hobby.
- Children, ages 6 to 72 months, who live near active lead smelters, battery recycling plants, or other industries likely to result in atmospheric lead release.

SCREENING METHOD

> Screening should be done using a blood lead test.

Since erythrocyte protoporphyrin (EP) is not sensitive enough to identify more than a small percentage of children with blood lead levels between 10 and 25 μg/dL and misses many children with blood lead levels ≥25 μg/dL (McElvaine et al., 1991), measurement of blood lead levels should replace the EP test as the primary screening method. Unless contamination of capillary blood samples can be prevented, lead levels should be measured on venous samples. Obtaining capillary specimens is more feasible at many screening sites. Contamination of capillary specimens obtained by finger prick can be minimized if trained personnel follow proper technique (see Appendix I for a capillary sampling protocol). Elevated blood lead results obtained on capillary specimens should be considered presumptive and must be confirmed using venous blood. At the present time, not all laboratories will measure lead levels on capillary specimens.

Programs will need to increase their capacity to perform blood lead testing. During the transition to the use of the blood lead test as the primary screening method, some programs will temporarily continue to use EP as a screening test. In addition, some nutrition programs (for example, the Supplemental Food Program for Women, Infants, and Children (WIC)) use the EP test to identify children with iron deficiency.

For a discussion of the units used to report EP results (Page 48). All EP test results of ≥35 μg/dL if standardized using 241 L cm-1 mmol-1, ≥28 μg/dL if standardized using 297 L cm-1 mmol-1, or ≥70 μmol ZnPP/mol heme, if the hematofluorometer reports in these units, must be followed by a blood lead test (preferably venous) and an evaluation for iron deficiency (Page 53). Work on developing easy-to-use, cheap, portable instruments for blood lead testing is ongoing.

ANTICIPATORY GUIDANCE AND ASSESSING RISK

> Anticipatory guidance helps prevent lead poisoning by educating parents on ways to reduce lead exposure.
>
> Questions about housing and other factors are used to identify which children are at greatest risk for high-dose lead exposure.
>
> Anticipatory guidance and assessment of risk should be tailored to important sources and pathways of lead exposure in the child's community.

Guidance on childhood lead poisoning prevention and assessment of the risk of lead poisoning should be part of routine pediatric care. Anticipatory guidance is discussed in more detail in Chapter 4. The guidance and risk assessment should emphasize the sources and exposures that are of greatest concern in the child's community (Chapter 3). Because lead-based paint has been used in housing throughout the United States, in most communities it will be necessary to focus on this source.

SCREENING SCHEDULE

The Screening Schedule

The screening schedule is based on the fact that children's blood lead levels increase most rapidly at 6-12 months and peak at 18-24 months.

Anticipatory guidance on preventing lead poisoning and assessing the risk for high-dose lead exposure should be part of routine pediatric care.

The urgency and type of follow-up depends on the screening blood lead test result.

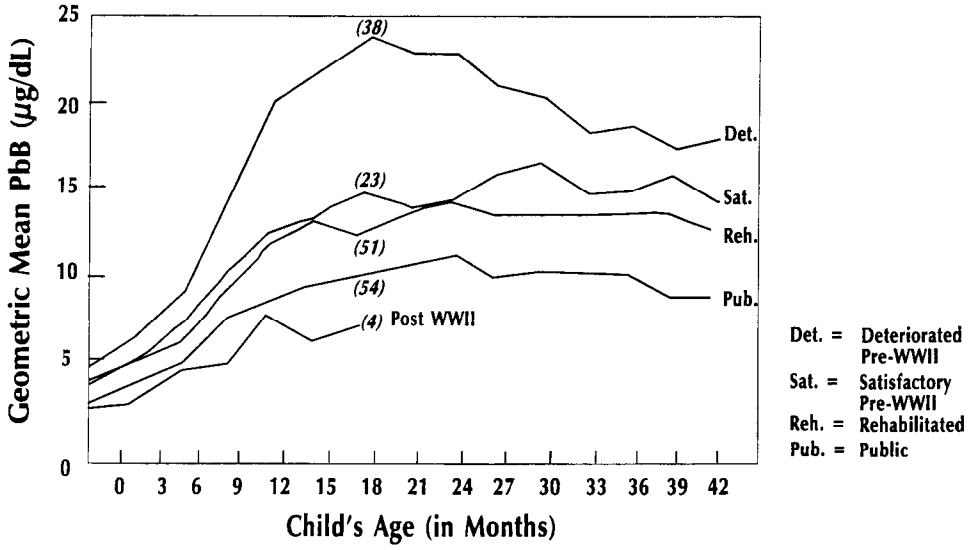
Background

The rationale for the screening schedule is based on data such as those shown in Figure 6-1. Those data were collected in a prospective study in Cincinnati (Clark et al., 1985). Blood lead levels were measured every 3 months from birth onward, and illustrate the trends in blood lead concentration in relation to the child's age and housing age and condition. Blood lead concentrations increase steadily up to at least 18 months of age. The most rapid rate of increase occurs between 6 and 12 months of age. The highest blood lead levels occur in children living in deteriorated older housing.

Assessment of Risk

Table 6-2 has sample questions. Starting at 6 months of age and at each regular office visit thereafter, pediatric health-care providers should discuss childhood lead poisoning and assess the child's risk for high-dose exposure. The questions asked should be tailored to the likely sources of exposure in the community. **The questions are not a substitute for a blood lead test.**

Figure 6-1. Relationship between children's blood lead levels and housing age and condition, Cincinnati



Note: Number of children at 18 months of age indicated in parentheses.
Source: Clark et al., 1985.

Table 6-2. Assessing the risk of high-dose exposure to lead—sample questionnaire

Does your child—

1. Live in or regularly visit a house with peeling or chipping paint built before 1960? This could include a day care center, preschool, the home of a babysitter or a relative, etc.
2. Live in or regularly visit a house built before 1960 with recent, ongoing, or planned renovation or remodelling?
3. Have a brother or sister, housemate, or playmate being followed or treated for lead poisoning (that is, blood lead ≥15 μg/dL)?
4. Live with an adult whose job or hobby involves exposure to lead (see Chapter 3)?
5. Live near an active lead smelter, battery recycling plant, or other industry likely to release lead?

Using Questionnaire Results

If answers to the questionnaire indicate that the child is not at high risk for high-dose lead exposure, the child should be screened at 12 months of age, and, if resources allow, at 24 months of age.

If answers to the questionnaire indicate that the child is at risk for high-dose lead exposure, the child should be screened starting at 6 months of age.

For children previously at low risk, any history suggesting that exposure to lead has increased should be followed up with a blood lead test.

On the basis of responses to questions such as those in Table 6-2, children can be categorized as low or high risk for high-dose lead exposure. If the answers to all questions are negative, the child is at low risk for high-dose lead exposure and should be screened by a blood lead test at 12 months and again, if possible, at 24 months (since blood lead levels often peak at ages greater than 12 months). If the answer to any question is positive, the child is potentially at high risk for high-dose lead exposure, and a blood lead test should be obtained. **For children previously at low risk, any history suggesting that exposure to lead has increased should be followed up with a blood lead test.**

Example: A pediatrician in southern California almost exclusively serves communities built after 1988. She is aware, however, that some of her patients' families store juice and punch in pottery imported from Mexico. In her guidance to parents, she warns them that lead can leach from improperly fired pottery. At every routine visit, she asks parents about the use of this pottery and screens any children whose parents use this pottery. In her anticipatory guidance and assessment of her patients' risks of lead poisoning, she emphasizes sources of exposure that are common in the community she serves.

Screening Schedule

The following sections provide a minimum screening schedule for children aged 6 up to 36 and 36 to 72 months. The schedule is not rigid. Rather, it is a guide for pediatric health-care providers and screening programs to use in conjunction with other pertinent information in determining when an individual child should be tested.

Children 6 up to 36 months of age:

A questionnaire should be used at each routine office visit to assess the potential for high-dose lead exposure and, therefore, the appropriate frequency of screening.

- ***Schedule if the child is at low risk for high dose lead exposure by questionnaire:***

  A child at *low risk* for exposure to high-dose lead sources by questionnaire should have an initial blood lead test at 12 months of age.

  If the 12-month blood lead result is **<10 μg/dL**, the child should be retested at 24 months if possible, since that is when blood lead levels peak.

  If a blood lead test result is **10-14 μg/dL**, the child should be retested every 3 to 4 months. After 2 consecutive measurements are <10 μg/dL or three are <15 μg/dL, the child should be retested in a year.

  If any blood lead test result is **≥15 μg/dL**, the child needs individual case management, which includes retesting the child at least every 3 to 4 months (Page 45).

- ***Schedule if the child is at high risk for high dose lead exposure by questionnaire:***

  A child at *high risk* for exposure to high-dose lead sources by questionnaire should have an initial blood lead test at 6 months of age.

  If the initial blood lead result is **<10 μg/dL**, the child should be rescreened every 6 months. After 2 subsequent consecutive measurements are <10 μg/dL or three are <15 μg/dL, testing frequency can be decreased to once a year.

  If a blood lead test result is **10-14 μg/dL**, the child should be screened every 3 to 4 months. Once 2 subsequent consecutive measurements are <10 μg/dL or three are <15 μg/dL, testing frequency can be decreased to once a year.

  If any blood lead test result is **≥15 μg/dL**, the child needs individual case management, which includes retesting the child at least every 3 to 4 months (Page 45).

Children ≥36 months and <72 months of age:

As for younger children, a questionnaire should be used at each routine office visit of children from 36 to 72 months of age. Any child at high risk by questionnaire who has not previously had a blood lead test should be tested. All children who have had venous blood lead tests ≥15 μg/dL or who are at high risk by questionnaire should be screened at least once a year until their sixth birthday (age 72 months) or later, if indicated (for example, a developmentally delayed child with pica). Children should also be rescreened any time history suggests exposure has increased. Children with blood lead levels ≥15 μg/dL should receive followup as described below.

Followup of children with blood lead levels ≥15 μg/dL

Followup of children with blood lead levels ≥15 μg/dL is discussed in more detail in later chapters and is briefly summarized below. In general, such children should receive blood lead tests at least every 3 to 4 months.

If the blood lead level is 15-19 μg/dL, the child should be screened every 3-4 months, the family should be given education and nutritional counselling as described in Chapter 4, and a detailed environmental history should be taken to identify any obvious sources or pathways of lead exposure. When the venous blood lead level is in this range in two consecutive tests 3-4 months apart, environmental investigation and abatement should be conducted, if resources permit.

If the blood lead level is ≥20 μg/dL, the child should be given a repeat test for confirmation. If the venous blood lead level is confirmed to be ≥20 μg/dL, the child should be referred for medical evaluation and followup as described in Chapter 7. Such children should continue to receive blood lead tests every 3-4 months or more often if indicated. Children with blood lead levels ≥45 μg/dL must receive urgent medical and environmental followup, preferably at a clinic with a staff experienced in dealing with this disease. Symptomatic lead poisoning or a venous blood lead concentration ≥70 μg/dL is a medical emergency, requiring immediate inpatient chelation therapy, as described in Chapter 7.

CLASSIFICATION ON THE BASIS OF SCREENING TEST RESULTS

On the basis of screening test results, children can be classified into categories according to their risk for adverse effects of lead. The urgency and type of followup are based on these risk classes. These classes are shown in Table 6-3.

MEASUREMENT OF BLOOD LEAD LEVELS

Venous blood is preferred for blood lead measurement.
Analytical variation in the laboratory can affect blood lead results.
Laboratories measuring blood lead levels should participate successfully in a blood lead proficiency testing program.

Several factors can influence the quality of blood lead measurements. The ubiquity of lead in the environment makes contamination of specimens during collection a major source of error. Analytical variation in the laboratory can affect results. Accuracy and precision of blood lead measurements, particularly at low concentrations, can be assured by the use of appropriate analytical standards, maintenance of equipment, training of personnel, and participation in external proficiency testing programs.

Since blood collected by venipuncture has a low likelihood of contamination compared to blood collected by fingerstick, venous blood is the preferred specimen for analysis and should be used for lead measurement whenever practicable. In addition, venous specimens provide a larger volume for analysis and are less prone to clotting and other problems that can be encountered with capillary specimens (DeSilva and Donnan, 1977; Mitchell et al., 1974).

Table 6-3. Class of child and recommended action according to blood lead measurement

Class	Blood Lead Concentration (μg/dL)	Action
I	≤9	Low risk for high-dose exposure: rescreen as described in text. High risk for high-dose exposure: rescreen as described in text.
IIA	10-14	Rescreen as described in text. If many children in the community have blood lead levels ≥10, community interventions (primary prevention activities) should be considered by appropriate agencies (see Chapter 9).
IIB	15-19	Rescreen as described in text. Take a history to assess possible high-dose sources of lead. Educate parents about diet, cleaning, etc. Test for iron deficiency. Consider environmental investigation and lead hazard abatement if levels persist.
III	20-44*	Conduct a complete medical evaluation. Identify and eliminate environmental lead sources.
IV	45-69*	Begin medical treatment and environmental assessment and remediation within 48 hours.
V	≥70*	Begin medical treatment and environmental assessment and remediation IMMEDIATELY.

*Based on confirmatory blood lead level.

Fingerstick specimens are acceptable for blood lead screening, provided that special collection procedures are followed to minimize the risk of contamination. Personnel must be thoroughly trained in collection procedures. A procedure for collecting fingerstick specimens is described in Appendix I. At the present time, not all laboratories will accept capillary samples for blood lead analysis.

Elevated blood lead results obtained on capillary specimens are presumptive and must be confirmed using venous blood. In general, children who have blood lead levels ≥15 μg/dL on capillary samples should have these levels confirmed on venous samples, according to the timetable in Table 6-4. A child with a blood lead level ≥70 μg/dL or with symptoms of lead poisoning should be treated immediately while the results of an immediate confirmatory test are awaited.

Additional Analytical Considerations

Blood lead levels can be determined by several analytic methods. The method used can affect the specimen volume required, the choice of anticoagulant (usually heparin or ethylenediaminetetraacetic acid (EDTA)), and other aspects related to specimen suitability. Specimen collection procedures and equipment must be checked for compatibility with laboratory

Table 6-4. Suggested timetable for confirming capillary blood lead results with a venous blood lead measurement

Blood Lead Level (μg/dL)	Time Within Which Blood Lead Level Should Be Obtained
<10	Not applicable
10-14	Not applicable
15-19	Within 1 month
20-44	Within 1 week
45-69	Within 48 hours
≥70	Immediately

requirements. Special lead-free evacuated tubes are available for blood collection, but standard tubes containing EDTA or heparin (lavender or green caps) can be acceptable after screening each lot to determine the lead content of the containers, needles, etc. Though reports of unsuitable levels of background lead in other collection materials are infrequent, all materials used should be determined to be lead-free before use.

Several analytical techniques available can be used to make accurate blood lead measurements at levels <25 μg/dL. These techniques are electrochemical techniques, usually anodic stripping voltammetry (ASV), and atomic absorption spectroscopy (AAS). Either of these techniques is capable of achieving detection limits <2 to 5 μg/dL. Success by these methods, however, requires careful and meticulous attention to the details of the analysis.

The reliability of a set of blood lead measurements is greatly enhanced by the use of high quality lead standard solution for instrument calibration. In the United States, the National Institute of Science and Technology has made such a material (SRM 3121) available. In addition, a set of whole blood reference materials (SRM 955A, Lead in Blood) provides a useful set of control materials over a wide range of concentrations—about 6 to 70 μg/dL.

Laboratories where blood is tested for lead levels should be successful participants in a blood lead proficiency testing program, such as the program conducted jointly by CDC, the Health Resources and Services Administration, and the University of Wisconsin. In interpreting laboratory results, it should be recognized that a proficient laboratory should measure blood lead levels to within several μg/dL of the true value (for example, within 4 or 6 μg/dL of a target value). The blood lead level reported by a laboratory, therefore, may be several μg/dL higher or lower than the actual blood lead level.

Analytical variability must be considered when interpreting blood lead results. Changes in successive blood lead measurements on an individual can be considered significant only if the net difference of results exceeds the limit of analytic variance that the laboratory allows. As a general rule, trends should not be considered significant unless the magnitude of the change is ≥5 μg/dL.

The degree of analytical variability between laboratories that employ different analytic methods usually exceeds that within a single laboratory. Therefore, a single laboratory using one analytical method should be used to best compare multiple blood lead results from an individual or a population.

ERYTHROCYTE PROTOPORPHYRIN (EP)

> EP is not a sensitive test for identifying children with blood lead levels below about 25 μg/dL.
>
> An EP level is elevated if it is ≥35 μg/dL when standardized using 241 L cm-1 mmol-1, ≥28 μg/dL when standardized using 297 L cm-1 mmol-1, or ≥70 μmol/mol when measured in μmol/mol units. All elevated EP results should be followed by a venous blood lead test.
>
> Laboratories measuring EP levels should be successful participants in an EP proficiency testing program.

Interpreting EP Results and Following Up on Children with High EP Levels

EP is not a sensitive test to identify children with blood lead levels below about 25 μg/dL, and therefore it is no longer the screening test of choice. Generally, EP is measured using a two-step extraction process followed by direct fluorometric measurement or by front-surface fluorometry (hematofluorometry). Most protoporphyrin in erythrocytes (about 90%) exists as zinc protoporphyrin (ZnPP) (Smith et al., 1980; Gotelli et al., 1980). This fraction is preferentially measured by hematofluorometers. Extraction methods measure all the protoporphyrin present, but strip the zinc from the ZnPP during the extraction process. For this reason, extraction results are sometimes referred to as [zinc] free erythrocyte protoporphyrin (FEP). Although the chemical forms measured by the two methods differ slightly, on a weight basis they are roughly equivalent, so results reported as EP, ZnPP, or FEP all reflect essentially the same analyte (Stanton et al., 1989).

In the past, an absorptivity of 241 L cm-1 mmol-1 has been used to determine EP levels. Recently, however, the correct absorptivity has been determined to be 297 L cm-1 mmol-1 (Gunter et al., 1989). Use of the correct absorptivity will result in EP values about 19% lower than those standardized using 241 L cm-1 mmol-1. Standardization of EP levels that are based on the correct absorptivity is expected to be widely adopted in 1992. Use of the correct standardization requires a change in calibration and is not simply a reduction of the screening cutoff value. Standardization criteria should also be considered when reviewing data in the literature.

An EP result of ≥35 μg/dL standardized using 241 L cm-1 mmol-1 or ≥28 μg/dL standardized using 297 L cm-1 mmol-1 is considered elevated. **All elevated EP results should be followed with a venous blood lead test to determine if lead poisoning is responsible for the elevation**. Elevated concentrations of EP also result from several health conditions other than lead intoxication, particularly iron deficiency (Reeves et al., 1984; Yip et al., 1983; Thomas et al., 1977). The iron status of children with elevated EP levels should always be determined, especially since iron deficiency and lead poisoning often coexist. In such cases, the EP may be disproportionately elevated in comparison to the blood lead level.

Some hematofluorometers report EP levels as μmol ZnPP/mol heme. For instruments that give results in these units, EP values ≥70 μmol/mol should be considered elevated and should be promptly investigated (Stanton et al., 1989).

Analytic Considerations

Only fresh blood is suitable for analysis by hematofluorometer (Blumberg et al., 1977). Complete oxygenation of sample hemoglobin is necessary to prevent low results in some instruments. The hemoglobin concentration in the sample can also affect hematofluorometer EP readings. Results obtained by extraction methods are not affected by these factors and can be used to confirm hematofluorometer EP results.

As with lead data, analytical variance must be considered when EP data are being interpreted. If trends in EP data are to be assessed correctly, analyses should preferably be performed by a single laboratory, and the variance of the method should be known when interpreting data. As with blood lead levels, interlaboratory variance usually exceeds intralaboratory variance. The observed variance for EP is wider than that for blood lead, underscoring the importance of analytical variance in the evaluation of EP data. In addition, because of substantial intermethod differences, extraction and hematofluorometer results should not be compared when assessing trends (Mitchell and Doran, 1985; Kaul et al., 1983; Peter et al., 1978). Laboratories that test patient specimens for EP levels should be participants in one or more external proficiency testing programs.

References

ATSDR (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: ATSDR, 1988.

Blumberg WE, Eisinger J, Lamola AA, Zuckerman DM. The hematofluorometer. Clin Chem 1977; 23:270-4.

Clark CS, Bornschein RL, Succop P, Que Hee SS, Hammond PB, Peace B. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. Environ Research 1985;38:46-53.

DeSilva PE, Donnan MB. Petrol venders, capillary blood lead levels, and contamination. Med J Aust 1977;1:794-5.

Gotelli GR, Wall JH, Kabra PM, Morton LJ. Simultaneous liquid-chromotagraphic determination of zinc protoporphyrin IX, protoporphyrin IX, and coproporphyrin in whole blood. Clin Chem 1980;26:205-8.

Gunter EW, Turner WE, Huff DL. Investigation of protoporphyrin IX standard materials used in acid-extraction methods, and a proposed correction for the millimolar absorptivity of protoporphyrin IX. Clin Chem 1989;35:1601-8.

HUD (U.S. Department of Housing and Urban Development). HUD Interim Guidelines on Lead-Based Paint. Washington (DC): HUD, 1990.

Kaul B, Slavin G, Davidow B. Free erythrocyte protoporphyrin and zinc protoporphyrin measurements compared as primary screening methods for detection of lead poisoning. Clin Chem 1983;29:1467-70.

McElvaine MD, Orbach HG, Binder S, Blanksma LA, Maes EF, Krieg RM. Evaluation of the erythrocyte protoporphyrin test as a screen for elevated blood lead levels, Chicago, Illinois, 1988-1989. J Pediatr (in press) 1991.

Mitchell DG, Aldous KM, Ryan FJ. Mass screening for lead poisoning: capillary blood sampling and automated Delves-cup atomic-absorption analysis. N Y State J Med 1974;74:1599-1603.

Mitchell DG, Doran D. Effect of bias in hematofluorometer measurements of protoporphyrin in screening programs for lead poisoning. Clin Chem 1985;31:386-90.

Peter F, Growcock G, Strunc G. Fluorometric determination of erythrocyte protoporphyrin in blood, a comparison between direct (hematofluorometric) and indirect (extraction) methods. Clin Chem 1978; 24:1515-57.

Reeves JD, Yip R, Kiley VA, Dallman PR. Iron deficiency in infants: the influence of mild antecedent infection. J Pediatr 1984;105:874-9.

Smith RM, Doran D, Mazur M, Bush B. High performance liquid chromatographic determination of protoporphyrin and zinc protoporphyrin in blood. J Chrom 1980;181:319-27.

Stanton NV, Gunter EW, Parsons PJ, Field PH. Empirically determined lead-poisoning screening cutoff for the protofluor-Z hematofluorometer. Clin Chem 1989;35:2104-7.

Thomas WJ, Koenig HM, Lightsey AL Jr, Green R. Free erythrocyte porphyrin: hemoglobin ratios, serum ferritin, and transferrin saturation levels during treatment of infants with iron-deficiency anemia. Blood 1977;49:455-62.

Yip R, Schwartz S, Deinard AS. Screening for iron deficiency with the erythrocyte protoporphyrin test. Pediatrics 1983;72:214-9.