Preventing Lead Poisoning in Young Children

U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control

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NOTE: 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.

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 rehabilitation. 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 revision of this document. I believe this document will be a major landmark in the effort to eliminate childhood lead poisoning from the United States.
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CONVERSION TO SYSTEME INTERNATIONAL (SI) UNITS

BLOOD LEAD

1.0 ug/dL = 0.04826 umol/L  
1.0 ug/dL = 20.72 ug/dL
0 ug/dL = 0.  umol/L
5 ug/dL = 0.241 umol/L
10 ug/dL = 0.483 umol/L
15 ug/dL = 0.724 umol/L
20 ug/dL = 0.965 umol/L
25 ug/dL = 1.206 umol/L
30 ug/dL = 1.448 umol/L
35 ug/dL = 1.689 umol/L
40 ug/dL = 1.930 umol/L
45 ug/dL = 2.172 umol/L
50 ug/dL = 2.413 umol/L

ERYTHROCYTE PROTOPORPHYRIN

1.0 ug/dL = 0.01778 umol/L  
1.0 umol/L = 56.25 ug/dL
28 ug/dL = 0.498 umol/L
35 ug/dL = 0.622 umol/L
70 ug/dL = 1.245 umol/L

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 ug/dL of whole blood.

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

A multitier approach to follow up 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 ug/dL. If many children in the community have blood lead levels > or = ug/dL, community wide interventions (primary prevention activities) should be considered by appropriate agencies. Interventions for individual children should begin at blood lead levels of 15 ug/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 looser and looser 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 ug/dL, although it was acknowledged that adverse effects occur below that level. In the past several years, however, the scientific evidence showing that
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some adverse effects occur at blood lead levels at least as low as 10 ug/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 ug/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 ug/dL to be evaluated definitively. As yet, no threshold has been identified for the harmful effects of lead.

Because 10 ug/dL is the lower level of the range at which effects are now identified, primary prevention activities community wide environmental interventions and nutritional and educational campaigns should be directed at reducing children's blood lead levels at least to below 10 ug/dL. Blood lead levels between 10 and 14 ug/dL are in a border zone. While the overall goal is to reduce children's blood lead levels below 10 ug/dL, there are several reasons for not attempting to do interventions directed at individual children to lower blood lead levels of 10-14 ug/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 ug/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 follow up 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. Community prevention activities should be triggered by blood lead levels > or = to 10 ug/dL. Medical evaluation and environmental investigation and remediation should be done for all children with blood lead levels > or = to 20 ug/dL. All children with blood lead levels > or = to 15 ug/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 ug/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 ug/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 still 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.

PRIMARY PREVENTION. Efforts need to be increasingly focused on preventing lead poisoning before it occurs. This will require community wide 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 ug/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 aid in 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 remains. 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 programs 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. 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

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 UG/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
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 \( \mu \text{g/dL} \) was considered toxic (Chisolm and Harrison, 1956). By 1978, the defined level of toxicity had declined 50% to 30 \( \mu \text{g/dL} \). Figure 2.2 shows how the federal definition of an elevated blood lead level has changed over the years.

RANGE OF EFFECTS OF LEAD

Very severe lead exposure in children (blood lead levels 380 \( \mu \text{g/dL} \)) 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 \( \mu \text{g/dL} \), 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 \( \mu \text{g/dL} \). Maternal and cord blood lead levels of 10-15 \( \mu \text{g/dL} \) appear to be associated with reduced gestational age and reduced weight at birth (ATSBR, 1988). Although researchers have not yet completely defined the impact of blood lead levels <10 \( \mu \text{g/dL} \) 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 \( \mu \text{g/dL} \) 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 \( \mu \text{g/dL} \). 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 at 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 of the more highly exposed children averaged 35 \( \mu \text{g/dL} \) (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 lower 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).
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 (Anastate, 1983)(Figure 25). 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).

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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 ug/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 REMODELED 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 remodeling 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/cm² 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 lead paint also may 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 ug/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, goose necks 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.

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 31. 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,
do 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 (OSSA) General Industry Lead Standard governing lead exposures (29 CAR 1910.1026) 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 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 OSSA comprehensive lead standard is inadequate (Landrigan, 1990; Maizlish, et al., 1990) even by those industries required to be in compliance. Furthermore, the current OSSA standard may not adequately protect the health of workers (Landrigan, 1990). OSSA 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 CERAMIC WARE, 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 ug/day in 1982, about 13 ug/day by 1985, and about 5 ug/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. Lead crystal should not be used to store food for consumption.
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.

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.

FOLK REMEDIES CONTAINING LEAD INCLUDE:
ALARCON
ALKOHOL
AZARCON
BALI GOLI
CORAL
GHASARD
GRETIL
LIGA
PAY-LOO-AH
RUEDA

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.
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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 discussed in Chapter 3.) Other education should be tailored to likely exposures in the community. For example, in some communities parents should be warned about the potential for lead exposure from improperly fired ceramic ware and imported pottery. Where water lead levels are a concern, parents could be advised to use only fully-flushed water (that is, water that has not been standing in pipes for a prolonged time) from the cold-water tap for drinking, cooking, or preparing infant formula.

SCREENING FOR CHILDHOOD LEAD POISONING

SCREENING FOR LEAD POISONING REQUIRES

Determining the child's risk for high-dose lead exposure by asking a few questions.

Measuring blood lead levels in children who are at the greatest risk for high-dose lead poisoning when they are 6 months old.

Measuring blood lead levels in children who are at lower risk for high-dose lead exposure at 12-15 months of age.

Conducting necessary followup blood lead testing of children.

The recommended screening schedule is discussed in detail in Chapter 1.

Since virtually all children are at risk for lead poisoning, universal screening is recommended, except in communities where large numbers or percentages of children have been screened and found not to have lead poisoning. (A more inexpensive and widely available blood lead test is under development.) Just as pediatric health-care providers ask screening questions about a child's development and eating habits, providers should also ask questions about a child's risk for high-dose lead exposure at every visit. (It is important to ask at every visit, since children's exposures may change over time.) On the basis of the parents' answers to these questions, the pediatric provider will be able to classify most children as being at either high or low risk for high-dose exposure to lead. The highest risk children should be screened starting when they are 6 months old, since that is when blood lead levels begin to rise. Lower risk children should be screened for the first time when they are 12-15 months old. Followup screening schedules should be based on the pediatric health-care provider's assessment of the child's risk for high-dose lead exposure and previous blood lead levels.

DOING APPROPRIATE DIAGNOSTIC BLOOD LEAD TESTING

Pediatric health-care providers should include lead poisoning in the differential diagnosis of a number of conditions. These include growth failure, developmental delays, hyperactivity, behavior disorders, hearing loss, and anemia. Children with parasites may be exhibiting pica, and the pediatric health-care provider should also consider measuring blood lead levels in such children.
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 ug/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 UG/DL. A blood lead level < 10 ug/dL is not considered to be indicative of lead poisoning.

BLOOD LEAD LEVEL 10-14 UG/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 ug/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 and decrease exposure to lead with some simple instructions. (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 ug/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 ug/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 ug/dL. Three months after that, when Johnny was 18 months old, his blood lead level was 5 ug/dL. His blood lead level was measured one year later and was 5 ug/dL and he received no further followup.

BLOOD LEAD LEVEL 15-19 UG/DL. Children with venous blood lead levels 15-19 ug/dL need more careful followup. The pediatric health-care provider should take a careful history, about sources of lead exposure (Chapter 3). Parents should receive guidance about interventions to reduce blood lead levels (Educating Parents about Reducing Blood Lead Levels). 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 greater than or equal to 15 ug/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 UG/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 greater than or equal to 45 ug/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 GREATER THAN OR EQUAL TO 70 UG/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 (Chapter 7 and Chapter 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 dish washing 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 ceramic ware 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**
- Liver
- Fortified cereal
- Cooked legumes
- Spinach

**CALCIUM**
- Milk
- Yogurt
- Cheese
- 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...