Protecting Workers Exposed to Lead-based Paint Hazards: A Report to Congress
Protecting Workers Exposed to Lead-Based Paint Hazards

A Report to Congress

Edited by:

Aaron Sussell, M.P.H., C.I.H.

Contributors:

Kevin Ashley, Ph.D.
Greg Burr, M.S., C.I.H.
Janie Gittleman, Ph.D., M.R.P.
Leroy Mickelsen, M.S., P.E.
Henryka Nagy, Ph.D.
Greg Piacitelli, M.S., C.I.H.
Robert Roscoe, M.S.
Aaron Sussell, M.P.H., C.I.H.
Elizabeth Whelan, Ph.D.
DISCLAIMER

Use of trade names and commercial sources is for identification only and does not imply endorsement by the National Institute for Occupational Safety and Health.

This document is in the public domain and may be freely copied or reprinted.

Copies of this and other NIOSH documents are available from

National Institute for Occupational Safety and Health
Publications Dissemination
4676 Columbia Parkway
Cincinnati, OH 45226–1998

Telephone number: 1–800–35–NIOSH (1–800–356–4674)
Fax number: (513) 533–8573
E-mail: pubstaff@cdc.gov

To receive other information about occupational safety and health problems, call 1–800–35–NIOSH (1–800–356–4674), or visit the NIOSH Home Page on the World Wide Web at http://www.cdc.gov/niosh

DHHS (NIOSH) Publication No. 98–112

Copies of the original document can be purchased from the National Technical Information Service for $25.00 plus handling. Please call (703) 487–4650 and ask for PB98–113319
EXECUTIVE SUMMARY

KEY RECOMMENDATIONS

- State surveillance programs should be expanded to all states where workers are exposed to lead-based paint (LBP) hazards to identify high-risk workplaces and conduct follow-up investigations where needed.

- Research and education are needed to assist small businesses involved in LBP activities in developing low-cost controls for reducing worker lead exposures and environmental releases of lead.

- Research is needed to determine better the extent of take-home lead exposures among workers who are exposed to low airborne lead levels, but who work in lead-contaminated environments. Until more data are available, protective clothing and hygiene facilities should be considered for workers in lead-contaminated workplaces, regardless of their airborne lead exposure levels.

- Research and education are needed to improve worker protection during maintenance and repainting of steel structures coated with LBP. This should include the use of improved engineering controls and design of highly protective respirators for abrasive blasting.

- Research is needed to provide a set of objective data that would be useful for employers’ initial exposure assessments of common residential lead abatement methods, and renovation and remodeling activities involving LBP.

- To reduce worker lead exposures during residential work, safer methods such as enclosure, encapsulation, and replacement should be used where possible instead of LBP removal by torch burning, heat gun, or abrasive methods.

- A system for evaluating the quality of analyses of lead in paint, dust, and soil, done in-place with portable instruments, is needed.

THE HEALTH EFFECTS OF LEAD EXPOSURE AND OCCUPATIONAL EXPOSURE CRITERIA

The toxic effects of lead are well documented in both children and adults. Workers’ exposure to lead can damage the central nervous system, cardiovascular system, reproductive system, hematological system, and the kidney. Workers’ lead exposure can also harm development of their children. Lead has been shown to be an animal carcinogen, and authors of recent studies suggest that occupational lead exposure increases the risk of cancer. Lead poisoning often goes

vii
undetected since many of the symptoms, such as stomach pain, headaches, anxiety, irritability, and poor appetite, are nonspecific and may not be recognized as symptoms of lead poisoning.

Because of national efforts to reduce environmental lead exposures, general population lead exposures in the United States have dropped significantly in the past two decades. In 1978, the Occupational Safety and Health Administration (OSHA) promulgated a lead standard to protect workers in general industry. In 1993, as required by Title X, OSHA provided an equivalent level of protection to workers in the construction industry. Lead exposures in the workplace, however, continue to be a significant public health problem.

Research studies on lead toxicity in humans indicate that current OSHA standards should prevent the most severe symptoms of lead poisoning, but these standards do not protect workers and their developing children from all of the adverse effects of lead. In recognition of this problem, voluntary standards and public health goals have been established to lower exposure limits for workers exposed to lead. The Department of Health and Human Services has established a national goal to eliminate, by the year 2000, all occupational lead exposures that result in blood lead levels (BLLs) greater than 25 µg/dL.

NIOSH SURVEILLANCE, INTERVENTIONS, AND EVALUATIONS

NIOSH conducts surveillance, intervention, and health hazard evaluation projects to identify and reduce occupational lead exposures. In the late 1980s, NIOSH started working with states to develop Adult Blood Lead Epidemiology and Surveillance (ABLES) programs at the state level. Currently, NIOSH is working with 34 states, with 25 states reporting adult BLLs regularly to NIOSH.

LEAD EXPOSURE OF WORKERS’ FAMILIES

Families of construction workers can be exposed to lead brought home from the workplace. NIOSH and New Jersey Department of Health studies indicate that a higher percentage of construction workers’ children, especially those under six years of age, have elevated BLLs when compared to age-specific averages for the United States and neighbors’ children.

METHODS TO CONTROL OCCUPATIONAL LEAD EXPOSURES DURING LEAD-BASED PAINT ACTIVITIES

Thousands of water storage tanks, fuel storage tanks, and other industrial steel structures coated with LBP are repainted annually. Typically, all of the existing LBP on the structures is removed with open abrasive blasting inside containment structures prior to repainting. This process exposes the workers to severe LBP hazards. Lead exposures are generally much lower during residential LBP work, but some tasks produce hazardous worker exposures. The work tasks and lead exposures during residential lead abatement and home renovation are similar.
METHODS FOR SAMPLING AND ANALYSIS OF ENVIRONMENTAL LEAD

To accurately identify the presence of lead in the workplace and occupational lead exposure hazards, appropriate standardized methods for sampling and analysis are essential. The sampling and analytical methods for assessment of lead in air, paint, soil, and surface dust, recommended by NIOSH in this report, are in many cases based on national consensus standards of the American Society for Testing and Materials (ASTM). Wherever possible, performance-based requirements for analytical testing are recommended.

EXPOSURE RISKS AMONG JANITORIAL AND CUSTODIAL WORKERS

NIOSH conducted an evaluation of lead exposures among custodial employees. Based on the results from this study, it would be reasonable to assume that routine janitorial tasks (such as sweeping, vacuuming, emptying trash receptacles, cleaning fixtures, and other related activities) in buildings with LBP generally would not produce hazardous worker lead exposures. However, one cannot conclude from this study that lead is never a hazard in janitorial and custodial work where LBP is present.
### ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABLES</td>
<td>Adult Blood Lead Epidemiology and Surveillance</td>
</tr>
<tr>
<td>ACGIH</td>
<td>American Conference of Governmental Industrial Hygienists</td>
</tr>
<tr>
<td>AIHA</td>
<td>American Industrial Hygiene Association</td>
</tr>
<tr>
<td>APF</td>
<td>assigned protection factor</td>
</tr>
<tr>
<td>ASTM</td>
<td>American Society for Testing and Materials</td>
</tr>
<tr>
<td>BLL</td>
<td>blood lead level</td>
</tr>
<tr>
<td>CDC</td>
<td>Centers for Disease Control and Prevention</td>
</tr>
<tr>
<td>ELPAT</td>
<td>Environmental Lead Proficiency Analytical Testing</td>
</tr>
<tr>
<td>EPA</td>
<td>U.S. Environmental Protection Agency</td>
</tr>
<tr>
<td>FTE</td>
<td>full-time equivalent (employee)</td>
</tr>
<tr>
<td>HEPA</td>
<td>high-efficiency air filter</td>
</tr>
<tr>
<td>HHE</td>
<td>health hazard evaluation</td>
</tr>
<tr>
<td>HUD</td>
<td>U.S. Department of Housing and Urban Development</td>
</tr>
<tr>
<td>LBP</td>
<td>lead-based paint</td>
</tr>
<tr>
<td>LEV</td>
<td>local exhaust ventilation</td>
</tr>
<tr>
<td>MDC</td>
<td>minimum detectable concentration</td>
</tr>
<tr>
<td>mg/m$^3$</td>
<td>milligrams per cubic meter</td>
</tr>
<tr>
<td>mg/cm$^2$</td>
<td>milligrams per square centimeter</td>
</tr>
<tr>
<td>MMWR</td>
<td>Morbidity and Mortality Weekly Report</td>
</tr>
<tr>
<td>MQC</td>
<td>minimum quantifiable concentration</td>
</tr>
<tr>
<td>ND</td>
<td>none detected</td>
</tr>
<tr>
<td>NHANES</td>
<td>National Health and Nutrition Examination Survey</td>
</tr>
<tr>
<td>NIOSH</td>
<td>National Institute for Occupational Safety and Health</td>
</tr>
<tr>
<td>NLLAP</td>
<td>National Lead Laboratory Analytical Proficiency</td>
</tr>
<tr>
<td>NTIS</td>
<td>National Technical Information Service</td>
</tr>
<tr>
<td>OSHA</td>
<td>Occupational Safety and Health Administration</td>
</tr>
<tr>
<td>PAPR</td>
<td>powered air-purifying respirator</td>
</tr>
<tr>
<td>PAT</td>
<td>Proficiency Analytical Testing</td>
</tr>
<tr>
<td>Pb</td>
<td>lead (symbol for the element)</td>
</tr>
<tr>
<td>PBZ</td>
<td>personal breathing-zone</td>
</tr>
<tr>
<td>PEL</td>
<td>Permissible Exposure Limit</td>
</tr>
<tr>
<td>PHS</td>
<td>U.S. Public Health Service</td>
</tr>
<tr>
<td>ppm</td>
<td>parts per million</td>
</tr>
<tr>
<td>REL</td>
<td>Recommended Exposure Limit</td>
</tr>
<tr>
<td>SHARP</td>
<td>Safety and Health Assessment and Research for Prevention</td>
</tr>
<tr>
<td>SIC</td>
<td>standard industrial classification</td>
</tr>
<tr>
<td>TWA</td>
<td>time-weighted average</td>
</tr>
<tr>
<td>µg/m$^3$</td>
<td>micrograms per cubic meter</td>
</tr>
<tr>
<td>µg/dL</td>
<td>micrograms per deciliter of (whole) blood</td>
</tr>
<tr>
<td>µg/ft$^2$</td>
<td>micrograms per square foot</td>
</tr>
<tr>
<td>µg/g</td>
<td>micrograms per gram</td>
</tr>
<tr>
<td>ZPP</td>
<td>zinc protoporphyrin</td>
</tr>
</tbody>
</table>
GLOSSARY

Some major definitions from Title IV of the Residential Lead-Based Paint Hazard Reduction Act of 1992 are presented here; additional definitions are contained in Title IV, Section 401.

- **“Lead-based paint”** (LBP) means paint or other surface coatings that contain lead in excess of 1.0 milligrams per square centimeter (mg/cm²) 0.5 percent by weight.

- **“Lead-based paint hazard”** means any condition that causes exposure to lead from lead-contaminated dust, lead-contaminated soil, lead-contaminated paint that is deteriorated or present in accessible surfaces, friction surfaces, or impact surfaces that would result in adverse human health effects.

- **“Abatement”** means any set of measures designed to permanently eliminate LBP hazards in accordance with established federal standards and includes removal, replacement, encapsulation, and all associated preparation, cleanup, and disposal activities.

- **“Lead hazard reduction”** means measures designed to reduce or eliminate human exposure to LBP hazards through methods including interim controls and abatement.
ACKNOWLEDGMENTS

The following individuals from NIOSH, other federal agencies, and nonfederal organizations provided review and comment of the report:

**NIOSH Reviewers**

Larry J. Elliot, M.S.P.H., C.I.H.
Lawrence J. Fine, M.D., Dr.P.H.
Jerome P. Flesch, B.S., M.S.
Ted Katz, B.A.
Mitch Singal, M.D., M.P.H.
Kathy Sykes, M.A., M.P.A.

**Other Reviewers**

Scott Clark, Ph.D., P.E., C.I.H. 
University of Cincinnati

Robert Herrick, Sc.D., C.I.H. 
Harvard School of Public Health

Thomas D. Matte, M.D., M.P.H. 
Environmental and Occupational Health Sciences Institute

Philip Landrigan, M.D., M.Sc. 
Mount Sinai School of Medicine

John D. Repko, Ph.D. 
United Brotherhood of Carpenters

Robert Goyer 
National Institute of Environmental Health Sciences

Sharon Harper 
U.S. Environmental Protection Agency

David E. Jacobs, C.I.H. 
U.S. Department of Housing and Urban Development
CHAPTER 1

HEALTH EFFECTS OF LEAD EXPOSURE AND OCCUPATIONAL EXPOSURE CRITERIA

INTRODUCTION

The health effects of lead have been previously extensively reviewed by the federal public health agencies: Agency for Toxic Substances and Disease Registry (ATSDR), Centers for Disease Control and Prevention (CDC), National Institute for Occupational Safety and Health (NIOSH). There are thousands of scientific articles on the adverse health effects of lead in either children or adults. This chapter is a synopsis of the cardinal adverse health effects of lead in adults.

Lead is a bluish-gray metal used since ancient times because of its useful properties, such as low melting point, pliability, and resistance to corrosion. The ancient Romans and Greeks first discovered its toxic effects. Hippocrates (370 B.C.) attributed a severe case of colic in a worker who extracted metals to lead exposure, and Pliny the Elder (A.D. 23–79) wrote that workers painting ships with native ceruse (white lead) wore loose bags over their faces to avoid breathing noxious dust. Lead is ubiquitous in older American homes and lead exposures in the workplace are common because of the widespread use, during the past century, of lead compounds in paints, gasoline, and industry.

Human lead exposure occurs when dust and fumes are inhaled and when lead is ingested via lead-contaminated hands, food, water, cigarettes, and clothing. Lead entering the respiratory and digestive systems is released to the blood and distributed throughout the body. More than 90 percent of total body burden of lead is accumulated in the bones, where it is stored for decades. Lead in bones may be released into the blood and re-exposes organ systems long after the original environmental exposure. This process can also expose the fetus to lead in pregnant women.

There are several biological indices of lead exposure. Lead concentrations in blood, urine, teeth, and hair can be used as biological indicators of lead exposure. Recent advances in the measurement of skeletal bone lead levels more accurately measure cumulative lead exposure and the total body burden of lead. At present, however, the best available method for monitoring biological exposure to lead is measurement of the blood lead level (BLL). The severity of symptoms associated with lead exposure generally increases as the BLL increases (see Table 1.1). No such relationship between symptoms and the other indices of lead exposure have been as well established.
A recent national survey found that the geometric mean BLL for the United States adult population (ages 20 to 74 yrs) declined significantly between 1976 and 1991, from 13.1 to 3.0 micrograms per deciliter (µg/dL). This decline was largely the result of stricter federal regulations and changes in regulated industries which reduced workplace exposures and the lead content of gasoline, paint, drinking water, and soldered food containers. To protect workers from lead poisoning, the Occupational Safety and Health Administration (OSHA) promulgated a lead standard for general industry in 1978 and an interim lead standard for the construction industry in 1993. More than 90 percent of adults now have a BLL < 10 µg/dL, and more than 98 percent have a BLL < 15 µg/dL.

Although much progress has been made in reducing lead exposures, exposures in the workplace continue to be a significant public health problem. Even with the federal regulations, thousands of adult elevated BLLs ≥ 25 µg/dL are reported each year to NIOSH by states participating in a NIOSH surveillance program (see Chapter 2 for a more complete discussion). Elimination of worker BLLs ≥ 25 µg/dL by the year 2000 is a health goal of the United States.

The toxic nature of lead is well documented. The most important aspects of lead toxicity are its effects on the central nervous system, which may be irreversible; however, lead affects all organs and functions of the body to varying degrees. The frequency and severity of symptoms among exposed workers depend upon the level of exposure. A summary of the lowest-observed-effect levels for key lead-induced health effects in adults is presented in Table 1.1.

The remainder of this chapter summarizes the NIOSH evaluation of the scientific literature regarding health effects of high- and low-level lead exposures and occupational exposure limits. In preparing this section, NIOSH consulted with the National Institute of Environmental Health Sciences.
Table 1.1 Summary of Lowest-observed-effect Levels for Key Lead-induced Health Effects in Adults*

| Lowest-observed-
<table>
<thead>
<tr>
<th>effect level (PbB)</th>
<th>Heme synthesis and hematological effects</th>
<th>Neurological effects</th>
<th>Effects on the kidney</th>
<th>Reproductive function effects</th>
<th>Cardiovascular effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>(µg/dL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>100–120</td>
<td>Encephalopathic signs and symptoms</td>
<td>Chronic nephropathy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>80</td>
<td>Frank anemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>Reduced hemoglobin production</td>
<td>Overt subencephalopathic neurological symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>Increased urinary ALA and elevated coproporphyrins</td>
<td>Peripheral nerve dysfunction (slowed nerve conduction)</td>
<td></td>
<td>Altered testicular function</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30</td>
<td></td>
<td></td>
<td></td>
<td>Elevated blood pressure (White males, aged 40–59)</td>
<td></td>
</tr>
<tr>
<td>25–30</td>
<td>Erythrocyte protoporphyrin (EP) elevation in males</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15–20</td>
<td>Erythrocyte protoporphyrin (EP) elevation in females</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 10</td>
<td>ALA–D inhibition</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Adapted from ATSDR 1990.

PbB = Blood lead concentration.

ATSDR indicates there may be no threshold for this effect.
NEUROTOXIC EFFECTS

One of the major targets of lead toxicity in adults is the nervous system, including the central and peripheral nervous systems. Lead damages the blood-brain barrier and, subsequently, brain tissues. Severe exposures resulting in BLLs > 80 µg/dL may cause coma, encephalopathy, or death. Historically, the most severe damage to the peripheral nervous system from high, chronic, workplace exposures to lead (two or more times higher than the current OSHA Permissible Exposure Limits [PEL] of 50 µg/m³) resulted in local paralysis described as “wrist drop” or “foot drop.” Because of the improved control of occupational lead exposures in recent decades, such overt symptoms of lead toxicity are rare today in the United States. Occupational lead exposures allowable under the current OSHA lead standards will not produce these obvious neurologic clinical symptoms; however, lead exposures permissible under the OSHA standards may be harmful to the central nervous system. Workers with BLLs of 40 to 50 µg/dL may experience fatigue, irritability, insomnia, headaches, and subtle evidence of mental and intellectual decline. BLLs as low as 30 to 40 µg/dL decrease motor nerve conduction velocity in workers, although these lead exposure levels are not associated with clinical symptoms. These subclinical symptoms represent early stages of neurologic damage to the central and peripheral nervous system.

HEMATOLOGIC AND RENAL EFFECTS

Anemia is one of the most characteristic symptoms of high and prolonged exposures to lead associated with BLLs > 80 µg/dL. This anemia results from the damaging effects of lead on the formation and functioning of red blood cells. Lead inhibits the synthesis of heme (the nonprotein, iron-containing component of hemoglobin) and damages the ion transport system in red blood cell membranes. Measurement of protoporphyrin (free or zinc protoporphyrin [ZPP]) concentration in red blood cells can be a good indicator of inhibition of heme synthesis by lead. There are, however, other causes (e.g., iron deficiency) of elevated protoporphyrin levels. Effects on heme synthesis can be observed at BLLs below 15 µg/dL, but the clinical significance of these effects at low BLLs is undetermined. As part of the medical evaluation for lead-exposed workers, OSHA requires measurement of blood lead and ZPP levels, hemoglobin and hematocrit determinations, red cell indices, and examination of the peripheral blood smears to evaluate red blood cell morphology.

Chronic high exposure to lead, above the OSHA PEL, may cause chronic nephropathy and, in extreme cases, kidney failure. There is substantially less evidence of kidney disease at lower exposures to lead.

REPRODUCTIVE AND DEVELOPMENTAL EFFECTS

Historical studies indicate that high exposures to lead produce stillbirths and miscarriages. Several studies conducted in the United States and abroad indicated that exposures to lower
concentrations of lead, with BLLs at or below 15 µg/dL may result in adverse pregnancy outcomes, such as shortened time of gestation and decreased fetal mental development and growth.14,15

The developing nervous system of the fetus is particularly vulnerable to lead toxicity. Neurological toxicity is observed in children of exposed female workers as a result of the ability of lead to cross the placental barrier and to cause neurological impairment in the fetus.16 A special concern for pregnant women is that some of the bone lead accumulation is released into the blood during pregnancy. Several studies conducted concurrently in the United States and other countries provided evidence that even low maternal exposures to lead, resulting in BLLs as low as 10 µg/dL, produce intellectual and behavioral deficits in children.17,18,19

BLLs of 60 µg/dL may be associated with male infertility.20 Studies in male workers indicate that exposures to lead resulting in BLLs as low as 40 µg/dL may cause decreased sperm count and abnormal sperm morphology.21,22 Several reports indicate that decreased sperm quality and hormonal changes can occur among male workers exposed to lead with BLLs of 30 to 40 µg/dL.23,24

In promulgating its general industry lead standard in 1978, OSHA recognized that children of lead-exposed workers are more likely to have birth defects, mental retardation, behavioral disorders or to die during the first year, and that these effects could occur at parental BLLs below the 50 µg/dL BLL allowed under the standard.25 At that time, OSHA determined it was not feasible to establish a lead standard that would protect workers from all physiologic changes, symptoms, and reproductive effects in men and women. As a result, OSHA said that men or women planning to have children should be advised to limit their BLLs ≤ 30 µg/dL. Subsequently, at least several large corporations developed “fetal protection” policies that excluded all fertile women from lead-exposed jobs, which were often high-paying. In March 1991, the U.S. Supreme Court (UAW, et al. v. Johnson Controls, Inc.) banned employers from barring women from hazardous jobs, finding that fetal protection policies constitute illegal sex discrimination in violation of the Civil Rights Act.

CARDIOVASCULAR EFFECTS

Chronic high exposures to lead that existed earlier in this century were associated with an increased incidence of hypertension and cardiovascular disease.26 Today these severe effects of lead exposure are rarely observed in the United States.27 Several studies reported modest increases in blood pressure among workers exposed to concentrations of lead allowable under the OSHA lead standards.28,29 Studies conducted in the general population, where lead exposures are much lower, have also indicated that increased BLLs are associated with small increases in blood pressure. This relationship appears to extend to BLLs below 10 µg/dL.30,31,32,33 A recent study suggests that long-term lead exposure, as measured by the bone lead level, is an independent predictor of development of hypertension in men in the general population.34
CARCINOGENIC EFFECTS

Lead has been shown to be an animal carcinogen. Animal studies clearly indicate that some lead compounds ingested or administered by subcutaneous or intraperitoneal injection, in quantities approaching the maximally tolerated dose, cause cancers in rodents.\textsuperscript{35,36}

Several studies have examined the relationship between workers' lead exposure and the occurrence of cancer among these workers.\textsuperscript{37,38,39} Results from two recent studies indicate that lead may increase the risk of cancer among workers exposed to high levels of lead.\textsuperscript{40,41}

The International Agency for Research on Cancer (IARC) has designated lead and inorganic lead compounds as possibly carcinogenic to humans (Group 2B), based on evidence for carcinogenicity in animals.\textsuperscript{42} The American Conference of Governmental Industrial Hygienists (ACGIH) has designated lead as an animal carcinogen, indicating that lead has been shown to be carcinogenic in animals.\textsuperscript{43}

OCCUPATIONAL EXPOSURE CRITERIA

Under the OSHA general industry lead standard (29 CFR 1910.1025), the PEL for personal exposure to airborne inorganic lead is 50 micrograms per cubic meter (µg/m\textsuperscript{3}) as an 8-hour time-weighted average (TWA). Maintaining the concentration of airborne particles of lead in the work environment below the PEL represents a preventive measure intended to protect workers from excessive exposure, which OSHA defines as a BLL > 40 µg/dL. The OSHA general industry lead standard requires lowering the PEL for shifts longer than 8 hours, medical monitoring for employees exposed to airborne lead at or above the action level of 30 µg/m\textsuperscript{3}, medical removal of employees whose average BLL is 50 µg/dL or greater, and pay retention for medically removed workers. Medically removed workers cannot return to jobs involving lead exposure until their BLL is below 40 µg/dL.

In the 1978 general industry standard, OSHA advised that men or women planning to have children should limit their exposure to maintain a BLL less than 30 µg/dL. At that time, OSHA said that feasibility constraints prevented it from establishing a lead standard that would prevent all physiologic changes, reproductive effects, and mild signs and symptoms in exposed workers.\textsuperscript{44} As required by Title X of the Residential Lead-Based Paint Hazard Reduction Act, in 1993 OSHA provided an equivalent level of protection to construction workers in an interim final rule for lead in the construction industry (29 CFR 1926.62). OSHA did not reexamine the feasibility of reducing the 1978 exposure limits for lead in this interim rule.

ACGIH has recommended that worker lead exposures be kept below 50 µg/m\textsuperscript{3} (as an 8-hour TWA), with worker BLLs to be kept ≤ 30 µg/dL. To protect lead-exposed workers, a World Health Organization study group recommended a biological exposure limit of 40 µg/dL in 1980, and further recommended that BLLs in women of reproductive ages should not exceed 30 µg/dL.\textsuperscript{45} In 1991, the U.S. Department of Health and Human Services established a national
goal to eliminate, by the year 2000, all occupational lead exposures that result in BLLs greater than 25 µg/dL.\textsuperscript{46}

CONCLUSIONS

Research studies on lead toxicity in humans indicate that current OSHA standards should prevent the most severe symptoms of lead poisoning, but these standards do not protect workers and their developing children from all of the adverse effects of lead. In recognition of this problem, voluntary standards and public health goals have established lower exposure limits for workers exposed to lead, which offer increased protection for workers and their children.
REFERENCES


