Chapter 21

OCCUPATIONAL AND ENVIRONMENTAL LEAD

CAROL I. TOBIAS, MBA, BSN*; RICHARD L. LACHIVER, MD, MPH†; AND HAN Q. BUI, MD, MPH‡

*Nurse Consultant, US Army Center for Public Health (Provisional), 1570 Wise Road, Building 1570, Aberdeen Proving Ground, Maryland 21010
†Formerly; Medical Director, Occupational Health Service of York Hospital, York, Pennsylvania; Major, US Army; Program Manager for Occupational Medicine and Residency Training Director, US Army Environmental Hygiene Agency, Aberdeen Proving Ground, Maryland
‡Commander, Medical Corps, US Navy; Chief, Occupational and Environmental Medicine, Naval Health Clinic Quantico, 3259 Catilin Avenue, Quantico, Virginia 22134

INTRODUCTION

This chapter is an update to Chapter 12, Lead, in the previous edition of this book.[1] Much of the chapter has been revised to reflect new policies related to workplace exposure limits. The references have been updated as well. This chapter recognizes efforts by the National Institute for Occupational Safety and Health (NIOSH) to review research on occupational lead exposures that noted health effects at levels half of the current permissible exposure limit (PEL), as well as Army efforts to lower the military exposure guideline to one-half of the current PEL. The US Environmental Protection Agency (EPA) initiated efforts to lower the current childhood exposure limit (10 μg/dL) based on new research that shows significant health effects occur at levels well below the limit. It is important for occupational health providers to be aware of the research basis for the proposed changes to lower workplace and environmental exposure limits.

Lead occurs naturally on Earth. The properties of lead make it very useful in commercial applications: it shields workers from radiation, resists corrosion, melts easily at low temperatures, and hardens quickly. Lead is a poor conductor of electricity and is extremely malleable, which makes it desirable in manufacturing and other job applications. However, lead exposure can lead to short- and long-term adverse health effects, so workers and young children with elevated biological indices should be monitored over time to detect any health effects. Lead can be ingested or inhaled, and air containing lead fumes or dust can lead to overexposure in unprotected workers. Medical surveillance for exposed workers should include monitoring of blood lead, zinc protoporphyrin (ZPP), and bone lead levels.

Public health measures including education and product substitution of lead for safer alternatives will reduce the average blood lead level (BLL) in the general population over time. Safety and occupational health professionals must lead the way by ensuring safer substitutes are found and safer work practices and administrative controls are adopted. Some manufacturing practices, such as lead recycling, can lead to increased industrial exposures. Lead exposures are also continuing in military applications where no substitutes for lead have been found, such as in the primer of explosive charges for ammunition. On many older military bases, lead leaches out of corroded pipes, causing drinking water contamination. Because military equipment, supplies, and training techniques have not completely substituted for lead or engineered around exposures, it is imperative that occupational and environmental medicine professionals be aware of the continued lead hazard.

HISTORY OF LEAD TOXICITY

Based on archeological and written evidence, Greeks and Romans were aware that lead had many useful properties but was toxic to humans. It is not clear how much they knew and to what extent they went to prevent lead exposures in the general population. Hippocrates was probably the first to report "lead colic" (spasmodic and recurrent episodes of abdominal pain), in 370 BCE, and Nicander described similar effects in the 2nd and 1st centuries BCE. These early healers gave no indication they understood how exposures occurred or how the disease could have been averted. Reports from the 1st century CE document that the historian Pliny the Elder warned mariners to protect themselves when painting their ships: "Cover yourselves with . . . animal bladder . . . lest you inhale this pernicious dust."[2]

Pliny the Elder warned about the risks associated with lead exposure; however, it is unlikely the hazards were common knowledge because lead's health effects take a long time to develop and often only occur after long periods of exposure. Only a few of those exposed may have suspected they were affected. Social factors played a role as well. The nobility may have surmised that, because "pernicious dust" was a problem encountered only by the working class, it did not concern them.

Lead poisoning was relatively forgotten after the collapse of the Roman Empire until the Middle Ages. Medieval Europeans may have poisoned themselves with wine contaminated with lead. Beginning in the Renaissance, rapidly expanding industrialization led to increased use of lead and corresponding increases in exposures. By the 1700s, Bernardo Ramazzini had made the connection between job, exposure type, and health effects in some trades. In particular, Ramazzini noted a high prevalence of lead exposure and lead poisoning among potters.[3]

During Prohibition, episodes of lead poisoning occurred around the country due to the consumption of illegally distilled whiskey that was contaminated with lead during the distillation process, when lead leached out of the solder in automobile radiators that were used as vapor condensers in stills.[4] Today, battery manufacturing and nonferrous metal production are two industries with the highest prevalence of lead poisoning in the United States.[5] Generally, the prevalence of lead poisoning in the US population (BLL above 25 μg/dL) is 5 per 100,000.[6]

MILITARY OCCUPATIONAL EXPOSURES

The US military has found many uses for lead. Lead musket balls and cannon balls were some of the earliest uses. Shot was produced by dropping molten lead from the top of a tall tower. As it drops, the molten lead solidifies into small spheres. Numerous historical shot towers still exist in the United States. During World War I and II, military personnel were exposed to lead in the metal of ship superstructures and on the painted surfaces of decking, walls, and overheads. After the wars, many cases of lead intoxication occurred during the large-scale disarmament of Navy ships. Lead smelting remains a common industrial operation in the private sector, and deployed soldiers in base camps situated adjacent to smelting facilities in Bosnia had high lead levels (see Chapter 6, History of Army Occupational Health, 1991–2015).

Lead is used today in munitions components, electrical solders, and ballast. Many workers at Army depots may be exposed to significant amounts of lead. Workers with the highest lead exposure levels include firing range instructors, welders, sand blasters, airline brake workers, maintenance mechanics, and pipefitters. Healthcare providers must be aware of the clinical signs and symptoms of lead poisoning. Workers exposed to lead must be enrolled in biological monitoring for blood lead as part of their ongoing medical surveillance program.

Paint Stripping and Welding

Lead is no longer permitted in paints, primarily due to the problem of environmental exposure in children through pica behaviors that involve eating chips of peeling paint on woodwork. However, large quantities of lead paint remained in the military supply system after the ban, and the Navy and other services continued to use the paint until it was depleted because of its excellent corrosion resistance in a wide range of environmental conditions, including on seagoing vessels and in the presence of chemical agents.

Healthcare personnel must be aware of the ongoing potential for lead exposure from older military hardware. This equipment is maintained at depots and will pose a risk of lead exposure as long as it continues to be used. Civilian and military personnel are commonly exposed to lead in the workplace during the removal of lead-based paint in building renovations and sheet metal work. Stripping of metal surfaces is done because (*a*) it is safer to weld on clean, unpainted metal; (*b*) equipment needs a new coat of paint; or (*c*) paint must be removed before equipment can be repaired.

Paint is usually stripped by spraying sand (or some other abrasive material such as bits of steel, walnut shells, or recycled plastic beads) forced from a compressed air source toward a painted target. Sand blasting and stripping paint by metal bead abrasion generates large quantities of highly respirable dust. Workers direct the flow of abrasive beads into many nooks and crannies to remove paint. Blasting the undersides of vehicles is problematic because of many hard-to-reach places, and blasters must direct their spray upward to strip the paint, causing abrasive material to be reflected back at the blaster. Even though paint strippers and sand blasters wear personal protective equipment (PPE), the abrasives that remove paint can also tear body suits if the spray reflects back. Stripping paint requires a significant amount of worker movement with bending, crouching, and stretching for prolonged periods in awkward positions, which can cause respirators to become dislodged.

Paint stripping continues to be a major source of lead exposure in the workplace. Depots now use robots to perform repetitive painting operations, but there are no robots available for paint stripping operations. Workers who handle waste material generated during the sand blasting operations are also at risk for lead exposure. At even greater risk are welders who work on metal surfaces containing lead. Welding is done at temperatures of between 1,000°C and 3,000°C. These temperatures vaporize lead,[7] and lead vapor is typically even more respirable than the dust produced by abrasive blasting.[8]

To reduce exposure to lead, education is needed to remind workers and service members that the old paint contains lead and that PPE must be worn properly. Supervisors must ensure welders wear adequate respiratory protection to keep exposure levels low. The US Coast Guard successfully implemented a lead poisoning prevention program to control lead exposures during ship maintenance. In this program, changes in paint removal work practices and equipment, and the proper use of air-line respirators, reduced BLLs by 50%.[9]

Firing Ranges

The Occupational Safety and Health Administration (OSHA) general industry lead standard was published in 1978.[10] Since then, a large body of literature on the health effects of lead has been published. In 2012 the US National Toxicology Program (NTP) released a monograph on the health effects of low-level lead exposure. The NTP defined low-level lead exposure as BLLs less than 10 μg/dL, and in some cases less than

5 μg/dL.[6] In 2013 the EPA released a final report of its *Integrated Science Assessment for Lead*.[11] The NTP and EPA reviews provide compelling evidence of health effects occurring at levels much lower than 10 μg/dL.

In light of the knowledge about the hazards posed by occupational lead exposure, the Department of Defense (DoD) asked the National Research Council (NRC) to evaluate potential health risks from recurrent lead exposure in firing range personnel. Specifically, NRC was asked to determine whether current exposure standards for lead on DoD firing ranges protect workers adequately. The NRC found that the OSHA PEL for lead, 50 $\mu g/m^3$, was frequently exceeded on Army, Navy, and Air Force firing ranges.[12] The NRC also reviewed the epidemiologic and toxicologic data and concluded there was overwhelming evidence that the OSHA standard provides inadequate protection for DoD firing range personnel and other worker populations covered by the lead standard.[12] The NRC recommended several engineering and administrative controls[12]:

1. **Ammunition substitution**. Exposure to airborne lead might be reduced by replacing traditional lead bullets with nylon-clad, copper-jacketed, zinc-based, or other forms of ammunition. Training requirements may limit the use of some ammunition, and the use of jacketed and alternative bullets may significantly increase the costs of training.
2. **Improvements in range design and ventilation**. The NRC recommended improving the HVAC systems in existing ranges to reduce or minimize airborne lead levels. Portable filtration units may be a cost-effective way to reduce airborne lead in indoor firing ranges.
3. **Range cleaning**. Housekeeping is also critically important for controlling lead contamination of surfaces in and around a shooting range. Even at ranges with good ventilation and use of ammunition with lead-free primers, poor housekeeping or failing to decontaminate a range thoroughly before switching primers may adversely affect lead exposures.[13] The Navy Environmental Health Center notes in its *Indoor Firing Ranges Industrial Hygiene Technical Guide*[14] that although there are no established limits for surface lead contamination in workplaces, OSHA has indicated in a compliance instruction for the construction industry that an acceptable lead load for non-lead work areas should be 200 $\mu g/ft^2$. Appendix D of the technical guide suggests clearance levels of 200 $\mu g/ft^2$ for interior floors and horizontal surfaces and 800 $\mu g/ft^2$ for exterior concrete.[14]
4. **Hygiene practices**. Strict adherence to OSHA recommendations for personal hygiene is critical. A cross-sectional study of 119 lead handlers in a battery recycling plant in Japan detected lead contamination on workers' hands even after they had washed their hands or bathed.[15]

More recent investigations have demonstrated that washing with soap and water is not effective in removing lead from skin. After workers in lead acid battery plants showed significant dermal-oral lead exposures despite washing with soap and water, NIOSH researchers developed a highly (nearly 100%) effective hand wipe method for removing lead from skin, using a mixture of isostearamidopropyl morpholine lactate and citric acid applied with a textured absorbent material. It is now clear that hand decontamination, rather than washing, is required to ensure complete removal of lead.[16] Preventing hand-to-mouth exposure to lead requires skin decontamination and colorimetric testing to detect residual contamination.[16] Worker education on lead must emphasize the importance of skin decontamination rather than emphasizing hand washing alone. Further, workers should be routinely tested for qualitative lead skin contamination with a colorimetric indicator.

Electrical Soldering and Ballast Handling

Several US Army depots employ personnel who solder electrical components, which generates small amounts of lead fume. However, the actual risk of lead exposure to personnel who solder is relatively low—much lower than the risk to welders. The risk from electrical soldering is greatest where ventilation is limited, such as inside enclosed or confined spaces. The European Union banned lead use in solder for electronics in 2011.[17]

Ballast is typically bulk metallic lead. Ballast handlers, who place weight on ships and planes to improve their stability, can inhale or ingest lead dust that sloughs off. Other workers who handle bare lead face similar hazards. Although the risk of exposure from handling bulk lead is not as high as the risk associated with inhaling lead fumes, exposure controls are necessary. The risk of exposure to lead ballast can be defined through an industrial hygiene survey. Controls include use of PPE and engineering measures such as adequate ventilation.

ENVIRONMENTAL EXPOSURES IN THE UNITED STATES

Air

The 1973 banning of tetraethyl lead in gasoline led to a substantial reduction of lead in the air around major US cities. Although the ban was phased in over many years, leaded gasoline is no longer available for use in on-road vehicles in this country. However, other countries around the world have not yet implemented this change, so troops may be exposed when they deploy to combat areas or provide humanitarian assistance in these places. Lead smelters have shut down as well; only one currently operates in the United States, in Herculaneum, Missouri.

Current sources of airborne environmental lead include small airplane engines, exempted from the law, that use leaded aviation gasoline, which accounts for 50% of the US lead pollution annually.[18] Other sources of lead pollution include smelting, mining, and workplaces where welding, paint stripping, and grinding of lead-based metal alloys occur. The EPA national ambient air quality standard for lead is 1.5 $\mu g/m^3$.[19] Most inhabitants of military installations are not at risk from airborne lead pollution; base industrial activities do not generate large enough quantities of airborne lead to pose a health hazard.

Water

Lead contaminates surface and ground water through intentional dumping, unintentional deposition, or washing out of ambient air by rain. Lead can contaminate surface and ground water when it leaches out of tailings from smelting, mining, and industrial wastes, as well as when industrial wastes are deliberately discharged into sewers.

EPA has set the maximum contaminant level of lead in drinking water at 0.015 mg/L.[20] Lead can enter drinking water as a contaminant when it leaches out of lead pipes bringing water into a house or worksite. Lead was banned in pipes in the construction industry in the 1990s, but houses built in the 1980s and earlier are at increased risk of having high lead levels in the pipes. Lead is only slightly soluble in water, but heat and an acidic pH increases its solubility. Further, if the plumbing system is used as an electrical ground, the resulting slight electrical charge increases the solubility of lead in drinking water. In addition, if water stands overnight in pipes, there is time for the lead to leach out and enter the tap water.[20]

Generally, plumbing that contains lead must be replaced. When it is not possible to replace the pipes, filtered or bottled water should be utilized. If the water's pH is acidic, neutralizing it can help prevent leaching. (These short-term fixes are intended to serve until lead-contaminated plumbing can be replaced.)

The city of Flint, Michigan, was taken over by the state and in an effort to cut costs, switched Flint's drinking water from Lake Huron to the Flint River in 2011. Unfortunately, the state chose not to pay the $100 per day cost for anti-corrosion treatment, which allowed lead to leach from old pipes into the water supply. In some locations, lead measured 10 times greater than the EPA limit for lead in drinking water. The city directed residents to use bottled water for the past 3 years, and only this year did the lead levels in the water supply fall to acceptable levels.[21]

Soil

The EPA has identified the three main sources of lead in soil: (1) Lead-based paint on exterior surfaces of buildings can flake off and enter the soil. (2) Local industrial pollution (eg, from smelters) can generate airborne lead that enters the soil. (3) Combustion of leaded gasoline in small planes and race cars can generate airborne lead that enters the soil.[22] Soil contamination, particularly around older buildings with lead-based paint, is fairly common. Children playing in the area may ingest lead when their hands or toys come in contact with their mouths. When soil contamination is present, the soil should be removed as soon as possible. In the interim, hand wipes and hygiene stations should be provided to permit removal of lead from hands.

ENVIRONMENTAL REGULATIONS

Regulations pertaining to environmental lead include the Safe Drinking Water Act[20]; the Comprehensive Environmental Response, Compensation, and Liability Act[23]; and the Clean Air Act.[24] These regulations cover exposure to lead from many sources including drinking water, air, food, and consumer items such as paint. The Resource Conservation Recovery Act covers the proper handling, storage, and disposal of lead.[25] Military preventive medicine specialists are responsible for the occupational and environmental health of the installation population. They ensure the post's drinking water is safe; that post buildings including housing and day-

care facilities are lead free; and that hazardous waste is handled properly. These specialists know the EPA and OSHA regulations pertaining to lead and work to ensure compliance and minimize lead exposures.

Drinking Water

The EPA regulates lead in drinking water and has proposed the long-term goal of zero lead in drinking water. The current lead regulations state that lead solder cannot be used in plumbing joints. Furthermore, the public must be notified if drinking water is contaminated with lead.

Ambient Air

The EPA used the Clean Air Act to require the complete phase-out of lead in gasoline, and a substantial reduction of airborne lead has been achieved. Currently, the law permits no more than 1.5 μg/m³ of lead in the air.[24] The reduction of lead in ambient air has markedly reduced the levels of lead in soil and surface water as well.

Food Contamination

Lead can contaminate food and drink. The Food and Drug Administration (FDA) recognized that food packaged in tin cans sealed with lead solder can become contaminated when the lead in the solder leaches into the food, particularly if the food is acidic.[26] Additionally, glaze used in the production of imported ceramic pottery occasionally contains lead, and if the pottery is used in meal production, food can become contaminated.[26] The FDA has focused on lowering lead content in canned foods and controlling the entry of contaminated pottery and pesticides into the United States.[26] In July 2010, the FDA examined foods for lead content and found that some commercial juice and food products contain lead, but the levels were below the FDA-allowable levels of 6 μg for children under age six, 25 μg for pregnant women, and 75 μg for other adults.[27]

Lead in Paint

The Consumer Product Safety Improvement Act of 2008 (Public Law 110-314) lowered the acceptable concentration of lead in paint from 0.06%, or 600 ppm, to 0.009%, or 90 ppm.[28] The Department of Housing and Urban Development has developed guidelines on lead abatement in homes, which apply to on-base housing and other buildings such as childcare centers that contain lead-based paint.[29] Base environmental engineers and preventive medicine personnel must become familiar with these guidelines to ensure compliance with changes in the law.

OCCUPATIONAL SURVEILLANCE

OSHA mandates that when the BLL is over 60 μg/dL, the individual requires medical management and medical removal from ongoing exposure at work.[10] For these individuals, the BLL should be checked monthly until the BLLs have returned to background levels. The regulations allow return to work when BLLs fall below 40 μg/dL.[10] The Centers for Disease Control and Prevention's state-based Adult Blood Lead Epidemiology and Surveillance program tracks laboratory-reported elevated BLLs. Data from 40 state programs was collected and analyzed in 2008 and 2009. When the prevalence rate in 2009 was compared to the prevalence rate in 1994, a drop of 7.7 cases per 100,000 was observed.[30] Workers in battery manufacturing, smelting, refining of nonferrous metals, painting, and paper hanging had the greatest number of exposures.[30] Additionally, people who shoot firearms and pack their own ammunition can be exposed to lead, and those who remodel and paint their own homes can run into difficulties with lead-based paint exposure. People who have been shot and retain fragments of the round in their bodies may be lead exposed as well.[30]

The California Department of Public Health requires all laboratories performing lead analyses on blood samples in California to report all results to the department. The department's Occupational Lead Poisoning Prevention Program collects and analyzes test results for adults (age 16 years and over) and enters the data into the California Occupational Blood Lead Registry.[31] This information is used to assist in lead poisoning prevention efforts. The data from the California registry from 2008 to 2011 showed that 10% of workers tested have BLLs above 10 μg /dL.[31] The BLL distribution for workers varied significantly by industry. Of those with BLL elevations, 58% of workers worked in manufacturing, 17% worked in construction, 5% worked in demolition, 4% worked in lead remediation, and 23% worked in other industries.[31] Storage battery manufacture including smelting during battery recycling had much higher percentages of workers with BLLs above 10 μg/dL than other industries.[31]

TESTS FOR LEAD EXPOSURE

Blood Lead Level

The BLL, which shows the amount of lead present in the blood, is the most frequently used test in lead medical surveillance. Lead has a half-life of 36 days in the blood.[32,33] Lead is rapidly deposited in mineralizing tissues and tends to accumulate in bone regions undergoing the most active calcification at the time of exposure.[6,34,35] BLLs can rise when bone is resorbed due to advanced age, broken bones, chronic disease, hyperthyroidism, kidney disease, menopause, osteoporosis, physiologic stress, pregnancy, and breastfeeding. Calcium deficiency worsens the bone-to-blood mobilization of lead.[6,34,35] The average US adult BLL is about 1.2 μg /dL.[30] A BLL over this baseline should be considered a sentinel event indicating a possible breakdown in workplace exposure controls.[30]

The Reference Chemistry Laboratory at the Department of Pathology and Area Laboratory Services at Ft Sam Houston, Joint Base San Antonio, performs the majority of BLL tests for Army medical treatment facilities. The laboratory reports elevated BBLs to the ordering physician, who contacts the patient regarding medical follow-up. The laboratory also notifies the preventive medicine department, which notifies the state public health office and assists in the investigation into the cause of the elevated BLLs.

For women who are pregnant or lactating, BLLs should be under 5 μg/dL. For everyone else, if the BLL is less than 10, no action is required. If the BLL is greater than 10 but less than 20, the individual should be counselled about the increased health risk of cardiovascular, renal, reproductive, and neurological disease.[36]

Zinc Protoporphyrin

The ZPP test, which measures the effect of lead on hemoglobin synthesis, should be obtained at the same time a BLL is ordered. ZPP is formed after free erythrocyte protoporphyrin (FEP) builds up in red blood cells when lead inhibits the enzyme ferrochelatase. Ferrochelatase catalyzes the insertion of a ferrous ion into protoporphyrin IX to form heme. If it is inhibited by lead, the protoporphyrin IX incorporates a zinc ion instead of a ferrous ion, producing ZPP.[37,38]

ZPP elevations in both acute and chronic exposure scenarios lag behind elevations in BLL by 8 to 12 weeks.[37,38] ZPP, when ordered together with BLL, provides useful clinical information on the duration of lead exposure because ZPP rises gradually over 8 to 12 weeks, and then drops off, while the BLL rises immediately. ZPP levels remain elevated in chronic exposure cases. In acute exposure cases when exposure stops, BLL drops off fairly quickly. Combined elevations in BLL and ZPP suggest that lead exposure began 8 to 12 weeks earlier or the individual has been chronically exposed to lead.[37,38] However, there is large variability in ZPP measurements and poor sensitivity at low lead exposure levels.[37,38]

Because of the limitations of the BLL at low lead levels, healthcare providers should consider requesting a complete blood count to look for evidence of basophilic stippling in red blood cells as early evidence of exposure when lead exposure levels are low or the lead exposure is suspected but not well documented.

Bone Lead Measurement

Lead is either excreted in the urine or stored in bone.[6,34,35,39] Skeletal bone contains approximately 95% of the lead body burden.[39] Blood lead has a half-life of about 35 days, so bone lead may be a better measure of the total lead burden.[6,34,35,39]

Bone lead is a measured by noninvasive x-ray fluorescence testing. The detector causes fluoresce in the lead atom and emission of x-rays that are proportional to the amount of lead present in bone.[40,41] The x-ray florescence produces low-level radiation and takes 30 minutes.[40,41] Unfortunately, x-ray fluorescence testing is available only at a few US research centers. Efforts are underway to develop a portable system capable of quickly measuring bone lead in a few minutes.[41]

Cumulative Blood Lead Index

Because of the dearth of x-ray fluorescence equipment, BLLs will remain the mainstay of biological monitoring for lead-exposed workers for the foreseeable future. The cumulative blood lead index (CBLI) is a measure of chronic lead exposure that can be calculated by multiplying the average BLL by the number of years of exposure. This measure may be helpful in determining how long chelation therapy might be needed in patients chronically exposed to lead.[33]

CLINICAL IMPLICATIONS OF LEAD EXPOSURE

When lead enters the body it serves no useful purpose. Toxic effects can occur, regardless of age, gender, or exposure pathway.

Effects in Children

In children, lead prevents the uptake of iron, zinc, and calcium, which are essential minerals needed for brain and nerve development. Often children show no signs of lead toxicity until the end of elementary school. Lead affects the nervous system more than any other organ system, and has been shown to cause behavioral problems and attention deficit hyperactivity disorder (ADHD). Acute exposures causing high BLLs (between 70 and 80 μg/dL) may produce encephalopathy and accompanying signs of ataxia, convulsions, hyperirritability, stupor, coma, and even death. These levels also produce long-term neurological and behavioral changes.[6,34,35] Childhood lead poisoning increases the risk of delayed learning, lower IQ, hypertension, renal failure, and reproductive problems later in life. Even children with BLLs less than 5 μg/dL have been shown to have behavioral problems, an increased risk of ADHD, and decreased cognitive performance based on lower IQ, academic achievement, and specific cognitive measures.[6,34,35]

Effects in Adults

In adults lead can cause signs and symptoms of exposure that include high blood pressure, joint and muscle pain, difficulties with memory or concentration, headache, abdominal pain, mood disorders, and problems in pregnancy. Lead can cause hypertension in both men and women and causes lower sperm counts in men.[6] Research has also shown that BLLs of 5 μg/dL have reduced renal function and that BLLs of 10 μg/dL have caused hypertension.[6,34,35]

Pregnancy

Sufficient evidence shows that maternal BLLs under 5 μg/dL are associated with reduced fetal growth or lower birth weight.[6,34,35] Maternal BLLs may be important predictors of the risk of developing eclampsia and preeclampsia.[6,34,35] There is also sufficient evidence showing that BLLs under 5 μg/dL are associated with decreased renal function, and that BLLs under 10 μg/dL are associated with increased blood pressure and hypertension during pregnancy.[6,34,35]

The risk for spontaneous abortion is elevated at lead levels between 40 and 60 μg/dL. A prospective cohort study of pregnant women in Mexico City noted a statistically significant dose-response relationship between maternal BLLs and risk for spontaneous abortion at BLLs between 0 and 30 μg/dL.[6,34,35] There is also limited evidence that maternal BLLs less than 10 μg/dL are associated with preterm birth and spontaneous abortion.[6,34,35]

Neurological Effects

Lead can cause neuropathy and encephalopathy at extremely high BLLs (over 80 μg/dL). Lead exposure can cause brain damage and problems with cognition, decision-making, and executive functioning. Lead-exposed adults have had problems with aggressive behavior and difficulties with fine motor control. Lower IQ, neuropsychological defects, and reduced hearing have been documented at BLLs less than 10 μg/dL.[6,34,35]

Renal Effects

Lead exposure was found to be strongly associated with renal effects in several studies.[6,34,35] Lead exposure in childhood has been observed to cause chronic renal disease and reduced renal function in adults who were exposed at high levels. BLLs of 1.5 μg/dL in children resulted in reduced kidney function in adolescents.[6,34,35] The glomular filtration rate (GFR) was reduced in adults who were exposed to high lead levels in childhood, and reduced GFR was found to be a risk factor for chronic renal failure. BLLs less than 5 μg/dL have been associated with both decreased GFR and chronic kidney disease.[6,34,35] BLLs less than 10 μg/dL in childhood have been associated with hypertension in adults. Repetitive lead exposures can cause chronic and irreversible chronic interstitial nephritis.[6,34,35]

Hematological Effects

Lead causes both anemia and basophilic stippling of red blood cells.[31,32] High lead levels can cause hemolytic anemia by decreasing red blood cell survival. Chronic lead poisoning can also cause hypochromic, normochromic, normocytic, or microcytic anemia.[32,33]

Endocrine Effects

High BLLs have been found to lower vitamin D levels and stop the conversion of vitamin D to 1,25-dihydroxycholecalciferol, which regulates calcium homeostasis. Lower 1,25-dihydroxycholecalciferol levels impair cell growth and tooth and bone development.[6,34,35]

Gastrointestinal Effects

Children and adults who have been exposed to lead may present with severe cramping and abdominal pain. The pain may be mistaken for an acute abdomen or appendicitis. Individuals with chronic lead poisoning often have chronic constipation as well.[6,34,35]

Cardiovascular Effects

Lead exposure is associated with the development of hypertension. Studies of people with BLLs of 40 to 60 μg/dL seen in the occupational setting show an increased risk of hypertensive heart disease and cerebrovascular disease in later life.[6,34,35] More recent studies noted an association with BLLs less than 10 μg/dL and hypertension.[6,34,35]

CHELATION

Chelation, which is the binding of lead to a binding agent, is the usual treatment for lead poisoning when BLLs are extremely high.[36,42] The FDA has approved the oral chelation agent 2,3-dimercaptosuccinic acid (DMSA), also known as succimer, for the treatment of lead poisoning in children. DMSA has also been found to be effective in treating lead poisoning in adults.[36,42] The baseline laboratory tests should include BLL, ZPP, complete blood count, neutrophil count, complete metabolic panel, and urinalysis to check for protein.[42] When BLLs are greater than 100 μg/dL, chelation should be performed in the intensive care unit, where individuals can be closely monitored. Individuals whose BLLs are between 80 and 100 μg/dL should be offered chelation if they are exhibiting symptoms of lead poisoning.[6,34–36] Chelation should be deferred until the absolute neutrophil count is above 1,200.

The recommended adult dosing of DMSA is 500 mg three times a day for 5 days, followed by 500 mg twice a day for 14 days. On day 6 and 20, the BLL, ZPP, complete blood count, complete metabolic panel, and urinalysis should be repeated.[42] The most common side effects of chelation therapy are gastrointestinal symptoms, nausea, transient rash, elevated liver transaminase enzymes, and neutropenia.

Calcium ethylenediaminetetraacetatic acid (EDTA), which increases renal excretion of lead, is clinically indicated when BLL is above 80 μg/dL and is administered as an alternative to DMSA by intravenous or intramuscular injection for 3 to 5 days. Treatment with calcium EDTA should be performed in an intensive care setting to monitor for and manage side effects.[6,34,35] The major site of potential toxicity is the kidney. Tubular necrosis is dose related, reversible, and associated with hematuria and proteinuria. Adequate hydration is important. Adverse side effects include hypotension and cardiac rhythm changes, including bradycardia, atrial-ventricular block, and ventricular dysrhythmias. Electrocardiograms must be monitored for the development of arrhythmias during infusion. The baseline laboratory tests BLL, ZPP, complete blood count, and complete metabolic panel should be repeated on day 3 and 5 of the treatment.[6,34,35]

CASE STUDY

A 33-year-old male active duty service member who immigrated to the United States in his early 20s developed acute abdominal pain while on a 2-week training mission. He was admitted to a local hospital and had a full workup that showed elevated lipase, amylase, and anemia. He was diagnosed clinically with pancreatitis. Over the next month he continued to have abdominal pain and experienced a 40-pound weight loss. He began to notice "bone pain" in his upper and lower extremities, fatigue, and insomnia. He was referred to hematology for evaluation of his anemia. Because of significant basophilic stippling, a BLL was ordered, which came back highly elevated: over 100 μg/dL. His ZPP was also markedly elevated, at over 400 μg/dL, which indicated chronic lead exposure. His personal history included a significant childhood exposure to construction sites in his country of origin. After coming to the United States, he worked for several more years in the construction industry. Lead is not considered an exposure hazard in his military occupational series.

The service member was admitted to a medical treatment facility and received lead chelation therapy in an inpatient setting. He was then followed as an outpatient for many months. Six months after presentation, his blood lead remained above 50 μg/dL. Even with this significant elevation, he had returned to full duty, was running nearly 15 miles per week, and had passed his physical training test. Ultimately the consulting occupational medicine physician (HQB) determined that the elevated blood lead was the result of a mixed exposure: chronic inhalation and ingestion exposure during childhood and as a young adult on construction sites, overlaid with an acute ingestion exposure of grown-in-China over-the-counter green tea supplements.

SUMMARY

Lead occurs naturally, and its properties make it an important commodity in manufacturing, particularly in less developed countries, where deployed service members may be exposed. Exposures in overseas locations have resulted from poor environmental controls where troops deploy, often without knowledge that lead is present in the environment. Military munitions still employ lead primers, so service members can be exposed in indoor ranges with inadequate ventilation.

Lead also remains an occupational health risk on military installations. Unprotected work with lead can cause inhalation or ingestion of fumes or dust, which must be monitored through BLLs. Workers with elevated biological indices should be considered for removal from exposure and monitored for possible short- and long-term adverse health effects.

With improved hazard communication, improved engineering controls, better utilization of PPE, and product substitutions, BLLs in the population have decreased over time. However, safety and occupational health professionals must remain aware that even with substitutions and improved work practices, exposures will continue. Breakdowns in engineering controls and new recycling initiatives will expose a new generation of workers to lead. Also, military equipment, supplies, and training techniques may still expose personnel to lead. It is imperative that occupational and environmental medicine professionals understand that lead remains a hazard in today's workplace. To reduce exposures in the workplace and in the general population, providers must maintain current knowledge of lead properties, regulations, evolving sources of exposure, diagnostics, medical surveillance protocols, and treatments.

REFERENCES

1. Lachiver RM. Lead. In: Deeter DE, Gaydos JC, eds. *Occupational Health: The Soldier and the Industrial Base*. Washington, DC: Borden Institute; 1993: Chap 12.

2. Hunter D. *The Diseases of Occupations.* 10th ed. London, England: Hodder and Stoughton; 2010.

3. Fuortes LJ. Health hazards of working with ceramics. Recommendations for reducing risks. *Postgrad Med.* 1989;85(1):133–136.

4. O'Neal B. Moonshining. Texas State Historical Association, Handbook of Texas online. http://www.tshaonline.org/handbook/online/articles/jbm01. Published June 15, 2010. Accessed August 29, 2017.

5. Centers for Disease Control and Prevention. Very high blood lead levels among adults - United States, 2002-2011. *MMWR Morb Mortal Wkly Rep.* 2013;62(47):967–971. https://www.cdc.gov/mmwr/preview/mmwrhtml/mm6247a6.htm. Accessed August 24, 2017.

6. US Department of Health and Human Services, National Toxicology Program. *Health Effects of Low-Level Lead*. Washington, DC: DHHS; 2012. http://ntp.niehs.nih.gov/NTP/ohat /Lead/Final/MonographHealthEffectsLowLevelLead_NewISSN_508.pdf. Accessed June 13, 2017.

7. Zenz C. *Occupational Medicine: Principles and Practical Applications*. Chicago, IL: Yearbook Medical Publishers; 1988.

8. Levy BS. *Occupational Health, Recognizing and Preventing Work-Related Disease*. Boston, MA: Little, Brown; 1983.

9. Hall FX. Lead in a Baltimore shipyard. *Mil Med.* 2006;171(12):1220–1222.

10. 29 CFR, Part 1910.1025. Lead in general industry. https://www.osha.gov/pls/oshaweb/owadisp.showdocument?p_table=standards&p_id=10030. Accessed August 23, 2017.

11. US Environmental Protection Agency. *Integrated Science Assessment for Lead*. Washington, DC: EPA; July 2013. http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=255721. Accessed March 3, 2014.

12. National Research Council Committee on Toxicology. *Potential Health Risks to DoD Firing-Range Personnel from Recurrent Lead Exposure*. Washington, DC: National Academies Press; 2012: 117–123.

13. Scott EE, Pavelchak N, DePersis R. Impact of housekeeping on lead exposure in indoor law enforcement shooting ranges. *J Occup Environ Hyg.* 2012;9(3):D45–51. doi: 10.1080/15459624.2011.648571.

14. Navy Environmental Health Center. *Indoor Firing Ranges Industrial Hygiene Technical Guide.* Portsmouth, VA; NEHC; 2002. Technical Manual NEHC–TM6290.99-10. http://www.med.navy.mil/sites/nmcphc/Documents/policy-and-instruction/ih-indoor-firing-ranges-technical-guide.pdf. Accessed June 13, 2017.

15. Sato M, Yano E. The association between lead contamination on the hand and blood lead concentration: a workplace application of the sodium sulphide (Na2S) test. *Sci Total Environ.* 2006;363(1-3):107–113. doi: 10.1016/j.scitotenv.2005.07.009.

16. Esswein EJ, Boeniger MF, Ashley K. Handwipe method for removing lead from skin. *J ASTM Int.* 2011;8(8):67–81.

17. QNET LLC. *Directive RoHS2 2011/65/EU.* Elk River, MN: QNET; 2013. http://www.ce-mark.com/RoHS2.pdf. Accessed August 16, 2017.

18. Kar A. Lead still found in gasoline? The answer for small airplanes is, surprisingly, yes. Natural Resources Defense Council expert blog. https://www.nrdc.org/experts/avinash-kar/lead-still-found-gasoline-answer-small-airplanes-surprisingly-yes. Published December 30, 2010. Accessed August 29, 2017.

19. 40 CFR, Parts 50, 51, 53, 58. National ambient air quality standards for lead: final rule. https://www.gpo.gov/fdsys/pkg/FR-2008-11-12/pdf/E8-25654.pdf. Accessed August 16, 2017.

20. The Safe Drinking Water Act of 1974, as amended. Pub L No. 93-523. https://www.gpo.gov/fdsys/pkg/STATUTE-88/pdf/STATUTE-88-Pg1660-2.pdf. Accessed August 17, 2017.

21. Franz J. It's been almost three years since Flint's water crisis began. What have we learned? [transcript]. *Science Friday*. Public Radio International. February 26, 2017. https://www.pri.org/stories/2017-02-26/it-s-been-almost-three-years-flint-s-water-crisis-began-what-have-we-learned. Accessed August 17, 2017.

22. US Environmental Protection Agency and Battelle Memorial Institute. *Sources of Lead in Soil: A Literature Review*. Washington, DC: EPA; 1998. EPA 747-R-98-001a. http://www.epa.gov/sites/production/files/documents/r98-001a.pdf. Accessed June 13, 2017.

23. 42 USC §9601. Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA). https://www.law.cornell.edu/uscode/text/42/chapter-103/subchapter-I. Accessed August 17, 2017.

24. 42 USC §7401 et seq. The Clean Air Act of 1970. https://www.epa.gov/laws-regulations/summary-clean-air-act. Accessed August 17, 2017.

25. The Resource Conservation and Recovery Act of 1979. Pub L No. 94-580. https://www.gpo.gov/fdsys/pkg/STATUTE-90/pdf/STATUTE-90-Pg2795.pdf. Accessed August 17, 2017.

26. US Food and Drug Administration. Guidance for industry: The safety of imported traditional pottery intended for use with food and the use of the term "lead free" in the labeling of pottery; and proper identification of ornamental and decorative ceramicware. http://www.fda.gov/Food/GuidanceRegulation/GuidanceDocumentsRegulatoryInformation/ChemicalContaminantsMetalsNaturalToxinsPesticides/ucm214740.htm. Published November 2010. Accessed June 13, 2017.

27. US Food and Drug Administration. Questions and answers on lead in foods. https://www.fda.gov/food/foodborneillnesscontaminants/metals/ucm557424.htm. Accessed August 17, 2017.

28. Consumer Product Safety Improvement Act of 2008. Pub L No. 110-314. https://www.congress.gov/110/plaws/publ314/PLAW-110publ314.pdf. Accessed August 24, 2017.

29. US Department of Housing and Urban Development. *Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing (2012 Edition).* Washington, DC: HUD; 2012. https://portal.hud.gov/hudportal/HUD?src=/program offices/healthy_homes/lbp/hudguidelines. Accessed August 24, 2017.

30. Centers for Disease Control and Prevention. Adult blood lead epidemiology and surveillance—United States, 2008-2009. *MMWR Morb Mortal Wkly Rep*. 2011;60:841–845. http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6025a2.htm. Accessed June 14, 2017.

31. California Department of Public Health. *Blood Lead Levels in California Workers, 2012–2014*. Sacramento, CA: CDPH; 2017. https://archive.cdph.ca.gov/programs/olppp/Documents/CABLLReport2012-14.pdf. Accessed August 18, 2017.

32. Centers for Disease Control and Prevention. National Biomonitoring Program, lead summary. https://www.cdc.gov/biomonitoring/Lead_BiomonitoringSummary.html. Accessed August 25, 2017.

33. Hu H, Shih R, Rothenberg S, Schwartz BS. The epidemiology of lead toxicity in adults: measuring dose and consideration of other methodologic issues. *Environ Health Perspect.* 2007;115(3):455–462. doi: 10.1289/ehp.9783.

34. Abadin H, Ashizawa A, Stevens YW, et al. *Toxicological Profile for Lead*. 2007. Washington, DC: US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry; 2007: 344–362. https://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=96&tid=22. Accessed August 25, 2017.

35. Tarragó O, Brown MJ. What are possible health effects from lead exposure? Agency for Toxic Substances and Disease Registry, case studies: lead toxicity. Published June 12, 2017. https://www.atsdr.cdc.gov/csem/csem.asp?csem=34&po=10. Accessed August 20, 2017.

36. Kosnett MJ, Wedeen RP, Rothenberg SJ, et al. Recommendations for medical management of adult lead exposure. *Environ Health Perspect*. 2007;115(3):463–471.

37. Martin CJ, Werntz CL 3rd, Ducatman AM. The interpretation of zinc protoporphyrin changes in lead intoxication: a case report and review of the literature. *Occup Med (Lond).* 2004;54(8):587–591. doi: 10.1093/occmed/kqh123.

38. Labbe RF, Vreman HJ, Stevenson DK. Zinc protoporphyrin: a metabolite with a mission. *Clin Chem.* 1999;45(12);2060–2072. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1849937/. Accessed June 13, 2017.

39. Rabinowitz MB. Toxicokinetics of bone lead. *Environ Health Perspect.* 1991;91:33–37.

40. Nie LH, Sanchez S, Newton K, Grodzins L, Cleveland RO, Weisskopf MG. In vivo quantification of lead in bone with a portable x-ray fluorescence system-methodology and feasibility. *Phys Med Biol.* 2011;56(3):N39–51. doi: 10.1088/0031-9155/56/3/n01.

41. Todd AC, Chettle DR. In vivo X-ray fluorescence of lead in bone: review and current issues. *Environ Health Perspect.* 1994;102(2):172–177.

42. Bradberry S, Sheehan T, Vale A. Use of oral dimercaptosuccinic acid (succimer) in adult patients with inorganic lead poisoning. *QJM.* 2009;102(10):721–732. doi: 10.1093/qjmed/hcp114.