Toxic Topics: Lead *by Michael Ichniowski, MD*

"Yes, there are two paths you can go by, but in the long run,

There's still time to change the road you're on."

-Robert Plant, Led Zeppelin

When guitarist Jimmy Page was assembling a new band in the late Sixties after the break-up of the British blues band, the Yardbirds, two members of The Who weighed in on the new band's prospects. Drummer Keith Moon stated they would "go down like a lead balloon"; Bassist John Entwistle went a step further and, alluding to the crash-and-burn demise of the Hindenburg, said it would be "more like a lead zeppelin." After their manager changed the spelling to "Led" to avoid the wrong pronunciation (how would you say lead guitarist for Lead Zeppelin?), the band, who actually used a picture of the Hindenburg disaster for their debut album, went on to turn Led into a great deal of gold and platinum. There is no indication that their *Stairway to Heaven* had any lead paint on its bannisters, or that anyone suffered any led poisoning from listening to their music.

The lines above from their classic song can apply to many environmental health issues: do we allow exposure to various toxic substances and treat the effects, or do we eliminate the exposure and prevent those toxic effects from occurring? Many substances, lead included, find their way into common usage before their toxicities are appreciated. Once the hazards are known, the arduous task of changing the road we're on and eliminating the use of the dangerous substance begins. The history of lead poisoning illustrates the time it can take to make such a change in course.

Historical Uses of Lead

Lead, in various forms and compounds, has been widely used for thousands of years. The ancient Egyptians used lead-containing kohl as an eye cosmetic. The ancient Romans (whose name for lead, plumbum, gives this element its symbol, Pb) used metallic lead for pipes and cooking utensils and used lead compounds in pottery glazes and as an additive to wines (perhaps contributing to the decline of their Empire). In more recent times, lead solder was commonly used as a sealant in food and drink containers. Lead carbonate, an opaque white pigment, was used to make paint that covered well, dried more quickly, gave a smooth finish and

resisted moisture. It was widely used in both housing and in industrial settings in the last century. Tetraethyl lead was used as a gasoline additive beginning in the 1920's to boost engine performance and fuel efficiency. This widespread use of lead paint and leaded fuel led to an enormous environmental lead burden. Though acute lead poisoning was being more widely recognized in the 1950's, it wasn't until 1977 that the used of lead-based paint in housing was banned. The phase-out of lead from gasoline did not occur until 1975 through 1986.

Routes and Sources of Exposure

Lead, in the many forms noted above, does not undergo degradation and persists unchanged in the environment. Oral ingestion of lead is by far the most common route of exposure. Deteriorating paint and renovation of older housing produces both large lead-containing particles and microscopic contamination of house dust. Decades of leaded gasoline use have contaminated soils, especially in urban and industrial areas and heavily-travelled roadsides. The hand-to-mouth habits of young children put them at great risk when exposed to lead in house dust and soils.

Lead ingestion may also occur from contaminated water (especially hot water) in lead pipes, and from metallic or alloyed lead in children's toys and jewelry, or such items painted with lead-based paints (usually imported from China). Lead glazing on pottery and ceramics (from Mexico and China), old pewter or soldered cookware and a variety of folk remedies are additional potential sources of lead ingestion.

Parental occupations and hobbies may also bring lead into the home. Lead smelting, automobile repair and battery recycling, industrial lead paint exposure, fishing weights, firearm ammunition and lead caming for stained glass are additional potential sources of lead exposures.

Airborne inhalation of lead is a less common route of exposure. This has been dramatically reduced since the elimination of lead from gasoline, but may also occur from lead-based industries (such emissions have also been strictly regulated), fumes from burning lead paint, and inhalation of fine particles from renovations and disturbance of lead-contaminated household dust.

Toxic Effects

Lead is a known neurotoxin whose effects appear to be irreversible, and the child's developing brain is especially susceptible to these toxic effects. These are believed to be due to interference with neurotransmission and disruption of cell migration during brain development. Symptoms of acute toxicity include headache with agitation or somnolence, accompanied by abdominal pain and constipation. This can progress to vomiting, stupor and convulsions and is a medical emergency. Before chelation therapy was available, lead encephalopathy (with blood lead levels $\geq$ 60 mcg/dL) resulted in death and permanent brain damage with seizures and intellectual disabilities in a large percentage of affected children. The elimination of lead from gasoline and paint has dramatically reduced the incidence of lead encephalopathy and symptomatic lead poisoning.

Other neurodevelopmental problems, including cognitive impairment, hyperactivity, inattention, school failure, and aggressive and delinquent behavior have been reported at lower blood lead levels, but there is no single finding or group of symptoms specific to lead toxicity. A decline in IQ scores with rising blood lead levels above 10 mcg/dL has been established in several studies, with recent evidence also suggesting a higher rate of decline at levels < 10 mcg/dL.

Lead exerts toxic effects on the kidneys, increasing the risk of renal dysfunction and hypertension later in life. Lead also interferes with hydroxylation and activation of Vitamin D, affecting bone metabolism and growth. Impaired heme synthesis is another effect of lead, leading to a microcytic hypochromic anemia.

Screening

A venous blood lead level is the preferred test to screen for significant lead exposure. Blood lead levels have been demonstrated to peak at age 2 years and then decline, even without intervention. As such, children at risk for lead exposure should be tested at age 1 year and 2 years. Federal policy requires these blood lead levels in all children receiving Medicaid insurance (and for 3-6 year olds not previously tested). Maryland law requires testing for children living in zip codes considered to be high-risk for lead exposure, based on the percentage of older housing. Additionally, all children should be screened for lead exposure, asking if they live or visit homes built before 1978, especially if peeling paint is present or

interior remodeling is being done; if there are any siblings or playmates with elevated lead levels; if anyone in the household has a work or hobby-related exposure to lead; or if the child lives near a lead-based industry. International adoptees, immigrants and refugees should also be screened upon entry to this country.

In May, 2012, the CDC revised their guidelines based on recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention. All children with a blood lead level of $\geq$ 5 mcg/dL should be monitored for changes in their lead levels until investigation for the source of exposure and mitigation have been completed. The previous level of concern was 10 mcg/dL.

Management

There is no form of treatment as effective as primary prevention of exposure to lead. Identification and elimination of sources of environmental exposure to lead will result in fewer and fewer children with significant lead levels and their associated toxicities.

In older homes where lead paint is likely to be present, keeping children from direct contact with painted surfaces, especially if flaking or peeling, is extremely important. Keeping children out of these homes during renovations is also crucial. Frequent washing of children's hands and toys will reduce the risk of hand-to-mouth exposure to lead in house dust, and regular wet-mopping and wet-wiping of floors and painted windows and door frames will help reduce the amount of lead-contaminated dust. Avoiding outdoor play on bare soil, particularly in urban/industrial neighborhoods, will decrease the likelihood of ingestion of lead-contaminated soil.

The following will summarize the recommendations for management of elevated lead levels, based on the blood lead level. At all levels, assessment for the source of lead is necessary, and steps should be taken to eliminate this source. Dietary evaluation to assure adequate intake of iron, zinc, calcium, protein and vitamin C should also be performed. A hemoglobin and/or hematocrit should be checked to identify a coexisting iron deficiency anemia, and iron supplementation should be given to correct a deficiency, if found.

Lead Level (mcg/dL)	Recommendations
5-9	Identify source if possible and eliminate exposure. Monitor blood lead level (BLL) within 3 months
10-14	Confirm BLL within 1 month, monitor q 3 months Notify local health dept.—a BLL of 10 triggers regulatory action for rental housing in Maryland
15-19	Confirm BLL within 1 month, repeat within 2 months Refer to Lead Poisoning Prevention Program (LPPP)
20-44	Complete medical/developmental/environmental eval. Confirm BLL within 1 week; consider abdominal X-ray if ingestion suspected. Refer to health dept. and LPPP for case management
45-69	Confirm BLL within 24-48 hours. Full evaluation as above. Remove child to lead-free location. Begin oral chelation in consultation with experienced clinician.
70 and above	Hospitalize immediately, confirm BLL and begin IM chelation with dimercaprol and EDTA in consultation with experienced clinician

Oral chelation uses succimer (dimercaptosuccinic acid or DMSA), which enhances urinary excretion of lead. This medication is administered three times a day for 5 days, then twice a day for an additional 14 days. Parenteral chelation begins with IM dimercaprol (BAL or British Anti-Lewisite), six divided doses per day for at least 3 days. Calcium disodium EDTA (CaNa2 edetate) is started 4 hours after the

first BAL dose, and continued for 5 days. Chelation is not recommended for BLL < 45 due to toxicity and lack of documented neurocognitive benefit. Lead levels should be checked 1 to 3 weeks after chelation for a rebound, as lead stored in bone mobilizes into the circulation.

Resources for guidance in managing children with lead poisoning include the Mt. Washington Children's Hospital Lead Clinic (410-367-2222) and the Mid-Atlantic Center for Children's Health and the Environment (MACCHE) (1-866-622-2431 or online at www.childrensnational.org/macche).

Great strides have been made in reducing exposure to lead, but environmental sources still exist, particularly in older housing units. The persistence needed to continue to reduce children's exposure to lead and work to eliminate this entirely preventable disease will require each of us "to be a rock, and not to roll."

References

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Website

CDC Lead Poisoning and Prevention Program: http://www.cdc.gov/nceh/lead/