Background

Lead, in both its organic and inorganic forms, can be toxic to humans. While toxicity from organic lead substances causes signs and symptoms virtually indistinguishable from those caused by inorganic lead, the entrance of the organic lead substances into the body and their rate of metabolism and excretion differ markedly from that of inorganic lead.

Lead is a ubiquitous substance found in low levels in food, water, and the ambient air; therefore, all humans have some lead in their bodies. In certain occupations (listed in Table 1), a worker can be exposed to much higher concentrations of inorganic lead primarily through the inhalation of lead fumes or lead-containing dust particles.

Table 1. Occupations at Risk for Lead Exposure

<table>
<thead>
<tr>
<th>Plumbers, Pipefitters</th>
<th>Policemen</th>
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<tbody>
<tr>
<td>Lead Miners</td>
<td>Steel Welders or Cutters</td>
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<tr>
<td>Auto Repairers</td>
<td>Construction Workers</td>
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<tr>
<td>Glass Manufacturers</td>
<td>Rubber Products Manufacturers</td>
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<tr>
<td>Shipbuilders</td>
<td>Gas Station Attendants</td>
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<tr>
<td>Printers</td>
<td>Battery Manufacturers</td>
</tr>
<tr>
<td>Plastics Manufacturers</td>
<td>Bridge Reconstruction Workers</td>
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<tr>
<td>Lead Smelters and Refiners</td>
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</tbody>
</table>

Inhaled lead deposited in the lower respiratory tract is completely absorbed. Inorganic lead can also enter the body through ingestion. The rate of absorption of ingested lead from the GI tract is dependent on the age and nutritional state of the individual. A fasting state and iron or calcium deficiency can increase the absorption rate of lead up to 50 percent of the amount ingested. But typically the amount of lead
absorbed from the GI tract of adults is 10 to 15 percent of the ingested quantity. Ingestion of lead is a much less significant route of entry in an occupational setting, especially if workers do not smoke, drink, or eat in lead-contaminated work areas. Once in the body, inorganic lead does not undergo biologic transformation.

In contrast, organic lead, found primarily in leaded gasolines as tetraethyl lead, can enter the body through either inhalation or absorption through the skin. Once in the body, organic lead compounds are metabolized in the liver. In 1976 and 1984, federal regulation drastically reduced the amount of lead in gasoline and, today, organic lead in gasoline is not a great environmental concern in the United States. Therefore, the primary mode of lead toxicity in an occupational setting is through the inhalation of inorganic lead dusts and fumes.

Once in the body, lead is distributed among three compartments:

1. **Blood.** Ninety-nine percent is associated with erythrocytes; 1 percent is in the plasma where it is available to transport to other tissues.
2. **Soft tissues;** kidneys, bone marrow, liver, and brain.
3. **Mineralizing tissues;** bones and teeth.

The excretion of lead is extremely slow, with the two primary routes of excretion being renal and hepatic. Other possible but less significant routes for lead excretion include sweat, milk, hair, nails, desquamating epithelia, and teeth. In single exposure studies, the half-life of lead in the blood is approximately 25 days; in soft tissues approximately 40 days; and in the non-labile portion of bone can be greater than 25 years.

For lead poisoning to develop, major acute exposures to lead need not occur. The body accumulates this metal over a lifetime and releases it slowly, so that even small doses over time can cause lead poisoning. It is the total body burden of lead that is related to the risk of adverse effects.

Lead toxicity in adults can cause:

1. Hematologic effects
2. Neurologic effects
3. Endocrine effects
4. Renal effects
5. Reproductive and developmental effects.

A summary of the physiologic toxic effects of lead in adults and children is summarized in Figure 1.

**Figure 1. Effects of Inorganic Lead on Children and Adults -- Lowest Observable Adverse Effect Levels**

<table>
<thead>
<tr>
<th>Children</th>
<th>Lead Concentration in Blood (μg Pb/dL)</th>
<th>Adults</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Death</td>
<td></td>
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<td></td>
<td>Encephalopathy</td>
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<td></td>
<td>Frank Anemia</td>
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<td></td>
<td>Decreased Longevity</td>
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<td></td>
<td>Colic</td>
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<td></td>
<td>Hemoglobin Synthesis</td>
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<td></td>
<td>Peripheral Neuropathies</td>
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<tr>
<td></td>
<td>Infertility (Men)</td>
<td></td>
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<tr>
<td></td>
<td>Nephropathy</td>
<td></td>
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<tr>
<td></td>
<td>Systolic Blood Pressure (Men)</td>
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<tr>
<td></td>
<td>Hearing Acuity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Erythrocyte Protoporphyrin (Men)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Erythrocyte Protoporphyrin (Women)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vitamin D Metabolism (??)</td>
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<tr>
<td></td>
<td>Developmental Toxicity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypertension (??)</td>
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<tr>
<td></td>
<td>Transplacental Transfer</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Increased function</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Decreased function</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from: ATSDR, Toxicology Profile for Lead (1989)
As displayed in the ATSDR’s "Case Studies in Environmental Medicine: Lead Toxicity," June, 1990 (p.7)
Hematologic Effects: Anemia is a prominent finding in lead poisoning caused by the combined effect of (1) inhibition of hemoglobin synthesis, and (2) the shortened life span of circulating erythrocytes. Lead inhibits several enzymes that are critical to the synthesis of hemoglobin. One of the substrates of hemoglobin synthesis is erythrocyte protoporphyrin which, in its "free" state, actually exists as zinc protoporphyrin (ZPP) and builds up to measurable levels in chronic, relatively high levels of lead poisoning. Lead in high levels has been associated with hemolytic anemia, but in lower levels, lead seems to cause anemia by shortening the life span of circulating erythrocytes by making the cell walls of red cells more fragile. The anemia in adults is usually mild to moderate. Early in the course it is often microcytic and hypochromic, but with lead poisoning, the red cells are often normocytic and normochromic. Basophilic stippling of the red cells on a peripheral smear is often seen in chronic lead poisoning.

Neurologic Effects: The most sensitive target of lead poisoning is the nervous system. In adults, the neurologic effects include subtle behavioral changes, fatigue, and impaired concentration. Peripheral nervous system damage, primarily motor, often begins with aching and tenderness of joints and muscles (especially forearm extensor muscles), increased fatiguability of the muscles, and development of a fine tremor which can progress to painless paresis of one or more muscle groups (the classic "wrist drop" of lead palsy).

Endocrine Effects: Lead interferes with a hormonal form of Vitamin D, which can impair cell maturation and skeletal growth. Lead can impair thyroid function by preventing the uptake of iodine, and can have a direct effect to decrease the pituitary and adrenal gland functions.

Renal Effects: A direct effect on the kidney from long-term lead exposure is nephropathy, characterized by a progressive, irreversible impairment of renal function often accompanied by hypertension. The hypertension, if untreated, can then cause secondary cardiovascular effects including left ventricle hypertrophy, myocardial infarction, and cerebrovascular accidents. Lead also interferes with the normal excretion of uric acid which can cause hyperuricemia and gout. Lead has also been implicated as a potential human renal carcinogen, having caused kidney tumors in rats.
Reproductive and Developmental Effects: There has been a long history of spontaneous abortions and stillbirths among women working with lead as early as the turn of the century. Lead can readily cross the placenta, which not only affects the viability of the fetus, but its development as well. Reduced birth weights and premature births have been reported. Lead is a known animal teratogen, and is believed to be able to cause at least minor malformations in human fetuses. In men, data is also available which suggests that chronic exposure to lead can reduce sperm counts and motility.

Purpose

The purpose of the Lead Surveillance Program at the NASA Lewis Research Center shall be to:

1. Identify any Lewis employee who is exposed to lead above the action level or at the physician’s discretion.

2. Educate that employee about the nature of lead, the proper use of respiratory protection and protective clothing, and appropriate sanitation practices to be used when exposed to lead-containing environments in the workplace.

3. Monitor those employees by obtaining an extensive past medical and occupational exposure history, and then performing period physical examinations with appropriate laboratory work to detect early signs and symptoms of lead poisoning. When lead exposure is anticipated, a baseline physical examination with the appropriate laboratory work shall be obtained.

Medical Surveillance

The specifics of the Medical Surveillance Program for Lead Exposure at the NASA Lewis Research Center will fulfill the criteria required by OSHA, as defined under 29 Code of Federal Regulations (CFR) 1910.1025, and will incorporate recommendations from NIOSH.
Since the primary occupational exposure to lead is through inhalation of inorganic lead fumes and dust particles, worker exposure limits are defined in terms of the concentration of lead in the ambient air and require air monitoring for measurement. The permissible exposure limit (PEL) for lead is 50 µg/m³ as an 8-hour time-weighted average (TWA). The action level (AL) for lead is 30 µg/m³ as an 8-hour TWA. Therefore, any worker who is exposed to the AL for lead for more than 30 days per year shall be included in the NASA Lewis Research Center Lead Medical Surveillance Program.

The maximum permissible limit for lead is calculated as:

Maximum Permissible Limit (in µg/m³) = 400 hours worked in the day.

If an employee is exposed to the maximum permissible exposure limit for lead in any given work day, then he/she shall be entered in the Lead Medical Surveillance Program.

Requirements for specific types of respiratory protection, protective clothing, engineering control, safety controls, and hygiene facilities and practices will be determined and enforced by the Office of Industrial Hygiene and the Safety Office.

Current OSHA recommendations seek to limit a lead-exposed employee's blood lead level to less than 40 µg/dl whole blood. However, as of the autumn of 1991, NIOSH now recommends limiting employee exposures to lead that result in blood lead levels less than 25 µg/dl whole blood. Given that the toxicity of lead is dependent on the total body burden of lead and that in chronic low level exposures there can be significant toxicity to lead even with normal blood lead levels, the NASA Lewis Research Center Medical Surveillance Program for Lead will utilize the NIOSH recommendations of limiting employee blood levels to less than 25 µg/dl.

NASA employees identified by the Office of Industrial Hygiene as having either the potential to being exposed to lead at or above the AL or having been exposed at or above the AL to lead will be included in the Lead Medical Surveillance Program. Medical surveillance baseline examinations will include:

1. **Medical and work histories**, with special attention to:
   - Previous occupational or recreational exposure
   - Occupational and recreational history of all home occupants
- Family history, including use of unusual medications
- Use of imported or glazed ceramics
- Use of leaded crystal for storing drinking beverages (especially alcoholic)
- Drinking water source and type of pipe
- Nutritional status
- Proximity to industrial facilities and hazardous waste sites
- Smoking history
- Personal hygiene and other habits
- Past or current GI problems
- Past or current reproductive problems (including pregnancy status)
- Past or current cardiovascular problems (especially history of hypertension)
- Past or current neurologic problems
- Past or current hematological or renal problems
- Past or current history of gout.

2. **A complete physical examination**, with special attention to:
   - Vital signs, especially blood pressure
   - Teeth and gums (purplish line on gums called a "lead line")
   - Renal System
   - Neurologic system (behavioral changes, fatigue, tremors, peripheral motor neuropathy, seizures)
   - Cardiovascular system (hypertension, LVH, remote MI, neurologic deficits suggestive of CVA)
   - Hematologic system (pallor, signs of anemia including tachycardia, CHF, etc.)
   - Gastrointestinal system (abdominal pain, nausea)
   - Pulmonary system if respiratory clearance is needed.

3. **Laboratory examination**, to include:
   - Chest X-ray (as a baseline test if not already available)
   - Spirometry (if respiratory clearance is indicated)
   - CBC with differential (anemia, low red cell indices, basophilic stippling on peripheral smear)
   - Blood lead level (less than 25 µg/dl. If \( > = 60 \) µg/dl, the employee shall be removed from any occupational exposure)
   - ZPP (Zinc Protoporphyrin) level (less than 35 µg/dl)
- Serum chemistries (attention to BUN, creatinine, uric acid, T4, electrolytes, and serum calcium/phosphorus)
- Urinalysis with microscopy
- EKG if not done within past 12 months (attention to changes associated with chronic, uncontrolled hypertension).

The periodic requirements for lead medical examinations shall include:

1. **Follow-up blood lead and ZPP tests:**
   - **Semi-annual** (every 6 months) for workers exposed to greater than the AL but whose blood levels have been < 25 µg/dl.
   - **Bimonthly** (every 2 months) for workers whose last blood level was >= 25 µg/dl. Continue until two consecutive tests are < 25 µg/dl.
   - **Monthly** for workers who have been removed from exposure to lead due to a blood level >= 60 µg/dl. After the first excess, repeat the blood lead and ZPP within two weeks.
   - **Every two weeks** for workers exposed to the maximum permissible exposure limit, as defined in the "Background" section, regardless of the worker’s past blood lead level.

2. **Complete lead examinations:**
   - **Annually** for workers exposed to the AL for lead (30 µg/m³, 8-hour TWA for more than 30 days/year).
   - **As soon as possible** after signs or symptoms of lead toxicity develop in anyone at any time.
   - **Annually** for workers whose blood lead was >= 25 µg/dl anytime during the previous 12 months.
   - **At the completion of any major lead abatement projects.**
   - **Upon retirement or termination of employment** of any worker who has been involved in an ongoing lead medical surveillance program.

References


