EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Volume: 8
Issue: 10
355-366
Year: 2021

A REVIEW ARTICLE ON ENVIRONMENTAL LEAD EXPOSURE: A PUBLIC HEALTH PROBLEM OF GLOBAL DIMENSIONS & RECENT ADVANCES ON LEAD POISONING TREATMENT

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Article Received on 14/08/2021 **Article Revised on 03/09/2021** **Article Accepted on 24/09/2021**

ABSTRACT

Lead is the most abundant of the heavy metals in the Earths crust. It has been used since prehistoric times, and has become widely distributed and mobilized in the environment. Exposure to and uptake of this non-essential element have consequently increased. Both occupational and environmental exposures to lead remain a serious problem in many developing and industrializing countries, as well as in some developed countries. In most developed countries, however, introduction of lead into the human environment has decreased in recent years, largely due to public health campaigns and a decline in its commercial usage, particularly in petrol. Acute lead poisoning has become rare in such countries, but chronic exposure to low levels of the metal is still a public health issue, especially among some minorities and socioeconomically disadvantaged groups. In developing countries, awareness of the public health impact of exposure to lead is growing but relatively few of these countries have introduced policies and regulations for significantly combating the problem.

KEYWORDS: lead; environmental exposure; biological fate, physiological effects, clinical assements,occupational diseases; clinical modalities,education,adverse effects; lead toxicity.

INTRODUCTION

LEAD

Lead is a soft, blue-gray metal, usually found as lead compounds, combined with other elements. Much of its presence in the environment stems from.

- Its historic use in paint and gasoline in the United States,
- Ongoing or historic mining/smelting,
- Commercial operations, and
- Lead contaminated consumer products.

IT'S PROPERTIES

->Lead is a

- Very soft,
- Dense, and
- Ductile (moldable) metal.

->Lead is very stable and resistant to corrosion, although acidic water may leach lead out of Pipes, Fittings, and Solder (metal joints).

Lead Found

- **Lead-based Paint in Homes and Buildings**

Lead-contaminated household dust is considered the major high-dose source of lead for children in the United States today.[2] Most literature refers to "Lead-based paint," or "Lead Paint" which used to contain higher amounts of lead.

In addition to degradation of interior paint, lead may be tracked into homes in significant quantities from exterior soil contaminated by historical use of lead in.

- Paint (exterior sources),
- Gasoline, or
- Industries (old smelters).

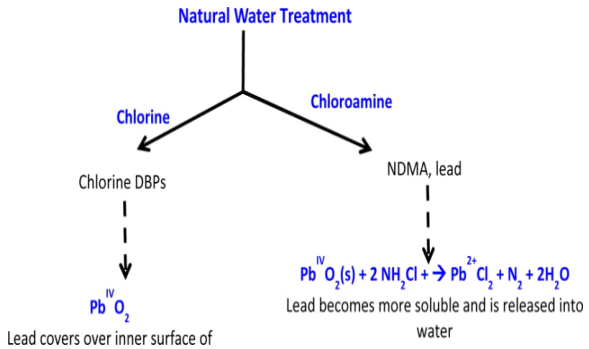
A secondary source of lead exposure for workers' families can take place if workers bring home lead-contaminated dust on their.

- Skin, Hair, Clothes, Shoes, orTools.

B) Lead in Drinking Water

Lead occurs in drinking water through leaching from lead-containing pipes, faucets, and solder frequently found in the plumbing of older buildings.

- "**Lead service lines**" (the water service pipes *that connect the water main in the street or "public water main"* to the household plumbing system can be made of lead) in the water supply systems of older neighborhoods can leach lead.
- If "lead service lines" are replaced, but the household plumbing remains as galvanized iron pipes, there can still be ongoing lead exposure in drinking water.



- *Corrosion* build up on the *inside of galvanized pipes can create the potential for lead to accumulate over time*. Acidic water can contribute to the leaching of lead from pipes into the drinking water supply.
- *Having a drinking water filtration system (filtered tap water/filtration pitchers)* certified by an independent testing organization such as the National Sanitation Foundation (NSF) to remove lead *can be a safe drinking water option* when instructions on how to use, maintain, and/or replace filters are followed.

- **Boiling water will not eliminate lead.**

C) Foods and Beverages Contaminated with Lead

Even when lead is not intentionally used in a product, it may contaminate items such as food, water, or alcoholic drinks.[17]

Lead may contaminate food during

- *Production,*
- *Processing,*
- *Packaging, and*
- *Storage.*

D) Commercial Products

While lead is prohibited from many products in the United States, imported or pre-regulation products may still pose a risk. Lead is still used in commercial products[23,24,25,52] and may be found in products such as

- Automotive batteries, Computers,
- Bridge paint, Curtain weights,
- Jewelry, Some ceramic glazes,
- Toys (especially antique or imported), Vinyl lunch boxes

E) Environmental and Industrial Sources

Lead is ubiquitous in the environment because of widespread human use. Environmental background levels vary depending on historic and ongoing uses in the area.

- Abandoned industrial lead sites, such as old mines or lead smelters, may continue to pose a potential public health hazard.
- Industries such as mining and lead smelting contribute to high levels of lead in the environment around such facilities.
- People living near hazardous waste sites, incinerators, landfills may be exposed to lead and chemicals that contain lead by breathing air, drinking water, eating foods, or swallowing dust or dirt that contains lead.

F) Occupational Exposures

Workers in the lead smelting, refining, and manufacturing industries experience the highest and most prolonged occupational exposures to lead.[7] Increased risk for occupational lead exposure occurs among workers in

- Battery manufacturing, Automobile brake repair, & Other manufacturing industries
- Companies that work with lead solder,
- Bridge maintenance and repair,

- Construction, especially renovation/rehabilitation,
- Pottery/ceramics companies and studios,
- Rubber products and plastics industries, andSteel welding/cutting operations.

Table 2. **Operations that may present lead hazards for workers** (Source ref. *37*).

Primary and secondary lead smelting	Lead mining
Welding and cutting of lead-painted metal constructions	Plumbing
Welding of galvanized or zinc silicate coated sheets	Cable making
Other welding	
Shipbreaking	Wire patenting
Nonferrous founding	Lead casting
Storage battery manufacture: pasting, assembling, welding of battery connectors	Type founding in printing shops
Production of lead paints	Sterotype setting
Spray painting	Assembling of cars
Mixing (by hand) of lead stabilizers into polyvinyl chloride	Shot making
Mixing (by hand) of crystal glass mass	
Sanding or scraping of lead paint	Lead glass blowing
Burning of lead in enamelling workshops	Pottery/glass making
Repair of automobile radiators	

Routes of Exposure to Lead

Today almost everyone is exposed to environmental lead. Exposure to lead and lead chemicals can occur through ***inhalation, ingestion, dermal absorption, absorption from retained or embedded leaded foreign body, and trans-placental (endogenous) routes.***

- **Ingestion**

Lead exposure in the general population (including children) occurs primarily through ingestion, making it the route that most commonly leads to elevated BLLs.

- Lead paint is the major source of higher lead level exposures in children.[7,1]
- As higher lead content *paint , Deteriorates, Peels, Chips*[Is removed (e.g., during renovation)] Or *crumbles due to friction* (e.g., in windowsills, steps, and doors), *house dust and surrounding soil may become contaminated*.[64,1]

ii. Ingestion of contaminated

- Food, **&** Water

iii. When fine particulate lead is inhaled, it can be absorbed directly through the lungs or could also be carried by the mucociliary tree to the throat where it can be swallowed and absorbed via the GI system.

B) Inhalation

Inhalation is the second major pathway of exposure for the general. The amount absorbed from the respiratory system depends on *particle size, respiratory volume, amount of deposition, and the mucociliary clearance of the inhaled lead.*

C) Dermal

Dermal exposure plays a role for exposure to organic lead among workers.

- Organic lead may be absorbed directly through the skin.
- Organic lead (tetraethyl lead) is more likely to be absorbed through the skin than inorganic lead.

D) Endogenous Exposure

- Once absorbed into the body, lead may be stored for long periods in mineralizing tissue (e.g., teeth and bones).
- The stored lead may be released again into the bloodstream, especially in times of calcium stress (e.g., pregnancy, lactation, osteoporosis) or calcium deficiency.

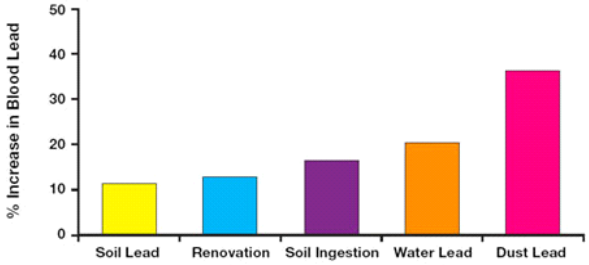
Risk of Lead Exposure

- Both children and adults are susceptible to health effects from lead exposure, although the typical exposure pathways and effects can be somewhat different.
- Developing fetuses are also at risk for adverse health outcomes (less than 1% of the mothers have levels greater than or equal to 5 micrograms per deciliter, or µg/dL), as levels that present risk to the fetus may not present risk to the mother.
- Smokers and their relatives (if exposed to secondhand smoke) are at a high risk for exposure due to the lead in tobacco smoke.

- **CHILDREN**

Because of their behavior and physiology, children are more affected by exposure to lead than adults.

- Children absorb more ingested lead than do adults.
- Children generally ingest lead-contaminated soil and house dust at higher rates than adults because of mouthing and hand-to-mouth behaviors.
- Being shorter than adults, children are more likely to breathe lead-contaminated dust and soil as well as lead-containing fumes close to the ground.



Pregnant Women, Lactating Women and Developing Fetuses

The mother's BLL is an important indication of risk to the fetus and neurological problems in newborns. Pregnant women with elevated BLLs may have an increased chance of

- Preterm labor,
- Miscarriage,
- Neurological effects and intrauterine growth restriction (IUGR),
- Spontaneous abortion or stillbirth, and/or
- Low birth weight.

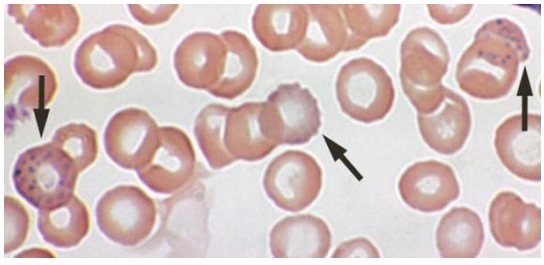
Biological Fate of Lead in the Body

The absorption and biological fate of lead once it enters the human body depends on a variety of factors.

- Absorbed lead that is not excreted is exchanged primarily among three compartments:
- Blood,
- Mineralizing tissues (bones and teeth), and
- Soft tissue (liver, kidneys, lungs, brain, spleen, muscles, and heart).

A) Lead in the Blood

- Although the blood generally carries only a small fraction of total lead body burden, it does serve as the initial receptacle of absorbed lead and distributes lead throughout the body, making it available to other tissues (or for excretion).
- The half-life of lead in adult human blood has been estimated as 28 days[43, 7] to 36 days.[62, 7]



- *Approximately 99% of the lead in blood is associated with red blood cells; the remaining 1% resides in blood plasma.*[7,31,32,36]
- The higher the lead concentration in the blood, the higher the percentage partitioned to plasma. This relationship is curvilinear - as blood lead levels (BLLs) increase, the high-end plasma level increases more.

B) Lead in Mineralizing Tissues (Bones and Teeth)

- Lead in mineralizing tissues is not uniformly distributed. It tends to accumulate in bone regions undergoing the most active calcification at the time of exposure.
- Known calcification rates of bones in childhood and adulthood suggest that lead accumulation will occur predominately in trabecular bone during childhood, and in both cortical and trabecular bone in adulthood.[7,11]

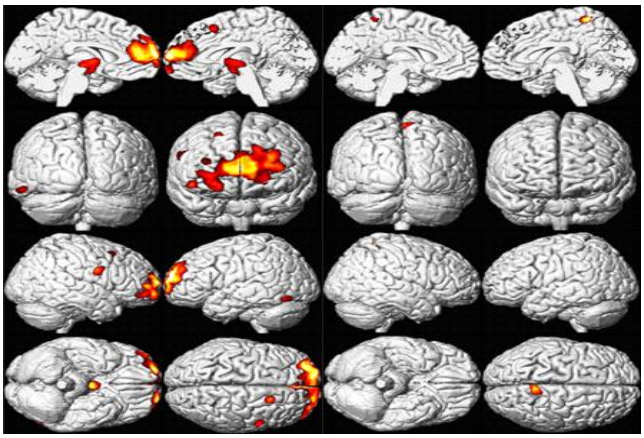
PHYSIOLOGICAL EFFECTS

A) Neurological Effects

Lead exposure has been linked with various types of brain damage. These include.

- Problems with thinking (cognition);
- Difficulties with organizing actions, decisions, and behaviors (executive functions);
- Abnormal social behavior (including aggression); and

Lead causes activation of protein kinase C (PKC) and binds to PKC more avidly than calcium (its physiologic activator). This creates problems with neurotransmitter release.



B) Renal Effects

- At ‹10 µg/dL there is increased blood pressure and increased risk of hypertension in adults.[58] However, continued or repetitive exposures can cause toxic stress on the kidney that, if unrelieved, may develop into chronic and often irreversible lead nephropathy (e.g., chronic interstitial nephritis).
- Lead nephrotoxicity is characterized by
- Proximal tubular nephropathy,
- Glomerular sclerosis, and
- Interstitial fibrosis.[41,53,78]

3. Most documented renal effects for occupational workers have been observed in acute high-dose exposures and high-to-moderate chronic exposures (BLL ›60 µg/dL).

C) Hematological Effects

- Lead inhibits the body's ability to make hemoglobin by interfering with several enzymatic steps in the heme synthesis pathway.
- Specifically, lead *decreases heme biosynthesis* by *inhibiting d-aminolevulinic acid dehydratase (ALAD) and ferrochelatase (FECH) activity*.A decrease in the activity of this enzyme results in an increase of the substrate erythrocyte protoporphyrin (EP) in the red blood cells (also found in the form of ZPP-bound to zinc rather than to iron).

D) Endocrine Effects

Studies of children with high lead exposure have found that a strong inverse correlation exists between BLLs and vitamin D levels.

- Lead impedes vitamin D conversion into its hormonal form, 1, 25-dihydroxyvitamin D, which is largely responsible for the maintenance of extra-and intra-cellular calcium homeostasis.
- Diminished 1, 25-dihydroxyvitamin D, in turn, may impair cell growth, maturation, and tooth and bone development.

E) Gastrointestinal Effects

In severe cases of lead poisoning, children or adults may *present with severe cramping abdominal pain (colic-like pain), which may be mistaken for an acute abdomen or appendicitis.* Lead colic is a symptom of chronic lead poisoning and is associated with obstinate constipation.

F) Cardiovascular Effects

A few population studies have shown a possible connection between lead exposure and other cardiovascular disorders including

- Ischemic coronary heart disease,
- Cerebrovascular accidents, and
- Peripheral vascular disease.[67]

Hypertension is a complex condition with many different causes and risk factors, including family history, age, weight, diet, and exercise habits.

G) Reproductive Effects

I) Male Reproductive Effects

Recent reproductive function studies in humans suggest that current occupational exposures may decrease sperm count totals and increase abnormal sperm frequencies.[58]
In men, there is sufficient evidence that BLLs ≥ 15 µg/dL are associated with adverse effects on sperm or semen.

II) Fertility

There is sufficient evidence *that BLLs ‹10 µg/dL show adverse health effects on reproduction in adult women, and BLLs ≥ 20 µ/dL are associated with delayed conception time.*[58,59]

III) Pregnancy Outcomes

Prenatal lead exposure has known influences on maternal health and infant birth and neurodevelopmental outcomes.

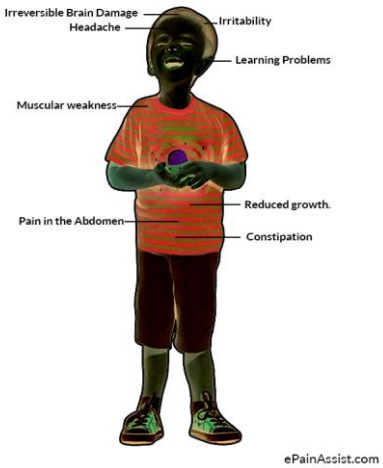
H) Other Potential Effects

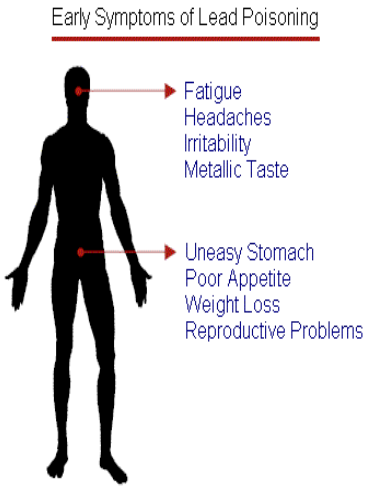
- *Lower Bone Mineral Density (BMD)*
- *Dental Health*
- *Cancer*

Signs and Symptoms

Typically, a sign is something the health care provider "sees" or "finds" during a physical exam. A symptom is experienced and reported by the patient.
Many patients who suffer from lead poisoning may be asymptomatic, hence the importance of exposure assessment and screening.

- **Lowest Exposure Dose Signs and Symptoms**: (patient may appear asymptomatic)
- Decreased learning and memory / Lowered IQ
- Decreased verbal ability & Impaired speech and hearing functions
- Low Exposure Dose Signs and Symptoms
- Irritability & Occasional abdominal discomfort
- Lethargy & Mild fatigue
- Myalgia or paresthesia





II Moderate Exposure Dose Signs and Symptoms.

- Constipation & Diffuse abdominal pain

- Difficulty concentrating/Muscular exhaustibility & General fatigue
- Headache , Vomiting & Weight loss

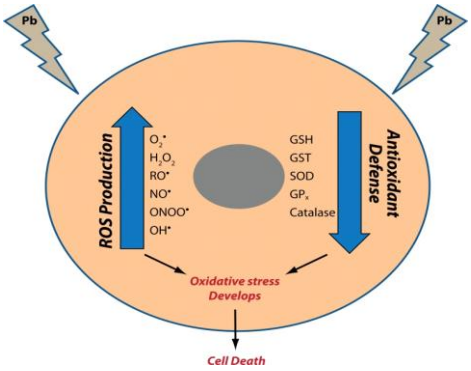
III High Exposure Dose Signs and Symptoms

- Colic (intermittent, severe abdominal cramps)
- Encephalopathy-may abruptly lead to seizure, change in consciousness, coma, and death
- Paresis or paralysis

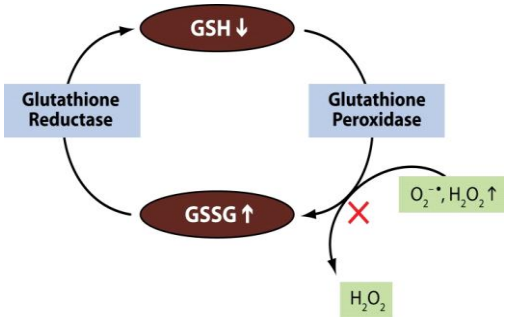
Mechanism of toxicity

Lead is probably the most extensively studied heavy metal. Studies carried out have reported the presence of various cellular, intracellular and molecular mechanisms.

- **Oxidative stress**
- *Oxidative stress represents an imbalance between the production of free radicals and the biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage*.[98]
- It has been reported as a major mechanism of lead induced toxicity.
- Under the influence of lead, onset of oxidative stress occurs on account of two different pathways operative simultaneously;
- first comes the generation of ROS, like hydroperoxides (HO2), singlet oxygen and hydrogen peroxide (H2O2), and
- second, the antioxidant reserves become depleted[98]



- The most important antioxidant found in cells is glutathione (GSH). It is a tripeptide having sulfhydryl groups and is found in mammalian tissues in millimolar concentrations.
- It is an important antioxidant for quenching free radicals.[97]
- *Glutathione exists in both reduced (GSH) and oxidized form (GSSG). The reduced state of glutathione donates reducing equivalents (H+ + e—) from its thiol groups present in cysteine residues to ROS and makes them stable. After donating the electron, it readily combines with another molecule of glutathione and forms glutathione disulfide (GSSG) in the presence of the enzyme glutathione peroxidase (GP X). GSH can be regenerated from GSSG by the enzyme glutathione reductase (GR)* .[in fig]



B) Ionic mechanism of lead toxicity

Ionic mechanism of action for lead mainly arises due to its ability to substitute other bivalent cations like Ca2+, Mg2+, Fe2+ and monovalent cations like Na+ (though bivalent cations are more readily substituted)

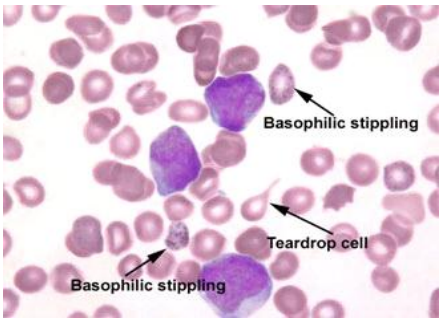
- [96]The ionic mechanism contributes principally to neurological deficits, as lead, after *replacing calcium ions, becomes competent to cross the blood-brain barrier (BBB) at* an appreciable rate.
- After crossing the BBB, lead accumulates in astroglial cells (containing lead binding proteins). Toxic effects of lead are more pronounced in the developing nervous system comprising immature astroglial cells that lack lead binding proteins.
- Lead easily *damages the immature astroglial cells* and *obstructs the formation of myelin sheath*, both factors involved in the development of BBB.

Imaging and Other Clinical Modalities

There are different clinical modalities available to further evaluate patients with elevated BLLs.

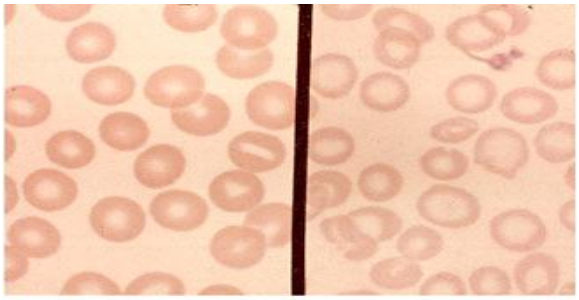
- Complete blood count (CBC) may be useful for patients with extensive lead exposure.
- In lead-exposed patients, the hematocrit and hemoglobin values may be slightly to moderately low in the CBC, and the peripheral smear may be either *normochromic and normocytic or hypochromic and microcytic.*

b. There may be basophilic stippling in patients who have been significantly poisoned for a prolonged period.



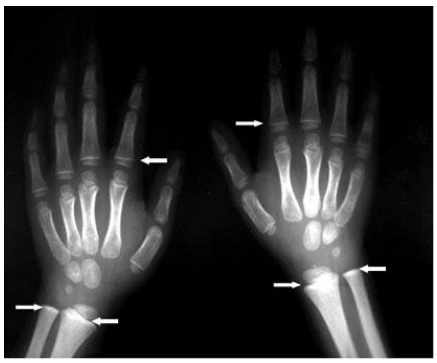
Basophilic stippling - Venous blood smear with basophilic stippling.

- A *hypochromic, microcytic anemia should be appropriately differentiated from* other causes, especially *iron-deficiency anemia by the use of testing for iron, iron binding capacity, and ferritin.*

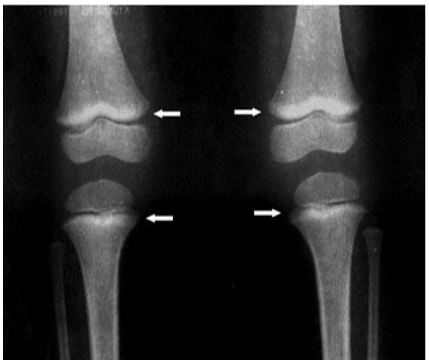


Microcytic hypochromic anemia can be associated with lead poisoning (Public domain)

c. Long bone radiographs can show "lead lines". These are lines of increased density on the metaphysis (growth plate) of the bone, showing radiological growth retardation. (Long bone radiographs are not recommended for diagnosing lead exposure[20], however, they are useful to determine growth retardation.)



Long Bone Radiograph of Hands - "lead lines"



Long Bone Radiograph of Knees - "lead lines"

d. Second tier tests (such as neurobehavioral/ psychological evaluation for children with indicative findings on exam)should be considered, as appropriate.

PREVENTION

A) ***Primary prevention:*** Primary prevention is defined as limiting the spread of illness to previously unaffected patients or populations. Preventing lead poisoning before its occurrence is crucial because lead-induced neurocognitive deficits are irreversible, and there are concerns about lead remobilization and adverse drug reactions. Even low concentrations of lead can cause permanent damage to developing the brain in children because of their immature blood-brain barrier[27][75] Since only 1-2% of the total amount of stored lead in the body is removed after a typical course of chelating therapy[49] source identification, and primary prevention play a pivotal role in the fight against lead poisoning.

B) Secondary prevention

- ***Pediatric screening***: Secondary prevention focuses on identifying asymptomatic people, particularly children with high BLC[69][14]According to recent studies, no threshold for the harmful effects of lead could be defined because childrens physical and mental development can be affected even at BLCs less than 10 µg/dL[75]At this concentration, detection and repair of lead hazards are not mandatory.[14]
- ***Occupational screening*** Workers who handle materials with a significant lead content are expected to have lead exposure through inhalation or by ingestion. It is thus recommended that a baseline medical history and physical examination as well as a measurement of BLC and serum creatinine be performed before commencement of the occupation and at a regular interval based on the level of lead exposure.
- ***Dietary management*** Some dietary deficiencies increase the risk of lead poisoning in children.

For example,

- iron deficiency is associated with elevated BLC. Therefore, CDC recommends assessment of iron status and placing ironrich diet on all children with lead poisoning.[69]
- Zinc deficiency worsens the toxicity caused by lead poisoning. Zinc, vitamin C, protein and phosphorus deficiency increase the absorption of ingested lead.[69,75]

PATIENT EDUCATION

Patients or parents should be advised to

- Eliminate source(s) of lead exposure,
- Flush standing water from the lines and faucet for several minutes before use, and use cold water for drinking (if they have older homes with galvanized lead or lead soldered pipes and/or lead service lines),
- Maintain a low-fat diet high in calcium, zinc, vitamin C and iron.

This information should be provided in appropriate cultural formats and reading level for the target audience.In addition, the clinician has a role in recognizing risks for potential lead exposures specific to

- Immigrant communities,Refugees, and Children adopted from foreign countries,

Treatment

- The first and most critical step in the treatment of lead poisoning is the removal of patients from the site of exposure and elimination of possible sources of lead.

- Dietary deficiencies should also be corrected as noted above especially in childhood plumbism. The indication and type of pharmacologic treatment are determined by patient's age, clinical symptomatology and BLC.[27,57]

For BLC ≥ 100µg/dL, chelating therapy is always necessary and should be started as parenteral in a hospital [49]On the other hand, in children, chelating therapy is indicated when lead concentrations in two venous blood samples are more than 45 µg/dL even the child is asymptomatic.

Chelating agents To date, the use of

- dimercaprol or British AntiLewisite (BAL),
- calcium disodium ethylenediaminetetraacetic acid (CaNa2EDTA),
- Dpenicillamine and
- Meso-2,3-dimercaptosuccinic acid (DMSA) named succimer is approved for the treatment, but not prevention[44] of acute and chronic lead poisoning.[27]

a) dimercaprol or British AntiLewisite: BAL is one of the oldest agents that were used in the treatment of lead poisoning.[76]

- It is administered intramuscularly (IM) 2.5 mg/kg per day in two divided doses. The duration of treatment is at least three days.
- BAL facilitates the excretion of zinc from urine.[27]
- Skin contact with BAL produces immediate pain and blistering. Eye contact can even result in blindness [42]

b) calcium disodium ethylenediaminetetraacetic acid. CaNa2 EDTA does not cross the blood—brain barrier to any major extent but mobilizes lead in kidneys, liver,and particularly in bones. Therefore, *CaNa2 EDTA is able to remove lead from the extracellular compartment.* Calcium ions displace lead cation.[13]

c) D-penicillamine : it is a reductive chelator. By reducing lead or copper cations, it decreases their binding affinity to proteins and finally stimulates their excretion in urine.

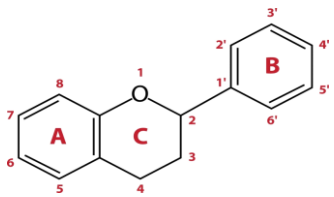
New and less toxic medications: Although other drugs than the above chelating agents have been recommended in the treatment of lead poisoning, more evidence is needed in favor of their safety and efficacy to advocate their use as an alternative or adjunct therapy for the standard pharmacological treatment.

The recommended medications are as follows.

- ***Thiamine*** (25-50 mg/kg/day) is a water-soluble vitamin that, in combination with CaNa2 EDTA (50 mg/kg/day) for 3 days, enhances the elimination of lead from liver and kidney[47,55] and decreases the oxidative damage caused by lead toxicity.[68]
- Similarly, *Vitamins C and E* reduce lead-induced oxidative stress.[63,68]
  The role of vitamins (particularly B, C and E) has been found to be extremely significant and competitive in fighting toxicological manifestations of lead poisoning. These vitamins may chelate lead from the tissues along with restoring the pro/antioxidant balance.
- ***N-acetylcysteine (NAC)*** is also considered an antioxidant chelator. It possesses reactive groups, such as thiol, hydroxyl, and amine, which can bind with lead and remove it from the body [12,16,28]
- A recent in vitro study has demonstrated that N-acetylcysteine amide (NACA) has a higher cellular permeability, better systemic bioavailability, and higher binding affinity to lead compared to NAC, and, therefore, is considered a preferable alternative[28]

4. ***Taurine*** is an organic acid and a major constituent of bile. Single therapy with taurine does not reduce lead burden; however, the concomitant administration of taurine (50-100 mg/kg once daily for 5 days) with a standard thiolchelator (DMSA 50 mg/kg once daily for 5 days) has been effective for the treatment of sub-chronic lead poisoning.[39]

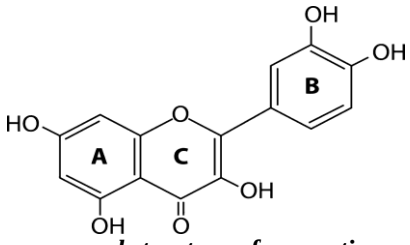
5. ***Flavonoids*** are naturally occurring polyphenolic compounds. They are the main constituents of fruits, vegetables and certain beverages[89] These compounds, like other anti-oxidants, can cure or prevent oxidative stress by chelating redox active metal ions and also by terminating the free radical chain reaction.[87,88]



general structure of flavonoids

6. ***Alpha-lipoic acid*** (50 mg/kg/day), methionine (100 mg/kg/day), homocysteine (25 mg/kg/day) are other sulfur-containing compounds which are able to reduce oxidative stress in lead-exposed tissues when administered for 5 weeks.[16]

7. ***Quercetin*** is a ubiquitously distributed and comprehensively explored bioflavonoid. Dietary sources of quercetin include fruits, vegetables and tea. *The chemical name for quercetin is 3,3,4,5,7-pentahydroxyflavone.* The presence of multiple hydroxyl groups in its chemical structure and conjugated electrons account for its antioxidant and metal chelating property.



general structure of quercetin

8. ***Garlic (Allium sativum)*** is an herbal product which is considered to have chelating effect in some animal studies[46,56,63] Furthermore, a number of neurological signs and symptoms such as irritability (P = 0.031), headache (P = 0.028) and decreased DTR (P = 0.019) were decreased[45] Dried powder garlic (400 mg; equivalent to 1200 µg allicin or 2 g fresh garlic) was administered 3 times daily for 4 weeks.

9. ***Curcumin*** is a yellow-colored polyphenolic compound and the principal active component of turmeric, which is obtained from the plant Curcuma longa. There are reports of antioxidant, radical scavenging and metal chelating effects of curcumin in metal toxicity.[83,84,85,86]

Conclusions and Relevance

Exposure to environmental lead is clearly a major public health hazard of global dimensions. As measures to control the transfer of lead to the environment are implemented in most developed countries through, for example, the phasing out of lead in fuel, paints and other consumer products. However, because of rapid industrialization and the persistence of lead in the environment, exposure is likely to remain a significant public health problem in most developing countries for many years. Much work needs to be done to identify and treat children with elevated blood lead levels and reduce lead exposure in the community. Screening, monitoring, intervention and evaluation are critical for the development of rational, cost-effective and science-based public health policies aimed at achieving these goals.

Various molecular, cellular and intracellular mechanisms have been proposed to explain the toxicological profile of lead that includes generation of oxidative stress, ionic mechanism and apoptosis. Of these oxidative stress has been found to be more pronounced and much more severe.

Public health measures should continue to be directed to the reduction and prevention of exposure to lead by reducing the use of the metal and its compounds and by minimizing lead-containing emissions that result in human exposures.

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