**Lead Toxicity**

**Introduction**

Lead is a toxic metal that has been known to mankind for thousands of years. It has been used in hundreds of ways, from pewter to water pipes. Only recently; however, has attention been focused on lead as a major source of ill health affecting millions of Americans. Some thirty important health conditions are linked to lead, many affecting children and the unborn.

Lead is widely distributed in the environment. Lead contamination has even been found in the Arctic ice caps and is increasing each decade.

A combination of factors contributes to the prevalence of lead toxicity today. In addition to its wide distribution in our environment, lead absorption is enhanced by a deficiency in the diet of vital trace elements such as manganese, zinc, copper, chromium, calcium and magnesium. Such deficiencies are common due to refined food diets and poor eating habits.

Lead has no known preferred function in the body. Lead can substitute for calcium in the bones and thus delay or possibly prevent osteoporosis. However, calcium is the preferred element and lead causes other metabolic problems when it replaces calcium or another preferred mineral.

Symptoms of lead toxicity may appear years after exposure, or even after exposure has ceased, as a result of the sudden release of stored lead due to illness, alcoholism, stress or other changes in one's metabolism.

**Sources Of Lead**

Naturally Occurring Lead

Lead is found in the earth's crust and is increasing with time due to the disintegration of radioactive elements such as uranium changing to lead.

Airborne Lead

Major sources of lead in our ambient air come from leaded gasoline and industries such as manufacture of storage batteries and ore smelting. Cigarette smoke is contaminated with lead due to lead arsenate insecticides sprayed on tobacco.

Food Sources

Food cans which are sealed with lead solder are a major source of ingested lead. This is especially the case with acidic foods such as tomatoes, okra, orange, grapefruit or cranberry juice. Canned baby foods such as evaporated milk may contain as much as 200 micrograms of lead per liter. Canned juices may contain 100 micrograms of lead per liter. Total food intake of lead may be as high as 300 micrograms per day.(1,2)

Foods Which May Be Contaminated With Lead

The following foods are high in lead content due to lead arsenate insecticides or because of lead accumulation, which occurs during the processing of foods. These foods should be avoided by anyone who is found to be lead intoxicated, until the lead level has returned to tolerable levels:

|  |  |  |
| --- | --- | --- |
|  | Anchovy fillets | Apple cider |
|  | Baking powder | Canned Sandwich meat (commercial nonorganic) |
|  | Claw meat | Corn, frozen |
|  | Gelatin, dried | Liverwurst |
|  | Lobster | Puffed rice (cereal) |
|  | Sardines | Sausage |
|  | Whole wheat flour |  |

Hair Colorings May Contain Lead

Several hair tints and dyes contain lead; including Grecian Formula and Youth Hair. Any individual who exhibits an extremely elevated tissue lead level should be questioned about the use of hair coloring products.

The lead contained in these products is absorbed through the scalp and is excreted through the feces and urine, as well as through the hair. Hindu women who traditionally use lead and vermilion to give their hair a reddish tint excrete lead in their urine within 24 hours of application to their hair.

Paints, Glazes and Household Dust

Since children are most prone to lead toxicity, household sources of lead should not be overlooked. Paints can contain lead and the glaze on ceramic cookware and plates is still a common source of lead toxicity. Expensive imported pottery frequently has lead-containing glaze.

"Lead in various forms, principally as lead-based paint, street dirt and household dust, is absorbed through the lungs, skin and intestinal tract.(1)

Drinking Water

Drinking water can be contaminated with lead from lead pipes, lead solder on pipe joints, or from other sources.

Drinking water and blood lead levels are only weakly correlated.(2,3) However, blood lead is not itself a measure of health, though it may be the best available guide to the clinical importance of current environmental sources of lead. The relation between water lead and blood lead probably differs with hard and soft waters(4) and this difference was predictable from the effect of calcium on lead absorption.(5)

**Congenital Lead Intoxication**

Another source of lead that is often overlooked is lead which is passed on from mothers to their unborn children. This would account for high lead levels in children when no known exposure has occurred.

**Excessive Exposure Not Necessary For Accumulation Of Lead**

Finally, it is important to remember that heavy exposure to a source of lead is not necessary for lead toxicity to develop, if a deficiency of vital protective nutrients exists.

Many children, for instance, require relatively high amounts of calcium in their diet because they have a rapid metabolic rate which causes them to excrete more calcium. The resulting calcium loss, together with excessive exposure to lead from environmental and food sources can lead to the accumulation of lead in children, which is absorbed when the child is calcium deficient.

**Detection Of Lead Poisoning**

Much of the chronic lead toxicity occurring today is either misdiagnosed or missed entirely by regular medical checkups. This occurs because physicians are generally unaware of the symptoms of chronic lead intoxication. In addition, adequate laboratory tests to diagnose lead toxicity are not routinely performed.

Blood Determination Of Lead Status

The determination of blood lead levels is not itself a measure of health.

Blood lead levels will frequently be normal in spite of chronic lead toxicity. This occurs because lead is deposited primarily in bones and the brain, so that only minimal levels of lead remain in the blood approximately thirty days after exposure to lead.

"...We must also recognize that blood lead really reflects recent exposure to lead...Basophilic stippling is not very useful in determining exposure to lead. It is nonspecific."(6)

If a person is exposed to lead, he may develop symptoms of lead poisoning three weeks later. If tested six weeks after exposure, the blood test would not reveal abnormal lead levels, because the lead has been cleared from the blood by this time.

Challenge Tests

Another method of assessing lead status is by giving a chelating agent such as EDTA and then collecting a 24-hour urine for lead. However, this test will only detect lead that is in the blood. It will reveal little about lead stored within body tissues.

Hair Analysis

The determination of lead in scalp hair provides additional direct evidence of chronic plumbism (lead toxicity) in children.(7)

Hair analysis is useful for detecting chronic lead toxicity because lead is quickly removed from the blood and stored away in tissues such as hair. Concentrations of lead in hair are often ten times greater than in blood and consequently are easier and more accurately measured.

More Than One Tissue Mineral Analysis May Be Require

Lead is often sequestered in bone and other tissues. For this reason, a high level of lead may not show up immediately on a hair mineral analysis until it is mobilized from storage organs. In certain cases, it may require a year or more of nutritional therapy before lead is released from tissue storage and is detectable on a hair mineral analysis test.

**Lead Levels May Rise And Fall As Elimination Progresses**

Lead is stored in at least five different tissue reservoirs. As lead is released from one storage site and eliminated through the hair tissue, the lead hair level rises and then falls, giving the impression that all the stored lead has been completely eliminated. A year later; however, another reservoir may be emptied, causing another rise in the hair lead level.

**Absorption Of Lead**

The toxicity of lead varies so much between individuals that no definite prediction can be made as to how much exposure will cause symptoms of lead poisoning to appear. It has been suggested by some researchers that continued absorption of more than 2 mg. of lead per day may produce intoxication.

Normally, from 1 to 10 percent of ingested lead is absorbed, depending on the form of the lead and depending upon the nutritional status of the individual. Lead uptake is greater in children than in adults, with children retaining up to 50% of ingested lead.

Absorption of Lead is Greater When Ingested Between Meals

Overall patterns of food intake influence absorption of lead. Lead ingested between meals was absorbed to a greater extent (70%) than was lead ingested with meals (6 to 14%).

Sunlight Increases Lead Absorption

A great majority of lead poisoning cases occur in summer. Animal experiments have demonstrated that vitamin D and the rays of the summer sun enhance the absorption of lead from the intestine.

Smith et al. (1978) have shown that vitamin D stimulated lead absorption in vitamin D-deprived animals. The possibility also exists that summer heat leads to dehydration and acidosis in young children. Vitamin D also increases the mobilization of lead from its storage depots in the bones and so precipitates the acute manifestations of the disease.

Summer is also a time of growth spurts, when the development of new bone calls for a fast turnover of calcium. Lead rides alongside the calcium into the blood stream, to attack the nervous system and the brain itself.

High Fat Diets Facilitate Lead Absorption

Baltrop and Khoo (1975) have shown in rats that high fat diets facilitate absorption and increase tissue lead concentrations. Reducing the dietary fat level to nearly zero; however did not reduce lead concentrations in the tissues.

Calcium/Phosphorus Ratio and Lead Absorption

The amount of lead incorporated into body tissues, particularly the skeleton, was greatly increased by low calcium and low phosphorus diets. The subsequent release of this incorporated lead was strongly inhibited by low calcium diets. This is significant because it provides a key to elimination of lead from storage sites. Adequate calcium in the diet is essential for the elimination of lead.

Calcium and Lead Absorption

Increasing dietary calcium intake has been associated with reduced absorption of lead from the gastrointestinal tract in children.(6)

In general, lead absorption from the intestine proceeds in a manner analogous to calcium, but also shows interactions with iron, zinc and copper. Both clinical and experimental studies indicate that moderately reduced dietary calcium intakes, even above the dietary requirements, increase lead absorption. Vitamin D also plays a role.

Deficiency Of Iron Causes Increased Absorption Of Lead

It is well known that an iron deficiency will increase the absorption of lead from the GI tract. This mechanism may be one of the causes for the apparent increase in lead body burden in those children that have some indication of being iron deficient. According to Lin-Fu, 1973 and Mahaffey, 1974, hyperactivity in children with increased lead may be due to an iron deficiency.

Iron And Lead Absorption

Iron deficiency recently was shown to enhance the absorption of lead as well as iron.(8) This mechanism may be one of the causes for the apparent increase in lead burden in those children that have some indication of being iron deficient.

Magnesium And Lead Absorption

Magnesium reduces lead absorption in puppies.(9)

Alginate And Lead Absorption

Alginate reduces lead absorption in humans.(10)

Children Absorb More Lead

The absorption of lead from the gastrointestinal tract in children is five to eight times as great as in adults, with increased retention.

**Retention Of Lead**

Calcium, Phosphorus And Lead Retention

Low dietary concentrations of calcium increase and high dietary concentrations decrease lead uptake and incorporation into the tissue, especially the skeleton and liver.

Once lead has been incorporated into the body, its removal is delayed by a low dietary calcium/phosphorus ratio or by high dietary vitamin D intake. More recently, dietary iron and zinc have been shown to influence the metabolism of lead. The extent to which dietary calcium can affect lead uptake is very great.

It has also been proven that a low calcium and phosphorus intake lead to increased absorption and retention of lead.

The levels of dietary calcium and phosphorus used in this experiment are not so low that they could not be achieved by many children or adults. They are about one-third to one-half of the N.R.C. recommended allowances for children.

Calcium/Phosphorus Ratio And Lead Retention

Often we find an elevated tissue lead level associated with both low calcium and phosphorus levels. More often than not an elevated lead level is associated with a low calcium/phosphorus ratio. A low calcium/phosphorus ratio is more conducive to lead retention than both a low calcium and phosphorus level. Lead elimination will not be complete until the calcium/phosphorus ratio is balanced.

Research indicates that almost 30% of patients exhibit either a low calcium or a low calcium/phosphorus ratio.

**Excretion Of Lead**

Aging Results In Decreased Lead Excretion

As one's age increases, lead excretion diminishes. This may be a function of a lowered rate of metabolism which often accompanies aging. As a result of diminished excretion, lead tends to accumulate in the body with age.

**Metabolic Effects Of Lead**

Effects Of Lead On Minerals:

* acts like calcium in its metabolism and storage - will be incorporated into bone in preference to calcium.
* interferes with iodine uptake by the thyroid.
* displaces and can cause deficiency or biounavailability of calcium, zinc, manganese, copper and iron. This may be due to interference with production of ceruloplasmin and ferritin, important copper and iron-binding proteins.
* often associated with increased copper and iron levels in the brain.

Effects Of Lead On Enzymes:

* inhibits enzymes associated with heme synthesis, including amino-levulinic acid dehydratase and ferrochelatase.(1)
* inhibits copper and iron dependent enzymes in the Krebs cycle.
* inhibits the oxidase enzyme systems.
* displacing zinc, copper, manganese, iron and chromium, lead can interfere with the function of hundreds of vital metallo-enzymes.

Effects Of Lead On Hormones And Glands:

* reduces the rate of protein binding of iodine in the thyroid gland.
* inactivates many hormones, particularly thyroxin.
* inhibits the copper-dependent biogenic amines (dopamine, epinephrine and norepinephrine.)

Effects Of Lead On Blood And Hemoglobin Synthesis:

* increases the rate of destruction of red blood cells.
* interferes with heme synthesis.

Effects Of Lead On Organs And Body Systems:

* impairs excretion of uric acid.
* may impair kidney function by reducing zinc levels.
* reduces the liver's ability to form new glycogen from added glucose.
* prolonged exposure is followed by intra-nuclear inclusion bodies, interstitial fibrosis and edema of the kidney (see reference: R.A. Goyer and B.C. Mehlman).

**Metabolic Dysfunctions Associated With  
Lead Toxicity By Body Systems**

Musculo-Skeletal System

* arthritis, osteo
* arthritis, rheumatoid
* gout
* low back pain, rickets

Nervous System

* brain function, abnormal
* blindness
* convulsions
* deafness
* dyslexia
* encephalitis
* encephalopathy
* epilepsy
* fatigue
* insomnia
* multiple sclerosis
* muscular dystrophy
* Parkinson's disease
* vertigo

Cardiovascular System

* arteriosclerosis
* atherosclerosis
* cardiovascular dysfunction

Digestive System

* abdominal pain
* colic
* constipation
* indigestion
* liver dysfunction
* weight loss

Reproductive System

* abortions, spontaneous
* impotency
* infertility
* libido, diminished
* menstrual difficulties
* sterility
* stillbirths

Endocrine and metabolic systems

* adrenal insufficiency
* hypopituitarism
* hypothyroidism

Excretory system

* nephritis
* renal dysfunction

Dental

* pyorrhea
* tooth decay

Metabolic dysfunctions

* diabetes
* hypoglycemia
* glycogen storage, impaired

Psychological

* anxiety
* concentration, poor
* depression, mental
* hallucinations
* hyperkinesis
* memory impairment
* mental retardation
* mood swings (manic-depressive disorder)
* nightmares
* psychotic behavior
* schizophrenia

Miscellaneous Dysfunctions

* anemia
* alopecia
* cancer
* cell integrity, impaired

Psychiatric Symptoms

* excitability
* restlessness
* insomnia
* nightmares
* hallucinations
* memory loss
* loss of mental concentration
* mental depression

Psychotic Behavior

The heavy metals, mercury and lead, are known to cause psychotic behavior. Lead poisoning can produce a variety of nervous and mental symptoms as a result of which the patient may be labeled hyperactive or schizophrenic.

Tooth Decay

Human studies have demonstrated increased lead (Pb) intake with increased dental caries.(1,2,8,10)

**The Effects Of Lead On Other Minerals**

Calcium

A low tissue calcium level is frequently associated with an elevated lead level. A low calcium to magnesium ratio is also frequently associated with an elevated lead level. An elevated lead level is frequently a causative factor of calcium biounavailability.

Copper

Lead in the diet inhibits copper metabolism and growth in a manner which was inversely related to copper intake. Lead adversely alters copper metabolism as one aspect of its toxicity.

Copper in the blood is primarily found in the plasma and 80-90% of this is usually found to be in the form of ceruloplasmin. The ceruloplasmin levels in animals exposed to 0.5% lead in their diets were markedly lower than those animals on corresponding control diets.

Lowered ceruloplasmin levels, or perhaps the lowering of zinc or iron, results in elevated hair copper levels. An elevated copper level is indicative of deposition of copper into the brain, liver and other tissues of the body.

Iron

A high tissue lead level is frequently associated with an elevated tissue iron level. Lead can apparently displace iron from the tissues. Lead inhibits heme synthesis in reticulocytes, causing an anemia that is morphologically similar to iron-deficiency anemia.

Zinc

An elevated tissue lead level is frequently associated with a low tissue zinc level. The displacement or biounavailability of zinc is associated with hypoglycemia, hypopituitarism, diminished libido and hardening of the arteries.

A high hair zinc with a high lead level is indicative that lead is interfering with zinc metabolism, resulting in a biounavailability of zinc and a consequent loss of zinc from the tissues.

Iodine

Lead appears to inhibit uptake of iodine by the thyroid and inhibit conversion of iodine to protein-bound iodine (Sandstead, 1967).

Sodium And Potassium

Lead intoxication appears to cause renal sodium loss. In addition to interference with sodium resorption by the kidney, lead appears to impair the energy-dependent mechanism of red blood cells for controlling sodium and potassium exchange.

**Effects Of Other Nutrients On Lead**

Calcium

An elevated tissue lead level may be due to a low tissue calcium level. Increased dietary calcium intake protects against the accumulation of tissue lead.

When lead is deposited in bones, these deposits take exactly the same pattern as calcium deposits. If both lead and calcium are present, the bone is more likely to take up the lead, because the lead compounds in the bone are less soluble than the corresponding calcium phosphates. However, if extra calcium is given before lead administration, less lead is taken up by the bones.

A complex relationship exists between calcium, phosphorus, vitamin D and lead. Vitamin D had a marked effect on the lead concentrations of blood and bone of young rats fed lead-containing diets.

Phosphorus

Phosphorus was found to be most protective against lead absorption. Groups fed vitamin D had higher lead in the bone ash on low calcium/low phosphorus (Diet A), high calcium/low phosphorus and high calcium/high phosphorus types of diets. Vitamin D raised the lead concentrations of the blood of the rats on all the diets except in the group on the high phosphorus-low calcium diet.

In the group on the high phosphorus/low calcium diet, the lead concentration of the blood was so much lower than in the other two groups that it could not be determined accurately. Any effect of vitamin D could not be demonstrated.

In rats fed high calcium/low phosphorus diets, the lead in the blood was markedly higher than in rats fed high phosphorus/low calcium diets, despite the fact that the latter diet contained three times as much lead as was present in the high calcium/low phosphorus diet.

The degree of calcification is significantly lower in the high calcium/low phosphorus group than in either of the two other groups, which have about the same degree of calcification. The degree of deposition of lead is higher in the low calcium/low phosphorus group than in either of the other groups.

The addition of either calcium or phosphorus to the low calcium/low phosphorus diet causes a decrease in lead deposition. The ratio of the deposition of lead over calcium, as shown by the mg% of lead in ash, does not change significantly when calcium is added to Diet A, but is significantly lower when phosphorus is added to Diet A.

Vitamin D

On a lead-containing, rickets producing diet, the addition of vitamin D increases bone lead.

Lead, phosphorus and vitamin D form a system of lead deposition analogous to the system of calcification. The addition of calcium tends to diminish phosphorus levels, resulting in increased lead deposition in the bones.(21)

In animals low in vitamin D, less lead is absorbed. This implies a competitive interaction between the two divalent cations, lead and calcium.

Vitamin D appears to increase absorption of lead (Sobel et al., 1940). (See above for the interaction between vitamin D, calcium, phosphorus and lead.)

Chromium

An elevated tissue lead level may be due in part, to a low tissue chromium level. Increasing chromium, if low, protects against increased lead levels.

Copper

A high tissue lead level is frequently associated with a low tissue copper level and/or a low zinc/copper ratio. It has been noted that adequate tissue copper protects against lead accumulation and that copper displaces lead from the tissues.

"Some nutrients are known to influence susceptibility to lead toxicity. Among these are vitamin E, selenium and copper."(6)

Iron

High levels of iron appear to decrease susceptibility to lead toxicity.

Magnesium

Magnesium and alginates reduce lead absorption.

Zinc

An elevated tissue lead level may be due in part, to a low tissue zinc level. Elevation of tissue zinc levels will serve to deter accumulation of lead.

Protein & Amino Acids

A 20% casein diet protected rats from lead more than a 6 or 13% casein diet. Cysteine and methionine appear protective.

Vitamin E

Vitamin E has been found to protect rats against the hemolytic effects of 250 ppm of lead in drinking water (Levander et al., 1974).

Vitamin C

200-800 mg of ascorbic acid protected lambs from lead in mine tailings (Clegg and Rylands, 1966). However citrus fruits are not protective, as citric acid increases lead absorption.

Niacin

Niacin has been reported to improve heme synthesis in lead-intoxicated rats (Kao and Forbes, 1973).

Detoxification of lead

Thorough detoxification of systemic lead toxicity takes time, because lead is deposited in tissues with a slow turnover time, such as bone. A detoxification program should include all the following components:

Cease Exposure

This is obvious and usually involves cessation of smoking, handling and breathing lead and removing other sources of lead contamination.

**Enhance Biochemical Energy Levels**

We consider the enhancement of biochemical energy levels as the most important single step that can be taken to promote detoxification, although it is the most often overlooked.

Energy is required for the organs of elimination to function, as well as for the replacement of the stored lead. An increased energy level allows the body's natural healing mechanisms to swing into high gear, significantly shortening the time period required for lead detoxification. On the other hand, if one's energy level remains low, lead can never be entirely eliminated.

The reason energy enhancement is not widely practiced is that it requires precise nutritional balancing to fine-tune body chemistry. To accomplish this, first an assessment must be made of the energy system, then a precise program must be designed and followed which moves the body toward balance and helps it to remain there.

We have specialized in the design of these programs for twelve years. We find that the hair element analysis, when properly performed and interpreted, is one of the few ways to assess the body's energy system and guide design of a program designed to enhance energy levels.

On the basis of the hair mineral test, a diet and supplement program is designed. Retests are necessary about every three months to keep the program appropriate so that the energy levels remain high in view of fluctuations in the body chemistry which will occur during correction.

**Administer Chelating Agents**

A chelating agent is one which binds to a metal and causes its removal. Substances which are known to be chelating agents for lead include vitamin C and sodium EDTA.

Vitamin C: Doses of 3 grams or more per day are required. Vitamin C enters all tissues of the body, so that the sites of lead storage are reached.

EDTA therapy: EDTA is a chemical which binds lead as well as approximately eight to twelve other essential minerals, facilitating their removal from the body. EDTA does not reach into the body tissues; however, and will only chelate from the blood and arterial walls, neither of which are major storage sites for lead.

A difficulty with the use of EDTA for lead detoxification is that EDTA also removes calcium. By removing calcium, deposition of lead is encouraged. Also, EDTA is not specific, so that other nutrient minerals are removed, altering the mineral balance. Use of EDTA is not a routine part of mineral balancing programs.

**Administer Biochemical Antagonists**

Lead antagonists include calcium, phosphorus, iron, copper, chromium and zinc. These minerals either compete for absorption with lead, in the gut, or replace lead in storage sites.

Use of antagonists is essential for effective lead detoxification, but are usually not sufficient if used alone.

**Improve Channels of Elimination**

At times, removal of toxic metals is prevented or impeded by inadequate liver, kidney, bowel or even skin activity. Therefore, it is helpful to enhance the activity of these organs. Methods include baths, enemas, bowel-cleansing programs.

Nutrients that may be given include; beet root powder, choline, inositol, sulfur, kidney glandular substance and herbs such as Russian Black Radish to stimulate or improve liver and kidney function. Bugleweed and yellow dock are herbs that may also assist elimination of metals.

The major eliminative channel for toxic metals is the intestinal tract and adequate bile is required for elimination of these metals through the colon. Stimulating bile production, facilitates removal of lead and other toxic metals.

All the above methods can have an important place in lead detoxification, especially if the activity of the organs of elimination are sluggish. Many times increasing colon activity and improving liver function at the beginning of a program can prevent reactions that may occur if the body cannot adequately eliminate lead.

Often several years are required for complete detoxification of lead. Commitment in both time and effort is essential for success of the program. By enhancing energy levels and combining this with the other methods described above; very satisfactory results can be obtained in most cases of lead toxicity.

**Diet For Lead Detoxification**

Calcium and phosphorus in the diet should be emphasized. Adequate iron, zinc, manganese and vitamin C are also helpful.

All metals are transported by proteins. Elimination of lead is aided by adequate protein levels.

**Symptoms Commonly Associated With Removal Of Lead From Tissues**

During a nutritional correction program, lead may be removed rapidly from storage. Such removal will often result in symptoms related to acute lead poisoning; these include:

|  |  |  |
| --- | --- | --- |
|  | abdominal pain | hyperactivity |
|  | arthritis | insomnia |
|  | constipation | inflammation |
|  | depression | aches & pains |
|  | digestive upsets | irritability |
|  | fatigue | headaches |

The Textbook of Medicine by Cecil and Loeb states that to reduce the symptoms associated with rapid removal of lead from the body, phosphorus intake should be increased. An increase in phosphorus intake causes lead to return to storage in the bones.

Lecithin is high in phosphorus and for this reason three 1200 mg capsules, three times a day (t.i.d.) may be given to ameliorate symptoms resulting from rapid elimination of lead from tissue storage sites.

**References**

* K.R. Mahaffey: Nutritional Factors in Lead Poisoning. Nutrition Reviews 39:353-362, 1981.
* Moore MR., Meredith PA., Campbell BC., Goldberg A., Pocock SJ., Contribution of lead in drinking water to blood lead, Lancet ii: 661-62, 1977.
* Thomas HF., Elwood PC., Welsby E., St. Leger AS., Relationship of blood lead in women and children to domestic water lead, Nature 282: 712, 1979.
* Thomas HF., Elwood PC., Toothill C., Morton M. Blood and water lead in a hard water area, Lancet i: 1047-48, 1981.
* Heard MJ., Chamberlain AC. Effect of minerals and food on uptake of lead from the gastrointestinal tract in humans. Hum. Toxicol (in press).
* J. Julian Chisolm, Jr., David M. O'Hara, Lead Absorption in Children, Urban & Schwarzenberg, Baltimore-Munich 1982.
* Kopito, L., Byers, R.K., and Shwachman, H., Lead in Hair of Children With Chronic Lead Poisoning, New Eng. J. Med. 276:949-953 (April) 1967.
* W.S. Watson, R, Hume and M.R. Moore: Oral Absorption of Lead and Iron. Lancet 2: 236-237, 1980.
* B.Fine, A. Barth, A. Sheffet and M. Lavenhar:Influence of Magnesium on the Intestinal Absorption of Lead., Environ. Res. 12:224-227, 1976.
* G.E. Harrison, T.E.F. Carr. A. Sutton, E.R. Humphreys and J. Rundo: Effect of Alginate on the Absorption of Lead in Man. Nature 224: 1115-1116, 1969.
* E.I. Dresel and J. El Falk: Studies on the Biosynthesis of Blood Pigments. 3. Haem and Porphyrin Formation from Aminolaevulic Acid and from Porphobilinogen in Haemolysed Chicken Erythrocytes. Biochem. J. 63: 80-87, 1956.
* S. Piomelli and J. Graziano, Pediatr. Clin. N.A. 27:843-853, 1980.
* M.R. Moore, P.A. Meredith and A. Goldberg in Lead Toxicity. R.A. Singhal and J.A. Thomas, Editors, pp. 79-117. Urban & Schwarzenberg, Baltimore, 1980.
* S. Piomelli in Low Level Lead Exposure: The Clinical Implications of Current Research. H.L. Needleman, Editor, pp. 67-74, Raven Press, New York, 1980.
* R.A. Goyer and B.C. Rhyne in International Review of Experimental Pathology, G. Richter and M.A. Epstein, Editors, vol. 12, pp. 1-77. Academic Press, New York, 1973.
* D.D. Choie and G.W. Richter and Lead Toxicity, R.L. Singhal and J.A. Thomas, Editors, pp. 187-212. Urban & Schwarzenberg. Baltimore, 1980.
* R.K. Byers and E.E. Lord, Am. J. Dis. Child, 66,471 (1943); C.D. Jenkins and R.B. Mellins, AMA Arch. Neurol. Psychiatry, 77, 70 (1957); D. L. Thurston, J.N. Middlecampe, E. Mason, J. Pediatr. 47, 413 (1955); J.J. Chisholm, Jr. Dev. Med. Child Neurol., 7, 529 (1965); G. Wiener, Public Health Rep., 85, 19 (1970).
* O.J. David, J. Clark, K. Voeller, Lancet 1972-II, 900 (1972); J. Perino and C.B. Ernhart, Proc. Annu. Conv. Am. Psychol. Assoc., 81, 719 (1973); O.J. David, S.P. Hoffman, J. Sverd, J. Clark, K. Voeller, Am. J. Psychiatry 133, 1155 (1976).
* D. Kotak, J. Pediatr, 80, 57 (1972); R.G. Lansdown, J. Shepherd, B.E. Clayton, H. T. Delves, P.J. Graham, W.C. Turner, Lancet 1974-I, 538 (1974).
* M. Webb, Br. Med. Bull. 31, 246 (1975); D.W. Fassett, Anual Rev. Pharmacol. 15, 425 (1975); R.L. Singhal, Z. Merali, P.D. Hrdina, Fed. Proc. Fed. Am. Soc. Exp. Biol., 35, 75 (1976).
* NUTRITION CLASSICS, The Journal of Biological Chemistry, Volume 132, 1940, pages 239-265, The Biochemical Behavior of Lead, by Albert E. Sobel, Henry Yuska, David D. Peters, and Benjamin Kramer.

**Recommended Readings**

* The Trace Elements and Man, Henry Schroeder, MD
* Mitchell and Aldors, l974. Lead Content of Foodstuffs. Environmental Health Prospectus.
* Alexander, 1974, The uptake of lead by children in differing environments. Environmental Health Prospectus.
* Smith, 1976, Metabolism and Toxicity of Lead. Trace Elements in Human Health and Disease.
* Goyer and Mehlman, Toxicology of Trace Elements, Hemisphere Publishing Corporation, Washington, 1977.
* Nutrition Reviews, Vol. 39, #10, October, 1981.

Copyright © 1989 - Analytical Research Laboratories, Inc.

This material is for educational purposes only. The preceding statements have not been evaluated by the Food and Drug Administration. This information is not intended to diagnose, treat, cure or prevent any disease.