

from e-book: Toxic Metals and Antidotes: The Chelation Therapy Handbook

Gentle detoxification – The use of natural chelating agents

When it comes to natural chelating products, we need to remember the old paradigm, 'If something is too good to be true, it isn't.' An open mind and the ability to engage logical and analytical thought helps us separate effective natural treatments from products that are no more than an illusion and money maker for those who market it.

We must also remember that powerful synthetic chelating agents are not always the best choice; the patient condition may warrant the use of gentle chelation.

The metal binding of nutrients

Nutritional chelation is one of the oldest means to detoxify the body. The sulfhydryl-containing amino acids are capable of binding heavy metals, just like any chelator of the thiol group.

A **thiol** is a compound that contains the functional group –SH, which is composed of a sulfur atom and a hydrogen atom. Being the sulfur analogue of an alcohol group (-OH), this functional group is referred to either as a *thiol group* or a *sulfhydryl group*. More traditionally, thiols are often referred to as **mercaptans**. The term *mercaptan* comes from the Latin *mercurius captans*, meaning 'laying hold of mercury.' As the name suggests, the –SH group binds tightly to the element mercury. It can, of course, bind to other elements such as lead.

Vitamin C, E and other antioxidants including the bioflavonoids also support metal binding. Dr. Earl B. Dawson of the University of Texas Medical Branch at Galveston found that adult smokers who took 1,000 milligrams daily of Vitamin C dramatically lowered lead levels in their blood within one week. Dr. Dawson reported that Vitamin C was given to 75 men aged 20 to 35 years. The men were randomly divided into three groups, receiving either 200 milligrams a day, 1,000 mg/day or a placebo which had no Vitamin C content. The study lasted one month, and a weekly evaluation by Dr. Dawson and colleagues found no changes in the placebo test group or in the group receiving only 200 milligrams daily. But the group receiving 1,000 milligrams a day saw blood levels of lead drop sharply after only one week of the vitamin supplementation. Their blood lead levels remained low throughout the remainder of the test period.¹

Scientists at the University of California at San Francisco also found that Vitamin C helps reduce dangerous blood levels of lead. Dr. Joel A. Simon and Dr. Esther Hudes revealed that high dosages of Vitamin C are associated with reduced blood levels of lead in both young children and adults. The researchers said they believe the results of their studies on lead in blood can have "public health implications" for controlling lead toxicity, particularly for children. Their studies indicated that high levels of Vitamin C in blood correlated with lower levels of lead in blood.²

"Vitamin C levels are an important independent correlate of blood lead levels among Americans," says Joel Simon, MD, MPH, SFVAMC staff physician and UCSF assistant professor of medicine, epidemiology & biostatistics. "To our knowledge, this report is the first population-based study to establish such an association. If a causal relation is confirmed, increased consumption of ascorbic acid may have public health implications for the prevention of lead toxicity."

I have used nutritional intervention in the treatment of chronic metal exposure long before I got involved in the teaching of synthetic chelation protocols. One of my most memorable cases was that of Oliver S., a Dutch teenager suffering from aplastic anemia. The cause was unknown. When I was first consulted, the 17-year old received weekly blood transfusions; his condition was considered serious. Through hair mineral analysis, we discovered a significant lead intoxication. His hair lead level was measured >80PPM (=>80mg/kg); the accepted reference range for adults is <3PPM. Blood levels turned out negative; not a surprise after many month of weekly blood transfusions. However, blood tests taken during the initial diagnostic evaluations had shown similar negative results, hence lead intoxication had been ruled out as a cause. Hair mineral analysis was never even considered.

Oliver and his family lived in a stately old Dutch home. Water testing revealed some lead content, though values did not seem high enough to cause a severe case of chronic lead intoxication. I asked the family to scratch paint off the walls inside his room, as lead paints were still in use at the time of Oliver's birth. Sure enough, we found extremely high levels of lead. Further investigation provided the answer. As a young child, Oliver had a habit of scratching paint off the wall, licking it. Subsequently, he had slowly intoxicated himself at an early age. It took nearly 16-year for the disease to develop.

For cases of acute or chronic lead intoxication, EDTA infusions are considered the treatment of choice. Oliver was chelated at a medical denter, but he did not respond favourably. Because of his condition, we decided to utilize nutritional therapy.

The patient received sulphur-containing amino acids, moderate amounts of vitamin C (1000mg 3-4x daily), 400 IE Vitamin E, and other antioxidants, a B-complex and a multivitamin/mineral complex. We did not include Lipoic acid or Glutathion, which are often used for detoxification therapies, simply because we did not know much about these nutrients' role in detoxification. Since glutathion can be synthesized from the amino acids L-cysteine, L-glutamate and glycine, it was, inadvertently, part of the program.

By today's standards, this oral nutritional program was moderate and rather simple, but within the first three month, Oliver's transfusion schedule could be stretched more and more until it was tapered off. After 1 ½ years of nutritional treatment, a repeat hair analysis showed a significant reduction in lead levels; within 2 years, his doctors released him from care, considering him healthy.

Table 1 shows the youngster with his sister and friends after health was regained.



In Oliver's case we did not utilize urine testing to document the metal-binding capacity of nutrients. In the late 1980's, it simply did not occur to us and the Dutch doctors. We have come a long way since then, but we still do not have sufficient data to prove or *disprove natural chelating agents*.

Detoxification of metals with Antioxidants and other Nutrients:

Vitamin C and other antioxidant nutrients are presumed to play a pivotal role in minimizing the damage from oxidative products, including free radicals. This protective function is twofold: the already-oxidized groups in prosthetic centers of enzymes are reduced and the oxidants and free radicals are removed. Much information has been accumulated by orthomolecular medicine to demonstrate that mineral replacement or detoxification through nutrients takes place.

As early as 1978 Prasad found that the daily ingestion of 150mg elemental zinc (as 660mg zinc sulfate) produced overt copper depletion with anemia in some patients (Prasad As eta l. JAMA 240:2166, 1978).

In 1986, Read et al reported that excessive supplementation of calcium decreases iron absorption. (Read MH et al. Mineral supplementation practices of adults in seven western states. Nut.Res. 6:375-83, 1986) and O'Donnell and Smith stated that excessive calcium impairs magnesium absorption probably due to competition for a common transport system. (O'Donnell JM, Smith DW. Uptake of calcium and magnesium by rat duodenal mucosa analyzed by means of competing metals. J.Physiol.229:733, 1973)

In 1983, Dr. Carl C Pfeiffer wrote that zinc may reduce blood manganese levels, (Pfeiffer CC, LaMola S. Zinc and Manganese in the schizophrenias, J.Orthomol.Psychiat. 12:215-34, 1983)

and as early as 1967, Van Campen reported that copper decreases zinc absorption. (Van Campen DR. Copper interference with intestinal absorption of zinc-65 by rats. *J.Nutr.* 473, 1967)

In 1998, Dr. Earl B. Dawson of the University of Texas Medical Branch at Galveston found that men who took 1,000 milligrams daily of Vitamin C dramatically lowered lead levels in their blood within one week. In an abstract prepared for the American College of Nutrition, Dr. Dawson reported on his study in which dietary ascorbic acid, or Vitamin C, was given to 75 men aged 20 to 35 years. The men were randomly divided into three groups, receiving either 200 milligrams a day, 1,000 milligrams or a placebo which had no Vitamin C content. The test lasted for one month. Studying the results each week, Dr. Dawson and his colleagues found no changes in the placebo test group or in the group receiving only 200 milligrams daily. But the group receiving 1,000 milligrams a day saw blood levels of lead drop sharply after only one week of the vitamin supplementation. Their blood lead levels remained low throughout the remainder of the test period.

Shinji Yoneda and Kazuo T. Suzuki of the Faculty of Pharmaceutical Sciences, Chiba University, Japan reported in *Toxicology and Applied Pharmacology*, Volume 143, Issue 2, April 1997, Pages 274-280 that the toxicity of mercury (Hg) can be reduced by coadministration with selenium. The study of Greenland animals by Dietz et al (see Abstract below) suggests that methyl mercury is detoxified by a chemical mechanism involving selenium.

The role of chelating agents for the prevention, intervention, and treatment of exposures to toxic metals was the topic of a conference held at the National Institute of Environmental Health Sciences, 22-23 September 1994. The objective of the conference was to review experimental and clinical studies concerned with the effectiveness and potential toxicity of chelating agents used to reduce the body burden of various metals and to identify research needs in the area of chelation. The conference was prompted by emerging evidence that low-level exposures to metals may result in toxic effects not previously recognized.

Discussed were synthetic and natural chelating methods such as the use of Metallothionin,

Metallothionein (MT) was discovered in 1957 as a cadmium-binding protein in the renal cortex of the horse and is characterized as a low molecular weight (>9000 Da), cysteine-rich, metal-binding protein. Mammalian MT contains 61 amino acids. The protein contains no aromatic amino acids or histidine. Twenty of the 61 amino acids are cysteine, and are arranged in a highly conserved sequence which is observed in vertebrate, invertebrate, yeast, and plant MT. Metals bind to MT in metal-thiolate complexes exhibiting tetrahedral (cadmium, zinc) or trigonal (copper) geometry. Two major isoforms of MT (designated MT I and II) have been identified in most species and are controlled separately by different genes. Humans contain subforms of the two isoforms. Recently, a third isoform (growth inhibitory factor, MT III) has been identified in the brain. The MT molecule is divided into two distinct metal-binding domains. The carboxyl terminal half of the molecule is designated the beta-domain and represents amino acids 30-61. It contains 11 cysteinyl residues and binds four atoms of zinc or cadmium or six atoms of copper. The β -domain is the amino-terminal half of MT and contains nine cysteinyl residues. This domain binds three ions of zinc or cadmium or six copper ions. MT exhibits numerous biological and physiological functions. It appears to have a role in zinc and copper metabolism. The induction of MT protects organisms from toxic metals such as cadmium. MT also exhibits free-radical

scavaging activity. Medically, the induction of intestinal MT by zinc therapy is correlated with normal to negative copper balance in patients with Wilson's disease. Renal MT induction also protects the intestine against the toxic effects of cisplatin treatment.

Research:

The following research abstracts speak for themselves (*italics provided by the author*); but clearly more analytical and clinical proof is needed.

Dietz R., Riget F, Born E.W. **An assessment of selenium to mercury in Greenland marine animals.** The Science of The Total Environment, Volume 245, Issues 1-3, 17 January 2000, Pages 15-24

Abstract

Information on mercury and selenium molar relation in muscle, liver and kidney tissue of Greenland marine animals is presented. In the majority of the samples selenium was present in a molar surplus to mercury. This was most clear in molluscs, crustaceans, fish and seabirds. A 1:1 molar ratio was found in tissues of marine mammals with high mercury concentrations (above approx. 10 nmol/g). This was most clearly demonstrated for liver and kidney tissue of polar bear and for ringed seal with high mercury concentration in the liver. These findings support previous results found in liver tissue of marine mammals, suggesting that methyl mercury is detoxified by a chemical mechanism involving selenium. If the anthropogenic release of mercury to the environment increases in the future due to increasing energy demands, species such as polar bears and seals with high tissue mercury concentrations should be monitored to elucidate whether this protective mechanism can be maintained in target organs.

Ercal N, Gurer-Orhan H, Aykin-Burns N. **Toxic metals and oxidative stress part I: mechanisms involved in metal-induced oxidative damage.** Curr Top Med Chem. 2001 Dec;1(6):529-39.

Abstract

Toxic metals (lead, cadmium, mercury and arsenic) are widely found in our environment. Humans are exposed to these metals from numerous sources, including contaminated air, water, soil and food. Recent studies indicate that transition metals act as catalysts in the oxidative reactions of biological macromolecules therefore the toxicities associated with these metals might be due to oxidative tissue damage. Redox-active metals, such as iron, copper and chromium, undergo redox cycling whereas redox-inactive metals, such as lead, cadmium, mercury and others deplete cells' major antioxidants, particularly thiol-containing antioxidants and enzymes. Either redox-active or redox-inactive metals may cause an increase in production of reactive oxygen species (ROS) such as hydroxyl radical (HO.), superoxide radical (O₂⁻) or hydrogen peroxide (H₂O₂). Enhanced generation of ROS can overwhelm cells' intrinsic antioxidant defenses, and result in a condition known as "oxidative stress". Cells under oxidative stress display various dysfunctions due to lesions caused by ROS to lipids, proteins and DNA. Consequently, it is suggested that metal-induced oxidative stress in cells can be partially responsible for the toxic effects of heavy metals. Several studies are underway to determine the effect of antioxidant supplementation following heavy metal exposure. Data suggest that antioxidants may play an important role in abating some hazards of heavy metals. In order to prove the importance of using

antioxidants in heavy metal poisoning, pertinent biochemical mechanisms for metal-induced oxidative stress should be reviewed.

Patrick L. **Toxic metals and antioxidants: Part II. The role of antioxidants in arsenic and cadmium toxicity.** *Altern Med Rev.* 2003 May;8(2):106-28.

Abstract

Exposure to toxic metals has become an increasingly recognized source of illness worldwide. Both cadmium and arsenic are ubiquitous in the environment, and exposure through food and water as well as occupational sources can contribute to a well-defined spectrum of disease. The symptom picture of arsenic toxicity is characterized by dermal lesions, anemia, and an increased risk for cardiovascular disease, diabetes, and liver damage. Cadmium has a significant effect on renal function, and as a result alters bone metabolism, leading to osteoporosis and osteomalacia. Cadmium-induced genotoxicity also increases risk for several cancers. The mechanisms of arsenic- and cadmium-induced damage include the production of free radicals that alter mitochondrial activity and genetic information. The metabolism and excretion of these heavy metals depend on the presence of antioxidants and thiols that aid arsenic methylation and both arsenic and cadmium metallothionein-binding. *S-adenosylmethionine, lipoic acid, glutathione, selenium, zinc, N-acetylcysteine (NAC), methionine, cysteine, alpha-tocopherol, and ascorbic acid have specific roles in the mitigation of heavy metal toxicity. Several antioxidants including NAC, zinc, methionine, and cysteine, when used in conjunction with standard chelating agents, can improve the mobilization and excretion of arsenic and cadmium.*