Toxic Heavy Metals Information

Aluminum (Al)

Common sources of bio-available aluminum include: aluminum cookware, flatware and especially coffee pots; aluminum hydroxide anti-acid formulations; some types of cosmetics, especially deodorants; some colloidal minerals and some herbs or herbal products. Aluminum cookware is particularly of concern if acid foods are cooked such as tomato paste (contains salicylates). In cosmetics and deodorants, aluminum chloride may be present as an astringent. In water purification, alum (sodium aluminum sulfate) may be used to coagulate dispersed solids and improve water clarity. Alumina or Al2O3 is very stable chemically and not bioavailable. Silica limits the solubility of aluminum and aluminum silicate is not very bioavailable. Clays, bentonite for example, contain aluminum that has poor bio-availability. Aluminum food containers are manufactured with polymer or plastic coatings that prevent direct food-aluminum contact provided such coatings are not damaged. In the GI tract, phosphates react with aluminum ions forming insoluble aluminum phosphates. If this phosphate-blocking were 100% efficient, then virtually no aluminum would be absorbed. Evidently, this phosphate-forming process is incomplete because body tissue levels (such as hair) usually contain measurable amounts of aluminum. In the body aluminum follows a path of increasing phosphate concentration: plasma, cytosol, cell nucleus. Once in the nucleus, it adversely affects protein formation. Long-lived cells such as neurons are susceptible to long-term accumulation. Aluminum is considered neurotoxic. Without intervention, aluminum accumulates continually in the body with the highest concentration occurring at old age or death. A hair element test can be used to corroborate increased body burden of aluminum. An oral provocation with the amino acid glycine, 80 mg/Kg body weight (in divided doses) 24 hours before a diagnostic EDTA chelation with subsequent urine collection can be done to confirm aluminum excess. (Eliminate food/beverage sources of Aluminum during this procedure.)

Barium (Ba)

Barium has not been established to be an essential nutritional element. Elevated levels of Barium often are observed after exposure to Barium which is used as a contrast agent during diagnostic medical tests such as "barium swallow", "upper GI series", "barium enema", etc. Elevated levels of Barium may interfere with calcium metabolism and potassium levels. Acutely high intake of soluble Barium salts (nitrates, sulfides, chlorides) can be toxic. Chronic exposure to Ba may be manifested by skeletal muscle and cardiac muscle stimulation, tingling in the extremities, and loss of tendon reflexes. Due to its high density, Barium is utilized to absorb radiation and is utilized in concrete shields around nuclear reactors and in plaster used to line x-ray rooms. The main use of Barium in medicine is as a contrast medium. Long-term retention of Barium can occur - granuloma of the traverse colon has been reported after diagnostic use of Barium sulfate. Crystalline Barium titanate is a ceramic compound which is used in capacitors and transducers. Barium is also used to produce pigments in paints and decorative glass. Soluble Barium compounds are highly toxic and may be used as insecticides. Barium aluminates are utilized for water purification, acceleration of concrete solidification, production of synthetic zeolites, and in the paper and enamel industries. Although Barium is poorly absorbed orally (<5%) it can be very high in peanuts and peanut butter (about 3,000 nanograms/gram) as compared to egg, frozen and fast foods such as burgers, fries, and hot dogs (400-500 nanograms/gram). It is noteworthy that Barium intake is much higher in children than adults (Health Canada 2005, www.atsdr.cdc.gov/toxprofiles/tp24-c6.pdf). Barium levels (and the levels of 16 other elements) in water can be assessed with water testing. A confirmatory test for elevated Barium is measurement of urine levels of Barium after a chelation provocation, and blood electrolytes should be checked as hypokalemia (low potassium) may be associated with elevated Barium.

Bismuth (Bi)

Bismuth (Bi) levels are measured primarily for investigational purposes. Bi is a non-essential element of low toxicity. However, excessive intake of insoluble, inorganic Bi containing compounds can cause nephrotoxicity and encephalopathy. Absorption is dependent upon solubility of the Bi compound, with insoluble Bi excreted in the feces while soluble forms are excreted in the urine. Sources of Bi include: cosmetics (lipstick), Bi containing medications such as rantidine Bi-citrate, antacids (Pepto Bismol), pigments used in colored glass and ceramics, dental cement, and dry cell battery electrodes.

Symptoms of moderate Bi toxicity include: constipation or bowel irregularity, foul breath, blue/black gum line, and malaise. High levels of Bi accumulation can result in nephrotoxicity (nephrosis, proteinurea) and neurotoxicity (tremor, memory loss, monoclonic jerks, dysarthria, dementia).

Urine elements analysis can be used to corroborate Bi absorption for a period of days or a few weeks after the exposure. Dithiol chelating/complexing agents (DMPS, DMSA) markedly reduced Bi levels in liver and kidneys, and increased Bi in urine in animal studies (J. Lab. Clin. Med.; 119:529-537,1992).

Cadmium (Cd)

is insidiously toxic with chronic accumulations affecting kidney function, lungs, cardiovascular tissues, bone, and the peripheral nervous system. Without intervention, the biological half-life of Cadmium in humans exceeds 20 years (Harrison’s Principles of Internal Medicine, 13th ed, pp 2463-64).Chronic manifestations associated with this degree of Cadmium excess include: hypertension, weight loss, microcytic-hypochromic anemia, lymphocytosis (excess white blood cells), proteinuria (protein in the urine) with wasting of beta2 microglobulin, emphysema and

pulmonary fibrosis (if inhalation was a route of contamination), atherosclerosis, osteomalacia, osteoporosis, lumbar (lower back) pain, and peripheral neuropathy. Acute inhalation of Cadmium dusts, fumes or soluble salts may produce cough, pneumonitis and fatigue. Manifestations of Cadium toxicity may be lessened or delayed by an individual's protective and detoxication capacities. Zinc and vitamin E are protective; metallothionein and glutathione bind Cadmium and help detoxify initially. Smoking can be a source for as much as 0.1 mcg Cadmium per cigarette (HEW Pub. No. NIOSH 76-192, US Govt. Printing Ofc., 1976). Some medical authorities consider Cadmium to be a carcinogen for lung cancer (Harrison's Principles, 13th ed, op. cit. pp 2463). Other occupational or environmental sources include: mining and smelting activities, pigments and paints, electroplating, electroplated parts (e.g., nuts and bolts), batteries (Ni-Cd), plastics and synthetic rubber, photographic and engraving processes, old drums from some copy machines, photoconductors and photovoltaic cells, and some alloys used in soldering and brazing. "Cadmium Red" as used in dental acrylics (dentures) could be a significant source of exposure for those making dentures or dentists and dental technitians making fine tune adjustments (grinding) to dentures chair side. Cadmium free acrylic dentures are now available.

Cesium (Cs)

Cesium is a naturally-occurring element found in rocks, soil and dust at low concentrations. It is present in the environment only in the stable form of 133 Cesium (the radioactive isotopes 134 Cesium and 137 Cesium are usually not measured or reported). Cesium can be absorbed after oral ingestion, upon breathing contaminated air and through contact with the skin. Cesium is readily absorbed across the brush border of the intestines in a manner similar to potassium and most is eventually excreted through the urine and feces. The biological half life of Cesium in humans ranges from 15 days in infants to 100-150 days in adults. Target organs of Cesium toxicity are the liver, intestine, heart, and kidneys. Physiological effects of Cesium include ventricular arrhythmias and displacement of potassium from muscle and erythrocytes. Cesium can have significant effects on both the central and peripheral nervous

systems. Cesium may cause epileptic seizures because it can share the same receptor as the inhibitory neurotransmitter amino acid glycine. Cesium can interfere with active ion transport by blocking potassium channels and also can interfere with lipid metabolism. Cesium may modify plasma membrane integrity, alter cytoplasmic components and cause cell damage. It is unlikely that children or adults would be exposed to enough 133 Cesium to experience any health effects that could be related to the stable cesium itself. Animals given very large doses of cesium compounds have shown changes in behavior, such as increased activity or decreased activity, but it is highly unlikely that a human would be exposed to enough stable cesium to cause similar effects. Cesium is not used extensively in industry but some uses are in the production of photoelectric cells, vacuum tubes, spectrographic instruments, scintillation counters and various optical and detecting devices. In biochemistry, cesium chloride is used to extract DNA from cells. The isotope 137 Cesium is used in radiation therapy for certain types of cancer. Other medical uses of Cesium are monitoring left ventricular function with 137 Cesium iodide probes and monitoring pulmonary (lung) endothelial permeability with 137 Cesium iodide crystal mini-detectors. It is emphasized that cesium measured is 133 Cesium, not 137 Cesium. Environmental contamination by 137 Cesium as a result of radioactive fallout could be a major concern, however, little data is available on this matter.

Blood testing is not an accurate indicators of tissue levels of Cesium.

Gadolinium (Gd)

Gadolinium is one of the most abundant ”rare-earth” elements but is never found as a free element in nature. Gadolinium has no known biological role in humans. Toxicity due to Gadolinium is rare due to its poor gastrointestinal absorption (it is suspected that very little Gadolinium is absorbed from the gastrointestinal tract (<0.05%), similar to other rare earth metals) and there is no information on the tissue distribution of Gadolinium. Most likely Gadolinium is excreted slowly through the fecal and urinary routes. If exposure to high enough doses and/or if absorption does occur,

symptoms of acute parenteral toxicity may develop, including abdominal cramps, diarrhea, lethargy, muscular spasms, and even eventual death due to respiratory collapse. Gadolinium salts can cause irritation of the skin and eyes and are suspected to be possible carcinogens. As reported by Perazella (2009) Gadollinium-based contrast (GBC) agents have been linked on occasion with a rare systemic fibrosing condition called nephrogenic systemic fibrosis (NSF) and their use in patients with advanced kidney disease should be avoided. Gadolinium is often used in alloys, improving the workability and resistance of metals (e.g. chromium, iron). Other technical uses include the phosphors of color cathode-ray television tubes and in making magnets and electronic components such as recording heads for video recorders and in the manufacture of compact disks and computer memory. In medicine Gadolinium, chelated with diethylenetriaminepentaacetic acid (DTPA), is used as a non-radioactive contrasting agent in magnetic resonance imaging (MRI) and has a half life in blood of about 90 minutes. It is also used in control rods for nuclear reactors and power plants, in making garnets for microwave applications. In vitro evidence suggests that EDTA may effectively bind to Gadolinium therefore EDTA would be a good choice as a chelator for Gadolinium.

Lead (Pb)

Sources of lead include: old lead-pigment paints, lead acid batteries, industrial smelting and alloying, some types of solders, ayruvedic herbs, some toys and products from China, glazes on (foreign) ceramics, leaded (antiknock compound) fuels, bullets and fishing sinkers, artist paints with lead pigments, and leaded joints in some municipal water systems. Most lead contamination occurs via oral ingestion of contaminated food or water or by children mouthing or eating lead containing substances. The degree of absorption of oral lead depends upon stomach contents (empty stomach increases uptake) and upon the body's mineral status. Deficiency of zinc, calcium or iron may increase lead uptake. Transdermal (skin) exposure is slight. Inhalation has decreased significantly with almost universal use of non-leaded automobile fuel. Lead accumulates extensively in bone and inhibits formation of

heme and hemoglobin in erythroid precursor cells (blood cells). Bone lead can be stored in bones for many years and is released to soft tissues with bone remodeling that can be accelerated with growth, menopausal hormonal changes and osteoporosis. Lead has physiological and pathological effects on body tissues that may be manifested from relatively low lead levels up to acutely toxic levels.In children, developmental disorders and behavior problems may occur at relatively low levels such as: loss of IQ, hearing loss, and poor growth. In order of occurrence with increasing lead concentration, the following can occur: impaired vitamin D metabolism, initial effects on erythrocyte and erythroid precursor cell enzymology, increased erythrocyte protoporphyrin, headache, decreased nerve conduction velocity, metallic taste, loss of appetite, constipation, poor blood hemoglobin synthesis, colic, frank anemia, tremors, nephrotoxic effects with impaired kidney excretion of uric acid, neuropathy and encephalopathy (altered brain function and structure. It is caused by diffuse brain disease). At relatively low levels, lead can participate in synergistic toxicity with other toxic elements (e.g. cadmium, mercury).

Mercury (Hg)

Presentation of symptoms associated with excessive mercury can depend on many factors: the chemical form of absorbed Hg and its transport in body tissues, presence of other synergistic toxics (lead and cadmium have such effects), presence of disease that depletes or inactivates lymphocytes or is immunosuppressive, organ levels of xenobiotic chemicals and sulfhydryl-bearing metabolites (e.g. glutathione), and the concentration of protective nutrients, (e.g. zinc, selenium, vitamin E). Early signs of mercury contamination include: decreased senses of touch, hearing, vision and taste, metallic taste in mouth, fatigue or lack of physical endurance, and increased salivation. Symptoms may progress with moderate or chronic exposure to include: anorexia, numbness and paresthesias, headaches, hypertension, irritability and excitability, and immune suppression, possibly immune system dysfunction. Advanced disease processes from mercury toxicity include: tremors and incoordination, anemia,psychoses, manic behaviors, possibly autoimmune disorders, kidney dysfunction or failure. Mercury is

commonly used in: dental amalgams, vaccines, explosive detonators; in pure liquid form for thermometers, barometers, and laboratory equipment; batteries and electrodes (”calomel”); and in fungicides and pesticides. The fungicide/pesticide use of mercury has declined due to environmental concerns, but mercury residues persist from past use.Methylmercury, the common, poisonous form, occurs by methylation in aquatic biota or sediments (both freshwater and ocean sediments). Methylmercury accumulates in aquatic animals and fish and is concentrated up the food chain reaching high concentrations in large fish and predatory birds. Except for fish, the human intake of dietary mercury is negligible unless the food is contaminated with one of the previously listed forms/sources. A daily diet of fish can cause 1 to 10 micrograms of mercury/day to be ingested, with about three-quarters of this (typically) as methylmercury. Depending upon body burden and upon type, duration and dosage of detoxifying agents, elevated urine mercury may occur after administration of: DMPS, DMSA, D-penicillamine, or EDTA. Elemental analysis of hair can be a secondary corroborating test for mercury burden. Blood and especially blood cell analyses are only useful for diagnosing very recent or ongoing organic (methyl) mercury exposure.

Nickel (Ni)

Urinary excretion of nickel bound to cysteinyl or thiol compounds (such as glutathione) or to amino acids (histidine, aspartic acid, arginine) is the predominant mode of excretion. With the exception of specific occupational exposures, most absorbed nickel comes from food or drink, and intakes can vary by factors exceeding 100 depending upon geographical location, food type, and water supply. Depending upon chemical form and physiological factors, from 1 to 10% of dietary nickel may be absorbed from the gastrointestinal tract into the blood. Urine reflects recent exposure to nickel and may vary widely in nickel content from day to day due to the above factors. Sources of nickel are numerous and include the following.

Cigarettes (2 to 6 mcg Nickel per average cigarette)

Diesel exhaust (particulates may contain up to 10 mg/gram Nickel)

Foods, especially: cocoa, chocolate, soya products, nuts, and hydrogenated oils

Nickel-cadmium batteries

Nonprecious, semiprecious dental materials

Nickel-containing prostheses

Electroplating, plated objects, costume jewelry

Pigments (usually for ceramics or glass)

Catalyst materials (for hydrogenation processes in the food, petroleum and petrochemical industries)

Arc welding

Nickel refining and metallurgical processes

Most clinically observed nickel contaminations are manifested as dermatoses – contact dermatitis and atopic dermatitis. However, Nickel hypersensitizes the immune system causing hyperallergenic responses to many different substances. Because Nickel can displace zinc from binding sites on enzymes, it can have inhibiting or activating effects on such enzymes. Nickel sensitivity is observed to be three to five times more frequent in women than in men. Other laboratory tests or clinical findings that would be indicative of Nickel excess are; hair element analysis, presentation of multiple allergic sensitivities, dermatitis, positive patch test for ”Ni allergy”, proteinuria, hyperaminoaciduria (by 24-hour urine amino acid analysis). Detoxification treatments with administration of EDTA or sulfhydryl agents (DMPS, DMSA, D-penicillamine) may increase urine nickel levels depending upon: body burden and mobility in tissues, duration of treatment, dosage and other factors.

Platinum (Pt)

Platinum is a nonessential element that can be found at elevated concentrations in urine with excessive exposure. Industrial workers

exposed to Platinum showed higher concentrations in the blood and urine (> 2 µg Platinum/24 hours) in comparison to non-exposed workers. Platinum is poorly absorbed in the gut but may be absorbed via inhalation. Since it is a relatively rare element, most Platinum exposures are of occupational origin. In recent years, there may have been a slight increase in environmental Platinum due to the use of Platinum as a catalyst in automobile exhaust converters. Platinum is a byproduct of copper refining and used as an alloy in dental and orthopedic materials. Symptoms of excess exposure to Platinum include: dermatitis, irritation of mucus membranes, shortness of breath and wheezing (for inhaled Platinum dusts or salts), development of chronic allergic reactions ("platinosis"), nephrosis, and immune system suppression (from Platinum diamine salts). Platinum containing drugs, such as cisplatin and carboplatin, are used as chemotherapeutic agents. Such drugs are extremely toxic and cause nephrotoxicity with associated magnesium wasting and hypomagnesemia (low magnesium), myelosuppression, inner ear toxicity, and neurotoxicity. Urinary Platinum can be significantly elevated for patients that have received the Platinum containing chemotherapeutic agents.

Thallium (Tl)

Thallium can be assimilated transdermally (through the skin), by inhalation, or by oral ingestion. Both valence states can have harmful effects: Thallium+1 may displace potassium from binding sites and influences enzyme activities; Thallium+3 affects RNA and protein synthesis. Thallium leaves blood plasma rapidly and is readily transported between body organs and tissues. It can be deposited in kidneys, pancreas, spleen, liver, lungs, muscles, neurons and brain. Blood is not a reliable indicator of thallium status. Symptoms of thallium contamination are often delayed. Early signs of chronic, low-level contamination may include: mental confusion, fatigue, and peripheral neurological signs: tingling sensations, muscle aches, tremors and ataxia (loss of voluntary muscle control, resulting in lack of balance and coordination). After 3 to 4 weeks, diffuse hair loss with sparing of pubic and body hair

and a decreased density of eye- brows usually occurs. Increased salivation occurs less commonly. Longer term or residual symptoms may include: hair loss, ataxia, tremor, memory loss, weight loss, protein in the urine, and possibly psychoses. Ophthalmologic neuritis and strabismus may be presented. Environmental and occupational sources of thallium include: contaminated drinking water, airborne plumes or waste streams from lead and zinc smelters, photoelectric, electrochemical and electronic components (photoelectric cells, semiconductors, infrared detectors, switches), pigments and paints, colored glass and synthetic gem manufacture, and industrial catalysts used in some polymer chemistry processes. Hair (pubic or scalp) element analysis is an excellent corroborative test for suspected thallium excess. Although urine is the primary natural route for excretion of thallium, the fecal route also contributes. Therefore, fecal metals analysis provides a confirmatory test for exposure to, and excretion of thallium. Other clinical findings that would be consistent are: albuminuria, EEG with diffuse abnormalities, hypertension, and elevated urine creatinine phosphokinase (CPK).

Thorium (Th)

Because most thorium salts are excreted via urine, a high urine thorium level indicates exposure and probably increased body burden of this element. Thorium is considered mildly toxic for two reasons, low-level radioactivity and slight biochemical toxicity. Thorium is a radioactive element having 7 isotopes with half lives that exceed one hour. Thorium 232 constitutes 99% of the naturally occurring thorium and this is the isotope measured. Thorium 232 has a halflife of 1.4x10 to the tenth years. It decays by alpha emission to produce radon, Radon 228. In turn Radon 228 (half life 6.7 years) decays to other radioactive isotopes, eventually reaching lead. This radioactive decay process produces alpha, beta and gamma emissions. Several decades ago, a thoria (Thorium O2) suspension (”Thorotrast”) was used diagnostically as a radiopaque agent. After a long period of latency, an unusually high proportion of individuals who received this procedure have developed leukemias, granulomas, and malignant liver tumors. These are slowly-developed diseases often with 20-30 year periods before

onset or definite diagnosis. The biochemical effects of thorium are mild. Reactive thorium salts at high levels may inhibit amylase and phosphatase enzymes. Most orally ingested thorium, if not excreted in urine, binds to bone tissue where it has a long biological half-life (years). There is a literature report for abnormal lymphocytes in animals following a thorium challenge. Thorium has about the same abundance in the earth as does lead and is encountered in mining activities for titanium and rare earth elements. Commercially, thorium is used in incandescent gas lantern mantles, refractory materials (thorium melts at 3300 degrees C), and as a coating for tungsten in electronic applications. It is present in nuclear fuels (Uranium 235 decays to Thorium 231). Thorium may also be present in tungsten-inert-gas (”TIG”) welding electrodes.

Tin (Sn)

Urine accounts for at least 80% of excreted Tin that is ingested and absorbed from the gastrointestinal tract. Ingested Tin is not significantly absorbed if it is an inorganic form. Oxide coatings readily form on metallic Tin, and salts can quickly oxidize making them insoluble. Organic Tin, however, is bioavailable and more readily absorbed. Some organic Tin compounds such as short-chain alkyltins can be absorbed transdermally and can cause degeneration of myelin. Food and drink usually provide small daily intakes of (nontoxic) Tin, with amounts depending upon type of food, packaging, quality of drinking water and water piping materials. Total daily intake is expected to vary from about 0.1 to 15 milligrams. Tin is present in many metal alloys and solders; bronze, brass and pewter contain the element. Dyes, pigments and bleaching agents often contain Tin. Anticorrosion plating of steel and electrical components may also use Tin. ”Tin cans” are Tin-plated steel with a thin outer oxide layer allowing the surface to be shiny but inert. Modern food-containing cans usually have polymer coatings that prevent food-metal contact. In the past some toothpastes contained stannous fluoride, a soluble fluoride source for strengthening tooth enamel. Currently most brands of fluoridated toothpastes contain sodium fluoride. Organic Tins, the usually toxic forms, are: biocides triphenyltin and alkyltins) used against rodents, fungi, insects and mites; curing agents for rubbers

and silicones (dialkyltin); and methyltin formed bacteriologically (similar to methylmercury). Mildly elevated levels of Tin in urine may reflect sporadic dietary intake and excretion; there may be no associated symptoms. A two- or three-fold increase in urine Tin levels is not uncommon following administration of EDTA or with sulfhydryl agents (DMSA, D-penicillamine, DMPS). Early signs of chronic organic Tin excess can be: reduced sense of smell, headaches, fatigue and muscle aches, ataxia and vertigo. Hyperglycemia and glucosuria are reported. Also, for organic Tin exposure, there can be irritation of contacted tissues (eyes, skin, bronchial tubes, or GI tract). Later, immune dysfunction may occur with reduced lymphocytes and leukocytes; mild anemia may occur. Tin is commonly elevated in urine from autistic patients following administration of EDTA, DMSA or DMPS.

Uranium (Ur)

Uranium is a radioactive element having 10 isotopes with half lives that exceed one hour. Uranium 238 constitutes about 99% of the naturally-occurring Uranium and this is the isotope measured. Uraunium 238 has a half life of 4.5 X 10 to the ninth years. Uranium 238 decays by alpha emission to produce thorium, Thorium 234, the initial step in a decay chain that eventually leads to lead. Alpha, beta and gamma emissions occur during this decay process. Because of the very long half life, the radioactivity danger is only slight. However, exposure to enriched or nuclear fuel grade Uranium (high in Uranium 235) does pose a health hazard. The measured result (Uranium 238) does not reflect or imply exposure to enriched Uranium. The major concern for (natural) Uranium excess is chemical toxicity rather than radiological. Uranium is a chemically-reactive element, has four valences (3,4,5 or 6), and may combine with: carbonate, phosphate, citrate, pyruvate, malate, lactate, etc. in body tissues. When not excreted in urine, Uranium may accumulate in the kidneys, spleen, liver, and in bone (substituting for calcium in hydroxyapatite). Uranium is nephrotoxic, causing damage to the glomeruli and proximal tubules of the kidneys. An early sign of Uranium excess is general fatigue. Kidney damage is reflected by excess protein, amino acids, or glucose in the urine. Albuminuria and urinary catalase are findings consistent with

Uranium excess. Elevated hair Uranium is a confirmatory finding; whole blood and fecal analyses may corroborate recent or ongoing exposures. Uranium is more common than mercury, silver or cadmium in the earth’s rock strata, and may be present, at low levels, in ground (drinking) water. Most commercial use of Uranium is for nuclear fuel, but it may be present in ceramics or colored glass, especially ancient or antique, yellow-colored glass.