Hair Toxics
Interpretation Guide

Content

Introduction

Hair is an excretory tissue that concentrates potentially toxic elements. In general, the amount of an element that is irreversibly incorporated into growing hair is proportional to the level of the element that has been circulating in blood. Therefore, the Hair Toxic Element Profile provides a screening test for EXPOSURE to potentially toxic elements such as methyl mercury, arsenic, lead, and cadmium.

The Hair Toxic Element Exposure Profile considers the relative toxicity per gram element and the relative frequency of occurrence of exposure to the elements. The reported elements are listed in descending order of importance in accordance with guidelines provided by the U.S. Centers for Disease Control and Prevention. Any metal found at levels equal to or exceeding the reference value (95th percentile) will generate interpretive text for that element in the body of the report that follows.

All screening tests have limitations that must be taken into consideration. The correlation between hair element levels and physiological disorders is determined by numerous factors. Individual variability and compensatory mechanisms are major factors that affect the relationship between the distribution of elements in hair and symptoms and pathological conditions. It is also very important to keep in mind that scalp hair is vulnerable to external contamination of elements by exposure to hair treatments and products. Likewise, some hair treatments (e.g. permanent solutions, dyes, and bleach) can strip hair of endogenously acquired elements and result in false low values. Careful consideration of the limitations must be made in the interpretation of results of hair analysis. The data provided should be considered in conjunction with symptomology, occupation, diet analysis and lifestyle, physical examination and the results of other analytical laboratory tests.

Caution: The contents of this report are not intended to be diagnostic and the physician using this information is cautioned against treatment based solely on the results of this screening test.

Aluminum

The Aluminum (Al) level in hair is a reliable indicator of assimilation of this element, provided that hair preparations have not added exogenous Al. Al is a nonessential element that can be toxic if excessively assimilated into cells.

Excess Al can inhibit the formation of alpha-keto glutarate and result in toxic levels of ammonia in tissues. Al can bind to phosphorylated bases on DNA and disrupt protein synthesis and catabolism. Al excess should be considered when symptoms of presenile dementia or Alzheimer's disease are observed. Hair Al is often elevated in children and adults with behavioral/learning disorders such as ADD, ADHD, and autism. Individuals with renal problems or on renal dialysis may have elevated Al.

Al is one of the most abundant metallic elements and due to its light weight, tensile strength and corrosion-resistant oxide coat, it is utilized in a wide variety of industrial and household applications (packing materials, containers, kitchen utensils, automobile and airplane components, and building materials). Commercial Al alloys commonly include copper, manganese, zinc, silicon, and magnesium. Inorganic aluminum compounds are found in drinking water, skin tanning solutions, cosmetics, mordants and coagulating agents. Al is used as a catalyst in the production of marble cement, concrete, and in the paper and enamel industries. Organoaluminum compounds are utilized to adjust the viscosity of varnishes, to impregnate textiles, and for antitransparents is cosmetics. Other sources of Al include antacids, baking powder, process cheese and other foods, and some vaccines. Analyses performed at DDI indicate extremely high levels of Al in the majority of "colloidal mineral" products.

Al has neurotoxic effects at high levels, but low levels of accumulation may not elicit immediate symptoms. Early symptoms of Al burden may include: fatigue, headache, and symptoms of phosphate depletion.

A post-Desferrioxamine or EDTA urine elements test can be used to corroborate Al exposure.

Al can be effectively complexed and excreted with silicon (J. Environ. Pathol. Toxicol. Oncol., 13(3):205-7, 1994). A complex of malic acid and Mg has been reported to be quite effective in lowering Al levels (DDI clients), and appears to be very effective in the treatment of fibromyalgia.

Antimony

Hair is a preferred tissue for analysis of Antimony (Sb) exposure and body burden. Elevated hair Sb levels have been noted as long as a year after exposure.

Sb is a nonessential element that is chemically similar to arsenic, but Sb compounds are generally less toxic than arsenic. Food and smoking are the usual sources of Sb. Thus cigarette smoke can externally contaminate hair, as well as contribute to uptake via inhalation. Gunpowder (ammunition) often contains Sb. Firearm enthusiasts often have elevated levels of Sb in hair. Other possible sources are textile industry (fire resistant fabrics), metal alloys, and some antihelminthic and antoprotozoic drugs. Sb is also used in the manufacture of paints, glass, ceramics, solder, batteries, bearing metals and semiconductors, rubberized goods and plastic.

Confirming a report from New Zealand, analysis performed at DDI revealed high levels of Sb and arsenic in sheepskin bedding designed for an infants crib. In addition, studies performed at DDI identified elevated levels of Sb in the hair of HAZMAT fire fighters who were previously wearing outdated flame retardant under garments.

Like arsenic, Sb has a high affinity for sulfhydryl groups on many enzymes. Sb is conjugated with glutathione and excreted in urine and feces. Therefore, excessive exposure to Sb has the potential to deplete intracellular glutathione pools.

Early signs of Sb excess include: fatigue, muscle weakness, myopathy, nausea, low back pain, headache, and metallic taste. Cardiac tissue Sb levels have been reported to be extremely high in patients with idiopathic dilated cardiomyopathy. Later symptoms include hemolytic anemia, myoglobinuria, hematuria and renal failure. Transdermal absorption can lead to "antimony spots" which resemble chicken pox. Respiratory tissue irritation may result from inhalation of

Sb particles or dust. Elevated levels of Sb in scalp hair are common in patients with

ADD/ADHD and autism. The clinical significance or physiological mechanism for increased uptake/retention of Sb in ADD/ADHD and autism are not known at this time.

Sb burden can be confirmed by urine elements analysis. Comparison of Sb levels pre and post provocation (DMPS, DMSA) permit differentiation between recent uptake and body stores.

Arsenic

In general, hair provides a good estimate of exposure to Arsenic (As). However, hair can be contaminated externally with As from air, water, dust, shampoos and soap. Inorganic As, and some organic As compounds, can cause toxicity. Some research suggests that As may be essential at extremely low levels but its function is not understood. Inorganic As accumulates in hair, nails, skin, thyroid gland, bone and the gastrointestinal tract. Organic As is rapidly excreted in the urine.

Common sources of As are insecticides (calcium and lead arsenate), drinking water, smog, shellfish (arsenobetaine), exterior wood preservatives, combustion of fossil fuel, copper smelting, and industrial exposure, particularly in the manufacture of electronic components (gallium arsenide).

As can cause malaise, muscle weakness, vomiting, diarrhea, dermatitis (hyperpigmentation), skin cancer, and peripheral neuropathies. As is a major biological antagonist to selenium.

As burden can be confirmed by urine elements analysis. Comparison of urine As levels pre and post provocation (DMPS, DMSA, D-penicillamine) permit differentiation between recent uptake and long-term accumulation in the body.

Barium

Hair may be used for biological monitoring of Barium (Ba). Exogenous contamination has been observed from bath water containing Ba. Elevated levels of hair Ba are often observed as a result of exposure to Ba for diagnostic medical tests.

Ba has not been established to be an essential element. Elevated levels of Ba may interfere with calcium metabolism and potassium retention. Acutely high intakes of soluble Ba salts (nitrates, sulfides, chlorides) can be toxic. Chronic exposure to Ba may be manifested by muscular and myocardial stimulation, tingling in the extremities, and loss of tendon reflexes. Due to its high density, Ba is utilized to absorb radiation and is utilized in concrete shields around nuclear reactors and in plaster to line x-ray rooms. The main use of Ba in medicine is as a contrast medium. Long-term retention of Ba can occur and granuloma of the traverse colon has been reported after diagnostic use of barium sulfate. Crystalline Ba titanate is a ceramic compound which is used in capacitors and transducers. Ba is also used to produce pigments in paints and decorative glass. Soluble Ba compounds are highly toxic and may be used as insecticides. Ba- aluminates are utilized for water purification, acceleration of concrete solidification, production of synthetic zeolites, and in the paper and enamel industries.

A confirmatory test for elevated Ba is measurement of blood electrolytes; hypokalemia may be associated with elevated Ba.

Beryllium

Beryllium (Be) has been found in the hair, but documentation correlating exposure, tissue levels and hair levels is lacking. Therefore Be is measured in hair primarily for investigational purposes. Be can be toxic to humans and animals. Be is a biological antagonist of magnesium. Be has a long-term effect of inducing abnormal activity in T lymphocytes, causing immune dysregulation and hypersensitivity reactions and chronic beryllium disease. The disease is characterized as a granulomatous intenstitial pneumonitis with lymphocyte infiltration and pulmonary fibrosis. In animals, Be has been shown to induce rickets and to damage liver, kidney, lungs, and skin. Be is poorly absorbed in the gastrointestinal tract but is readily absorbed across the skin and lungs. Inhalation is the primary route of exposure to Be and chronic uptake results in dyspnea, cough and pulmonary distress. It appears that once inhalation of Be occurs, it can never be completely eliminated.

Possible sources of Be are: electronic components, metal alloys used in aircraft and aerospace applications (especially aluminum-copper-beryllium alloys), bearing sleeves, optical lens coatings, and some phosphors in fluorescent lights. Tobacco contains Be, and smoking immediately increases the Be levels in the blood and urine.

Confirming tests for Be exposure are urine and fecal elements analyses. Oral administration of DMPS and DMSA may

significantly increase fecal levels of Be. Be is slowly excreted in urine and may be found elevated many months after the exposure. An intravenous DMPS challenge can be used to enhance urinary excretion of Be.

Bismuth

No published studies correlate Bismuth (Bi) exposure with hair Bi levels, therefore, hair Bi levels are measured primarily for investigational purposes. However, observations at DDI clearly indicate elevated Bi in hair for patients that use Bi containing antacids or have been given Bi prior to EPD therapy.

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. Organobismuth compounds are extremely toxic and may be used in some bactericidal and fungicidal applications. Other less toxic forms of Bi are found in 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, irritation of mucosal membranes 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). In the same study, EDTA increased brain Bi levels.

Cadmium

Hair Cadmium (Cd) levels provide an excellent indication of mild to moderate body burden. Very high exposure and assimilation of Cd destroys the hair follicle. Cd is a toxic heavy metal that has no metabolic function in the body. Moderately high Cd exposure may be associated with hypertension, while very severe Cd toxicity may cause hypotension. Cd adversely affects the kidneys, lungs, testes, arterial walls, and bones and interferes with many enzymatic reactions. Chronic Cd excess can lead to microcytic, hypochromic anemia and proteinuria with excretion of beta-2-microglobin, and functional zinc deficiency. Cd excess is also commonly associated with fatigue, weight loss, osteomalacia, and lumbar pain.

Occupationally, inhalation of Cd is the primary route of exposure. Otherwise the most significant source of exposure is due to contaminated food and water. Cd occurs at relatively high levels in human biosolids that are used as fertilizer. Cd absorption is reduced by zinc, calcium, and selenium. Oral absorption of Cd is generally higher in females than in males due to differences in iron stores. Cd is found in varying amounts in foods, from .04 æg/g for some fruits to 3-5 æg/g in some oysters and anchovies. Refined carbohydrates have very little zinc in relation to Cd. Cigarette smoking significantly increases Cd intake. Other sources of Cd include drinking water, fungicides, rubber products (tires), welding rods, and silver solders, and interestingly, old metal refrigerator shelves that have been utilized as grills for outdoor cooking. Cadmium toxicity is common among welders and construction workers (cement dust).

If hair zinc is not abnormal, external contamination may have caused the elevated hair Cd level.

A confirming test for elevated body burden of Cd is urine analysis following administration of appropriate chelating

agents: EDTA, DMPS. Excretion of Cd via the feces is about 90%; therefore, fecal Cd levels are useful as indication of oral Cd intake and an approximation of assimilation. Serum alkaline phosphatase activity is commonly elevated with cadmium toxicity.

Cesium

This individuals urine Cesium (Cs) level is higher than expected, reflecting exosure to Cs but symptoms may not be evident. 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 Cs133; the radioactive isotopes 134Cs and 137Cs are not measured or reported by Doctor's Data. Natural deposits of Cs ores occur in Main, South Dakota and

Manitoba (Bernic Lake), Canada. Cesium may bio-accumulate in aquatic food chains; higher levels of cesium have been found in Pacific deep-sea fish and local shellfish since the 2011 Fukoshima reactor accident. Cesium may be used in high-density drilling fluids (oil and gas industry) and may contaminate local water and vegetation; Cs has been found in cow's milk. Cesium may occur naturally in mineral waters; one study analyzed the Cs concentration in 163 mineral and thermal waters and found the level ranged from 4.5 to 148 µg per liter.

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 Cs in humans ranges from 15 days in infants to 100-150 days in adults.

The cesium-137 isotope is used in cancer treatments, for ventricular function and pulmonary imaging studies, industrial radiology, and for food and instrument sterilization; Cs137 agents may contain small amounts of Cs133. Non-radioactive cesium chloride may be advertised on the internet as "high pH therapy." Currently there is no support in the scientific literature for that purpose as advertised. Radioactive Cs isotopes may contaminate soil at nuclear waste sites. Cesium may be used in industry for the production of photoelectric cells, vacuum tubes, spectrographic instruments, scintillation counters, DNA biochemistry, in various optical or detecting devices.

Target organs of potential toxic effects of Cs are the liver, intestine, heart, and kidneys. Physiological effects of excessive Cs include ventricular arrhythmias and displacement of potassium from muscle cells 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 excitatory bioamine glycine. Cesium can interfere with active ion transport by blocking potassium channels and also can interfere with lipid metabolism. Excessive Cs may modify plasma membrane integrity, alter cytoplasmic components and cause cytogenetic damage.

It is unlikely that children or adults would be exposed to enough Cs133 to experience any health effects that could be related to the stable Cs itself. Animals given very large doses of Cs compounds have shown changes in behavior, such as increased activity or decreased activity, but it is unlikely that a human would be exposed to enough stable Cs to cause similar effects.

The isotope Cs137 is used in radiation therapy for certain types of cancer. Other medical uses of Cs are monitoring left ventricular function with Cs137 iodide probes and monitoring pulmonary endothelial permeability with Cs137 iodide crystal mini-detectors. Again, it is emphasized that Cs measured at Doctor's Datais Cs133, not Cs137. Environmental contamination by Cs137 as a result of radioactive fallout could be a concern. Exposure to Cs may be assessed by hair elemental analysis.

Commonly used chelating agents are not effective binders of Cs.

Resources:

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Chromium

A high hair Chromium (Cr) level is likely to indicate excess exposure to Cr. Hair Cr levels do not appear to be affected by permanent solutions, dyes, or bleaches, but external contamination is possible. Trivalent Cr is considered to be an essential trace element with a low order of toxicity.

Cr toxicity via oral ingestion is not likely. However, it is noteworthy that self-supplementation has been reported to be associated with insomnia and increased unpleasant dream activity in some individuals (J. Nutr. Med.; 3(43), 1992). Phytates decrease oral assimilation of Cr+3, whereas nicotinic acid and vitamin C increase absorption of Cr+3, zinc, vanadium and iron compete with Cr for absorption.

In contrast, hexavalent Cr compounds are considerably more toxic and are primarily absorbed via inhalation as a result of industrial exposure. Industrial exposure to high amounts of Cr has been reported to be associated with allergic dermatitis, skin ulcers, bronchitis, and lung and nasal carcinoma. Elevated hair Cr levels have also been observed in patients with cerebral thrombosis and cerebral hemorrhage.

Sources of exposure to hexavalent Cr include: manufacture and use of ferrochromium and stainless steel, chromium plating (plumbing, electrical appliances, automotive parts), welding, commercial spray painting, wood finishing and leather tanning industries, and handling of cement. Extensive mining of Cr and disposal of spent ore presents a serious environmental problem in certain regions.

Tests to confirm excess exposure to Cr include analysis of Cr in plasma (trivalent) versus packed red blood cells (hexavalent); both analyses are more indicative of recent exposure than of body burden. A urine elements analysis

will confirm recent exposure and serum hyaluronidase activity is reported to be elevated with excessive exposure to Cr.

Cobalt

Hair may be used for monitoring excess exposure to Cobalt (Co). However, hair is occasionally contaminated by external Co from some hair treatments. If an individual's hair has been treated with permanent solutions, dyes, or bleach, the Co levels may not be indicative of body Co accumulation.

Humans absorb Co both as inorganic Co and as vitamin B-12; the body pools of each fluctuate independently. Humans cannot convert inorganic Co to vitamin B-12, and vitamin B-12 provides the only documented function of Co in humans. Thus, a high hair Co level does not mean that vitamin B-12 levels are high or even adequate.

Co is utilized in the manufacture of metal alloys with high melting point, strength and resistance to oxidation. Alloys with chromium, nickel, molybdenum, and copper are utilized in the electrical, aeronautical, and automobile industries. Co salts and oxides are used in the glass industry, for paints and colored pottery. They are also used as fertilizers, catalysts in chemical reactions and in some dental amalgams. Co salts are not longer utilized as foam stabilizers in the brewing of malt beverages due to cardiovascular diseases that the metal induced.

The primary clinical manifestations of chronic Co toxicity include weight loss, pulmonary syndrome, allergy, nausea, cardiomyopathy, electrolyte imbalance, hematological disorders, and thyroid lesion.

The dietary content of Co is highly variable and depends upon types of foods eaten, geographical location, and type of soil. Toxicity has been noted with ingestion of 250-400 mg/day. Recent animal studies indicate that excess Co can cause marked impairment of myocardial metabolism that results in metabolic acidosis (Clin. Chem.; 43(6): 5192, 1997).

Post-EDTA urine elements analysis can be utilized to further assess Co status in the body.Hair analysis cannot be used to assess vitamin B-12 status. Appropriate tests for determination of B-12 status are the measurement of urine levels of methylmalonic acid (elevated with B-12 coenzyme deficiency/dysfunction), quantitative blood assay for vitamin B-12, and urine amino acids analysis (several metabolic steps require vitamin B-12).

Copper

An elevated level of copper (Cu) in hair may be indicative of excess Cu in the body. However, it is important first to rule out exogenous contamination sources: permanent solutions, dyes, bleaches, swimming pool/hot tub water (very common), and washing hair in acidic water carried through Cu pipes. In the case of contamination from hair treatments, other elements (aluminum, silver, nickel, titanium) may also be elevated.

Copper is used extensively in sanitation and in the production of kitchen utensils, and thermal and electric conductors. Copper solutions are used in industrial processes such as electroplating, printed circuit production, textile production, and as catalysts in chemical processes. Albeit reduced, Cu-sulfate is sometimes used in agriculture (vineyards, orchards). Other sources of Cu exposure include contaminated food or drinking water, and excessive Cu supplementation, particularly in combination with low intake of zinc or molybdenum. Insufficient intake of competitively absorbed elements such as zinc or molybdenum can lead to, or worsen Cu excess. Cu toxicity significantly compromises zinc homeostasis.

Medical conditions that may be associated with excess Cu include: biliary obstruction (reduced ability to excrete Cu), liver disease (hepatitis or cirrhosis), and renal dysfunction. Symptoms associated with excess Cu accumulation

are muscle and joint pain, depression, irritability, tremor, hemolytic anemia, learning disabilities, and behavioral disorders.

Confirmatory tests for Cu excess are a comparison of Cu in pre- vs. post-provocation (D- penicillamine, DMPS) urine elements tests and a serum, whole blood or blood cell elements analysis. Fecal Cu levels can be measured as indication of exposure and approximation of assimilation.

Germanium

The relationship between the levels of Ge in hair and other tissues has not been established and there is currently no published documentation linking elevated hair Ge levels to Ge toxicity. However, recent observations at DDI indicate that hair Ge levels are increased by supplementation/therapeutic use of Ge compounds.

Ge is generally considered to be a nonessential element that has chemical properties similar to that of silicon. Pure organic Ge compounds (e.g. carboxyethyl Ge sesquioxide, lactate/citrate salts of Ge) of relatively low toxicity and have been used experimentally as anti-viral agents and immunostimulants. However, studies in humans are not extensive or conclusive. Inorganic Ge could to be nephrotoxic and neurotoxic if present in excess.

Ge is utilized in the manufacture of transistors, diodes, and fluorescent lamps. It is also utilized in the preparation of metal alloys and in medical imaging devices. Silver-copper-Ge alloys are used in some dental restorations materials. Low concentrations of Ge are found in foods.

Gold

Normal levels of gold (Au) reported in hair are < 0.15 æg/g. Analysis at DDI indicates that 95% of hair samples have Au levels of < 0.2 æg/g. Levels are clearly elevated in patients receiving gold for therapeutic reasons. Dietary gold intake is estimated at below

7 æg/day. Intestinal and dermal absorption is reported to be poor with exception to oral therapeutic Auranofin which is lipid soluble (25% absorbed). Maximal absorption is obtained with aurothiomalate (intamuscular).

Gold (Au) poisoning is not common and occurs mainly as a result of long-term gold therapy. Acute toxic effects of Au include severe diarrhea, nausea, vomiting, gastritis, colitis, erythema, and severe exfoliative dermatitis. The therapeutic use of Au in rheumatoid arthritis should be carefully monitored. Adverse effects of Au therapy include blood disorders (leukopenia, agranulocytosis, aplastic anemia, thrombocytopenia and pancytopenia) and vasomotor reactions (weakness, dizziness, nausea, vomiting, sweating, and facial flushing). Gold may illicit autoimmune responses/diseases in a significant percentage of patients. Nephrotic syndrome/immune complex glomerulonephritis, similar to penicillamine-induced nephropathy, has been reported in association with Au therapy. Liver toxicity, including cholestatic jaundice and hepatitis, may also occur.

Major uses of Au are in dentistry, jewelry, coin and utensil making. Gold is also used in electrical contact and plating materials. In medicine, due to its anti-inflammatory and immunological activities, Au is used in the treatment of rheumatoid arthritis (injectible aurothiomalate acid or aurothioglucose or oral auranofin). In cancer treatment, gold complexes have been used as an experimental cytotoxic/antitumor drug.

In gold therapy, levels are often monitored in hair. DMPS is used for the chelation of Au. In laboratory animals DMPS increased the urinary and fecal excretion of gold, and decreased the levels of gold in the liver, kidneys and skin.

Lead

Generally, hair is an excellent indicator of the body burden of lead (Pb). However, elevated levels of Pb in head hair are occasionally an artifact of hair darkening agents, or dyes, e.g. lead acetate. Although these agents can cause exogenous contamination, some transdermal absorption can contribute to body burden. When scalp hair is contaminated with such products, hair Pb levels are typically extremely high.

Pb has neurotoxic and nephrotoxic effects and interferes with heme biosynthesis. Pb may also affect the body's ability to utilize the essential elements calcium, magnesium, and zinc. At moderate levels of body burden, Pb may have adverse effects on memory, cognitive function, nerve conduction, and metabolism of vitamin D. Pb is transported through the placenta to the fetus and is found in human breast milk. Children with hair Pb levels greater than 1 æg/g have been reported to have a higher incidence of hyperactivity than those with less than 1 æg/g. Children with hair Pb levels above 3 æg/g have been reported to have more learning problems than those with less than 3 æg/g. Hair lead levels are commonly elevated is association with ADD/ADHD. Detoxification therapy by means of chelation results in transient increases in hair lead. Eventually, the hair Pb level will normalize after detoxification is complete.

Symptoms associated with excell Pb are somewhat nonspecific, but include: anemia, immune dysregulation, headaches, gastric distress, fatigue, weight loss, cognitive dysfunction, decreased coordination, depression, anxiety, and aberrant behavior.

Occupation exposure to Pb occurs in the fields of mining, refineries, and the production of storage batteries, ammunition, solder, building materials, cable sheeting and foils. Other sources of exposure to Pb include: welding, old leaded paint (chips/dust), drinking water, some fertilizers, industrial pollution, lead-glazed pottery, newsprint, and some candles. A recent study indicates that silicofluoridation of water enhances the assimilation of Pb in humans.

Confirmatory tests for Pb excess are: urine elements analysis following provocation with intravenous EDTA, DMPS, or oral DMSA. Whole blood analysis only reflects recent or ongoing exposures and may not correlate with total body burden. Fecal lead analysis provides an excellent indication of dietary exposure and an approximation of assimilation. Increased blood or urine protoporphyrins is a finding consistent with Pb excess, but may occur with other toxic elements as well.

Manganese

Hair Manganese (Mn) levels generally reflect actual body stores, and external contamination can influence hair Mn. Since particulate manganese-containing dust is the most common source of Mn toxicity, hair is considered to be an excellent tissue for the assessment of Mn exposure. However, high hair Mn can be an artifact of contamination from hair treatments/products or well water (containing high Mn). These possibilities should definitely be considered and ruled out before proceeding with therapies to alleviate an apparent excess Mn.

Mn is an essential element which is involved in the activation of many important enzymes. However, Mn excess is postulated to result in glutathionyl radical formation, reduction of the free glutathione pool, and increased exposure of adrenal catecholamines (e.g. dopamine) to free radical damage. Excess Mn causes degeneration of myelin pigmented dopaminergic neurons which results in abnormally low levels of serotonin and dopamine in the brain. This is hypothesized to be a reason behind the neurotoxic effects attributed to Mn overload.

The brain is particularly affected by Mn excess. Symptoms or conditions consistent with excessive Mn include: lethargy, disorientation, memory loss, anxiety, emotional instability, and bipolar-like behaviors (laughing and crying),

aberrant or violent behaviors, and tremor or Parkinson-like symptoms.

Occupationally, the greatest sources of exposure to Mn dust and fumes occurs in mining of the element, and in the production and fabrication of iron and steel. In addition, various Mn compounds are widely used in fertilizers, animal feeds, pharmaceutical products, dyes, paint dryers, catalysts, wood preservatives, and ceramic production. Mn is also an air pollutant derived from the gasoline additive MMT.

Other sources of exposure include contaminated teas, contaminated drinking water, some street drugs, and smoking. Conditions predisposing to Mn excess are: iron or calcium deficiency, chronic infection, and impaired liver function. Mn excess is occasionally associated with alcoholism (hepatic dysfunction), and biliary cirrhosis/ obstruction.

A confirmatory test for Mn excess is packed red blood cell elements analysis. Dithiol chelators are relatively weak with regard to enhancing Mn excretion.

Mercury

Mercury (Hg) is toxic to humans and animals. The accumulation of Hg in the body is generally reflected by the hair Hg levels, but hair Hg levels can occasionally be high in association with the use of certain hair dyes and sprays.

The concentration of Hg in hair is typically 200-300 times greater than that in blood. Organic Hg, such as methylmercury derived from fish, is incorporated into hair at a much higher rate than is inorganic Hg (dental amalgams). Therefore, very elevated levels of hair Hg are most often associated with high end fish consumption or occupational exposure. As a result of DDI experience in a large multi-center trial of Hg detoxification, it is apparent that hair Hg may be deceptively low or even nondetectable in some individuals who do not have adequate endogenous detoxification capacity. Apparently, such individuals are unable to efficiently mobilize/excrete Hg.

Individuals vary greatly in sensitivity and tolerance to Hg exposure. At hair levels below 3 æg/g, Hg can suppress biological selenium function and may cause or contribute to immune dysregulation. Hallmark symptoms of excess Hg include: loss of appetite, decreased senses of touch, hearing, and vision, fatigue, depression, emotional instability, peripheral numbness and tremors, poor memory and cognitive dysfunction, and neuromuscular disorders. Hair Hg has been reported to correlate with fish consumption and acute myocardial infarction. On average each 1 æg/g of hair Hg was found to correlate with a 9% increase in AMI risk (Circulation 1995; 91:645-655).

Sources of Hg include dental amalgams, fish, contaminated water supplies, some hemorrhoidal preparations, some vaccines, skin lightening agents, instruments (thermometers, electrodes, batteries), combustion of fossil fuels and hospital wastes, some fertilizers, and the paper/pulp and gold industries. After dental amalgams are installed or removed a transient (several months) increase in hair Hg is often observed. Also, "baseline" hair Hg levels for individuals with dental amalgams are higher (about 1 to 2 æg/g) than are baseline levels for those without (below 1 æg/g).

Confirmatory tests for elevated Hg are measurement of Hg in packed red blood cells as an indication of recent/ ongoing exposure (does not correlate with whole body accumulation), fecal mercury levels, and measurement of urine Hg following use of a dithiol chelating or mobilizing agent such as DMPS/DMSA (an indication of total body burden). Greater than 90% of Hg is naturally excreted into the feces via the biliary route.

Nickel

Hair is a reasonable tissue for monitoring accumulated body stores of Nickel (Ni). However, hair is OFTEN

contaminated with Ni from hair treatments, dyes, and hair products.

There is substantial evidence that Ni is an essential element which is required in extremely low amounts. However, excess Ni has been well established to be nephrotoxic, and carcinogenic. Elevated Ni is often found in individuals who work in the electronic and plating, mining, and steel manufacture industries. A cigarette typically contains from 2 to 6 mcg of Ni; Ni is absorbed more efficiently in the lungs (~35%) than in the gastrointestinal tract (~5%). Symptoms of chronic Ni exposure include dermatitis, chronic rhinitis, and hypersensitivity reactions. Ni can hypersensitize the immune system, subsequently causing hyperallergenic responses to many different substances.

Symptoms of Ni toxicity are dermatitis and pulmonary inflammation (following exposure to Ni dust, smoke). Long term or chronic Ni toxicity may lead to liver necrosis and carcinoma.

A confirmatory test for elevated Ni is the measurement of urine Ni before and after administration of complexing agents that mobilize Ni (e.g., DMPS, EDTA).

Palladium

The relationship between the levels of Palladium (Pd) in hair and exposure to the metal has yet to be determined. Pd compounds are rarely encountered by most people. Palladium compounds should be regarded as toxic and carcinogenic. There have been numerous reports of allergic contact dermatitis to Pd. The main contact sources are jewelry and dental materials (gold alloys).

The characteristics of Pd (ductile, malleable, resistant to corrosion, easily fused and welded) make it an acceptable material for jewelry making and dentistry. Palladium is used in the field of communications in facing electrical contacts in automatic switch gear. The nonmagnetic springs in clocks and watches as well as special coatings for mirrors are also made of Pd. The chemicals industry uses Pd as a catalyst. Palladium is often found associated with platinum in Australia, Brazil, Russia, Ethiopia, and North and South America, as well as with nickel and copper deposits in Canada and South Africa.

Palladium was formerly used as a treatment for tuberculosis (0.065 g per day). DMPS, like other chelating agents studied, demonstrate no effects on mortality of mice with acute Pd poisoning.

Platinum

Platinum (Pt) is a nonessential element that is sometimes detected in hair. However, the clinical significance of hair Pt has not been well studied. Hair treatments may contribute to artifactual contamination of scalp hair.

Pt is poorly absorbed in the gut but may be absorbed via inhalation. Since it is a relatively rare element, most Pt exposures are of occupational origin. In recent years, there may have been a slight increase in environmental Pt due to the use of Pt as a catalyst in automobile exhaust converters. Pt is a byproduct of copper refining and used as an alloy in dental and orthopedic materials. Symptoms excess exposure to Pt include: dermatitis, irritation of mucus membranes, dyspnea and wheezing (for inhaled Pt dusts or salts), development of chronic allergic reactions ("platinosis"), nephrosis, and immunosuppression (from Pt diamine salts).

Pt containing drugs, such as cisplatin and carboplatin, are used as chemotherpeutic agents. Such drugs are extremely toxic and cause nephrotoxicity with associated magnesium wasting and hypomagnesemia, myelosupression, ototoxicity, and neurotoxicity. Hair Pt levels will be very high in individuals exposed to the mentioned chemotherapeutic agents. It has been reported that DMPS did not decrease kidney Pt concentrations in

animals that had previously been treated with cisplatin.

Selenium

Selenium (Se) is a nutritionally essential element, but it is also one of the most toxic essential elements when exposure is excessive (Modern Nutrition in Health and Disease, ed. Shils, Olson and Shike, 1994). Barring exogenous contamination (which is frequent for Se), hair Se levels accurately reflect tissue levels. Hair is subject to considerable Se contamination by some anti- dandruff shampoos.

Elevated hair Se is almost always from external contamination. One exposure to a Se containing anti-dandruff shampoo will elevate the Se levels. Similarly, sulfur containing shampoos are often contaminated with Se and can also cause artifactually high hair Se levels.

Se toxicity usually results from industrial exposure. Se is utilized in the electronics and related industries. Se is utilized in xerography, semiconductors, photoelectric cells and infrared optic materials. Se is also utilized in the manufacture of pink and red glasses, and for head-resistant red pigments in plastics, enamels, paints and inks. Se can be toxic when ingested at levels exceeding 15 times that needed for essential functions.

Se toxicity (selenosis) can result in: interference with the metabolism of sulfur-bearing amino acids, structural changes and red pigmentation in hair and nails, garlic breath, metallic taste in the mouth, discoloration of teeth and skin, gastroenteritis, and neurological disorders. Some Se compounds have been reported to have carcinogenic activity and promote the carcinogenic effects of certain chemicals.

Confirmatory tests for Se excess are measurements of Se level in whole blood or packed red blood cells and urine. For urine Se measurement, diagnostic provocation with oral doses of N- acetylcysteine, glutathione, or DMSA may mobilize tissue Se and increase excretion (Toxicology of Metals, ed. Chang, 1996).

Silver

Hair Silver (Ag) levels have been found to reflect environmental exposure to the element. However, hair is commonly contaminated with Ag from hair treatments such as permanents, dyes, and bleaches.

Ag is not an essential element and is of relatively low toxicity. However, some Ag salts are very toxic.

Sources of Ag include seafood, metal and chemical processing industries, photographic processes, jewelry making (especially soldering), effluents from coal fired power plants and colloidal silver products.

The bacteriostatic properties of Ag have been long recognized and Ag has been used extensively for medicinal purposes; particularly in the treatment of burns. There is much controversy over the long term safety of consumption of colloidal silver. Very high intake of colloidal silver has been reported to give rise to tumors in the liver and spleen of animals (Metals in Clinical and Analytical Chemistry, eds. Seiler, Segel and Segel, 1994). However, these data may not have relevance to the effects of chronic, low level consumption by humans.

Dithiol chelators effectively bind Ag and DMPS increased survival in rats that received injections of silver nitrate. However, there are currently no labs that can accurately measure Ag in urine due to technical difficulties.

Tellurium

Little is known about the toxicity of Tellurium (Te) and the relationship between the levels of Tellurium (Te) in hair and total body accumulation. There have been reports of humans occupationally exposed to as little as 0.01 mg/m3 of air or less developing "tellurium breath", which has a garlic-like odor.

Te deposits are found in Mexico, South America, Western Australia, Ontario, Canada, and in the US (small rock deposits are found in Colorado and California), however Te rarely occurs in its native state. Te is usually associated with gold, silver, copper, lead, mercury, and bismuth. Commercial sources of Te are found as by products in lead and copper refineries. The alloying of Te with copper and stainless steel improves the machinability of metals (improves strength/hardness, decreases corrosive action of sulfuric acid on lead). Other uses of Te include the manufacturing of rectifiers, thermoelectric devices, semi-conductor research, and anti-knock compounds for gas. Te is also responsible for the blue color in glass. Colloidal Te is used as an insecticide, germicide, and fungicide.

Thallium

Thallium (Tl) is a highly toxic element which, like lead and mercury, accumulates in many body tissues. Hair levels reflect chronic accumulation of Tl, but alopecia occurs about two weeks after ACUTE Tl poisoning.

Thallium occurs naturally in some minerals, and magmatic and sedimentary rock, consequently in soil, water, and air. Industrially, Tl is used in lenses and prisms, as an alloy with mercury in low temperature thermometers, and in the preparation of high density liquids. Rodenticides and pesticides (thallium sulfate) are a major source of exposure to Tl.

Other sources of Tl are: foods (marine organisms concentrate Tl up to 700 times), tobacco, contaminated water, electronic components, fly ash, cement dust, and some fertilizers. Tl is rapidly and completely absorbed when ingested, inhaled or brought into contact with skin.

Symptoms of Tl excess include: sleep disturbances, and cardiac, optical, dermatatological, liver, GI, and kidney dysfunctions. Albuminuria and alopecia are consistent with Tl excess. Potassium, selenium and sulfhydryl compounds (e.g. glutathione) diminish Tl retention and toxicity. Tl toxicity can have a long latency period before clinical symptoms become apparent. Tl inhibits Na/K ATPase and thereby disrupts intracellular K homeostasis.

Tl is primarily excreted in the urine and feces. Fecal Tl and cesium excretion appear to be enhanced by DMSA and oral Prussian blue (potassium ferric cyanoferrate II) as a results of inhibition of enterohepatic resorption (Toxicology of Metals, ed. Chang, 1996). EDTA and BAL have been reported to be ineffective for chelation of Tl.

Thorium

Correlations among the levels of Thorium (Th) in hair and other tissues have yet to be established. However, recent studies performed at DDI found significantly elevated levels of Th in hair in sub-populations in India where levels of Th are naturally high in the environment. This a radioactive element with 13 known isotopes; 232Th constitutes 99% of the naturally- occurring Th. 232Th is the isotope measured at DDI and reported for this sample. 232Th has a half-life of 1.410 years. It decays by alpha-emission to produce radon, 228Ra. In turn 228Ra (half-life 6.7 years) decays to other radioactive isotopes, and eventually converts to lead. This radioactive decay process produces alpha, beta, and gamma emissions.

This considered mildly toxic for two reasons, the low-level radioactivity and slight biochemical toxicity. Th salts at high levels may inhibit amylase and phosphatase enzymes. Most orally ingested Th, if not excreted in urine, binds to bone tissue where it has a long biological half-life (years).

Th has about the same abundance in the earth as does lead and is encountered in mining for titanium and rare

earth elements. Commercially, Th is used in incandescent gas lantern mantles, refractory materials, and as a coating for tungsten in electronic applications. Th may also be present in tungsten-inert-gas (TIG) welding electrodes.

A urine or fecal elements analysis can be performed to assess further the extent of exposure to Th.

Tin

Hair Tin (Sn) levels have been found to correlate with environmental exposure. Depending on chemical form, Sn is a potentially toxic element. Inorganic Sn has a low degree of toxicity, while organic Sn has appreciable toxicity.

Inorganic Sn is used as flame-proofing treatment in textiles, as a wood preservative, and has various uses in the glass industry. Sn is also used in tin plate electrolysis for Sn alloy coatings. Stannous fluoride is found in some toothpastes and stannous octanoate is a catalyst utilized in the production of flexible polyurethane foam.

Food is a common source of Sn. Other possible sources are: dental amalgams, cosmetics, preservatives, food and beverage containers, pewter, bronze, and anticorrosive platings. Symptoms of excess Sn include: skin, eye, and GI tract irritation, muscle weakness, anemia, and neurodegenerative disease (demyelinization).

A confirmatory test for excessive accumulation of Sn is the measurement of Sn in urine before and after provocation with DMPS, DMSA or EDTA. Urinary Sn is often high post DMSA/DMPS in autistic patients. Dietary exposure to Sn can be assessed by fecal elements analysis.

Titanium

Titanium (Ti) is measured in hair to assist in the identification of external contamination of hair by treatments and products. Shampoos, dyes, and "highlighting" are the primary sources of Ti, which binds tenaciously to hair.

Ti dioxide is the most common form of Ti used as a whitening agent (toothpaste, conditioners, shampoos, etc.). It is also used for industrial purposes; e.g., coating of welding rods and as white pigment in paints, dyes, and paper fillers. Ti dioxide and other Ti containing compounds have extremely low toxicity. The elevated level of Ti in the hair sample is most likely without clinical significance. Do consider surgical or dental implants as a possible source of elevated Ti in hair.

Tungsten

The relationship between the levels of Tungsten (W) in hair and exposure/body burden has yet to be established. W has no known biological role. Long-term chronic exposures have been associated with lung disease (pneumoconiosis or "hard metal lung disease") and lung cancer. Skin contact with W may produce contact eczema, pruritis, folliculitis, and neurodermatitis. Tungsten appears to have an antagonistic relationship to Mo decreasing hepatic Mo concentration and reducing the effectiveness of sulfite and xanthine oxidases.

Tungsten is a silvery-white lustrous element usually obtained as a grey powder and is mainly utilized as tungsten carbide in metallurgy, mining and petroleum industries. Calcium and magnesium tungstates are widely used in as filaments for electric lamps, electron tubes and television tubes. Since Tungsten has the highest melting point of all metals it is used for high-speed and hot-worked steels. Other sources of W include catalysts and reagents in biological analysis, fire and waterproof materials, and industrial lubrifications.

Acute environmental exposures have been detected in hair (4.26 æg/g) up to two months after ingestion of a

tungsten containing beverage. Other limited data suggests mean values of .015 æg/g in pubic hair for nonexposed persons and 5.2 æg/g in pubic hair for exposed persons. Intestinal absorption of tungsten is rapid and seemingly significant. W is rapidly transported to the blood and then to the kidneys for filtration and eventual excretion from the body. Pulmonary absorption of W-tungstic oxide has been studied in dogs. 60% of W is rapidly deposited in the respiratory tract and 33% of that fraction reaches systemic circulation. Tungsten is also easily transferred from mother to fetus, usually later in gestation.

Confirmatory tests for W accumulation and exposure, respectively, are (DMPS/DMSA) urine provocation or Fecal Elements testing.

Uranium

The levels of Uranium (U) in hair usually reflect levels of U in other tissues. However, hair may be externally contaminated by shampoos or hair products that contain U.

U is a nonessential element that is very abundant in rock, particularly granite, lignite, monazite sands, and phosphate rocks. U is present at widely varying levels in drinking water, root vegetables, and present in high phosphate fertilizers. Other sources of U include: ceramics, some colored glass, many household products and tailings from U mines. Spent U rods have been milled into armor piercing bullets and missile heads.

Uranyl cations bind tenaciously to protein, phosphate, nucleotides, and bone, where it substitutes for Ca. Published data are sparse, but there appears to be a correlation between U exposure, nephrotoxicity and cancer. Kidney and bone are the primary sites of U accumulation.

All isotopes of U are radioactive; 238U is the most abundant and lowest energy emitter. It is important to note that the measured result, which is 238U, does NOT necessarily indicate or imply exposure to highly enriched 235U, which is used in nuclear power and weaponry.

Chronic fatigue is often reported in association with hair U levels >0.5 æg/g (DDI observations). U is rapidly cleared from blood and deposited in tissues. Currently, DTPA is the only effective chelating agent for ACUTE U poisoning. However, it must be administered immediately and is not effective once U has transferred from blood to tissues. Currently there are no available chelators or complexing agents that have been established to be effective for ameliorating U retention associated with long-term, low-level exposure to U.

Urine or fecal elements analysis can be performed to confirm recent or ongoing exposure to U. Because U is such a potent nephrotoxin, one might test for urinary wasting of amino acids and low molecular weight proteins (B-2-microglobulin) in patients with markedly elevated hair U levels.

Vanadium

High levels of Vanadium (V) in hair may be indicative of excess absorption of the element. It is well established that excess V can have toxic effects in humans depending on the chemical form. Although it appears that V may have essential functions, over zealous supplementation is not warranted.

Excess levels of V in the body can also result from chronic consumption of fish, shrimp, crabs, and oysters derived from water near offshore oil rigs. Industrial/environmental sources of V include: processing of mineral ores, phosphate fertilizers, combustion of oil and coal, production of steel, and chemicals used in the fixation of dyes and print (Metals in Clinical and Analytical Chemistry, 1994). V is used in producing rust-resistant, spring and high speed tool steels. Vanadium pentoxide and other vanadates are used as catalysts in the production of sulfuric acid and

formaldehyde. Urban airsheds in industrialized areas have been reported to have high levels of V.

Symptoms of V toxicity vary with chemical form and route of absorption. Inhalation of excess V may produce respiratory irritation and bronchitis. Excess ingestion of V can result in decreased appetite, depressed growth, diarrhea/gastrointestinal disturbances, nephrotoxic and hematotoxic effects. Pallor, diarrhea, and green tongue are early signs of excess V and have been reported in human subjects consuming about 20 mg V/day (Modern Nutrition in Health and Disease, 8th edition, eds. Shils, M., Olson, J., and Mosha, S., 1994).

Confirmatory tests for excess V are red blood cell elements analysis, and urine V which reflects recent intake. EDTA but not DMPS, is an effective chelator of V.

Zinc

A high level of zinc (Zn) in hair may be indicative of low Zn in cells, and functional Zn deficiency. Zn can be displaced from proteins such as intracellular metallothionein by other metals, particularly cadmium, lead, copper, and mercury (Toxicology of Metals, 1994), resulting in paradoxically elevated hair Zn. Zn may also be high in hair as a result of the use of Zn- containing anti-dandruff shampoo. Rough or dry, flaky skin is a symptom of Zn deficiency, so it is not uncommon for Zn deficient patients to use an anti-dandruff shampoo. A result of high hair Zn warrants further testing to assess Zn status.

Zn is an essential element that is required in many very important biological processes. However, Zn can be toxic if exposure is excessive. Although uncommon, high hair Zn might be indicative of Zn overload which could result from Zn contaminated water (galvanized pipes), welding or gross, chronic over-supplementation (100 mg/day). Other sources of Zn exposure include: manufacture of brass, bronze, white paint, pesticide production, galvanization of steel and iron products, dry cell batteries, and use in rubber, textile, and ceramic industries. Symptoms of Zn excess include: gastrointestinal disorders, decreased heme synthesis (copper deficiency), tachycardia, blurred vision, and hypothermia.

Confirmatory tests for Zn status are whole blood or packed red blood cell elements analysis, and serum ceruloplasmin (low with Zn induced copper deficiency). Urinary Zn will be elevated to some extent post EDTA/DMPS even in patients who do not have Zn overload.

Nordic Laboratories

PATIENT:	**Sample Report**			TEST REF:	###-##-#####
TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy		
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
GENDER:	Female	TESTED:	dd/mm/yyyy	ADDRESS:	
AGE:	48				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Hair Elements

Toxic & Essential Elements; Hair

TOXIC METALS		RESULT µg/g	REFERENCE INTERVAL	PERCENTILE 68th – 95th
Aluminum	(Al)	5.0	< 7.0	
Antimony	(Sb)	0.011	< 0.050	
Arsenic	(As)	0.12	< 0.060	
Barium	(Ba)	0.44	< 2.0	
Beryllium	(Be)	< 0.01	< 0.020	
Bismuth	(Bi)	1.3	< 2.0	
Cadmium	(Cd)	0.029	< 0.050	
Lead	(Pb)	5.0	< 0.60	
Mercury	(Hg)	0.50	< 0.80	
Platinum	(Pt)	< 0.003	< 0.005	
Thallium	(Tl)	< 0.001	< 0.002	
Thorium	(Th)	0.001	< 0.002	
Uranium	(U)	0.029	< 0.060	
Nickel	(Ni)	0.08	< 0.30	
Silver	(Ag)	0.32	< 0.15	
Tin	(Sn)	0.23	< 0.30	
Titanium	(Ti)	0.37	< 0.70	
Total Toxic Representation				

ESSENTIAL AND OTHER ELEMENTS		RESULT µg/g	REFERENCE INTERVAL	PERCENTILE 2.5th – 16th – 50th – 84th – 97.5th
Calcium	(Ca)	224	300– 1200	
Magnesium	(Mg)	21	35– 120	
Sodium	(Na)	48	20– 250	
Potassium	(K)	24	8– 75	
Copper	(Cu)	74	11– 37	
Zinc	(Zn)	180	140– 220	
Manganese	(Mn)	0.31	0.08– 0.60	
Chromium	(Cr)	0.39	0.40– 0.65	
Vanadium	(V)	0.026	0.018– 0.065	
Molybdenum	(Mo)	0.026	0.020– 0.050	
Boron	(B)	0.24	0.25– 1.5	
Iodine	(I)	1.1	0.25– 1.8	
Lithium	(Li)	< 0.004	0.007– 0.020	
Phosphorus	(P)	157	150– 220	
Selenium	(Se)	1.3	0.55– 1.1	
Strontium	(Sr)	0.41	0.50– 7.6	
Sulfur	(S)	48900	44000– 50000	
Cobalt	(Co)	0.004	0.005– 0.040	
Iron	(Fe)	5.7	7.0– 16	
Germanium	(Ge)	0.037	0.030– 0.040	
Rubidium	(Rb)	0.013	0.007– 0.096	
Zirconium	(Zr)	0.039	0.020– 0.42	

SPECIMEN DATA

COMMENTS:

Date Collected: mm/dd/yyyy
Date Received: mm/dd/yyyy
Date Completed: mm/dd/yyyy
Methodology: ICP/MS

Sample Size: 0.200 g
Sample Type: Head
Hair Color:
Treatment:
Shampoo:

RATIOS

ELEMENTS	RATIOS	RANGE
Ca/Mg	10.7	4– 30
Ca/P	1.43	1– 12
Na/K	2	0.5– 10
Zn/Cu	2.43	4– 20
Zn/Cd	> 999	> 800

Nordic Laboratories

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