TEST REPORT

8605 SW Creekside Place
Beaverton, OR 97008
Phone: 503-466-2445 Fax: 503-466-1636

ZRT LABORATORY

 # 2018 07 01 211 BU

Ordering Provider:
Jane Getuwell, MD

Samples Received
07/09/2018

Report Date
07/12/2018

Samples Collected
Blood Spot - 07/03/18 07:40
Urine - 07/03/18 07:00
Urine - 07/03/18 22:00

Patient Name: Comprehensive Toxic & Essential Elements
Patient Phone Number: 555 555 5555

Gender	Height	Waist
Male	5 ft 9 in	36 in
DOB	**Weight**	**BMI**
4/12/1980 (38 yrs)	173 lb	25.5

TEST NAME	RESULTS | 07/03/18	RANGE
Toxic & Essential Elements - Blood		
Zinc	7.10	5.04-8.46 mg/L
Copper	0.73	0.59-1.03 mg/L
Ratio: Zn/Cu	9.7	6.6-10.8
Magnesium	45	27-49 mg/L
Selenium	297 H	116-314 µg/L
Cadmium	0.45	<1.04 µg/L
Mercury	22.92 H	<5.29 µg/L
Toxic & Essential Elements - Urine		
Iodine	112	100-380 µg/g Cr
Bromine	738	700-4800 µg/g Cr
Selenium	59	34-220 µg/g Cr
Lithium	15	10-218 µg/g Cr
Arsenic	65 H	<42 µg/g Cr
Cadmium	0.04	<0.72 µg/g Cr
Mercury	1.49	<1.58 µg/g Cr
Creatinine	2.47 H	0.3-2.0 mg/mL

<dL = Less than the detectable limit of the lab. N/A = Not applicable; 1 or more values used in this calculation is less than the detectable limit. H = High. L = Low.

Therapies

10mg oral DHEA (OTC) (1 Days Last Used)10mg oral Pregnenolone (OTC) (1 Days Last Used)

The above results and comments are for informational purposes only and are not to be construed as medical advice. Please consult your healthcare practitioner for diagnosis and treatment.

David T. Zava, Ph.D.
Laboratory Director

Alison McAllister, ND.
(Ordering Provider unless otherwise specified on page 1)

Disclaimer: Symptom Categories below show percent of symptoms self-reported by the patient compared to total available symptoms for each category. For detailed information on category breakdowns, go to www.zrtlab.com/patient-symptoms.

SYMPTOM CATEGORIES	RESULTS | 07/03/18
Estrogen / Progesterone Deficiency	0%
Estrogen Dominance / Progesterone Deficiency	3%
Low Androgens (DHEA/Testosterone)	5%
High Androgens (DHEA/Testosterone)	8%
Low Cortisol	5%
High Cortisol	2%
Hypometabolism	10%
Metabolic Syndrome	6%

SYMPTOM CHECKLIST	MILD	MODERATE	SEVERE
Acne			
Aggressive Behavior			
Allergies			
Anxious			
Apathy			
Blood Pressure High			
Blood Pressure Low			
Blood Sugar Low			
Body Temperature Cold			
Bone Loss			
Burned Out Feeling			
Chemical Sensitivity			
Cholesterol High			
Constipation			
Depressed			
Dizzy Spells			
Erections Decreased			
Fatigue - Evening			
Fatigue - Mental			
Fatigue - Morning			
Flexibility Decreased			
Forgetfulness Increased			
Goiter			
Hair - Dry or Brittle			
Hair or Skin Oily			
Headaches			
Hearing Loss			
Heart Palpitations			
Hoarseness			
Hot Flashes			
Infertility			
Irritable			
Joint Pain Increased			
Libido Decreased			
Mental Sharpness Decreased			
Muscle Size Decreased			
Muscle Soreness			
Nails Breaking or Brittle			
Neck or Back Pain			
Nervous			
Night Sweats			

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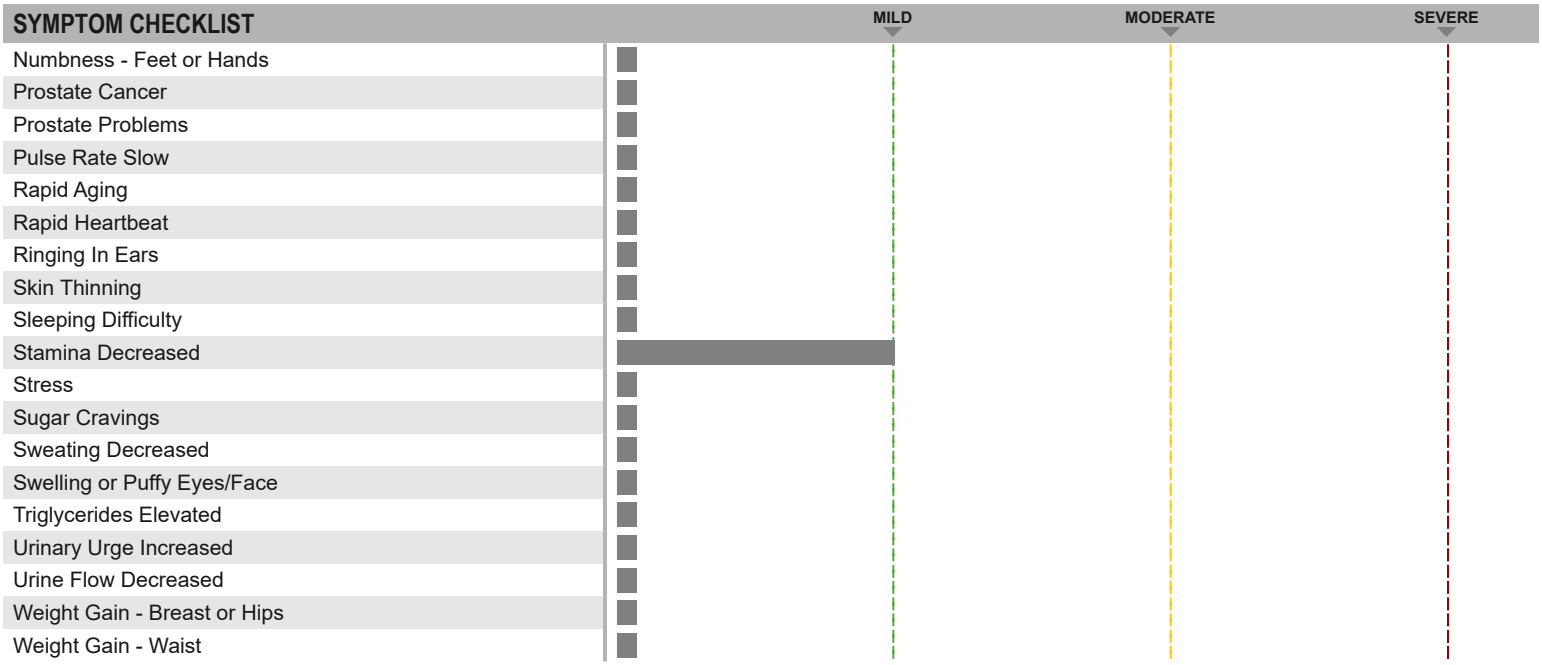

Lab Comments

ZINC

Whole blood zinc is within normal reference range.

Zinc is an essential element that is a co-factor in over 300 enzymes, and is required for cell growth and division, DNA synthesis, wound healing, taste, immune and thyroid function, blood clotting, reproduction, tissue growth, prevention of oxidative damage, and many other catalytic, structural and regulatory functions. Proper zinc nutrition has been shown to reduce the absorption of lead and prevent kidney damage caused by cadmium. Generally, zinc absorption is greater when animal protein intake (e.g., eggs, beef, cheese) is high because released amino acids help to keep zinc in solution allowing optimal absorption. Phytates (present primarily in legumes and whole grains) chelate zinc and inhibit its absorption. Vegetarians and vegans, who consume elevated levels of plant-based phytates and low levels of animal proteins in foods, are more likely to be zinc deficient and often require more supplemental zinc in their diet. Alcohol consumption can also prevent zinc absorption due to reduced uptake and increased urinary excretion.

The current RDA for zinc is 8 mg/day for women and 11 mg/day for men while requirements are lower for children and higher during lactation or pregnancy. Zinc should always be well balanced with copper (see below). The primary sources of dietary zinc are red meat and poultry, with other good sources being oysters, beans, nuts, seafood, whole grains, fortified cereals, and dairy products.

For more information, you can find a review of zinc and the zinc/copper balance at: http://www.omicsonline.org/copper-and-zinc-biological-role-and-significance-of-copper-zincimbalance-2161-0495.S3-001.pdf

COPPER

Whole blood copper is within normal reference range.

Copper is an essential element required for antioxidant defense, immune function, neuron formation, iron metabolism, and as a cofactor of critical enzymes and proteins. The body contains around 100 mg copper, with the highest concentrations in the brain and liver. Copper absorption occurs primarily in the small intestine and stomach where a high pH causes copper to break apart from dietary macromolecules. In the bloodstream copper is transported by albumin and transcuperin to the liver where it binds to the copper binding protein ceruloplasmin. Adrenal hormones promote ceruloplasmin production, so liver and adrenal gland dysfunction can cause copper to accumulate in tissues and organs. Typically, copper homeostasis is well maintained and toxicity is prevented via biliary excretion.

The current RDA for copper is 0.9 mg/day for both men and women, although an argument has been made for a higher intake of 2.3 mg/day. Common sources of dietary copper include animal products, legumes, grains, and vegetables. Copper water pipes, cookware, drinking water, birth control, fungicides, and dietary supplements are all potential sources of copper exposure. Drinking water contributes about 6-13% of the average daily intake of Copper. Most diets contain enough copper (1-5 mg) to prevent a deficiency.

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MAGNESIUM

Whole blood magnesium is within normal reference range.

Magnesium is an essential element and co-factor in approximately 600 enzyme systems. It is required for protein synthesis, reproduction, DNA and RNA synthesis, cellular energy production and storage, muscle and nerve function, blood glucose control, blood pressure regulation, along with many other vital bodily functions. Significant evidence shows that magnesium intake is inversely associated with the risk of stroke. The human body contains between 21-28 g of magnesium; approximately 53% is in bone, 27% in muscle, 19% in soft tissues, 0.5% in erythrocytes, and 0.3% in serum. After oral intake, around 40-50% of dietary magnesium is absorbed in the small intestine. Dietary intake of calcium, phosphate, and potassium can competitively inhibit gut absorption of magnesium. It is estimated that 60% of Americans do not consume the daily recommended amount of magnesium, with the elderly the most vulnerable population due to decreased gut absorption and renal excretion. Magnesium homeostasis is primarily controlled by the kidney, aiding in prevention of deficiency or toxicity.

The current recommended dietary allowance (RDA) for magnesium is 420 mg/day for men and 320 mg/day for women in adults. Magnesium content of soil has decreased 20-30% over the last 60 years, and it is estimated that 80-90% of magnesium is lost during food processing of whole grains. Foods highest in magnesium are whole grains, nuts, legumes, potatoes, and dark leafy vegetable.

For an excellent and easy-to-read online mini-review on magnesium published in April 2016 in Food & Nutrition please search: Magnesium: The Missing Mineral? by Julia Greenwald Jay.

For online reviews on magnesium please see:
https://ods.od.nih.gov/factsheets/Magnesium-HealthProfessional/
http://advances.nutrition.org/content/4/3/378S.long
http://physrev.physiology.org/content/95/1/1.long

SELENIUM

Whole blood selenium is higher than the reference range which may be toxic. Whole blood selenium levels represent long-term exposure while urine selenium levels reflect recent intake. High selenium is usually from supplementing with selenium, but can also be from eating foods and drinking water in an area high in selenium. Symptoms of toxicity include: diarrhea, fatigue, hair loss, joint pain, nail changes, and nausea.

Selenium is an essential element that has an important role in thyroid hormone metabolism, antioxidant function (i.e. through glutathione), and redox status. Selenium supplementation has been shown to increase the effectiveness of cancer therapy or help prevent certain types of cancer such as lung, colon, bladder, and prostate. Low selenium is closely associated with thyroid diseases such as Hashimoto's thyroiditis as well as decreased conversion between T4 and T3.

Immediate discontinuation of selenium supplementation is suggested as well as discussion with a healthcare provider.
For more information, you can find a review of selenium at: http://www.nature.com/ejcn/journal/v58/n3/full/1601800a.html

CADMIUM

Whole blood cadmium is within the normal reference range, which should be considered beneficial as it indicates low recent exposure to cadmium. High-normal cadmium should be cross-checked with urinary cadmium which better reflects long-term exposure to cadmium. Cadmium bioaccumulates in the body, meaning that at birth levels are low, but by age 30 the body burden may reach toxic levels that adversely affect health. The half-life of cadmium in the kidneys is 15-30 years making urine an ideal body fluid to assess lifetime exposure to cadmium.

Cadmium is a non-essential toxic element and a kidney toxin, peripheral nerve toxin, an estrogen mimic, and a group 1 carcinogen. Elevated levels of cadmium are believed to play a role in the development of lung, prostate, breast, endometrial, testicular, kidney, bladder, pancreatic and gall bladder cancer. The major sources of cadmium exposure are from vegetables, grains, tobacco, seafood, organ meats, and root crops all which take up and accumulate cadmium from the soil. Lung absorption can be up to 50%, which is why cadmium in the urine of smokers is double that of non-smokers. Human activities and products such as mining, smelting, artisan glass manufacturing, waste disposal, fertilizer, pesticides, nickel-cadmium batteries, and vehicle exhaust all contribute to environmental and occupational cadmium exposure.

Avoiding sources of cadmium esp smoking, and maintaining adequate zinc, selenium and fiber intake can continue to prevent cadmium toxicity.
For more information, you can find a review of cadmium at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3686085/

MERCURY

Whole blood mercury is higher than the reference range.

Mercury is a potent toxin. Mercury is found in 3 basic forms in the body: elemental mercury (HgO), inorganic mercury (Hg2+), and organic mercury (MeHg). High mercury exposure can cause symptoms which include balance problems, hearing loss, speech issues, and damage to peripheral nerves (tingling sensation). If selenium and/or zinc levels are low in concert with high whole blood mercury, it is recommended that they be increased to protect against antioxidant functions. The half-life of mercury in the brain is estimated at 20 years.

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There mercury is bound strongly to sulfur and selenium groups. Metallothioneins are proteins rich in sulfur residues and upregulated by zinc intake. They preferentially bind heavy metals such as mercury and cadmium, preventing them from causing further damage. Natural sources of mercury are volcanoes, weathering of rock, oceans, soil, and burning vegetation. It is estimated that 50-75% of environmental mercury comes from human activity; with the largest sources of mercury being coal fired power plants, gold mines, and metal and cement production.

Elemental Mercury (Hg0)

There is very little absorption of elemental mercury in the GI tract, but nearly 80% is absorbed by the lungs as a vapor. Absorbed elemental mercury is oxidized to inorganic forms of mercury, but remains a vapor long enough in the blood for a significant amount to penetrate the blood-brain barrier. Sources of elemental mercury include light bulbs, mines, industrial manufacturing, dental amalgams, and thermometer production. Dental amalgams, which are 50% mercury, gas off between 2-28 µg elemental mercury/day, of which 80% is absorbed. Elimination of elemental mercury, which is converted to inorganic mercury in the body, is through urine and feces.

Inorganic Mercury (Hg2+)

Inorganic mercury can reach most organs, but primarily accumulates in the kidneys where it does the most damage. Most pharmaceutical and agricultural uses of inorganic mercury have been discontinued, but mercury chloride is still used as a pesticide and disinfectant. Essentially all mercury in urine is inorganic, whereas that in whole blood, mostly found in red blood cell membranes, is organic (e.g., methylmercury).

Organic Mercury (Methylmercury)

Methylmercury is the most common and toxic form of mercury. It is nonpolar and accumulates in fatty tissues such as the plasma membranes of red blood cells and other fatty tissues like the brain. Methylmercury is purported to be 100 times more toxic than elemental or inorganic mercury. Atmospheric elemental and inorganic mercury is converted by microorganisms in water to organic mercury, which works its way up the food chain and bioaccumulates. Fish at the top of the food chain (tuna, shark, swordfish) have the highest levels of mercury, with 95-97% present as organic mercury. Nearly all methylmercury consumed in foods such as fish is absorbed by the GI tract. Once in the blood a majority of methylmercury binds to sulfur or selenium groups, with up to 10% accumulating in the brain. Most of the toxic effects of methylmercury are on the CNS, although the immune system and kidneys are affected as well. About 95% of mercury in blood is methylmercury, with the majority residing in red blood cells. This makes whole blood an ideal matrix to evaluate methylmercury burden. The half-life of methylmercury in blood is about 50 days, so whole blood analysis represents recent and past exposure to mercury.

For more information, you can find a review of mercury at:
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3253456/
https://www3.epa.gov/ttn/atw/hlthef/mercury.html

IODINE:

Urinary iodine/creatinine falls into the reference range that is considered optimal for thyroid hormone synthesis (100-380 ug/g creatinine). The iodine test result represents an average of your urinary iodine for a single day, and is reflective of your dietary/supplement iodine consumption over the last several days. If your daily diet is representative of the day you tested, and you are not suffering from symptoms of iodine deficiency (mostly related to thyroid dysfunction-hypothyrodism, hyperthyroidism, goiter); then you are likely iodine sufficient. Even though iodine levels may be sufficient, if levels of other halogens, particularly fluorine and/or bromine, or natural goitrogens, such as soy and cruciferous vegetables, are high, these can compete with iodine or inhibit its incorporation into thyroid hormones and contribute to hypothyroid symptoms.

Natural sources of iodine are highest in seafood (fish, seaweed) with lesser amounts found in milk products and eggs. For an excellent and brief NIH-sponsored Medline review on iodine dosage recommendations and potential side effects of iodine supplementation please view: www.nlm.nih.gov/medlineplus/druginfo/natural/35.html.

BROMINE:

Bromine excretion is lower than the 5-95% reference range. Bromine has no know role in the human body, so low levels are of no consequence and only indicate very low exposure to foods, beverages, or other products containing bromine. Dietary bromine is well absorbed in the gut and is mostly excreted in urine, making urinary bromine a good indicator of bromine intake. In the United States, bromine intake from grains, nuts and fish is estimated to be 2-8mg/day. Bromine belongs to the same family of elements termed halogens, which also include iodine, chlorine, and fluorine. Because of their structural similarity with iodine, excessive levels of these other halogens like bromine, compete with iodine and block its uptake into the thyroid gland. In the presence of adequate iodine, bromine has little effect on iodine uptake and thyroid hormone synthesis; however, when iodine is low and bromine levels are elevated this can lower both iodine uptake and thyroid hormone synthesis. Bromine is present at high concentration in many different commercial products that result in significant exposure to humans (e.g., brominated vegetable oil [soft drinks], polybrominated diphenyl ether [fire retardant], sodium bromate [dough conditioner], methyl bromide [soil fumigation] and hypobromous acid [pool/spa disinfectant].

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SELENIUM:

Selenium excretion in urine is within the optimal reference range (> 50-200 µg/g creatinine) seen in regions with adequate dietary selenium intake. Intake of selenium in the U.S. has been estimated at 135 µg/day for men and 92 µg/day for women, which is consistent with the reported average urinary level of selenium in the U.S. of about 40-60 µg/g creatinine range (assuming about 50-70% of selenium ingested is excreted in urine). The RDA for selenium in adults is around 55 µg/day http://ods.od.nih.gov/factsheets/Selenium-HealthProfessional/; however, this may be insufficient in individuals with excessive oxidative stress and overexposure to environmental toxins. The therapeutic window for optimal selenium supplementation is quite narrow, with tolerable upper intake levels recommended at about 400 µg/day. Higher levels (up to 800 µg) have been used in cancer patients without significant side effects. Chronic high selenium is associated with symptoms such as hair and nail loss and brittleness. Food is the major source of selenium intake for the general population, which is highly dependent on the selenium content of the soil and water. Local foods grown in selenium-deficient soils, as found in some regions around the world, can lead to selenium deficiency. Seafood, eggs, grains, vegetables, red meat and chicken are the primary food sources of selenium. The minimum requirement is suggested to be 40 µg/day; intake lower than 11 µg/day results in selenium deficiency disorders. Around 50-70% of selenium ingested is excreted in urine, therefore the amount of selenium in urine is proportional to the amount ingested.

Selenium is an essential nutrient found in the form of a unique amino acid, selenocysteine, in over 25 different proteins involved in redox reactions associated with antioxidant enzymes, thyroid hormone synthesis, and thyroid deiodinases involved in the intracellular conversion of bio-inert thyroxine (T4) to active T3 or inactive reverse T3 in all tissues throughout the body. The antioxidant glutathione peroxidase plays an important role throughout the body in removing oxidants such as hydrogen peroxide (H202) and oxidized lipids that form during normal metabolism. In the thyroid gland glutathione peroxidase, in concert with glutathione, plays an essential role in protecting the thyroid from the strong oxidant H202, necessary for activation of iodine and synthesis of thyroid hormones T4 and T3. In this regard, selenium plays an important protective role in Hashimoto's thyroiditis, an autoimmune disease that results in persistent destruction of the thyroid gland and eventual fibrosis and hypothyroidism. Hashimoto's is strongly associated with selenium deficiency and lower intracellular levels of selenium-containing antioxidants like glutathione peroxidase and thioredoxin reductase, which are present at very high levels in cells (thyrocytes) of the thyroid gland in healthy individuals. Hashimoto's is an autoimmune disease associated with antibodies against thyroid peroxidase, the enzyme that uses H202 to activate iodine for thyroid hormone synthesis. Low levels of selenium result in less protection of the thyroid against H202. Selenium's ability to decrease thyroid antibodies in individuals with Hashimoto's thyroiditis is well documented.

Selenium is also present in the catalytic site of the 3 thyroid deiodinases that convert T4 to active T3 or rT3 in all tissues throughout the body. About half of the T3 used by the body for cellular metabolism is from direct intracellular conversion of T4 to T3, mostly by deiodinase 2. Even normal (optimal) urinary levels of selenium can be insufficient when oxidant stress is high, caused by exposure to excessive levels of environmental toxins (e.g., oxidized lipids, heavy metals, chemical pollutants). Arsenic and mercury form extremely tight complexes with selenium, effectively preventing it from incorporation into selenoproteins like glutathione peroxidase and thyroid deiodinases, thus compromising thyroid hormone formation and metabolism. This reduces the body's ability to detox oxidized lipids and optimally synthesize thyroid hormones and convert T4 to T3, essential for normal metabolic activity and creation of energy. Thus, selenium levels should be viewed in light of arsenic and mercury levels, and if these toxic metals are high more supplemental selenium may be necessary to meet the needs of the selenoproteins. This is particularly true in autoimmune diseases such as Hashimoto's thyroiditis. High exposure to arsenic and mercury and consequent reduction in selenium bioavailability in selenoproteins can be countered by selenium supplementation beyond the recommended RDA of 55 µg/day (see above).

LITHIUM:

Lithium excretion is within the normal reference range. Lithium is almost completely absorbed through the GI tract, and the majority is excreted in urine within 24 hours [Freeman et al. 2006], making urine lithium a good indicator of recent intake. Sources of lithium include well water, meat, dairy, grains and vegetables. There is no established recommended daily amount (RDA). Lithium is being researched for mood stabilization, for anxiety, memory and suicidology prevention. Lithium is dosed in low doses (OTC 1 microgram to 100mg) to pharmacologic (prescription 100mg) dosages; discuss with your healthcare provider.

ARSENIC:

Arsenic excretion is higher than the reference range (< 42 ug/g creatinine).. Results above this range indicate acute and possible chronic exposure to high levels of arsenic. Recent consumption of food products high in arsenic may cause a temporary rise in arsenic levels. Consider identifying and eliminating sources of arsenic exposure and selenium supplementation to prevent arsenic from reducing levels of selenoproteins. The most common cause of arsenic toxicity is constant exposure to contaminated drinking water from wells. The World Health Organization and Environmental Protection Agency have set a maximum level of arsenic in drinking water to 10µg/L. Even with regulations in place to limit arsenic in drinking water; private wells may contain high levels of arsenic. Food sources of arsenic include fish, shellfish, rice, fruit, beer and wine, flour, corn and wheat. Ocean fish and shellfish generally have high levels of arsenic and may cause a transient rise in urinary arsenic levels for several days. Consumption of shellfish such as lobster, which can have high levels of organic (nontoxic) arsenic, should be avoided for several days prior to urine testing. Seaweeds are unable to convert inorganic to organic arsenic, with certain species such as hijiki containing very high levels. Normal urine arsenic levels will vary from about 5-41 µg/g creatinine; Acute toxicity can occur at levels >100µg/g creatinine. Around 80% of arsenic is excreted in the urine after three days, making urine arsenic a good indicator of intake. Arsenic exists in inorganic and organic forms, with inorganic arsenic exposure being highly toxic compared to organic arsenic. It is not possible to differentiate the more toxic inorganic forms of arsenic from the less toxic organic forms in urine using inductively coupled plasma mass spectrometry alone. However, anyone with arsenic above the 5-40 ug/day range should attempt to identify and eliminate the possible source of the arsenic, which is usually well water or foods (mostly rice) grown in water contaminated by arsenic.

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Arsenic is known to disrupt over 200 enzymes in humans. Arsenic acts on the human body by inducing oxidative stress, altering DNA, suppressing and amplifying genes and causing chromosomal abnormalities. One of the principle mechanisms of arsenic toxicity is through its tight binding with selenium, effectively removing it from incorporation into selenoproteins essential as antioxidants (e.g. glutathione peroxidase and thioredoxin reductase) and thyroid deiodinases. In regions with very high levels of arsenic in well water and foods irrigated with this water (mostly rice), such as Bangladesh, arsenic toxicity is extremely problematic and closely associated with diabetes, hypertension, cardiovascular disease, vascular changes, neuropathy, memory loss and hormonal regulation modifications Human studies using selenium supplementation to combat the toxic effects of arsenic exposure have been successful. Patients in Bangladesh suffering from arsenicosis caused by contamination of their well water were treated successfully with 100 µg of selenomethionine a day for 12 months, resulting in greater reduction of hair, nail and urine arsenic levels compared to a placebo group. Similar studies in Bangladesh and Mongolia showed improvement of skin lesions in arsenicosis patients treated with selenium.

Chronic arsenic toxicity symptoms include ataxia, cognitive deficits, fatigue, muscular weakness, anorexia, jaundice, nausea, vomiting, eczema, pigmentation, keratosis, scaling, brittle nails, white lines in nails and localized subcutaneous edema. High arsenic exposure, particularly when selenium is low, is linked to cancer of the lung, prostate, bladder and skin.

CADMIUM:

Urinary cadmium is within normal reference range.

Cadmium is a toxic heavy metal that enters the body mostly through food consumption and tobacco smoke. Average cadmium intake per day is around 8-25 µg. While only about 5% of cadmium consumed orally in foods and liquids is absorbed by the gastrointestinal tract (about 1-2 ug), more than 90% is absorbed by the lungs on inhalation of cigarette smoke or polluted air. Those who smoke one pack of cigarettes per day (made from tobacco leaves) will take in an additional 1 to 3 µg.

High cadmium levels have been linked to cancers of the reproductive organs, including the breasts, prostate, and uterus. Cadmium is believed to increase cancers of estrogen-sensitive tissues by binding to and activating cellular estrogen receptors that increase gene products associated with increased cell proliferation. Like other heavy metals cadmium also increases cellular Reactive Oxygen Species (ROS), which increase DNA mutations that can lead to increased cancer risk.

Cadmium is slowly eliminated from the body with a half-life of 10-20 years. Cadmium will primarily affect the kidneys, but also damages the nervous and cardiovascular systems, liver, lungs, pancreas, bones, and reproductive organs. The adverse effects of cadmium are more pronounced when selenium and zinc levels are low; therefore, supplementation with these essential elements should be considered if they are found to be low.

MERCURY:

Mercury excretion is in the upper quartile of the reference range. Urine excretion at this level indicates relatively high mercury exposure. This may be more problematic if other heavy metals are elevaled, particularly arsenic, or selenium is low. Mercury may be present from normal environmental exposure, dental amalgams, diet or prior tissue accumulation.

Mercury is primarily excreted in urine and feces, with other routes of elimination being sweat, saliva, breast milk, and expired air. The excretion route depends primarily on whether the mercury is elemental, inorganic or organic. The most reliable determinant of long-term elemental, inorganic and organic mercury exposure is urine content due to mercury's accumulation in the kidneys, which also estimates total body burden. Urine mercury levels >10 µg/L indicates that a person has had mercury exposure, while neurological signs may be present at levels >100 µg/L. Urine mercury levels do not represent fish consumption (methylmercury).

An estimated 50-75% of environmental mercury comes from human sources. In 2000, global mercury emissions were from fossil fuel combustion (65%), gold production (11%), non-ferrous metal production (7%) and cement production (6%). Mercury can be found in common household items such as lights bulbs, thermometers, barometers, switches, medicines, paint, antiques, and cosmetics. Thimerosal, a vaccine preservative, contains 50% mercury by weight and has been used since the 1930's. The highest source of organic mercury (methylmercury) exposure in the United States is from fish, with fish tissue containing up to 95-97% of this mercury species.

The possible health effects of mercury exposure in an environmental or occupational setting depends on the form of mercury (elemental, inorganic or organic), toxicology of the form, and characteristics of the exposure (route, frequency, duration and magnitude). The principal reaction of mercury in biological systems is with sulfhydryl (-SH) and selenium groups present in the amino acids cysteine, selenocysteine and selenomethionine. Mercury inactivates sulfur and selenium containing residues in enzymes and structural proteins, a primary cause of mercury toxicity. Because mercury forms an exceptionally strong bond with selenium, it has the potential of causing thyroid dysfunction at multiple levels by reducing available glutathione peroxidase, thioredoxin, thyroid deiodinases and other selenium containing proteins. Although selenium and sulfur share similar chemical properties, selenium's binding affinity with mercury is around one million times greater than sulfur's, promoting formation of HgSe adducts.

Mercury interferes with DNA transcription and protein synthesis, resulting in destruction of endoplasmic reticulum and disappearance of ribosomes. One of the first symptoms of mercury toxicity is tremor, indicating impairment of the area of the brain involved in coordination and voluntary movements. Extended exposures to mercury can result in symptoms such as tremor, vision changes, hearing loss, gingivitis, neurocognitive or behavioral disturbances, irritability, depression, fatigue, memory loss and sleep disturbances.

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Dental amalgams contain about 50% by weight of elemental mercury. Amalgams continuously release mercury vapor which is inhaled and absorbed by the body. As much as 50% of mercury in fillings has been found to have vaporized after 5 years, and 80% by 20 years. Around 80% of mercury vapor outgassing from dental amalgams is absorbed. The number of dental amalgam surfaces has been correlated to the total mercury levels in a number of human tissues, with highest levels observed in the frontal cortex (part of the brain responsible for behavior, motor skills and problem solving). In general, patients with amalgam fillings show a small but statistically significant increase in blood and urine mercury levels; levels can increase by about 1 µg/L per 10 amalgam surfaces. The level of mercury in breast milk is significantly correlated with the number of dental amalgam fillings in the mother. Subjects with the highest level of urine mercury in a human study showed the best recovery rates from neuropsychological complaints after removing their amalgam fillings. The amount of mercury accumulated in the thyroid and pituitary is strongly associated with the number of dental amalgam surfaces. In patients that have a mercury allergy, the removal of dental amalgams resulted in significantly decreased levels of thyroid peroxidase antibody (TPOAb) and thyroid thyroglobulin antibody (TgAb).

Elemental mercury is able to cross the blood-brain and placental barriers and distribute widely in the body. The brain and kidney are particularly susceptible to the effects of elemental mercury. Elemental mercury is lipophilic and around 80% is absorbed when inhaled. Besides the brain and kidneys, elemental mercury concentrates in the liver, skin, sweat glands, pancreas, enterocytes, lungs, salivary glands, testes, thyroid and prostate, and may be associated with dysfunction in those organs. Inorganic mercury is not readily absorbed through the skin, but is water soluble and is easily absorbed after ingestion. Around 10-30% of inorganic mercury is absorbed in the GI tract. Organic mercury includes compounds in which mercury is bonded to a structure containing carbon atoms (methyl, ethyl, phenyl, or similar groups). The most common form of organic mercury encountered is methylmercury. Around 95% of methylmercury is absorbed in the GI tract. Once methylmercury enters the body, it is readily absorbed and stored, slowly demethylating to inorganic mercury which has a prolonged half-life. Concentration of methylmercury occurs in the brain, liver, kidneys, placenta, fetus (especially the fetal brain), peripheral nerves and bone marrow. Methylmercury is the most dangerous mercury species due to its stability and lipid solubility, leading to high membrane penetration in living organisms.

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