Test Results

ZRT LABORATORY
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Samples Arrived: 02/16/2017
Date Closed: 02/16/2017

Samples Collected:
Blood Spot: 02/14/17 06:00
Urine: 02/14/17 06:15
Urine: 02/14/17 22:00

Ordering Provider:

Getuwell
8605 SW Creekside Pl
Beaverton, OR 97008

Laura Leddie
123 Main Ln
Portland, OR 97207

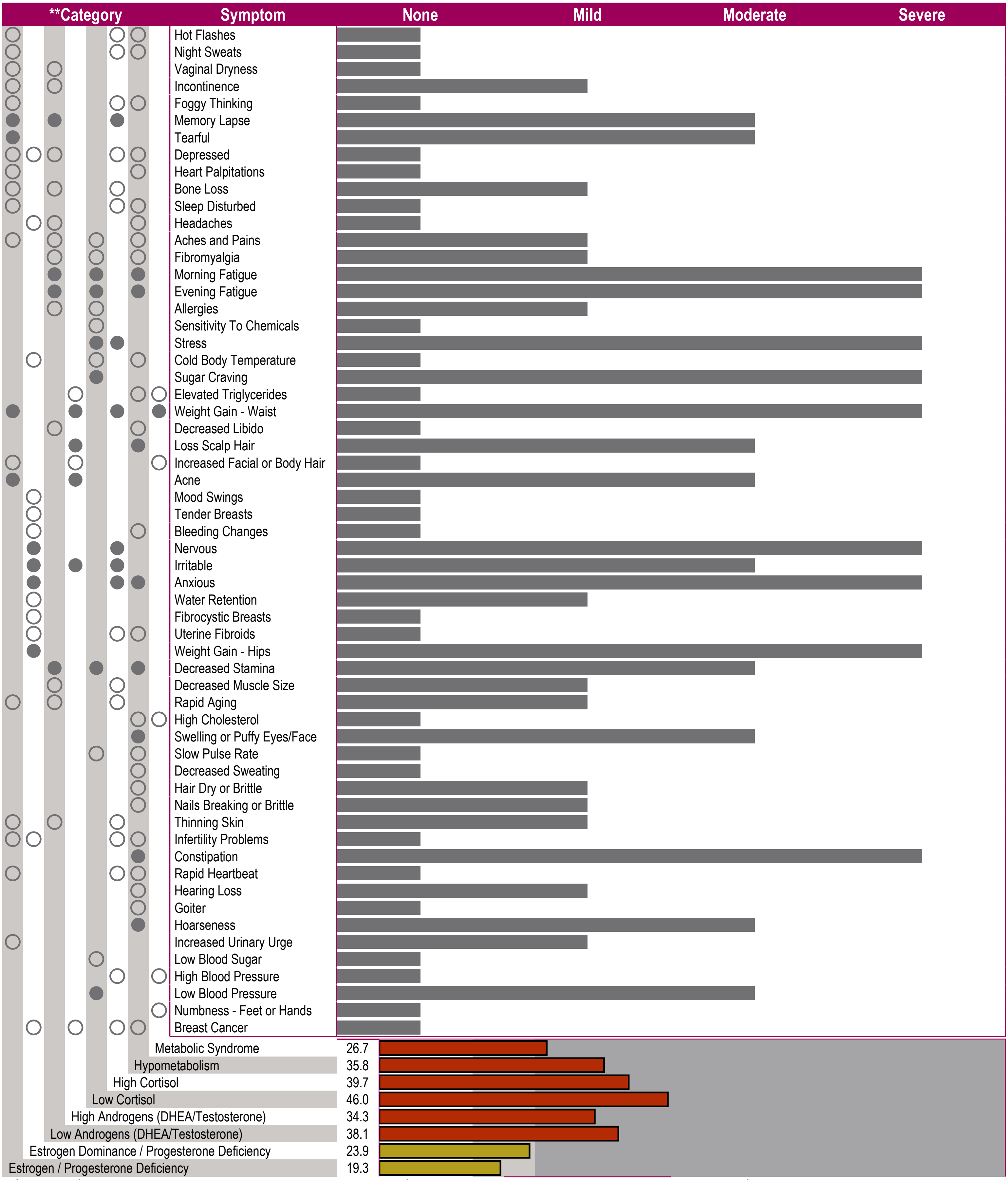
Menses Status: Pre-Menopausal
Gender: Female
Last Menses: Unspecified
DOB: 7/3/1974 (42 yrs)
Patient Ph#: 555 555 5555
Height: Unspecified
Weight: Unspecified
Waist: Unspecified

Test Name	Result		Units	Range
Zinc (Blood Spot)	6.75		mg/L	5.37-7.94
Copper (Blood Spot)	0.8		mg/L	0.64-0.96
Ratio: Zn/Cu (Blood Spot)	8.4			6.6-10.8
Magnesium (Blood Spot)	35		mg/L	28-46
Selenium (Blood Spot)	256	H	µg/L	132-247
Cadmium (Blood Spot)	0.67		µg/L	<1.04
Lead (Blood Spot)	1.37		µg/dL	<2.23
Mercury (Blood Spot)	10.24	H	µg/L	<5.29
Iodine (Urine)	107		µg/g Cr	100-380
Bromine (Urine)	8674	H	µg/g Cr	700-4800
Selenium (Urine)	68		µg/g Cr	34-220
Arsenic (Urine)	31		µg/g Cr	<42
Cadmium (Urine)	1.47	H	µg/g Cr	<0.72
Mercury (Urine)	2.35	H	µg/g Cr	<1.58
Creatinine (Urine)	0.37		mg/mL	0.3-2

<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.

Therapies

None



***Category refers to the most common symptoms experienced when specific hormone types (eg estrogens, androgens, cortisol) are out of balance, i.e., either high or low.*

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 pg1)

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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 high 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.

For more information, you can find a review of copper 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

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 vegetables.

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. 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. While normal selenium levels are essential for health, excessively high levels can be toxic.

Selenium is an essential element that has an important role in thyroid hormone metabolism, antioxidant function, and redox status. Selenium replaces sulfur in amino acids cysteine and methionine to form selenocysteine- and selenomethionine-containing proteins. 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, which is linked to lower levels of the selenium-containing enzyme glutathione peroxidase that protects against free radical production during normal thyroid hormone formation with activated iodine. Selenium supplementation above the recommended guidelines has been show to significantly decrease anti-TPO antibodies in Hashimoto disease patients.

Selenium in an essential part of at least 24 selenoproteins; the most widely studied are glutathione peroxidases, thioredoxin reductases, and iodothyronine deiodinases. Glutathione peroxidases (GPx) protect against free radicals and oxidative stress caused by heavy metals and oxidized lipids. Thioredoxin reductases (TRx) are essential for cell development and proliferation. Iodothyronine deiodinases (ID) are essential for converting inactive thyroxine (T4) to triiodothyronine (T3) in all tissues throughout the body.

Selenium serves as a detoxifying agent, preventing tissue damage, by forming tight ionic complexes with heavy metals such as mercury, arsenic, lead, and cadmium. The selenium-heavy metal complex neutralizes heavy metals preventing them from creating reactive oxygen species (ROS) that damage tissues. If heavy metal exposure is high, it is essential that selenium intake is high enough maintain adequate levels of selenium-containing anti-oxidant enzymes and neutralizing complexes.

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 as the latter more closely reflects long term exposure to cadmium.

Cadmium is a non-essential toxic element and a nephrotoxin, estrogen mimic, and a group 1 carcinogen according to the International Agency for Research on Cancer. High 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. Cadmium will accumulate in the renal cortex and cause tubular damage, preventing re-absorption of nutritional elements. Oxidative stress caused by cadmium can cause irregular gene expression, DNA damage, and cell death. Unlike mercury, cadmium does not readily cross the blood brain barrier, so neurotoxic effects are more common in the peripheral than the central nervous system. 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. Blood cadmium levels are more representative of recent exposure, while urine cadmium levels represent long term exposure and kidney burden. The half-life of cadmium in the kidneys is 15-30 years making urine an ideal body fluid to assess lifetime exposure to cadmium.

Interactions of essential elements with cadmium play a large role in preventing cadmium toxicity. Zinc has been shown to reduce cadmium absorption, compete with cadmium for enzyme binding sites, and induce synthesis of metallothionein, a metal binding protein synthesized in the liver and kidneys that has a high affinity for heavy metals such as cadmium. Proper iron intake is important because it competes with and lowers cadmium absorption in the intestines. Also, selenium can directly bind to cadmium, creating biologically inactive selenium-cadmium complexes, which helps lower cadmium burden, but also lowers the bioavailable levels of selenium necessary for antioxidant enzymes. High fiber will also inhibit cadmium's absorption due to insoluble phytate-cadmium complexes formed in the intestine.

The major sources of cadmium exposure are from vegetables, grains, and tobacco, all of which take up and accumulate cadmium from the soil. In the absence of heavy environmental exposure, diet usually contributes to most of the cadmium exposure in non-smokers. Vegans and vegetarians are particularly susceptible to cadmium because of their high consumption of cadmium-containing plant foods and increased likelihood of zinc or iron deficiency. Absorption of dietary cadmium in the intestines is relatively low, with only 3-5% being absorbed from a normal daily intake of 8-25 µg/day. In comparison, lung absorption can be up to 50%, which is why cadmium in the urine of smokers is double that of non-smokers. Cadmium content in cigarettes from different countries varies by up to 10-fold depending on the content of cadmium in the soil where the tobacco is grown. Other sources of cadmium include seafood, organ meats, and root crops. 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.

For more information, you can find a review of cadmium at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3686085/

LEAD

Whole blood lead is within the normal reference range.

Lead is a non-essential toxic element that can affect all organs in the body, including the nervous, skeletal, urinary, cardiovascular, immune, gastrointestinal and reproductive systems. High lead levels have been linked to an increased risk of stroke and heart disease along with higher mortality rate. Documented as a probable human carcinogen, lead has been associated with cancers of the brain, kidney, stomach, lung, and meninges. Lead's toxic action is a result of its ability to mimic and replace other essential elements such as calcium, zinc, copper, magnesium, sodium, and iron. Lead also binds to sulfhydryl groups found in the catalytic site of many enzymes, inactivating them. The brain is the most sensitive organ to lead exposure due to damage to neurons and interference with neurotransmitters, specifically glutamate which is required for development and learning.

The CDC considers blood lead to be elevated at 10 µg/dL for adults and 5 µg/dL for children. Once lead is ingested (10% absorption) or inhaled (50% absorption) it is bound to hemoglobin in red blood cells and transported and deposited in different organs throughout the body. Children and pregnant women absorb around 50% of ingested lead, making them more susceptible to lead toxicity. Lead's half-life in blood is around 40 days, which is about the same as the half-life of a red blood cell. Approximately 95% of lead that is absorbed will be stored in the bones with a half-life of around 25 years. Even after the exposure has ceased, lead can be re-introduced into the bloodstream from bone, meaning that blood levels indicate both current and past exposure. As women enter menopause bone resorption increases, which can increase lead exposure to other organs.

Over the past 3 centuries environmental levels of lead have increased 1000 fold as a result of human activities. The prime contributors to this increase are leaded gasoline (banned in 1996), lead paint (banned in 1978) and lead-soldered copper pipes (banned in 1986), mining operations, and other industrial applications. About 1 in 3 housing units in the U.S. has lead-based paint hazards. The amount of lead in soil and dust in cities is proportional to the historical traffic flow volumes when lead was used in gasoline. Other uses of lead include ammunition, paints, ceramics, artisan glassware, hair dye, and cosmetics. In the U.S. occupational exposure is the main cause of lead poisoning in adults. About 15-20% of total lead exposure is attributed to lead released from old pipes used to deliver drinking water.

For more information, you can find a review of lead at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3485653/pdf/ITX-5-047.pdf

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 (Hg^{2+}), 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. 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 (Hg^{2+})

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 is in the lower half of the iodine range (100-150 µg/g creatinine) and is considered optimal for thyroid hormone synthesis. However, some patients may have symptoms and feel best when iodine levels are greater than 150. According to the CDC and other agencies that have studied the relationship of thyroid function to iodine deficiency and iodine excess in large population groups, cutoffs for degrees of iodine deficiency, sufficiency, and excess in µg/L urine (very similar when expressed as µg/g creatinine) are: < 20 = severe iodine deficiency; 20-49 = moderate iodine deficiency; 50-99 = mild iodine deficiency; 100-300 = no iodine deficiency; > 300 = iodine excess (Zimmerman MB, Endocrine Reviews 2009, 30(4): 376-408). Iodine is an essential component of thyroid hormones T3 and T4, and when urinary iodine levels drop below about 50 µg/g creatinine the thyroid gland is less able to synthesize adequate thyroid hormones. The presence of goitrogens in common foods (e.g., soyfoods and cruciferous vegetables) as well as environmental toxins (perchlorate, polybrominated biphenols, bromine, fluroride, arsenic, mercury) can exacerbate a low iodine condition by inhibiting iodine uptake and thyroid hormone synthesis.

Your iodine test result represents an average of the urinary iodine excreted for a single day, and is reflective of your dietary/supplemental iodine consumption over the last several days. Consider increasing intake of foods that contain iodine (e.g., seafoods, seaweed, dairy, eggs) or take a supplement containing at least the RDA for iodine to place your levels in the upper ranges of iodine. It is important to note that this test, and any other 24 h urine iodine test, cannot be used to determine if you have a chronic iodine deficiency, which requires multiple testing over at least 10 days or blood testing of other markers of iodine deficiency (i.e., blood levels of total T4, TSH, and thyroglobulin). Iodine deficiency over weeks and months results in lower blood levels of total T4 and higher levels of thyroglobulin and TSH. Prolonged deficiency over months and years results in thyroid gland enlargement in the form of thyroid nodules or goiter. Thyroid hormone and thyroid marker testing, in combination with urinary iodine, help confirm a chronic iodine deficiency problem. Since the iodine in this test result is lower end of the normal range and this individual has self-reported iodine deficiency symptoms, the thyroid deficiency blood markers should be evaluated to determine if the low iodine is affecting thyroid hormone synthesis.

Thyroid hormone production is optimal when dietary iodine consumption is within the 150-300 µg range, which results in urinary iodine levels of about 100-300 µg/L or µg/g creatinine range (note: this is based on 80-90% of dietary iodine excreted in urine, and an average urine volume and g of creatinine daily of approximately 1 liter and 1 g, respectively). In the U.S., the Institute of Medicine (IOM) considers daily iodine consumption > 1100 µg as excessive for adults and likely to lead to a higher incidence of underlying thyroid problems, particularly in those individuals with preexisting conditions (e.g., subclinical or overt hypothyroidism, hyperthyroidism, Hashimoto's thyroiditis, autonomous thyroid nodules, goiter). This upper limit of tolerance is disputed by other groups who believe much higher (> 10-fold) iodine consumption is needed to protect the breasts and tissues of the lower reproductive tract. In Japan, where the average daily dietary intake is about 10-fold higher (about 1-3 mg with average about 1.2 mg) (Zava TT, Thyroid Research, 2011) the incidence of breast and prostate cancers are about 1/5th that of the U.S. and other countries who consume much less iodine in the diet. The Japanese Health Ministry has set the upper tolerable limit of daily iodine consumption higher at 3 mg (3000 µg).

Iodine is highest in seafoods (fish, seaweed); lower amounts are found in milk products and eggs. Vegetarians who do not eat sea vegetables or take iodine supplements are more likely to suffer from iodine deficiency and associated iodine deficiency disorders (e.g., thyroid problems). If symptoms of thyroid deficiency are problematic, consider testing thyroid hormones and supplementation with iodine and/or thyroid hormones. 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 higher than the normal reference range. 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 U.S., bromine intake from grains, nuts and fish is estimated to be 2-8 mg/day. Bromine belongs to the halogen family of elements, which also includes 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 levels above the median plasma level were shown to increase plasma TSH in patients with subclinical hypothyrodism (normal T4, elevated TSH), indicating a minor inhibitory effect on thyroid activity (Allain P, J Clin Pathol 46: 456-458, 1993). If thyroid hormone levels are low (low to low-normal total T4, free T4, free T3, and/or elevated TSH) consider identifying and eliminating the source of excessive bromine exposure to improve thyroid function. Bromine is present at high concentration in many different commercial products that result in significant exposure to humans (e.g., brominated vegetable oils found in some soft drinks, polybrominated diphenyl ether used as fire retardants, sodium bromate as a dough conditioner, methyl bromide for soil fumigation, and hypobromous acid to disinfectant pools and spas).

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.

ARSENIC:

Arsenic excretion is within range. Ideally, arsenic levels should be within the lower end of the reference range, indicating normal exposure, while results at the higher end of the normal reference range (30-41) can be more toxic when selenium is low and levels of other heavy metals (e.g., mercury, cadmium) are high.

The most common cause of arsenic toxicity is constant exposure to contaminated drinking water. The WHO and EPA have set a maximum level of arsenic in drinking water to 10 µg/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 5-40 µg/day with acute toxicity possible at levels >100 µg/day. Around 80% of arsenic is excreted in the urine after 3 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 ICP-MS alone. However, anyone with arsenic above the 5-40 µg/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.

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 principal mechanisms of arsenic toxicity is through its tight binding with selenium, effectively preventing it from incorporation into selenoproteins that are 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), arsenic toxicity is extremely problematic and closely associated with diabetes, hypertension, cardiovascular disease, vascular changes, neuropathy, memory loss and hormonal regulation modifications.

High arsenic exposure, particularly when selenium is low, is linked to cancer of the lung, prostate, bladder and skin.

CADMIUM:

Urinary cadmium is higher than the reference range suggesting overall higher lifetime exposure to this heavy metal.

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 in foods and liquids is absorbed by the gastrointestinal tract (about 1-2 µg), 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 above the reference range. Urine excretion at this level indicates high mercury exposure (note: this assumes no mercury chelating agents were used at the time of urine collection). 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 light bulbs, thermometers, barometers, switches, medicines, paint, antiques, and cosmetics. Thimerosal, a vaccine preservative, contains 50% mercury by weight and has been used since the 1930s. The highest source of organic mercury (methylmercury) exposure in the U.S. 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 depend 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.

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, which 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 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.