Test Results

ZRT LABORATORY
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2015 08 00 001 U

Samples Arrived: 08/19/2015
Date Closed: 08/22/2015

Samples Collected: Urine: 08/16/15 06:38
Urine: 08/16/15 21:37

Dr ZRT
8605 SW Creekside Pl
Beaverton, OR 97008

Ellen E Elements

BMI: 22.3
Height: 5 ft 1 in
Weight: 118 lb
Waist: 27 in

Menses Status: Pre-Menopausal - Irregular
Gender: Female
Last Menses: Unspecified
DOB: 5/12/1970 (45 yrs)
Patient Ph#: 555 555 5555

Test Name	Result		Units	Range
Iodine (Urine)	46	L	µg/g Cr	100-380
Bromine (Urine)	2027		µg/g Cr	700-4800
Selenium (Urine)	159		µg/g Cr	34-220
Arsenic (Urine)	28		µg/g Cr	<42
Mercury (Urine)	4.28	H	µg/g Cr	<1.58
Cadmium (Urine)	0.29		µg/g Cr	<0.72
Creatinine (Urine)	1.23		mg/mL	0.3-2

Therapies

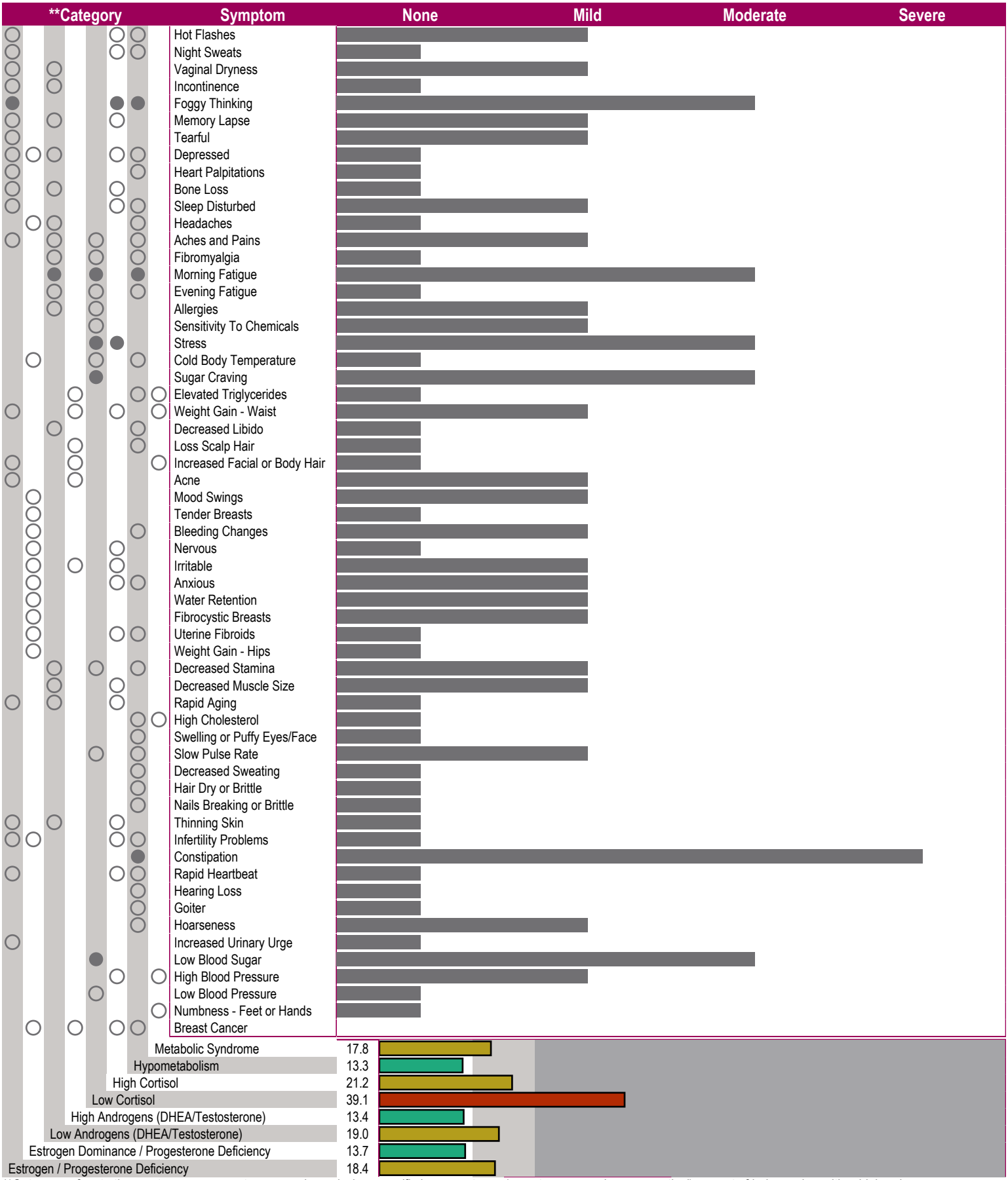
None

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

David T. Zava, Ph.D.
(Laboratory Director)

CLIA Lic # 38D0960950
Composed by: 1165029919 at 9/8/2015 2:09:26 PM



***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.*

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Lab Comments

IODINE:

Urinary iodine/creatinine falls into the reference range that is considered mildly deficient (50-99 ug/g creatinine) on the day tested. According to the Center for Disease Control (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 ug/L urine (very similar when expressed as ug/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 ug/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/supplement iodine consumption over the last several days. If your daily diet is representative of the day you tested then you are likely iodine insufficient, and should 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.

Natural sources of iodine are highest in seafoods (fish, seaweed) with lesser amounts found in milk products and eggs. Vegans 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). 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 is within 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 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].

SELENIUM:

Selenium excretion in urine is within the optimal reference range (> 50-200 ug/g creatinine) seen in regions with adequate dietary selenium intake. Intake of selenium in the United States has been estimated at 135µg/day for men and 92µg/day for women, which is consistent with the reported average urinary level of selenium in the US of about 40-60 ug/g creatinine range (assuming about 50-70% of selenium ingested is excreted in urine). The RDA for selenium in adults is around 55 micrograms/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 micrograms/day. Higher levels (up to 800 micrograms) 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 40µg/day; intake lower than 11µg/day results in selenium deficiency disorders.

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 bio-inert thyroxine (T4) to active T3 or inactive reverse T3 in all tissues throughout the body. Hashimoto's is strongly associated with selenium deficiency and lower intracellular levels of the selenium-containing antioxidants like glutathione peroxidase and thioredoxin reductase.

Even normal (optimal) urinary levels of selenium can be insufficient when the 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 removing it from incorporation into selenoproteins like glutathione peroxidase

and thyroid deiodinases, thus compromising thyroid hormone formation and metabolism.

ARSENIC:

Arsenic excretion is within range. Ideally, Arsenic levels are withing the lower end (5-40 ug/g creatinine) of the reference range. Results at the lower end of this range indicate normal exposure, while results at the higher end (>40-138 ug/g creatinine) of the normal reference range can indicate possible acute toxicity.

The most common cause of arsenic toxicity is constant exposure to contaminated drinking water. 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 urnine 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 5-40µg/day with acute toxicity possible at levels >100µg/day. Around 80% of arsenic is excreted in the urine after three days, making urine arsenic a good indicator of intake.

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.

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 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.

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; 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.

CADMIUM:
Cadmium is within range. Cadmium is known to contribute to conditions including learning disabilities, hypertension, kidney disease and lung issues, and cancer. Cadmium may induce estrogenic changes in the uterus and breast. Cadmium is accumulated in the body due to exposure to industry polluted water which is used to water crops or in industry using soldering or welding metals. In developed nations the accumulation in tobacco plants from ground water still impacts tobacco smokers but policies generally control the release by industry. 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 acting as a metalloestrogen to activate estrogen-regulated genes. Continuing to avoid cadmium exposure is required for excellent health.