Test Results

ZRT LABORATORY
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Beaverton, OR 97008
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2016 02 00 001 BU

Samples Arrived: 02/23/2016
Date Closed: 02/28/2016

Samples Collected:
Blood Spot: 02/17/16 07:15
Urine: 02/17/16 04:00
Urine: 02/17/16 21:30

Ordering Provider:

Jane Smith MD
8605 SW Creekside Pl
Beaverton, OR 97008

Ellen T Elements
111 N Fake St
Beaverton, OR 97008

BMI: 21.6
Height: 5 ft 7 in
Weight: 138 lb
Waist: 28 in

Menses Status: Pre-Menopausal - Irregular
Gender: Female
Last Menses: Unspecified
DOB: 3/10/1972 (43 yrs)
Patient Ph#: 555 555 5555

Test Name	Result		Units	Range
Thyroglobulin (Blood Spot)	25.2		ng/mL	3-40 (optimal 3-10) ng/ml
Total T4 (Blood Spot)	5.7		µg/dL	5-10.8 ug/dL
Free T4 (Blood Spot)*	1.0		ng/dL	0.7-2.5
Free T3 (Blood Spot)	2.5		pg/mL	2.5-6.5
TSH (Blood Spot)	5.2	H	µU/mL	0.5-3.0
TPOab (Blood Spot)*	48		IU/mL	0-150 (70-150 borderline)
Iodine (Urine)	103		µg/g Cr	100-380
Bromine (Urine)	4526		µg/g Cr	700-4800
Selenium (Urine)	53		µg/g Cr	34-220
Arsenic (Urine)	193	H	µg/g Cr	<42
Cadmium (Urine)	0.29		µg/g Cr	<0.72
Mercury (Urine)	1.72	H	µg/g Cr	<1.58
Creatinine (Urine)	0.62		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.
**For research purposes only.*

Therapies

50mcg oral Synthroid (T4) (Pharmaceutical) (1 Days Last used)

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

Alison McAllister, ND
(Ordering Provider unless otherwise specified on pg1)

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**Category	Symptom	None	Mild	Moderate	Severe
	Hot Flashes				
	Night Sweats				
	Vaginal Dryness				
	Incontinence				
	Foggy Thinking				
	Memory Lapse				
	Tearful				
	Depressed				
	Heart Palpitations				
	Bone Loss				
	Sleep Disturbed				
	Headaches				
	Aches and Pains				
	Fibromyalgia				
	Morning Fatigue				
	Evening Fatigue				
	Allergies				
	Sensitivity To Chemicals				
	Stress				
	Cold Body Temperature				
	Sugar Craving				
	Elevated Triglycerides				
	Weight Gain - Waist				
	Decreased Libido				
	Loss Scalp Hair				
	Increased Facial or Body Hair				
	Acne				
	Mood Swings				
	Tender Breasts				
	Bleeding Changes				
	Nervous				
	Irritable				
	Anxious				
	Water Retention				
	Fibrocystic Breasts				
	Uterine Fibroids				
	Weight Gain - Hips				
	Decreased Stamina				
	Decreased Muscle Size				
	Rapid Aging				
	High Cholesterol				
	Swelling or Puffy Eyes/Face				
	Slow Pulse Rate				
	Decreased Sweating				
	Hair Dry or Brittle				
	Nails Breaking or Brittle				
	Thinning Skin				
	Infertility Problems				
	Constipation				
	Rapid Heartbeat				
	Hearing Loss				
	Goiter				
	Hoarseness				
	Increased Urinary Urge				
	Low Blood Sugar				
	High Blood Pressure				
	Low Blood Pressure				
	Numbness - Feet or Hands				
	Breast Cancer				

Metabolic Syndrome	48.9	
Hypometabolism	52.9	
High Cortisol	49.4	
Low Cortisol	65.5	
High Androgens (DHEA/Testosterone)	58.2	
Low Androgens (DHEA/Testosterone)	53.3	
Estrogen Dominance / Progesterone Deficiency	46.2	
Estrogen / Progesterone Deficiency	49.1	

***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.

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Lab Comments

Thyroglobulin is within normal range. Thyroglobulin is considered a good marker of the average iodine status over the past few weeks or longer. Thyroglobulin levels in blood usually are inversely related to the iodine status; when urinary iodine levels are sufficient, thyroglobulin levels will usually be < 10 and > 3 ng/ml, and when iodine is insufficient thyroglobulin levels rise in the blood in response to higher TSH stimulating thyroglobulin synthesis in the thyroid gland in an attempt to increase thyroid hormone synthesis.

Total T4 is within the expected reference range with T4 (thyroxine-Synthroid) therapy. In the absence of T4 therapy total T4 is a good marker of the thyroid glands ability to synthesize thyroid hormones. This individual indicates use of T4 therapy, which raises blood T4 and lowers TSH.

Free T4 is within normal range.

Free T3, the most potent bioactive thyroid hormone, is low-normal and TSH is high, indicating a clinically hypothyroid state. Normal T4 and low T3 usually results from poor hepatic conversion of T4 to T3, which suggests one or more of the following: nutrient deficiency (e.g., zinc and/or selenium), heavy metal toxicity (mercury, lead, cadmium), liver damage (caused by viruses, alcohol, etc.), or steroid hormone imbalances (e.g., high cortisol). Testing for steroid hormones (estradiol, progesterone, testosterone, DHEAS, cortisol am/pm) also is worthwhile considering if symptoms of their imbalances are problematic. If conventional T4 therapy does not resolve symptoms of thyroid deficiency, consider combination T4/T3 replacement therapy or slow release T3 therapy alone.

Thyroid peroxidase antibodies (TPO) are low indicating that Hashimoto's thyroiditis is unlikely.

IODINE:

Urinary iodine/creatinine falls into the reference range that is considered "sufficient" (100-150 ug/g creatinine). 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.

Thyroid hormone production is optimal when dietary iodine consumption is within the 150-300 ug range, which results in urinary iodine levels of about 100-250 ug/l or ug/g creatinine range (note: this is based on 80-90% of dietary iodine excreted in urine and average urine volume and g of creatinine daily is approximately 1 liter and 1 g, respectively). In the US, the Institute of Medicine (IOM) considers daily iodine consumption > 1100 ug 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).

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 is within high-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 levels above the median plasma level 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). 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 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. 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. 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 bio-inert thyroxine (T4) to active T3 or inactive reverse T3 in all tissues throughout the body. The selenium containing 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 which 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 the 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.

ARSENIC:

Arsenic excretion is higher than the reference range. 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 (referred to as speciation) and eliminating sources of arsenic exposure. Selenium supplementation should be considered as a means to prevent arsenic from reducing levels of selenoproteins.

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.

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 5-40 µg/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 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 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.

CADMIUM:

Urinary cadmium is within normal reference range.

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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 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 due to mercury's accumulation in the kidneys, which also estimates total body burden. 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 (ethyl mercury), 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 adverse 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.

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). Individuals 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,

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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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