TEST REPORT

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ZRT LABORATORY

 # 2018 08 28 061 U

Ordering Provider:
John Getuwell, ND

Samples Received
08/28/2018

Report Date
08/30/2018

Samples Collected
Urine - 08/23/18 07:30
Urine - 08/23/18 22:00

Patient Name: Toxic & Essential Elements - Urine
Patient Phone Number: 555 555 5555

Gender	Last Menses	Height	Waist
Female	Unspecified	5 ft 6 in	28 in
DOB	**Menses Status**	**Weight**	**BMI**
2/11/1967 (51 yrs)	Postmenopausal	145 lb	23.4

TEST NAME	RESULTS | 08/23/18	RANGE
Urinary Elements		
Iodine	137	100-380 µg/g Cr
Bromine	1754	700-4800 µg/g Cr
Selenium	79	34-220 µg/g Cr
Lithium	45	10-218 µg/g Cr
Arsenic	87 H	<42 µg/g Cr
Cadmium	0.17	<0.72 µg/g Cr
Mercury	0.54	<1.58 µg/g Cr
Creatinine	1.50	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

topical Estrogel (estradiol) (compounded) (1 Days Last Used)

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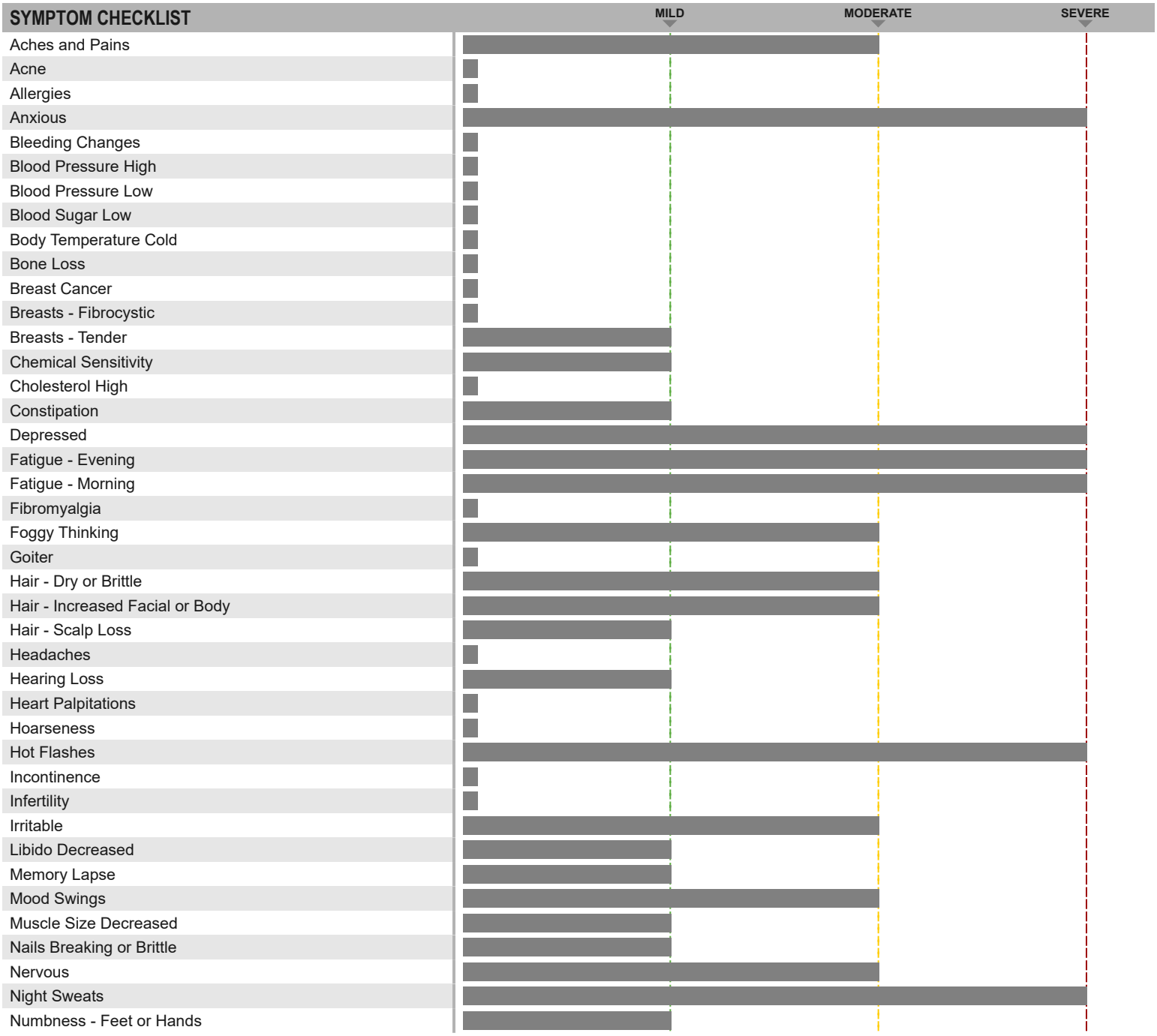
Disclaimer: Symptom Categories below show percent of symptoms self-reported by the patient compared to total available symptoms for each category. For detailed information on category breakdowns, go to www.zrtlab.com/patient-symptoms.

SYMPTOM CATEGORIES	RESULTS | 08/23/18
Estrogen / Progesterone Deficiency	49%
Estrogen Dominance / Progesterone Deficiency	35%
Low Androgens (DHEA/Testosterone)	42%
High Androgens (DHEA/Testosterone)	32%
Low Cortisol	39%
High Cortisol	56%
Hypometabolism	36%
Metabolic Syndrome	33%



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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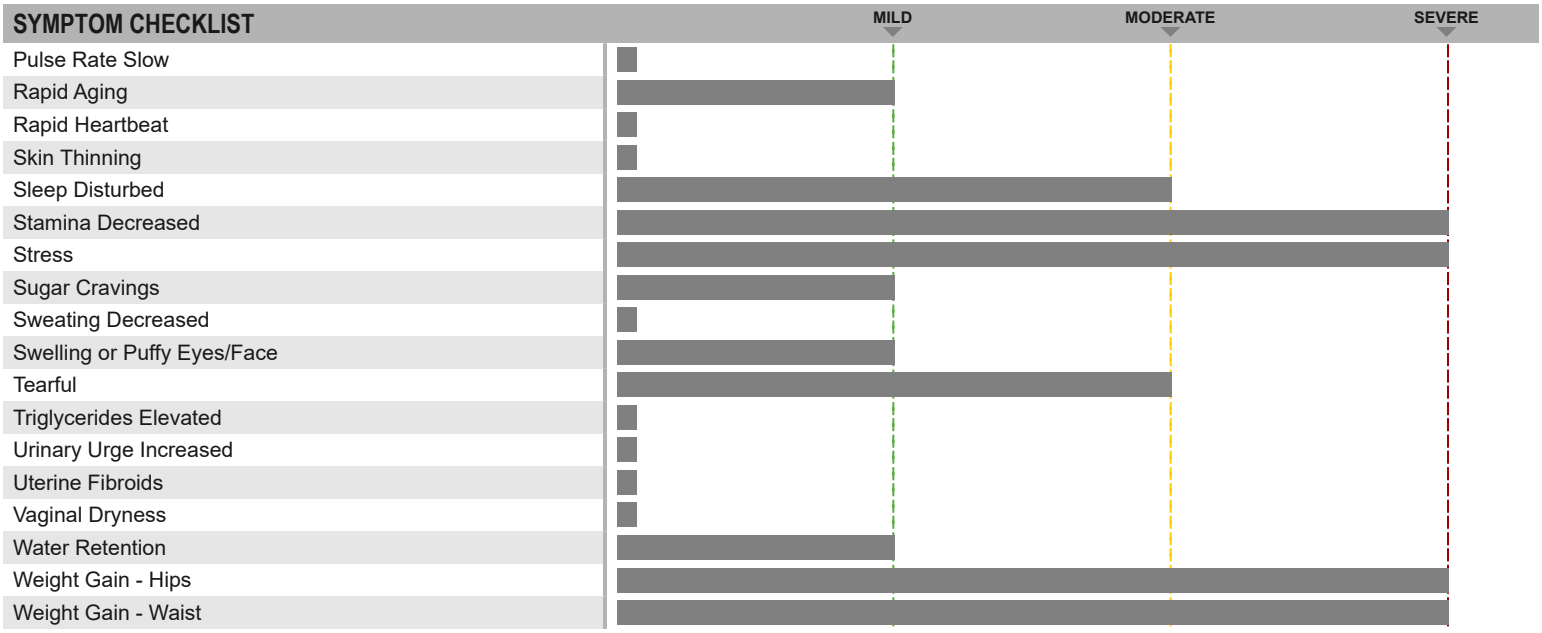

Lab Comments

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

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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 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). 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 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

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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.

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 (lower than median level of 0.27 ug/g creatinine) suggesting overall lower 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 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 in this individual's urine is greater than the median of the reference range (median 0.29; 5-95% range: 0.01-1.58 ug/g creatinine), indicating some exposure to low levels of mercury.

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.

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)

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exposure in the United States is from fish, with fish tissue containing up to 95-97% of this mercury species.

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