

TEST REPORT

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D2026 05 06 013 BU

Ordering Provider:
Getuwell

Samples Received
05/06/2026
Report Date
05/18/2026

Samples Collected
Blood Spot - 04/18/26 09:00
Urine - 04/18/26 07:00
Urine - 04/18/26 17:00

Patient Name: Blood Spot and Dried Urine ICPMS Toxic and Essential Elements
Patient Phone Number:

Gender Female	Last Menses Unspecified	Height Unspecified	Waist Unspecified
DOB 1/1/1975 (51 yrs)	Menses Status Postmenopausal	Weight Unspecified	

TEST NAME	RESULTS 04/18/26	RANGE
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Blood Spot Toxic & Essential Elements

Zinc	6.14	5.06-8.57 mg/L
Copper	0.79	0.64-1.10 mg/L
Ratio: Zn/Cu	7.8	6.16-10.30
Magnesium	31	29-51 mg/L
Selenium	97 L	130-362 µg/L
Cadmium	<0.22	<0.75 µg/L
Mercury	0.58	<6.98 µg/L

Urinary Toxic & Essential Elements

Iodine	138	100-380 µg/g Cr
Bromine	2161	700-4800 µg/g Cr
Selenium	31 L	34-220 µg/g Cr
Lithium	9 L	10-218 µg/g Cr
Arsenic	12	<42 µg/g Cr
Cadmium	0.17	<0.72 µg/g Cr
Mercury	0.13	<1.58 µg/g Cr

Urinary Creatinine

Creatinine	0.87	0.3-2.0 mg/mL
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<dI = 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

25mcg oral Levothyroxine (T4) (Pharmaceutical) (12 Hours Last Used)

TEST REPORT | Patient Reported Symptoms

👤 Blood Spot and Dried Urine ICPMS Toxic and Essential Elements
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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 04/18/26
Estrogen / Progesterone Deficiency	23%
Estrogen Dominance / Progesterone Deficiency	31%
Low Androgens (DHEA/Testosterone)	19%
High Androgens (DHEA/Testosterone)	16%
Low Cortisol	31%
High Cortisol	29%
Hypometabolism	25%
Metabolic Syndrome	0%

SYMPTOM CHECKLIST	MILD	MODERATE	SEVERE
Aches and Pains			
Acne			
ADD/ADHD			
Addictive Behaviors			
Allergies			
Anxious			
Autism Spectrum Disorder			
Bleeding Changes			
Blood Pressure High			
Blood Pressure Low			
Blood Sugar Low			
Body Temperature Cold			
Bone Loss			
Breast Cancer			
Breasts - Fibrocystic			
Breasts - Tender			
Chemical Sensitivity			
Cholesterol High			
Constipation			
Depressed			
Developmental Delays			
Eating Disorders			
Fatigue - Evening			
Fatigue - Morning			
Fibromyalgia			
Foggy Thinking			
Goiter			
Hair - Dry or Brittle			
Hair - Increased Facial or Body			
Hair - Scalp Loss			
Headaches			
Hearing Loss			
Heart Palpitations			
Hoarseness			
Hot Flashes			
Incontinence			
Infertility			
Irritable			
Libido Decreased			
Mania			

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 vegetable.

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

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

SELENIUM

Whole blood selenium is lower than the reference range. Whole blood selenium levels represent long-term exposure while urine selenium levels reflect recent intake.

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

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.

Consider increasing selenium intake through supplements or consuming foods that contain higher levels of selenium. The current recommended dietary allowance (RDA) for selenium is 55 mcg/day. Selenium supplementation above 600 mcg/day can be toxic in some individuals. Foods that are rich in selenium are shellfish, organ meat, dairy products, eggs, garlic, nuts (Brazil nuts very high), broccoli, fish, onion, ginseng, grains, and mushrooms.

For more information, you can find a review of selenium at: <https://www.nature.com/articles/1601800>

CADMIUM

Whole blood cadmium is within the normal reference range, which should be considered beneficial as it indicates low recent exposure to cadmium. High-normal cadmium should be cross-checked with urinary cadmium which better reflects long-term exposure to cadmium. Cadmium bioaccumulates in the body, meaning that at birth levels are low, but by age 30 the body burden may reach toxic levels that adversely affect

health. The half-life of cadmium in the kidneys is 15-30 years making urine an ideal body fluid to assess lifetime exposure to cadmium.

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

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

MERCURY

Whole blood mercury is within normal reference range. If mercury is within the high-normal range this may indicate excessive exposure and need to identify and eliminate the source.

Mercury is a potent toxin. Mercury is found in three basic forms in the body: elemental mercury (Hg₀), inorganic mercury (Hg₂₊), and organic mercury (MeHg). High mercury exposure can cause symptoms which include balance problems, hearing loss, speech issues, and damage to peripheral nerves (tingling sensation). If selenium and/or zinc levels are low in concert with high whole blood mercury, it is recommended that they be increased to protect against antioxidant functions. The half-life of mercury in the brain is estimated at 20 years. 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 lightbulbs, mines, industrial manufacturing, dental amalgams, and thermometer production. Dental amalgams, which are 50% mercury, gas off between 2-28 µg elemental mercury/day, of which 80% is absorbed. Elimination of elemental mercury, which is converted to inorganic mercury in the body, is through urine and feces.

Inorganic Mercury (Hg₂₊)

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

Organic Mercury (Methylmercury)

Methylmercury is the most common and toxic form of mercury. It is nonpolar and accumulates in fatty tissues such as the plasma membranes of red blood cells and other fatty tissues like the brain. Methylmercury is purported to be 100 times more toxic than elemental or inorganic mercury. Atmospheric elemental and inorganic mercury is converted by microorganisms in water to organic mercury, which works its way up the food chain and bio accumulates. 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 central nervous system, 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, fluoride, 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 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 hypothyroidism (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 below the reference range. Consider increasing dietary intake of foods that contain selenium, or use of a selenium supplement. 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 µg/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. 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 antioxidant glutathione peroxidase plays an important role throughout the body in removing oxidants such as hydrogen peroxide (H2O2) 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 H₂O₂, 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 H₂O₂ to activate iodine for thyroid hormone synthesis. Low levels of selenium result in less protection of the thyroid against H₂O₂. 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 three 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). Low selenium is particularly problematic 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. 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. 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 micrograms/day (see above). <https://www.nature.com/articles/1601800>

LITHIUM:

Lithium excretion is lower than the reference range. Lithium is not considered to be an essential element and there is no established recommended daily amount (RDA). 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.

Lithium is being researched and used for mood stabilization and slow the progression of dementia [Nunes et al. 2013]. Lithium has been associated with increased telomere length [Squassina et al. 2016], decreases in neurological disorders and cardiovascular diseases [Prosser et al. 2016], lower incidence of some cancers [Huang et al. 2016], stabilized neuronal activities and neuroprotection [Chiu 2011], improved visual memory [Bersani et al. 2016], increased brain gray matter [Sassi et al. 2002], regulated serotonin transport [Chenu et al. 2006], and increased levels of glutathione in the brain [Cui et al. 2007]. Lithium can cause problems with kidney and thyroid issues especially at high dosages. Lithium is dosed in low doses (OTC - micrograms) to pharmacologic (prescription >100mg) dosages; discuss with your healthcare provider.

ARSENIC:

Urinary arsenic levels are within the lower quadrant of the reference range. Results at the lower end of this range indicate normal exposure.

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 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 5-40µg/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 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 low (within the lower quadrant of the reference range-0.01-1.58 ug/g creatinine). Urine excretion at this level is consistent with low mercury exposure.

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