Nordic Laboratories

PATIENT:	**Sample Report**			TEST REF:	**TST-##-#####**
TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy		
GENDER:	Female	TESTED:	dd/mm/yyyy	ADDRESS:	
AGE:	42		dd/mm/yyyy		
DATE OF BIRTH:	dd-mm-yyyy		dd/mm/yyyy		
			dd/mm/yyyy		
			dd/mm/yyyy		
			dd/mm/yyyy		
			dd/mm/yyyy		

TEST NAME: Complete Iodine Thyroid w/ elements & Adrenal stress

TEST NAME	RESULTS | 01/01/19	RANGE
Salivary Steroids		
DHEAS	2.1	2-23 ng/mL (Age Dependent)
Cortisol	8.2	3.7-9.5 ng/mL (morning)
Cortisol	2.6	1.2-3.0 ng/mL (noon)
Cortisol	2.0 H	0.6-1.9 ng/mL (evening)
Cortisol	0.7	0.4-1.0 ng/mL (night)
Blood Spot Thyroids		
Thyroglobulin	3.7	3-40 ng/mL (optimal 3-10)
Total T4	6.2	5-10.8 µg/dL
Free T4*	1.1	0.7-2.5 ng/dL
Free T3	3.0	2.4-4.2 pg/mL
TSH	8.5 H	0.5-3.0 µU/mL
TPOab*	71	0-150 IU/mL (70-150 borderline)
Urinary Elements		
Iodine	63 L	100-380 µg/g Cr
Bromine	921	700-4800 µg/g Cr
Selenium	29 L	34-220 µg/g Cr
Lithium	12	10-218 µg/g Cr
Arsenic	75 H	<42 µg/g Cr

Nordic Laboratories

PATIENT:	**Sample Report**			TEST REF:	**TST-##-#####**
TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy		
GENDER:	Female	TESTED:	dd/mm/yyyy	ADDRESS:	
AGE:	42		dd/mm/yyyy		
DATE OF BIRTH:	dd-mm-yyyy		dd/mm/yyyy		
			dd/mm/yyyy		
			dd/mm/yyyy		
			dd/mm/yyyy		
			dd/mm/yyyy		

TEST NAME: Complete Iodine Thyroid w/ elements & Adrenal stress

TEST REPORT | Results *continued*

Sample Test Report
2019 01 01 999 ABC

TEST NAME	RESULTS | 01/14/19	RANGE
Urinary Elements		
Cadmium	0.08	<0.72 µg/g Cr
Mercury	0.80	<1.58 µg/g Cr
Urinary Creatinine		
Creatinine	1.06	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. * For research purposes only.*

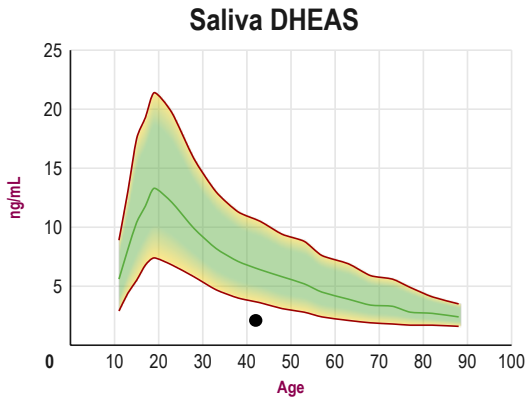
Therapies

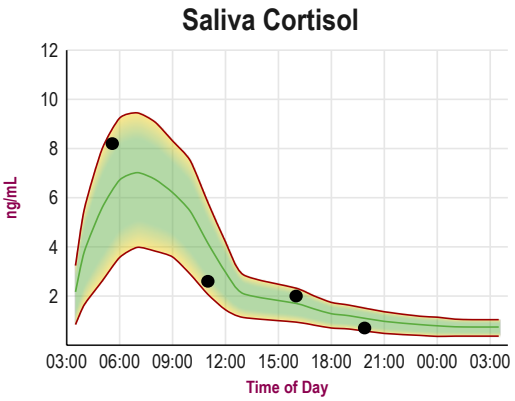
None Indicated

Graphs

Disclaimer: Graphs below represent averages for healthy individuals not using hormones. Supplementation ranges may be higher. Please see supplementation ranges and lab comments if results are higher or lower than expected.

Average ▼▲ Off Graph





Nordic Laboratories

PATIENT:	**Sample Report**			TEST REF:	**TST-##-#####**
TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy		
GENDER:	Female	TESTED:	dd/mm/yyyy	ADDRESS:	
AGE:	42		dd/mm/yyyy		
DATE OF BIRTH:	dd-mm-yyyy		dd/mm/yyyy		
			dd/mm/yyyy		
			dd/mm/yyyy		
			dd/mm/yyyy		
			dd/mm/yyyy		

TEST NAME: Complete Iodine Thyroid w/ elements & Adrenal stress

TEST REPORT | Reference Ranges

Sample Test Report
2019 01 01 999 ABC

Disclaimer: Supplement type and dosage are for informational purposes only and are not recommendations for treatment. For a complete listing of reference ranges, go to www.zrtlab.com/reference-ranges.

TEST NAME	WOMEN
DHEAS	2-23 ng/mL (Age Dependent)
Cortisol	3.7-9.5 ng/mL (morning); 1.2-3.0 ng/mL (noon); 0.6-1.9 ng/mL (evening); 0.4-1.0 ng/mL (night)
Thyroglobulin	3-40 ng/mL (optimal 3-10)
Total T4	5-10.8 µg/dL
Free T4	0.7-2.5 ng/dL
Free T3	2.4-4.2 pg/mL
TSH	0.5-3.0 µU/mL
TPOab	0-150 IU/mL (70-150 borderline)
Iodine	100-380 µg/g Cr
Bromine	700-4800 µg/g Cr
Selenium	34-220 µg/g Cr
Lithium	10-218 µg/g Cr
Arsenic	<42 µg/g Cr
Cadmium	<0.72 µg/g Cr
Mercury	<1.58 µg/g Cr
Creatinine	0.3-2.0 mg/mL

Lab Comments

DHEAS is within low-normal expected age range. Chronic low DHEAS may suggest HPA axis dysfunction, particularly if cortisol is also low and symptoms are indicative of low adrenal function. DHEAS is highest during the late teens to early twenties (10-20 ng/ml) and drops steadily with age to the lower end of range by age 70-80. Consider adrenal adaptogens or DHEA supplements if symptoms of androgen deficiency are problematic.

Cortisol is near expected range throughout most of the day and is following a normal circadian rhythm; however, some symptoms of low cortisol are reported. When cortisol levels are within normal range but symptoms of low cortisol are reported, this may suggest that the adrenal glands are overworking to keep up with the demands of the stressors, which could eventually lead to adrenal exhaustion. HPA axis dysfunction is most commonly caused by stressors which include: psychological stress (emotional), sleep deprivation, poor diet (low protein-particularly problematic in vegetarians), nutrient deficiencies (particularly low vitamins C and B5), physical insults (surgery, injury), diseases (cancer, diabetes), chemical exposure (environmental pollutants, excessive medications), low levels of cortisol precursors (pregnenolone and progesterone) and pathogenic infections (bacteria, viruses and fungi). A normal daily output of cortisol is essential to maintain normal metabolic activity, help regulate steady state glucose levels (important for brain function and energy production), and optimize immune function. Depletion of adrenal cortisol synthesis by a chronic stressor, sleep deprivation, and/or nutrient deficiencies (particularly vitamins C and B5) often leads to symptoms such as fatigue, allergies (immune dysfunction), chemical sensitivity, cold body temp, and sugar craving. For additional information about strategies for supporting adrenal health and reducing stress(ors), the following books are worth reading: "Adrenal Fatigue", by James L. Wilson, N.D., D.C., Ph.D.; "The Cortisol Connection", by Shawn Talbott, Ph.D.; "The End of Stress As We Know It" by Bruce McEwen; "Awakening Athena" by Kenna Stephenson, MD.

Thyroglobulin is within normal range. In contrast to urinary iodine, which provides information on the iodine status over the past 24 hr, 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. Exceptions occur when TSH is low despite low thyroid hormone levels and when anti-thyroglobulin antibodies are present. Low TSH, despite low thyroid hormone levels, can result from high levels of glucocorticoids (e.g. endogenous cortisol caused by stressors or exogenous anti-inflammatory glucocorticoids) . Anti-thyroglobulin antibodies interfere with the thyroglobulin test, and can cause a false-low result. Individuals with Hashimoto's thyroditis (postive TPO antibodies), are very likely (50%) to also have antibodies to thyroglobulin, which would interfer with the thyroglobulin measurement, causing false low levels.

TEST NAME: Complete Iodine Thyroid w/ elements & Adrenal stress

TEST REPORT | Comments *continued*

Total T4 is within the expected reference range, but low-normal. At baseline in the absence of T4 therapy (T4 alone or T4/T3 combination therapy) the total T4 is a good marker of the thyroid glands ability to synthesize thyroid hormones. Outside of frank hypothyroidism, lower T4 synthesis by the thyroid gland can be caused by one or more of the following: 1) T3 therapy (e.g. Cytomel) that suppresses TSH and thus thyroid gland production of T4; 2) Hashimoto's thyroiditis (seen as elevated TPO antibodies), which causes destruction and fibrosis of the thyroid gland; 3) low iodine, which is essential for thyroid hormone synthesis; 4) low iodine uptake and iodine organification by the thyroid gland caused by excessive consumption of goitrogens (e.g. soy isoflavones, cruciferous vegetables); 5) low catalytic activity of thyroid peroxidase (enzyme that catalyzes thyroid hormone synthesis from iodine and thyroglobulin-bound tyrosine) due to iron deficiency/low ferritin); 6) and/or overall inhibition of thyroid hormone synthesis caused by excessive iodine supplementation (Wolff-Chaikoff Effect). If the low total T4 is associated with symptoms of thyroid deficiency, consider thyroid hormone therapy or increasing dosage if already taking.

Free T4 is within normal range but reported symptoms indicate thyroid deficiency. This might suggest that hepatic conversion of T4 to T3 is impaired or T4 is being converted to reverse T3 (both conditions increased under conditions of high stress/cortisol). It would be worthwhile to evaluate steroid hormones by saliva and correct any hormonal imbalances (eg. high estradiol, low progesterone, low testosterone, high or low cortisol) that might impede optimal thyroid function.

Free T3 is within normal range but symptoms indicate thyroid deficiency. A normal T3 does not exclude the possibility of a "functional" thyroid deficiency caused by other hormonal imbalances such as excess estrogen, low progesterone, low testosterone, low or high cortisol, and low growth hormone (IGF-1). Testing for these hormones is recommended.

TSH is high. Although most laboratories have a TSH range of 0.35-5.50, new studies are finding that the mean and median values are 1.0-1.5mU/l . TSH levels >3.0 are now considered abnormal due to changes by the endocrinology association - see www.aace.com for more information. Some experts believe that TSH should be kept below 2.0 for optimal health. Elevated TSH is often associated with symptoms of hypothyrodism, which include fatigue, decreased stamina, depression, rheumatic pain, sleep disturbances, cold extremities or feeling cold, reduced body temperature, brittle nails, dry coarse hair, hair loss, infertility, low libido, puffy eyes and face, decreased sweating, menorrhagia, and/or constipation. Periodic TSH monitoring is recommended if clinical symptoms of thyroid deficiency persist. T3 results may help guide treatment decisions. Thyroid therapy may be worthwhile considering if T4 and/or T3 are low and symptoms of thyroid deficiency are problematic.

Thyroid peroxidase (TPO) antibodies are borderline positive, suggesting a possible evolving issue with Hashimoto's autoimmune thyroiditis. If symptoms of thyroid dysfunction become more problematic it would be worthwhile to recheck TPO levels. Antibodies to this enzyme may cause an increase in autoimmune dysfunction around the thyroid causing an increase in inflammatory cytokines, increased T cells, and NK cell function. The autoimmune reaction to the thyroid tissue results in destruction of the thyroid cells with consequent release of high levels of thyroid hormones (T4 and to a lesser extent T3), which results in a hyperthyroid state. Continued destruction of the thyroid gland results in fibrosis and eventual depletion of the thyroid hormone, thus causing a hypothyroid state. Clinical studies show that selenium supplementation is helpful in decreasing TPO antibody levels and thus helps prevent autoimmune destruction of the thyroid gland (Duntas et al. Eur J Endocrinology 148: 389-393, 2003).

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. It is important to note that this test, and any other 24 hr urine iodine test, can not 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 mildly deficient, the thyroid deficiency blood markers should be evaluated to determine if the low iodine is affecting thyroid hormone synthesis.

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

TEST REPORT | Comments *continued*

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 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 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. 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 (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.

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).

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 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.

TEST REPORT | Comments *continued*

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.

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 is in the upper quartile of the reference range. Urine excretion at this level indicates relatively high mercury exposure. This may be more problematic if other heavy metals are elevated, particularly arsenic, or selenium is low. 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.

TEST REPORT | Comments *continued*

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. 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; 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.