TEST REPORT

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ZRT LABORATORY

 # 2019 03 15 001 U

Ordering Provider:
Dr Getuwell

Samples Received
03/15/2019

Report Date
03/18/2019

Samples Collected
Urine - 03/11/19 03:45
Urine - 03/11/19 22:44

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

Gender	Last Menses	Height	Waist
Female	02/01/2010	5 ft 3 in	Unspecified
DOB	**Menses Status**	**Weight**	**BMI**
7/1/1969 (49 yrs)	Hysterectomy (ovaries removed)	160 lb	28.3

TEST NAME	RESULTS | 03/11/19	RANGE
Iodine	224	100-380 µg/g Cr
Bromine	1507	700-4800 µg/g Cr
Selenium	50	34-220 µg/g Cr
Lithium	60	10-218 µg/g Cr
Arsenic	11	<42 µg/g Cr
Cadmium	0.15	<0.72 µg/g Cr
Mercury	0.10	<1.58 µg/g Cr
Gadolinium	6.5 H	<0.33 µg/g Cr
Thallium	0.67	<0.72 µg/g Cr
Uranium	0.03	<0.10 µg/g Cr
Urinary Creatinine		
Creatinine	1.09	0.3-2.0 mg/mL
Creatinine	1.12	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

50mg SC Pellets Estrogen (type not indicated) (Pharmaceutical) (3 Weeks Last Used);100mg troche Progesterone (compounded) (1 Days Last Used);25mg SC Pellets Testosterone (Pharmaceutical) (3 Weeks Last Used);0.025mg oral Synthroid (T4) (Pharmaceutical) (1 Days Last Used); Clonazepam

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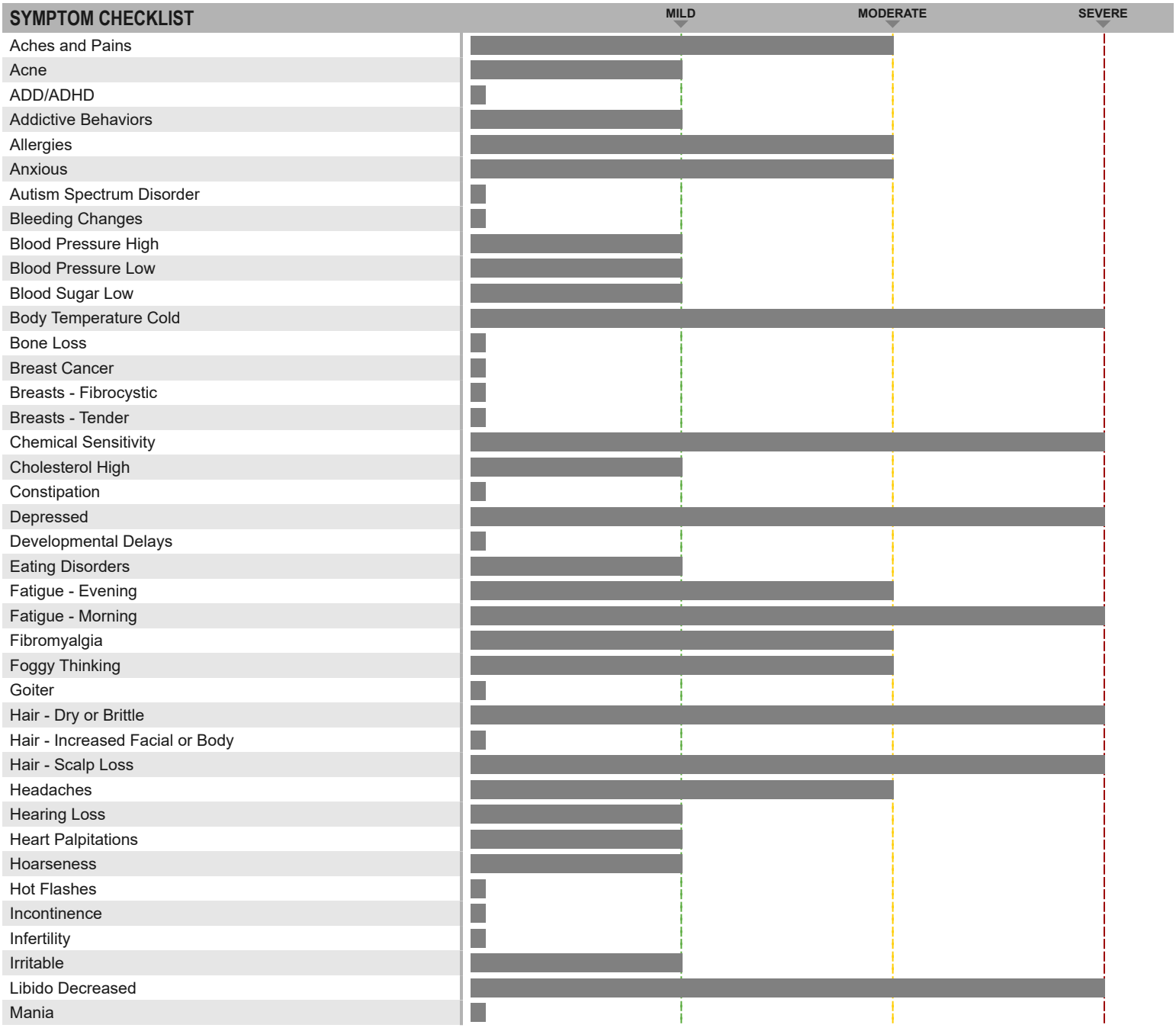
Disclaimer: Symptom Categories below show percent of symptoms self-reported by the patient compared to total available symptoms for each category. For detailed information on category breakdowns, go to www.zrtlab.com/patient-symptoms.

SYMPTOM CATEGORIES	RESULTS | 03/11/19
Estrogen / Progesterone Deficiency	39%
Estrogen Dominance / Progesterone Deficiency	38%
Low Androgens (DHEA/Testosterone)	57%
High Androgens (DHEA/Testosterone)	28%
Low Cortisol	74%
High Cortisol	51%
Hypometabolism	66%
Metabolic Syndrome	51%



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Lab Comments

IODINE:

Urinary iodine/creatinine falls into the reference range that is considered optimal for thyroid hormone synthesis (100-380 ug/g creatinine). The iodine test result represents an average of your urinary iodine 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, and you are not suffering from symptoms of iodine deficiency (mostly related to thyroid dysfunction-hypothyrodism, hyperthyroidism, goiter); then you are likely iodine sufficient. Even though iodine levels may be sufficient, if levels of other halogens, particularly fluorine and/or bromine, or natural goitrogens, such as soy and cruciferous vegetables, are high, these can compete with iodine or inhibit its incorporation into thyroid hormones and contribute to hypothyroid symptoms.

Natural sources of iodine are highest in seafood (fish, seaweed) with lesser amounts found in milk products and eggs. 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 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:

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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:

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.

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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:

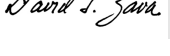
Mercury excretion in this individual's urine is low (within the lower quadrant of the reference range-0.01-1.58 ug/g creatinine). Urine excretion at this level is consistent with low mercury exposure.

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.

GADOLINIUM:

Gadolinium is higher than the expected range. Exposure to gadolinium generally comes from MRIs done with contrast dye done recently. This elevated level may also be due to multiple MRIs with contrast dye done years ago. Additional sources of exposure of individuals to gadolinium comes from industry; although gadolinium contamination of drinking water has also been raised as a concern. When used in contrast dyes, gadolinium is part of a complex called a chelate. This gadolinium chelate is much safer than free gadolinium and able to be excreted more rapidly by the body. Gadolinium has only recently been recognized to be stored in the skin and brain, but is also stored in the liver, kidney, and bone for many years (duration unknown at this time). Osteoporosis may contribute to rising gadolinium through bone degradation releasing gadolinium into circulation. For patients with kidney disorders, there is concern about the dosage of gadolinium given because of compromised excretion leading to the rare condition Nephrogenic Systemic Fibrosis (NSF - scarring in the kidney causing renal failure). Although the scientific community is still analyzing data, other patients will report symptoms within minutes to 1 month after having a gadolinium contrast dye including: skin symptoms (e.g. intense burning, tightness, tingling, and pins and needles), bone or joint pain, foggy thinking, muscle twitches, head pain (reported as different than headache - tight feeling or burning sensation), joint stiffness, gastrointestinal disorders, heart rhythm changes and/or



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metallic taste in the mouth. Most of these individuals appear to have self-resolution within 3-6 months. Symptoms appear most likely if multiple MRIs with dye are used in a short time span. If symptoms are noted by the individual, further gadolinium dye exposure is considered contraindicated. Fortunately, most patients who have an MRI with contrast will experience no symptoms, even if high levels of gadolinium are stored in their tissues. At this time, no treatment is recommended for asymptomatic individuals. Individuals who are experiencing symptoms should seek a healthcare provider familiar with gadolinium.

THALLIUM

Urinary thallium is within the normal reference range, consistent with low thalium exposure.

Thallium is a rare but very toxic heavy metal. The primary source of thallium is coal burning and smelting operations. Thallium can be ingested through fruits and vegetables grown in contaminated soil. Small amounts of thallium can also enter the body from tobacco products.

Thallium is primarily excreted in urine and feces. In the body, thallium accumulates in the bones, kidneys, and the nervous system. Comparably to other heavy metals, thallium increases oxidative stress and disrupts glutathione metabolism and function, thus inhibiting the body's natural defense mechanisms. Signs of acute toxicity are hair loss and nail irregularity, gastrointestinal and respiratory effects, and neurological symptoms like numbness/pain in hands and feet, which are usually reversible when exposure is eliminated.

URANIUM

Urinary uranium is within normal reference range, suggesting lower exposure to this heavy metal.

High uranium levels can come from coal combustion or phosphate fertilizers. The most common sources of exposure to uranium are well water, shellfish and root vegetables. Uranium can also leach from yellow to green-colored ceramics or glassware. Higher uranium levels are naturally found in the mountainous areas of the United States.

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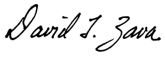

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