



Sample Report TEE03

CLIENT #: 38596

DOCTOR:

Regenerus Laboratories Ltd

Aero 14 Redhill Aerodrome Kings Mill Lane
Redhill, Surrey, RH1 5YP UNITED KINGDOM

SEX: Female

DOB: 12/02/1972

AGE: 46

Toxic Metals; Urine

TOXIC METALS						
		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE	OUTSIDE REFERENCE	
Aluminum	(Al)	4.8	< 35			
Antimony	(Sb)	< dl	< 0.2			
Arsenic	(As)	7.7	< 80			
Barium	(Ba)	1.6	< 7			
Beryllium	(Be)	< dl	< 1			
Bismuth	(Bi)	< dl	< 4			
Cadmium	(Cd)	0.2	< 1			
Cesium	(Cs)	6	< 10			
Gadolinium	(Gd)	< dl	< 0.8			
Lead	(Pb)	4.7	< 2			
Mercury	(Hg)	3.2	< 4			
Nickel	(Ni)	2.1	< 10			
Palladium	(Pd)	< dl	< 0.3			
Platinum	(Pt)	< dl	< 0.1			
Tellurium	(Te)	< dl	< 0.5			
Thallium	(Tl)	0.4	< 0.5			
Thorium	(Th)	< dl	< 0.03			
Tin	(Sn)	0.6	< 5			
Tungsten	(W)	< dl	< 0.4			
Uranium	(U)	< dl	< 0.04			

URINE CREATININE						
	RESULT mg/dL	REFERENCE INTERVAL	-2SD	-1SD	MEAN	+1SD +2SD
Creatinine	29.6	30- 225				

SPECIMEN DATA			
Comments: Vial and requisition are marked pre; sample appears to be post.			
Date Collected: 07/10/2019	pH upon receipt: Acceptable	Collection Period: timed: 6 hours	
Date Received: 07/12/2019	<dl: less than detection limit	Volume: 1500 ml	
Date Reported: 07/16/2019	Provoking Agent: DMSA 500MG	Provocation: PRE PROVOCATIVE	
Method: ICP-MS	Creatinine by Jaffe Method		
Results are creatinine corrected to account for urine dilution variations. Reference intervals and corresponding graphs are representative of a healthy population under non-provoked conditions. Chelation (provocation) agents can increase urinary excretion of metals/elements.			

V13



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 SEX: Female
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Essential Elements; Urine

ESSENTIAL AND OTHER ELEMENTS								
	RESULT/UNIT per creatinine	REFERENCE INTERVAL	PERCENTILE					
			2.5 th	16 th	50 th	84 th	97.5 th	
Sodium (Na)	140 mEq/g	45- 200						
Potassium (K)	110 mEq/g	20- 110						
Phosphorus (P)	540 µg/mg	180- 1100						
Calcium (Ca)	65 µg/mg	30- 350						
Magnesium (Mg)	73 µg/mg	25- 230						
Zinc (Zn)	0.38 µg/mg	0.1- 1.5						
Copper (Cu)	0.051 µg/mg	0.007- 0.06						
Sulfur (S)	770 µg/mg	275- 1200						
Manganese (Mn)	0.001 µg/mg	0.0004- 0.007						
Molybdenum (Mo)	0.016 µg/mg	0.013- 0.15						
Boron (B)	1.3 µg/mg	0.5- 4						
Chromium (Cr)	< dl µg/mg	0.0003-0.0025						
Lithium (Li)	0.022 µg/mg	0.009- 0.2						
Selenium (Se)	0.047 µg/mg	0.03- 0.25						
Strontium (Sr)	0.076 µg/mg	0.045- 0.5						
Vanadium (V)	< dl µg/mg	0.0001-0.0017						
			68 th		95 th			
Cobalt (Co)	< dl µg/mg	< 0.008						
Iron (Fe)	< dl µg/mg	< 1						

URINE CREATININE					
RESULT mg/dL	REFERENCE INTERVAL	-2SD	-1SD	MEAN	+1SD +2SD
29.6	30- 225				

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Date Reported: 07/16/2019	Provoking Agent: DMSA 500MG	Provocation: PRE PROVOCATIVE	
Method: ISE;Na, K Spectrophotometry; P ICP-MS; B, Ca, Cr, Co, Cu, Fe, Mg, Mn, Mo, Se, Sr, S, V, Zn Creatinine by Jaffe method			
Results are creatinine corrected to account for urine dilution variations. Reference intervals and corresponding graphs are representative of a healthy population under non-provoked conditions. Chelation (provocation) agents can increase urinary excretion of metals/elements.			
V13			

INTRODUCTION

This analysis of urinary elements was performed by ICP-Mass Spectroscopy following acid digestion of the specimen. Urine element analysis is intended primarily for: diagnostic assessment of toxic element status, monitoring detoxification therapy, and identifying or quantifying renal wasting conditions. It is difficult and problematic to use urinary elements analysis to assess nutritional status or adequacy for essential elements. Blood, cell, and other elemental assimilation and retention parameters are better indicators of nutritional status.

1) 24 Hour Collections

"Essential and other" elements are reported as mg/24 h; mg element/urine volume (L) is equivalent to ppm. "Potentially Toxic Elements" are reported as µg/24 h; µg element/urine volume (L) is equivalent to ppb.

2) Timed Samples (< 24 hour collections)

All "Potentially Toxic Elements" are reported as µg/g creatinine; all other elements are reported as µg/mg creatinine. Normalization per creatinine reduces the potentially great margin of error which can be introduced by variation in the sample volume. It should be noted, however, that creatinine excretion can vary significantly within an individual over the course of a day.

If one intends to utilize urinary elements analysis to assess nutritional status or renal wasting of essential elements, it is recommended that unprovoked urine samples be collected for a complete 24 hour period. For provocation (challenge) tests for potentially toxic elements, shorter timed collections can be utilized, based upon the pharmacokinetics of the specific chelating agent. When using EDTA, DMPS or DMSA, urine collections up to 12 hours are sufficient to recover greater than 90% of the mobilized metals. Specifically, we recommend collection times of: 9 - 12 hours post intravenous EDTA, 6 hours post intravenous or oral DMPS and, 6 hours post oral bolus administration of DMSA. What ever collection time is selected by the physician, it is important to maintain consistency for subsequent testing for a given patient.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. Because renal excretion is a minor route of excretion for some elements, (Cu, Fe, Mn Zn), urinary excretion may not influence or reflect body stores. Also, renal excretion for many elements reflects homeostasis and the loss of quantities that may be at higher dietary levels than is needed temporarily. For these reasons, descriptive texts are provided for specific elements when deviations are clinically significant. For potentially toxic elements, a descriptive text is provided whenever levels are measured to be higher than expected. If no descriptive texts follow this introduction, then all essential element levels are within acceptable range and all potentially toxic elements are within expected limits.

Reference intervals and corresponding graphs shown in this report are representative of a healthy population under non-provoked conditions. Descriptive texts appear in this report on the basis of measured results and correspond to non-challenge, non-provoked conditions.

Chelation (provocation) agents can increase urinary excretion of metals/elements. Provoked

reference intervals have not been established therefore non-provoked reference intervals shown are not recommended for comparison purposes with provoked test results. Provoked results can be compared with non-provoked results (not reference intervals) to assess body burden of metals and to distinguish between transient exposure and net retention of metals. Provoked results can also be compared to previous provoked results to monitor therapies implemented by the treating physician. Additionally, Ca-EDTA provoked results can be used to calculate the EDTA/Lead Excretion Ratio (LER) in patients with elevated blood levels.

CAUTION: Even the most sensitive instruments have some detection limit below which a measurement cannot be made reliably. Any value below the method detection limit is simply reported as "< dl." If an individual excretes an abnormally high volume of urine, urinary components are likely to be extremely dilute. It is possible for an individual to excrete a relatively large amount of an element per day that is so diluted by the large urine volume that the value measured is near the dl. This cannot automatically be assumed to be within the reference range.

LEAD HIGH

This individual's urine lead (Pb) is higher than expected which means that Pb exposure is higher than that of the general population. A percentage of assimilated Pb is excreted in urine. Therefore the urine Pb level reflects recent or ongoing exposure to Pb and the degree of excretion or endogenous detoxification processes.

Sources of Pb include: old lead-based paints, batteries, industrial smelting and alloying, some types of solders, Ayurvedic herbs, some toys and products from China and Mexico, glazes on (foreign) ceramics, leaded (anti-knock compound) fuels, bullets and fishing sinkers, artist paints with Pb pigments, and leaded joints in municipal water systems. Most Pb contamination occurs via oral ingestion of contaminated food or water or by children mouthing or eating Pb-containing substances. The degree of absorption of oral Pb depends upon stomach contents (empty stomach increases uptake) and upon the essential element intake and Pb status. Deficiency of zinc, calcium or iron increases Pb uptake. Transdermal exposure is significant for Pb-acetate (hair blackening products). Inhalation has decreased significantly with almost universal use of non-leaded automobile fuel.

Lead accumulates extensively in bone and can inhibit formation of heme and hemoglobin in erythroid precursor cells. Bone Pb is released to soft tissues with bone remodeling that can be accelerated with growth, menopausal hormonal changes, osteoporosis, or skeletal injury. Low levels of Pb may cause impaired vitamin D metabolism, decreased nerve conduction, and developmental problems for children including: decreased IQ, hearing impairment, delayed growth, behavior disorders, and decreased glomerular function. Transplacental transfer of Pb to the fetus can occur at very low Pb concentrations in the body. At relatively low levels, Pb can participate in synergistic toxicity with other toxic elements (e.g. cadmium, mercury).

Excessive Pb exposure can be assessed by comparing urine Pb levels before and after provocation with Ca-EDTA (iv) or oral DMSA. Urine Pb is higher post-provocation to some extent in almost everyone. Whole blood analysis reflects only recent ongoing exposure and does not correlate well with total body retention of Pb. However, elevated blood Pb is the standard of care for diagnosis of Pb poisoning (toxicity).

BIBLIOGRAPHY FOR LEAD

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Potassium High

The level of potassium (K) is higher than expected in this sample. Symptoms of elevated K may include mental confusion, weakness, numbness, tingling in the extremities, brady-cardia or irregular heart rhythm and ventricular fibrillation.

K is an electrolyte and a potentiator of enzymatic reactions in the body. Elevated K in hair may reflect overall retention of K by the body or maldistribution of this element. In adrenocortical insufficiency, K is increased in blood, while it is decreased in urine; cellular K may or may not be increased.

Appropriate tests to confirm excess K in body tissues may include measurements of packed red blood cell K; serum or whole blood K and sodium/K ratio. An assessment of adrenocortical function may be indicated for symptomatic patients with a confirmed elevation in serum K.

CALCIUM LOW

This individual's urine calcium is lower than one standard deviation below the mean of the reference population and corresponds to the lower 17% (approximately) of that population.

Low urinary calcium may be the result of: insufficient dietary intake, insufficient gastric acidification, inadequate vitamin D (or vitamin D function), or excessive phosphates, oxalates

(spinach) or phytates (cereal grains) which may form insoluble calcium salts in the intestine. Intestinal absorption of calcium also is hindered in cases of lipid malabsorption; undigested fats can form insoluble calcium compounds. A very low protein diet or an overly alkaline intestine (pH > 7.5 approx.) can result in poor calcium uptake. Insufficient acidophilic flora, such as Lactobacilli, can impair calcium uptake (Harper, Rev. Phys.Biochem. 17th ed.p.576). Correction of dietary imbalances typically normalizes calcium uptake within several days; urine levels may take longer to normalize if there is need for calcium deposition in body issues.

Use of thiazide diuretics decreases calcium concentration in urine.

Pathological conditions that may feature subnormal urine calcium include: hypoparathyroidism, gastric hypochlorhydria, gastrointestinal malabsorption featuring impaired vitamin D uptake, lack of sunlight for vitamin D activation, steatorrhea, fatty acid metabolism disorder, some types of hypertension, tetany (serum calcium ion concentration also low), pre-eclampsia, genetic hypocalciuric hypercalcemia (elevated blood Ca), renal osteodystrophy, and vitamin D-resistant rickets.

Dietary deficiency or poor absorption of calcium increases the absorption of lead, increases blood and tissue levels of lead and, enhances the adverse effects of lead on cognitive function and behavior.

Urine analysis is not a preferred way to assess body calcium stores, and nutritional sufficiency of calcium should be assessed through dietary analysis. Whole blood calcium level, serum calcium level, serum vitamin D level (1,25-dihydroxy), parathyroid hormone determinations, and bone density measurement are tests that are more indicative of calcium status.

BIBLIOGRAPHY FOR CALCIUM

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COPPER HIGH

Significantly elevated copper in urine can be secondary to provocative challenge with sulfhydryl (-SH) bearing agents such as D-penicillamine ("Cuprimine"), DMSA, or DMPS. Large, multi-gram doses of vitamin C (ascorbic acid), administered orally or intravenously, may slightly or moderately increase excretion of copper.

Increased urinary copper can be an artifact of nutritional supplementation with copper or come from drinking water that is high in copper content. Acidic water carried in copper pipes can dissolve some copper which increases the copper intake if used for drinking or cooking.

Molybdenum supplementation at high levels or if inappropriate may cause increased copper excretion; molybdenum and copper are mutually antagonistic in terms of body retention.

Bacterial or other infections may cause hypercupremia with attendant or delayed hypercuprinuria. This is transient and follows the inflammatory stage of the disease. Published studies such as Vivoli, *Sci Total Environ*, 66 p. 55-64, 1987 have correlated increased urinary copper with increased blood pressures in hypertensives. Biliary obstruction or insufficiency can decrease normal excretion of copper via the bile while increasing blood and urinary levels. Proteinuria also may feature increased copper levels.

Hyperaminoacidurias that include histidinuria can result in urinary copper wasting because histidine is a powerful chelator of copper. Hyperaminoacidurias that include histidine can be of many origins including: genetic factors, chemical or elemental toxicities, infectious agents, hyperthyroidism, sugar intolerances, nephrotic syndromes, etc.

In Wilson's disease, urinary copper is generally increased (above 100 micrograms/24 hours) without provocation or chelation. Use of D-penicillamine or DMPS as a provocative diagnostic procedure can yield a 5 - 10X increase in urinary copper levels in normal individuals. In contrast, Wilson's disease patients may then excrete 50-100 times the normal levels or 1000 to 2000 mcg/24 hr. (Walshe, *J. Rheumatology (supp/7)* 8 p.3-8, 1981).

Urine analysis (unprovoked) is not an adequate procedure to assess copper stores or copper metabolism. Blood levels, erythrocyte copper content, erythrocyte superoxide dismutase activity, and serum ceruloplasmin are other more indicative measurements for copper status.

BIBLIOGRAPHY FOR COPPER

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MOLYBDENUM LOW

This individual's molybdenum level is lower than one standard deviation below the mean of the reference population which means that this individual's urine molybdenum level corresponds to the lowest 17% (approximately) of that population.

Molybdenum is an essential activator of some important enzymes in the body: sulfite oxidase (catalyzes formation of sulfate from sulfite), xanthine oxidase (formation of uric acid and superoxide ion from xanthine), and aldehyde oxidase (processes aldehydes). Over 50% of absorbed Mo is normally excreted in urine; the remainder is excreted via bile to the feces or is excreted in sweat.

The level of molybdenum in urine may be a transient finding and may not reflect body tissue or

liver levels. In copper deficiency, retention of molybdenum is increased (tissue levels could be normal or high), while urine levels might be subnormal. In renal insufficiency, molybdenum (and other elements) can be low in urine. Creatinine clearance and blood metabolite levels should be measured if a renal transport disorder is suspected.

Individuals receiving prolonged total parenteral nutrition ("TPN") may have low body tissue and urine levels of molybdenum because it is occasionally omitted from TPN formulations.

Molybdenum in foods is mostly in soluble complexes, and only a small amount is required daily (100 to 200 micrograms, adults). Therefore, frank molybdenum deficiency is uncommon. However, GI dysfunctions, poor-quality diet, and stressors can combine to produce inadequate molybdenum. Tungsten is a powerful antagonist of molybdenum retention, copper less so. Episodic exposures to high levels of either may result in periods of low Mo excretion that follow prior periods of high Mo excretion. Sulfites, aldehydes and high amounts of purines in the diet may increase need for and retention of molybdenum. Prolonged use of dithiol chelators (DMPS, DMSA) or MSM can result in poor molybdenum status as indicated by low levels in red blood cells (DDI observations).

A multielement hair analysis plus analyses for serum and urine uric acid can be used to confirm or rule out molybdenum insufficiency.

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CHROMIUM LOW

The chromium level in this urine sample is low. Chromium (Cr) is essential for proper metabolism of glucose in humans. It potentiates the action of insulin via glucose tolerance factor (GTF) which is Cr+3 bound in a dinicotinic acid-glutathione complex. Other functions of Cr include aiding in lipid metabolism and assisting with HDL/LDL cholesterol balance.

Significance of Low Chromium: Clinical findings consistent with Cr deficiency are those of GTF insufficiency including diabetes, hyperglycemia, and possibly transient hyper/hypoglycemia. Excessive LDL cholesterol also may be consistent with Cr deficiency. Some investigators have linked Cr deficiency to ischemic heart disease and atherosclerosis.

Other Useful Analyses: Urine Toxic Metals and Essential Elements provocative testing with EDTA can be used to assess Cr stores.

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Vanadium Low

A low level of Vanadium (V) was found in this urine sample. Excessively low urinary V excretion may reflect a deficiency state due to poor dietary intake and/or poor absorption (less than 5% of dietary V is absorbed).

Dietary vanadium is found in seafood, eggs, black pepper, mushrooms, dill seed, parsley, soy, corn, olive oil, radishes and other root vegetables, lettuces, nuts, strawberries and gelatin. A balanced diet may provide 10 to 30 mcg of V per day. This trace element is important in cellular metabolism, bone and tooth formation, reproduction and growth. Also, V appears to be involved in glucose metabolism.

There are no known symptoms of V deficiency. Although trace amounts of V may have essential metabolic functions, over-zealous supplementation of V is not warranted. There is no RDA for V but, if supplementation is warranted, a common daily dose of tetravalent vanadyl sulfate is 20 to 30 mcg per day.

Diabetics should not use supplemental V as the sole intervention in the management of their diabetes and should only use it with the advice of their attending practitioner. People with hypoglycemia should not use supplemental V as it may further lower blood glucose.

A more direct confirmatory test for V deficiency is the Doctor's Data whole blood vanadium test.

