Nordic Laboratories

PATIENT:	**Sample Report**			TEST REF:	**TST-##-#####**
TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy		
GENDER:	Male	TESTED:	dd/mm/yyyy	ADDRESS:	
AGE:	2				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Urine Toxic and Essential Elements (timed or 24 hour) Pre

Toxic Metals; Urine

TOXIC METALS		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	**16**	< 100	
Antimony	(Sb)	**< dl**	< 0.7	
Arsenic	(As)	**120**	< 120	
Barium	(Ba)	**9.2**	< 8	
Beryllium	(Be)	**< dl**	< 1	
Bismuth	(Bi)	**< dl**	< 2	
Cadmium	(Cd)	**< dl**	< 0.5	
Cesium	(Cs)	**10**	< 15	
Gadolinium	(Gd)	**71**	< 0.5	
Lead	(Pb)	**3.7**	< 3	
Mercury	(Hg)	**1.2**	< 4.5	
Nickel	(Ni)	**3.1**	< 18	
Palladium	(Pd)	**< dl**	< 0.3	
Platinum	(Pt)	**< dl**	< 0.1	
Tellurium	(Te)	**< dl**	< 0.5	
Thallium	(Tl)	**1**	< 1	
Thorium	(Th)	**< dl**	< 0.1	
Tin	(Sn)	**0.7**	< 15	
Tungsten	(W)	**0.6**	< 1	
Uranium	(U)	**1.3**	< 0.05	

URINE CREATININE	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	**59.4**	15– 120	

SPECIMEN DATA		
Comments: **results checked**		
Date Collected: **dd/mm/yyyy**	pH upon receipt: **Acceptable**	Collection Period: **Random**
Date Received: **dd/mm/yyyy**	<dl: **less than detection limit**	Volume:
Date Completed: **dd/mm/yyyy**	Provoking Agent:	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.

Nordic Laboratories

PATIENT:	**Sample Report**			TEST REF:	**TST-##-#####**
TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy	ADDRESS:	
GENDER:	Male	TESTED:	dd/mm/yyyy		
AGE:	2				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Urine Toxic and Essential Elements (timed or 24 hour) Pre

Essential Elements; Urine

ESSENTIAL AND OTHER ELEMENTS		RESULT/UNIT per creatinine		REFERENCE INTERVAL	PERCENTILE 2.5th 16th 50th 84th 97.5th
Sodium	(Na)	**220**	mEq/g	45– 350	
Potassium	(K)	**150**	mEq/g	30– 250	
Phosphorus	(P)	**1870**	µg/mg	300– 2200	
Calcium	(Ca)	**53**	µg/mg	30– 300	
Magnesium	(Mg)	**130**	µg/mg	30– 350	
Zinc	(Zn)	**0.69**	µg/mg	0.2– 3	
Copper	(Cu)	**0.12**	µg/mg	0.008– 0.07	
Sulfur	(S)	**1460**	µg/mg	330– 2100	
Manganese	(Mn)	**0.005**	µg/mg	0.0008– 0.025	
Molybdenum	(Mo)	**0.15**	µg/mg	0.025– 0.3	
Boron	(B)	**2**	µg/mg	1– 10	
Chromium	(Cr)	**0.003**	µg/mg	0.0007– 0.005	
Lithium	(Li)	**0.071**	µg/mg	0.012– 0.3	
Selenium	(Se)	**0.089**	µg/mg	0.04– 0.35	
Strontium	(Sr)	**0.089**	µg/mg	0.06– 0.65	
Vanadium	(V)	**0.004**	µg/mg	0.0002–0.0025	
					68th 95th
Cobalt	(Co)	**< dl**	µg/mg	< 0.007	
Iron	(Fe)	**< dl**	µg/mg	< 1	

URINE CREATININE	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	**59.4**	15– 120	

SPECIMEN DATA			
Comments:	**results checked**		
Date Collected:	**dd/mm/yyyy**	pH Upon Receipt: **Acceptable**	Collection Period: **Random**
Date Received:	**dd/mm/yyyy**	<dl: **less than detection limit**	Volume:
Date Completed:	**dd/mm/yyyy**	Provoking Agent:	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

TEST NAME: Urine Toxic and Essential Elements (timed or 24 hour) Pre

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.

Barium High

Barium (Ba) has not been established to be an essential element. Elevated levels of Ba often are observed after exposure to Ba (a contrast agent) during diagnostic medical tests (e.g. "barium swallow", "upper GI series", "barium enema", etc.). Elevated levels of Ba may interfere with calcium metabolism and potassium retention. Acutely high intake of soluble Ba-salts (nitrates, sulfides, chlorides) can be toxic. Chronic exposure to Ba may be manifested by muscular and myocardial stimulation, tingling in the extremities, and loss of tendon reflexes.

Brazil nuts and peanuts/peanut butter are very high in Ba so urine Ba may be elevated shortly after consumption of these foods; toxic effects would not be anticipated under such conditions. Although Ba is poorly absorbed orally (<5%) it can be very high in peanuts and peanut butter (about 3,000 nanograms/gram), frozen and fast foods such as burgers, fries, and hot dogs (400-500 nanograms/gram). It is noteworthy that Ba intake is much higher in children than adults (Health Canada 2005, www.atsdr.cdc.gov/toxprofiles/tp24-c6.pdf).

Ba is surprisingly abundant in the Earth's crust, being the 14th most abundant element. High amounts of Ba may be found in soils and in food, such as nuts (e.g. brazil nuts), seaweed, fish and certain plants. Because of the extensive use of barium in industry, human activitiesadd greatly to the release of barium in the environment. As a result barium concentrations in air, water and soil may be higher than naturally occurring concentrations in many locations. It can also enter the air during coal and oil combustion. Barium compounds are used by the oil and gas industries to make drilling mud. Drilling mud simplifies drilling through rocks by lubricating the drill. Barium compounds are also used to make paint, brics, tiles, glass, and rubber. Soluble Ba compounds are highly toxic and may be used as insecticides. Ba-aluminates are utilized for water purification, acceleration of concrete solidification, production of synthetic zeolites, and in the paper and enamel industries.

Ba levels (and the levels of 16 other elements) in water can be assessed with water testing as provided by DDI. A possible confirmatory test for excessive Ba is measurement of blood electrolytes as hypokalemia may be associated with excessive Ba in the body. Hair elements

analysis may provide further evidence of exosure to Ba.

Gadolinium High

This individual's urine level of Gadolinium (Gd) is higher than expected. Gadolinium is one of the most abundant "rare-earth" elements but is never found as a free element in nature. Gadolinium has no known biological role in humans.

Toxicity is rarely associated with Gd due to its poor gastrointestinal absorption (it is suspected that very little Gd is absorbed from the gastrointestinal tract (<0.05%). If exposure to high enough doses and/or if absorption does occur, symptoms of acute toxicity may develop, including abdominal cramps, diarrhea, lethargy, muscular spasms, and even eventual death due to respiratory collapse. Gadolinium salts can cause irritation of the skin and eyes and are suspected to be possible carcinogens. As reported by Perazella (2009) Gadollinium-based contrast (GBC) agents have been linked on occasion with a rare systemic fibrosing condition called nephrogenic systemic fibrosis (NSF) and their use in patients with even mild kidney disease should be avoided (parenteral administration).

Gd is often used in alloys (e.g. chromium, iron). Other technical uses include the phosphors of color television tubes and in making magnets and electronic components such as recording heads for video recorders and in the manufacture of compact disks and computer memory. In medicine Gd, chelated with diethylenetriaminepentaacetic acid (DTPA), is used as a non-radioactive contrasting agent in magnetic resonance imaging and has a half-life in blood of about 90 minutes. However, residual Gd is retained in tissues for quite some time. It is also used in control rods for nuclear reactors and power plants, in making garnets for microwave applications.

EDTA effectively chelates Gd therefore urinary Gd might be higher than average post-Ca-EDTA provocation, particularly in patients who have had Gd-enhanced MRIs.

References:

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Perazella M. Current Status of Gadollinium Toxicity in Patients with Kidney Disease. Clin J Am Soc Nephrol 4: 461-69, 2009.

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, Ayruvedic 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 in 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).

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URANIUM HIGH

This individual’s urine uranium (U) is markedly higher than that of the general population. Renal excretion is the primary route of U excretion. This finding is consistent with an excessive exposure to uranium or to an unusually high body burden of this element.

TEST NAME: Urine Toxic and Essential Elements (timed or 24 hour) Pre

Uranium is a radioactive element having 10 isotopes with half lives that exceed one hour. U238 constitutes about 99% of the naturally-occurring uranium and this is the isotope measured at DDI and reported for this individual. U238 has a half life of 4.5 X 10 to the ninth years. It decays by alpha emission to produce thorium, Th234, the initial step in a decay chain that eventually leads to lead. Alpha, beta and gamma emissions occur during this decay process. Because of the very long half life, the radioactivity danger is only slight. However, exposure to enriched or nuclear fuel grade U (high in U235) does pose a health hazard. The measured result (U238) does not reflect or imply exposure to enriched U235.

The major concern for (natural) uranium excess is toxochemical rather than radiochemical. Uranium is a chemically-reactive element, has four valences (3,4,5 or 6), and may combine with: carbonate, phosphate, citrate, pyruvate, malate, lactate, etc. in body tissues. When not excreted in urine, it may accumulate in the kidneys, spleen, liver, and in bone (substituting for calcium in hydroxyapatite). Uranium is nephrotoxic, causing damage to the glomeruli and proximal tubules. An early sign of U excess is general fatigue. Renal damage is reflected by proteinuria, hyper-aminoaciduria and glucosuria. Albuminuria and urinary catalase are findings consistent with U excess. Elevated hair U may provide further information regarding U exposure. Whole blood analysis may corroborate very recent or ongoing exposure. There are no currently available metal binding/chelating agents to assess the net retention of U that may have occurRed over time.

Uranium is more common than mercury, silver or cadmium in the earth's rock strata, and may be present, at low levels, in ground (drinking) water. Most commercial use of U is for nuclear fuel, but it may be present in ceramics or colored glass, especially ancient or antique, yellow-colored glass.

BIBLIOGRAPHY FOR URANIUM

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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 metabo- lism 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.

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Vanadium High

A high level of Vanadium (V) was found in this urine sample. Increased V, especially in an unprovoked urine sample, reflects recent excessive exposure/intake and absorption to V.

Vanadium can be highly toxic. Excess levels of V can result from over-zealous V supplementation. It may also result from chronic consumption of fish, shrimp, crabs, and oysters that have been harvested near offshore oil rigs. Industrial/environmental sources of V include: processing of mineral ores, phosphate fertilizers, combustion of oil and coal, production of steel, and chemicals used in the fixation of dyes and print (Metals in Clinical and Analytical Chemistry, 1994). V is used in producing rust-resistant, spring and high speed tool steels. Vanadium pentoxide and other vanadates are used as catalysts in the production of sulfuric acid and formaldehyde. Urban air in industrialized areas may have higher levels of V than in rural areas.

Symptoms of V toxicity vary with chemical form and route of assimilation. Inhalation of excess V may produce respiratory irritation and bronchitis. Excess ingestion of V can result in decreased appetite, depressed growth, diarrhea/gastrointestinal disturbances, nephrotoxic and hematotoxic effects. Pallor, diarrhea, and green tongue are early signs of excess V and have been reported in human subjects consuming about 20 mg V/day (Modern Nutrition in Health and Disease, 8th edition, eds. Shils, M., Olson, J., and Mosha, S., 1994).

Nordic Laboratories

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PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy	ADDRESS:	
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TEST NAME: Urine Toxic and Essential Elements (timed or 24 hour) Pre

A confirmatory test for excess exposure to V is the Doctor's Data the whole blood vanadium test. EDTA (but not DMPS or DMSA) is an effective chelator of V. Therefore excessive retention (body burden) of V can be assessed by comparing pre- and post-Ca-Na2-EDTA urine V levels.