Nordic Laboratories

Test #	Practitioner Name
Patient #	Practitioner Address
TST #	
Patient Name	
Sex Age	

Toxic Metals; Urine

TOXIC METALS		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	5.5	< 35	
Antimony	(Sb)	0.2	< 0.2	
Arsenic	(As)	7.6	< 80	
Barium	(Ba)	1.7	< 7	
Beryllium	(Be)	< dl	< 1	
Bismuth	(Bi)	< dl	< 4	
Cadmium	(Cd)	< dl	< 1	
Cesium	(Cs)	9.6	< 10	
Gadolinium	(Gd)	< dl	< 0.8	
Lead	(Pb)	43	< 2	
Mercury	(Hg)	1	< 4	
Nickel	(Ni)	3.6	< 10	
Palladium	(Pd)	< dl	< 0.15	
Platinum	(Pt)	< dl	< 0.1	
Tellurium	(Te)	< dl	< 0.5	
Thallium	(Tl)	0.7	< 0.5	
Thorium	(Th)	< dl	< 0.03	
Tin	(Sn)	0.3	< 5	
Tungsten	(W)	0.1	< 0.4	
Uranium	(U)	< dl	< 0.04	

URINE CREATININE	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	37.6	30– 225	

SPECIMEN DATA

Comments:

Date Collected: 06/28/2015
Date Received: 07/02/2015
Date Completed: 07/06/2015
Method: ICP-MS

pH upon receipt: Acceptable
<dl: less than detection limit
Provoking Agent: Creatinine by Jaffe Method

Collection Period: timed: 6 hours
Volume: 1250 ml
Provocation: POST PROVOCATIVE

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.

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Nordic Laboratories

Test #	Practitioner Name
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Essential Elements; Urine

ESSENTIAL AND OTHER ELEMENTS		RESULT/UNIT per creatinine		REFERENCE INTERVAL	PERCENTILE 2.5th 16th 50th 84th 97.5th
Sodium	(Na)	49	mEq/g	45 – 200	
Potassium	(K)	94	mEq/g	20 – 110	
Phosphorus	(P)	500	µg/mg	180 – 1100	
Calcium	(Ca)	45	µg/mg	30 – 350	
Magnesium	(Mg)	69	µg/mg	25 – 230	
Zinc	(Zn)	0.49	µg/mg	0.1 – 1.5	
Copper	(Cu)	0.099	µg/mg	0.007 – 0.06	
Sulfur	(S)	620	µg/mg	275 – 1200	
Manganese	(Mn)	0.014	µg/mg	0.0004 – 0.007	
Molybdenum	(Mo)	0.015	µg/mg	0.013 – 0.15	
Boron	(B)	2.2	µg/mg	0.5 – 4	
Chromium	(Cr)	0.002	µg/mg	0.0003–0.0025	
Lithium	(Li)	0.027	µg/mg	0.009 – 0.2	
Selenium	(Se)	0.035	µg/mg	0.03 – 0.25	
Strontium	(Sr)	0.085	µg/mg	0.045 – 0.5	
Vanadium	(V)	< dl	µg/mg	0.0001–0.0017	
					68th 95th
Cobalt	(Co)	< dl	µg/mg	< 0.008	
Iron	(Fe)	0.18	µg/mg	< 1	

URINE CREATININE	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	37.6	30 – 225	

SPECIMEN DATA

Comments:

Date Collected: 06/28/2015
Date Received: 07/02/2015
Date Completed: 07/06/2015
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

pH Upon Receipt: Acceptable
<dl: less than detection limit
Provoking Agent:

Collection Period: timed: 6 hours
Volume: 1250 ml
Provocation: POST PROVOCATIVE

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

Nordic Laboratories

Test #	Practitioner Name
Patient #	Practitioner Address
Patient Name	
Sex Age	

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 exceeds three times the upper expected limit per the reference population. Because a percentage of absorbed or assimilated lead is excreted in urine, the urine lead level reflects recent or ongoing exposure to lead and the degree of excretion or detoxification.

Sources of lead include: old lead-pigment paints, batteries, industrial smelting and alloying, some types of solders, ayruvedic herbs, some toys and products from China, glazes on (foreign) ceramics, leaded (antiknock compound) fuels, bullets and fishing sinkers, artist paints with lead pigments, and leaded joints in some municipal water systems. Most lead contamination occurs via oral ingestion of contaminated food or water or by children mouthing or eating lead-containing substances. The degree of absorption of oral lead depends upon stomach contents (empty stomach increases uptake) and upon the body's mineral status. Deficiency of zinc, calcium or iron may increase lead uptake. Transdermal exposure is slight. Inhalation has decreased significantly with almost universal use of non-leaded automobile fuel.

Lead accumulates extensively in bone and inhibits formation of heme and hemoglobin in erythroid precursor cells. Bone lead is released to soft tissues with bone remodeling that can be accelerated with growth, menopausal hormonal changes and osteoporosis. Lead has physiological and pathological effects on body tissues that may be manifested from relatively low lead levels up to acutely toxic levels. In children, developmental disorders and behavior problems may occur at relatively low levels: loss of IQ, hearing loss, poor growth. In order of occurrence with increasing lead concentration, the following can occur: impaired vitamin D metabolism, initial effects on erythrocyte and erythroid precursor cell enzymology, increased erythrocyte protoporphyrin, headache, decreased nerve conduction velocity, metallic taste, loss of appetite, constipation, poor hemoglobin synthesis, colic, frank anemia, tremors, nephrotoxic effects with impaired renal excretion of uric acid, neuropathy and encephalopathy. At relatively low levels, lead can participate in synergistic toxicity with other toxic elements (e.g. cadmium, mercury).

Excessive retention of lead can be assessed by urinalysis after provocation with Ca-EDTA (iv)

or oral DMSA. Whole blood analysis can be expected to reflect onlyrecent exposures and does not correlate well with total body burden of lead.

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THALLIUM HIGH

This individual's urine thallium (Tl) is higher than expected, but associated symptoms or toxic effects may or may not be presented. Presentation of symptoms can depend upon several factors including: chemical form of the Tl, mode of assimilation, severity and duration of exposure, and organ levels of metabolites and nutrients that effect the action of Tl in the body.

Thallium can be assimilated transdermally, by inhalation, or by oral ingestion. Both valence states can have harmful effects: Tl+1 may displace potassium from binding sites and influences enzyme activities; Tl+3 affects RNA and protein synthesis. Tl is rapidly cleared from blood and is readily taken up by tissues. It can be deposited in kidneys, pancreas, spleen, liver, lungs, muscles, neurons and the brain. Blood is not a reliable indicator of Tl exposure.

Symptoms that may be associated with excessive Tl exposure are often delayed. Early signs of chronic, low-level Tl exposure and retention may include: mental confusion, fatigue, and peripheral neurological signs: paresthesias, myalgias, tremor and ataxia. After 3 to 4 weeks, diffuse hair loss with sparing of pubic and body hair and a lateral fraction of eye- brows usually occurs. Increased salivation occurs less commonly. Longer term or residual symptoms may include: alopecia, ataxia, tremor, memory loss, weight loss, proteinuria (albuminuria), and possibly psychoses. Ophthalmologic neuritis and strabismus may be presented.

Environmental and occupational sources of Tl include: contaminated drinking water, airborne plumes or waste streams from lead and zinc smelting, photoelectric, electrochemical and electronic components (photoelectric cells, semiconductors, infrared detectors, switches), pigments and paints, colored glass and synthetic gem manufacture, and industrial catalysts used

in some polymer chemistry processes. Thallium is present in some ”weight loss” supplements (e.g. Active 8) at undisclosed levels (”trade secret”).

Hair (pubic or scalp) element analysis may be used to test for suspected Tl exposure. Although urine is the primary natural route for excretion of thallium, the biliary/fecal route also contributes. Therefore, fecal metals analysis provides a confirmatory test for chronic ongoing exposure to Tl. Clinical findings that might be associated with excessive Tl are: albuminuria, EEG with diffuse abnormalities, hypertension, and elevated urine creatinine phosphokinase (CPK). No provocation agents are currently available to estimate Tl retention by means of urinalysis.

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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.

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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.

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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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MANGANESE HIGH

This individuals urine manganese (Mn) is higher than expected. High urinary MN may or may not correspond to global Mn excess or Mn loss from body tissues because the normal route for Mn excretion is via the bile (feces). Typically, less than onehalf of one percent of total manganese excretion occurs via urine, 3-5% occurs in sweat; the remainder (approx. 95%) occurs via intestinal transport (bile) and feces. Hence urinary Mn may be increased in patients with biliary obstruction or cirrhosis. Urinary Mn levels may fluctuate without reflecting or influencing body stores.

Elevated urinary Mn in increased following intravenous administration of EDTA; levels increase as much as 15-X compared to pre-EDTA levels in healthy adults without evidence of manganese overload (unpublished observations, DDI). D-penicillamine. DMSA and DMPS administration also may result in increases in urinary Mn levels.

Manganese excesses in urine (without provocative challenge) are featured in renal wasting syndromes, nephritis, biliary insufficiency or obstruction, and dietary overload or inappropriate or excessive supplementation. Some hormones and drugs inhibit biliary excretion of manganese resulting in increased urinary excretion.

Environmental or industrial sources of Mn include: mining, refining and processing metals or ores, metal alloying, welding, some types of batteries, glazes and pigments, catalysts (petrochemical, plastics and synthetic rubber industries), and the gasoline additive, "MMT". Ground water used as drinking water may contain Mn, and a 1975 USEPA survey of city drinking waters showed variability from < 5 to 350 mcg/L ("Drinking Water and Health", U.S. Printing & Publishing Office, Nat. Acad. of Sci., Washington DC, 1977). Some herbs and teas may containhigh concentrations of Mn.

Relative to other essential trace elements, excessive Mn retention can be neurotoxic. Inhalation, as a result of occupational exposure, is the route of assimilation that is most harmful. Some symptoms and manifestations of excess Mn exposue include:psychiatric disturbances (hyperirritability, hallucinations, violence), tremor,Parkinson's-like symptoms, anorexia, sexual impotence, and speech disturbance.

Because urine is not a reliable indicator of manganese status, other laboratory tests are advised if Mn excess is suspected. These are: whole blood elemental analysis, red blood cell elements analysis, and possibly hair elemental analysis.

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Patient Name
Sex Age
Practitioner Name
Practitioner Address

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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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SELENIUM LOW

Urine accounts for about one-half of the total body excretion of dietary selenium when normal amounts are ingested. Seafood, organ meats, cereal grains, and seleniferous vegetables (garlic, onions) are good dietary sources. Selenium is also excreted in sweat, and lesser amounts are present in fecal matter. Because diets are highly variable in selenium content, urine is not a reliable indicator of selenium adequacy or function.

Low urinary selenium may be a consequence of: junk food diet or highly-processed food diet, gastrointestinal dysfunctions, renal insufficiency (in which case other elements will be subnormal in urine but possibly elevated in blood), and long-term parenteral nutrition or special diets that are low in selenium.

Selenium is a necessary element for proper activity of two enzymes in human metabolism: glutathione peroxidase (GPx) and iodothyronine deiodinase (ITD). Selenium deficiency may cause weakness or rate limitation for one or both of these enzymes. GPx oxidizes glutathione while reducing oxidized lipids. Weak GPx activity may allow excessive inflammation to occur. ITD deiodinates thyroxine prohormone and catalyzes T4 _ T3. Selenium deficiency may be a cause of insufficient T3 and thyroid dysfunction (Berry J.M. Nature 349, 1991 pp.438-40).

Symptoms consistent with selenium deficiency include: myalgia, increased inflammatory responses, hypothyroidism with low T3. Cardiomyopathy and Keshan disease can occur in cases of severe, chronic Se deficiency. Subnormal selenium may accentuate the effects of cadmium, mercury or arsenic overload. Confirmatory tests for selenium status include packed red blood cell elements, and hair elemental analysis (provided that antidandruff shampoos have not been used.

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