URINE TOXIC METALS

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LAB#: U080708-0445-1
PATIENT: Servando Perez-Dominguez
SEX: Male
AGE: 40

CLIENT#: 22844
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POTENTIALLY TOXIC METALS

METALS	RESULT µg/g CREAT	REFERENCE RANGE	WITHIN REFERENCE RANGE / ELEVATED / VERY ELEVATED
Aluminum	**< dl**	< 25	
Antimony	**0.2**	< 0.6	
Arsenic	**200**	< 120	
Beryllium	**< dl**	< 0.5	
Bismuth	**0.6**	< 10	
Cadmium	**0.4**	< 2	
Lead	**6.4**	< 5	
Mercury	**76**	< 3	
Nickel	**0.4**	< 10	
Platinum	**< dl**	< 1	
Thallium	**0.9**	< 0.7	
Thorium	**< dl**	< 0.3	
Tin	**6.4**	< 9	
Tungsten	**< dl**	< 0.7	
Uranium	**< dl**	< 0.1	

CREATININE

	RESULT mg/dL	REFERENCE RANGE	2SD LOW / 1SD LOW / MEAN / 1SD HIGH / 2SD HIGH
Creatinine	**47**	45- 225	

SPECIMEN DATA

Comments:

Date Collected: **7/1/2008**
Date Received: **7/8/2008**
Date Completed: **7/10/2008**

Method: **ICP-MS**
<dl: **less than detection limit**
Provoking Agent: **DMPS**

Collection Period: **timed: 6 hours**
Volume:
Provocation: **POST PROVOCATIVE**

Toxic metals are reported as µg/g creatinine to account for urine dilution variations. **Reference ranges are representative of a healthy population under non-challenge or non-provoked conditions.** No safe reference levels for toxic metals have been established.

V10.00

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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.

For essential elements, the mean and the reference ranges apply to human urine under non-challenge, non-provocation conditions. Detoxification therapies can cause significant deviations in essential element content of urine. For potentially toxic elements, the expected range also applies to conditions of non-challenge or non-provocation. Diagnostic or therapeutic administration of detoxifying agents frequently raise the urinary levels content of potentially

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toxic elements. Descriptive texts appear in this report on the basis of measured results and correspond to non-challenge, non-provocation conditions.

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.

ARSENIC HIGH

This inidividual's urine arsenic is higher than expected. Because urine is the major mode of excretion for arsenic, an elevated level reflects increased intake or body burden of this element. Occasional ingestion of arsenic in seafood is not uncommon and is not serious if the body can detoxify and clear this arsenic before it accumulates and inhibits metabolic processes. Very low levels of arsenic are postulated by some authors to be essential or at least beneficial. Arsenobetaine and arsinocholine, commonly found in shellfish are relatively non-toxic and 90% is excreted in the urine.

Sources of arsenic include: contaminated foods (especially seafood), water or medications. Industrial sources are: ore smelting/refining/processing plants, galvanizing, etching plating processes. Tailing from or river bottoms near gold mining areas (past or present) may contain arsenic. Insecticides, rodenticides and fungicides (Na-, K- arsenites, arsenates, also oxides are commercially available). Commercial arsenic products include: sodium arsenite, calcium arsenate, lead arsenate and "Paris green" which is cupric acetoarsenite, a wood preservative (pressure treated wood).

Chronic exposure to or ingestion of arsenic causes tissue levels to gradually increase as the element binds to sulfur, phosphorus and selenium. An important detrimental effect is inactivation of lipoic acid,a vitamin cofactor needed for metabolism of pyruvate and alpha-ketoglutarate.

Symptoms consistent with mild or moderate arsenic exposure include: fatigue, malaise, eczema or allergic-like dermatitis, and garlic-like breath. Increased salivation may occur. Hair element analysis can be done for corroborative evidence of arsenic excess. Blood arsenic levels are not dose related and may or may not reflect arsenic exposure or body burden. Following detoxification treatments with sulfhydryl agents (D-penicillamine, DMSA, DMPS, urine levels of arsenic may exceed the expected range by 2X or 3X depending upon body burden and dietary intake. This does not necessarily indicate arsenic excess to the point of toxicity or physiological impairment.

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LEAD HIGH

This individual's urine lead is higher than expected which means that lead intake or body burden is higher than that of the reference population.

Sources of lead include: old lead-pigment paints, batteries, industrial smelting and alloying, some types of solders, glazes on (foreign) ceramics, leaded (anti-knock 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 in bones and inhibits formation of heme and hemoglobin in erythroid precursor cells. Before this happens, however, lower levels of lead can cause other problems. These are: impaired vitamin D metabolism, decreased nerve conduction rates, and developmental problems for children including: loss of IQ, hearing impairment, delayed growth, and behavior disorders. Transplacental transfer of lead to the fetus can occur at very low lead concentrations in the body. At relatively low levels, lead can participate in synergistic toxicity with other elements (cadmium, mercury).

Confirming tests for lead excess are: urinary lead following provocation with intravenous EDTA, or DMPS, or oral DMSA and hair element analysis. Whole blood analysis can be expected to reflect only recent exposures and does not correlate well with total body burden of lead (Carson, Ellis and McCann, Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, p. 130, 1987). Preliminary studies performed at DDI indicate significantly increased fecal lead following I.V. vitamin C.

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MERCURY HIGH

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This individual's urine mercury equals or exceeds twice the maximum expected level. Presentation of symptoms associated with excessive mercury can depend on many factors: the chemical form of absorbed Hg and its transport in body tissues, presence of other synergistic toxics (Pb, Cd have such effects),presence of disease that depletes or inactivates lymphocytes or is immunosuppressive, organ levels of xenobiotic chemicals and sulfhydryl-bearing metabolites (e.g. glutathione), and the concentration of protective nutrients, (e.g. zinc, selenium, vitamin E).

Early signs of mercury contamination include: decreased senses of touch, hearing, vision and taste, metallic taste in mouth, fatigue or lack of physical endurance, and increased salivation. Symptoms may progress with moderate or chronic exposure to include: anorexia, numbness and paresthesias, headaches, hypertension, irritability and excitability, and immune suppression, possibly immune dysregulation. Advanced disease processes from mercury toxicity include: tremors and incoordination, anemia, psychoses, manic behaviors, possibly autoimmune disorders, renal dysfunction or failure.

Mercury is commonly used in: dental amalgams, explosive detonators; in pure liquid form for thermometers, barometers, and laboratory equipment; batteries and electrodes ("calomel"); and in fungicides and pesticides. The fungicide/pesticide use of mercury has declined due to environmental concerns, but mercury residues persist from past use.

Methylmercury, the common, poisonous form, occurs by methylation in aquatic biota or sediments (both freshwater and ocean sediments). Methylmercury accumulates in aquatic animals and fish and is concentrated up the food chain reaching high concentrations in large fish and predatory birds. Except for fish, the human intake of dietary mercury is negligible unless the food is contaminated with one of the previously listed forms/sources. A daily diet of fish can cause 1 to 10 micrograms of mercury/day to be ingested, with about three-quarters of this (typically) as methylmercury.

Depending upon body burden and upon type, duration and dosage of detoxifying agents, elevated urine mercury may occur after administration of: DMPS, DMSA, D-penicillamine, or EDTA. Blood and especially blood cell analyses are only useful for diagnosing very recent or ongoing organic (methyl) mercury exposure.

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THALLIUM HIGH

This individual's urine thallium 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 thallium and mode of assimilation; severity and duration of exposure; and organ levels of metabolites and nutrients that effect the action of thallium in body tissue.

Thallium (Tl) 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. Thallium leaves blood plasma rapidly and is readily transported between body organs and tissues. It can be deposited in kidneys, pancreas, spleen, liver, lungs, muscles, neurons and brain. Blood is not a reliable indicator of thallium status.

Symptoms of thallium contamination are often delayed. Early signs of chronic, low-level contamination 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 thallium include: contaminated drinking water, airborne plumes or waste streams from lead and zinc smelters, 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.

Hair (pubic or scalp) element analysis is an excellent corroborative test for suspected thallium excess. 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 exposure to, and excretion of thallium. Other clinical findings that would be consistent are: albuminuria, EEG with diffuse abnormalities, hypertension, and elevated urine creatinine phosphokinase (CPK).

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