URINE TOXIC METALS

DOCTOR'S DATA INC

LAB#: U999999-9999-9
PATIENT: Sample Patient
SEX: Female
AGE: 51

CLIENT#: 12345
DOCTOR:
Doctor's Data, Inc.
3755 Illinois Ave.
St. Charles, IL 60174

POTENTIALLY TOXIC METALS

METALS	RESULT µg/g CREAT	REFERENCE RANGE	WITHIN REFERENCE RANGE / ELEVATED / VERY ELEVATED
Aluminum	**11**	< 35	
Antimony	**0.8**	< 1	
Arsenic	**25**	< 130	
Beryllium	**< dl**	< 0.5	
Bismuth	**< dl**	< 15	
Cadmium	**0.3**	< 2	
Lead	**6.6**	< 5	
Mercury	**8.1**	< 4	
Nickel	**3.8**	< 12	
Platinum	**< dl**	< 1	
Thallium	**0.2**	< 0.8	
Thorium	**< dl**	< 0.3	
Tin	**4.7**	< 10	
Tungsten	**0.08**	< 1	
Uranium	**< dl**	< 0.2	

CREATININE

	RESULT mg/dL	REFERENCE RANGE	2SD LOW / 1SD LOW / MEAN / 1SD HIGH / 2SD HIGH
Creatinine	**53**	35- 225	

SPECIMEN DATA

Comments:
Date Collected: **11/29/2005**
Date Received: **12/1/2005**
Date Completed: **12/3/2005**

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

Collection Period: **timed: 8 hours**
Volume:
Provocation:

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

URINE ESSENTIAL ELEMENTS

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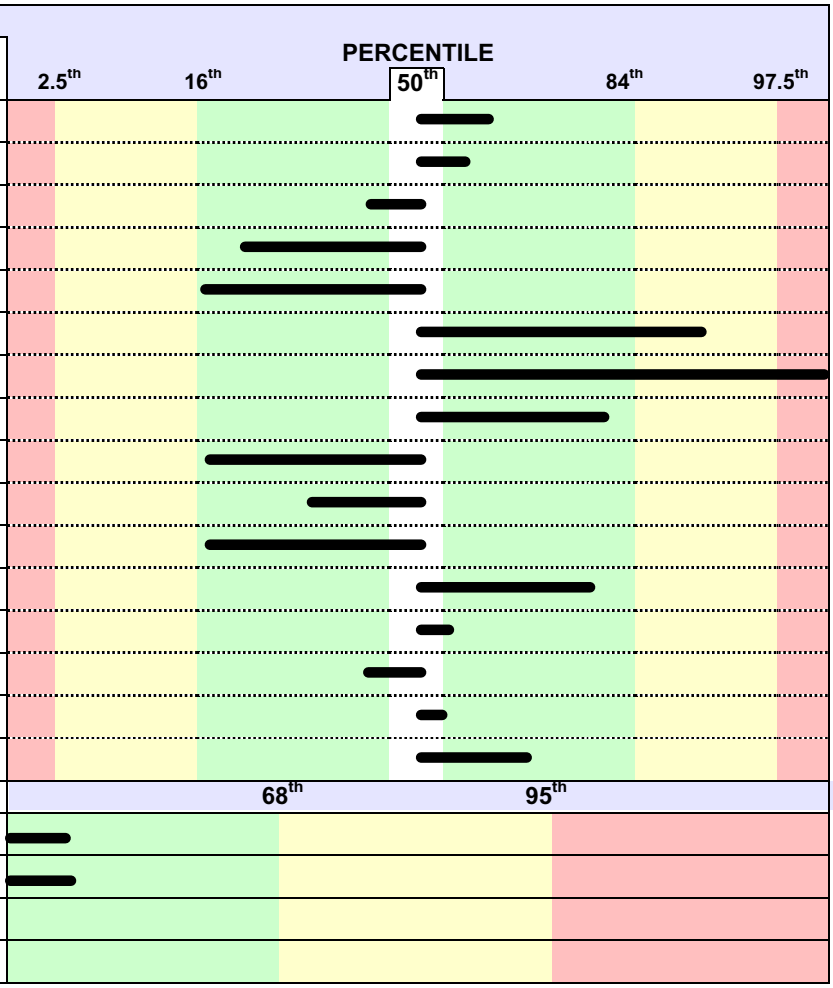
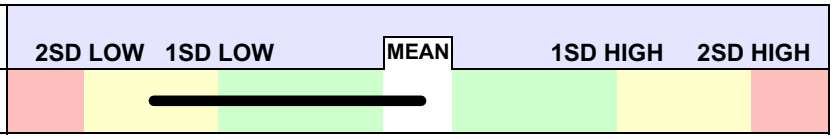
ESSENTIAL ELEMENTS

ELEMENTS	RESULT µg/mg CREAT	REFERENCE RANGE
Sodium	**2820**	1000- 5200
Potassium	**1850**	850- 3200
Phosphorus	**580**	250- 1300
Calcium	**120**	55- 400
Magnesium	**74**	45- 230
Zinc	**1.5**	0.1- 2
Copper	**0.42**	0.01- 0.09
Sulfur	**960**	280- 1500
Manganese	**0.001**	0.0005- 0.01
Molybdenum	**0.046**	0.016- 0.18
Boron	**1.2**	0.8- 5.7
Chromium	**0.092**	0.01- 0.15
Lithium	**0.032**	0.008- 0.2
Selenium	**0.11**	0.05- 0.35
Strontium	**0.19**	0.06- 0.45
Vanadium	**0.025**	0.004- 0.045
Barium	**0.001**	< 0.015
Cobalt	**0.0005**	< 0.04
Iron	**< dl**	< 0.45
Zirconium	**< dl**	< 0.005



CREATININE

	RESULT mg/dL	REFERENCE RANGE
Creatinine	**53**	35- 225



SPECIMEN DATA

Comments:
Date Collected: **11/29/2005**
Date Received: **12/1/2005**
Date Completed: **12/3/2005**

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

Collection Period: **timed: 8 hours**
Volume:
Provocation:

Essential elements are reported as µg/mg creatinine to account for urine dilution variations. **Reference ranges are representative of a healthy population under non-challenge or non-provoked conditions.** Detoxification therapies can cause significant elevations of certain essential element levels (e.g. Cu, Zn). V10.00

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

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.

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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6. Shubert J. et al. "Combined Effects in Toxicology - a Rapid Systematic Testing Procedure: Cadmium, Mercury and Lead" - J. Toxicology and Environmental Health, 4:763-776, 1978.

MERCURY HIGH

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.

BIBLIOGRAPHY FOR MERCURY

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2. World Health Organization: "Methylmercury" Environ. Health Criteria 101 (1990); "Inorganic Mercury" Environ. Health Criteria 118 (1991) WHO, Geneva, Switzerland.
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5. Pelletier L. "Autoreactive T Cells in Mercury-Induced Autoim- munity", J. Immunology, 140 no.3 (1988) pp 750-54.
6. Werbach M.R. Nutritional Influences on Illness, 2nd ed, Third Line Press, Tarzana CA, pp 249, 647, 679, 1993.

MAGNESIUM LOW

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

In renal insufficiency, magnesium (along with other elements) can be low in urine but elevated in blood. Creatinine clearance and blood metabolite levels should be measured if a renal transport disorder is suspected.

24-hour urine levels of magnesium are considered by some authors to be a sensitive indicator of magnesium status (Galland, Magnesium 8 no.2, 1988, pp 78-83). Less than 24 mg/hr urinary Mg excretion suggests deficiency (Lauler, Am. J. Cardiology 63 no 14, 1989, 16).

Homeostatic regulation of blood magnesium levels is normally maintained within close limits. There are, however, many possible nutritional, metabolic, and hormonal factors which can result in subnormal urine levels of magnesium. These are listed below.

. Junk food diet, consumption of magnesium-deficient foods
. Malabsorption syndromes resulting in magnesium deficiency
  a. Gluten enteropathy, sprue
  b. Immune dysregulation, food reactivities with villous atrophy in the small intestine
  c. Intestinal dysbiosis
  d. Intestinal fistulas, bypass or resection surgery
  e. Radiation enteritis
  f. Gastric hypochlorhydria
  g. Pancreatic insufficiency
  h. Biliary insufficiency, steatorrhea
. Hypocalcemia with increased retention of Mg
. Hypothyroidism
. Alkalosis
. Alcoholic withdrawal
. Prolonged diarrhea

Magnesium status can be difficult to assess; whole blood and blood cell levels are more indicative than serum/plasma levels. The magnesium challenge method may be most indicative: baseline 24-hour urine mg measurement, followed by 0.2 mEq/Kg intravenous Mg, followed by 24-hour Mg measurement. A deficiency is judged to be present if less than 80% of Mg challenge is excreted. Ref. Jones et al. "Magnesium Requirements in Adults", Med. Journal Clin. Nutr., 20 (1967) pp. 632-35.

BIBLIOGRAPHY FOR MAGNESIUM

1. Knochel J.P. "Disorders of Magnesium Metabolism", Chapt 360 in Harrison's Principles of

Internal Medicine, 13th ed., McGraw-Hill pp. 2187-90, 1994.
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5(a) Halpern M.J. and J. Durlach eds., Magnesium Deficiency Karger (Basel and New York), esp. pp. 146-180, 1985.
5(b) See also Magnesium and Trace Elements, official journal of the Am. Soc. for Magnesium Research, B.M. Altura (Brooklyn NY), Ed.-in- Chief, S. Karger A.G. Postfach CH-4009 Basel, Switzerland.
6. Galland L. ”Magnesium and Inflammatory Bowel Disease” Magnesium 7 no. 2, pp. 78-83, 1988.
7. Rea W.J. ”Magnesium Deficiency in Patients with Chemical Sensitivity” Clinical Ecology 4 no. 1, pp 17-20, 1986.

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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