DOCTOR'S DATA INC.

Toxic Metals; urine

TOXIC METALS				
		RESULT µg/g Creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	1.3	< 15	
Antimony	(Sb)	0.028	< 0.18	
Arsenic	(As)	4.4	< 40	
Barium	(Ba)	3.3	< 5	
Beryllium	(Be)	<dl	< 0.01	
Bismuth	(Bi)	0.008	< 0.8	
Cadmium	(Cd)	0.09	< 0.6	
Cesium	(Cs)	1.4	< 9	
Gadolinium	(Gd)	<dl	< 0.5	
Lead	(Pb)	0.14	< 1.1	
Mercury	(Hg)	0.31	< 0.8	
Nickel	(Ni)	0.5	< 4	
Palladium	(Pd)	0.03	< 0.3	
Platinum	(Pt)	<dl	< 0.1	
Tellurium	(Te)	<dl	< 0.5	
Thallium	(Tl)	0.07	< 0.4	
Thorium	(Th)	<dl	< 0.015	
Tin	(Sn)	0.50	< 3	
Tungsten	(W)	0.12	< 0.4	
Uranium	(U)	0.008	< 0.03	

URINE CREATININE			
	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	256	35 – 240	

SPECIMEN DATA

< dl: less than detection limit
Results are creatinine corrected to account for urine dilution variations. **Reference intervals are based upon NHANES (cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.

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Essential Elements; urine

ESSENTIAL ELEMENTS				
		RESULT mEq/g Creat	REFERENCE INTERVAL	PERCENTILE 2.5th 16th 50th 84th 97.5th
Sodium	(Na)	29.1	40 – 200	
Potassium	(K)	10.2	20 – 90	
		RESULT µg/mg Creat		
Phosphorus	(P)	475	150 – 1000	
Calcium	(Ca)	91	20 – 250	
Magnesium	(Mg)	39.3	20 – 200	
Zinc	(Zn)	0.37	0.09 – 1.3	
Copper	(Cu)	0.0051	0.003 – 0.022	
Sulfur	(S)	398	250 – 900	
Molybdenum	(Mo)	0.0322	0.01 – 0.11	
Boron	(B)	0.54	0.5 – 3.8	
Lithium	(Li)	0.0297	0.008 – 0.18	
Selenium	(Se)	0.046	0.03 – 0.2	
Strontium	(Sr)	0.173	0.035 – 0.26	

		RESULT µg/g Creat	REFERENCE INTERVAL	68th 95th
Cobalt	(Co)	0.11	< 1	
Iron	(Fe)	5	< 50	
Manganese	(Mn)	0.02	< 0.4	
Chromium	(Cr)	0.089	< 1.5	
Vanadium	(V)	0.17	< 0.6	

URINE CREATININE				
		RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine		256	35 – 240	

SPECIMEN DATA

< dl: less than detection limit
Results are creatinine corrected to account for urine dilution variations. **Reference intervals are based upon NHANES (cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.

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.

- 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.
- 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.

This analysis of urinary essential elements was performed by ICP-Mass Spectroscopy. Analysis of essential and other elements in urine is used primarily to identify and characterize renal wasting conditions. Analysis of essential elements in urine is not a direct approach for assessing nutritional status or adequacy. Blood, cell, and other assimilation and retention parameters are optimal direct indicators of essential element status.

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 24 hour urine collections essential elements are reported as mg/24 h. For timed or first morning urine collections, elements are normalized per gram creatinine to avoid the potentially great margin of error which can be introduced by variation in the sample volume (concentration). It should be noted that creatinine excretion for an individual may vary to some extent over the course of a day, and from day to day.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. If there are no descriptive texts following this introduction, all essential element levels are within acceptable range. All reference ranges are age and sex specific.

This analysis of urinary toxic metals and essential elements was performed by ICP-Mass Spectroscopy. Analysis of metals in urine is traditionally used for evaluation of very recent or ongoing exposure to potentially toxic metals. The urinary excretion of certain metals is known to be increased (provoked) to a variable extent after administration of specific chelating agents. Reference values and corresponding graphs are representative of a healthy population under non-provoked conditions; reference values have not been established for provoked urine samples.

Analysis of essential and other elements in urine is used primarily to identify and characterize renal wasting conditions. Analysis of essential elements in urine is not a direct approach for assessing nutritional status or adequacy. Blood, cell, and other assimilation and retention parameters are optimal direct indicators of essential element status.

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 24 hour urine collections essential elements are reported as mg/24 h, and toxic metals are reported as µg/24 h. For timed, random or first morning urine collections, elements and metals are normalized per gram creatinine to avoid the potentially great margin of error that can be introduced by variation in the sample volume (concentration). It should be noted that creatinine excretion for an individual may vary to some extent over the course of a day, and from day to day.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. For potentially toxic elements, a descriptive text is provided whenever levels are measured to be higher than the unprovoked reference values. If there are no descriptive texts following this introduction, all essential element levels are within acceptable range and all potentially toxic metals are at levels below the unprovoked reference values. All reference ranges and reference values are age and sex specific.

Boron Low

Boron (B) is introduced to the body mainly through food (noncitrus fruits, leafy vegetables, nuts, legumes, wine, cider, beer) and drinking water but is also found in food preservatives (sodium borate), and insecticides (boric acid). Although there is an absolute requirement for B in vascular plants, evidence for biological essentiality in animals (including man) is limited. It has been proposed that boron contributes to living systems by acting indirectly as a proton donor and that it exerts a particular influence on cell membrane and structure and function. Boron is rapidly absorbed and excreted largely in the urine. Based on urinary recovery findings, more than 90% of ingested B is usually absorbed. Boron is distributed throughout the tissues and organs of animals and humans at concentrations mostly between 4.6 and 55.5 nmol (0.05 and 0.6 µg)/g fresh weight. Among the organs that contain the highest amounts of B are bone, spleen, and thyroid.

Boron influences macromineral metabolism and steroid hormone metabolism (testosterone, estrogen, DHEA, and 1,25 dihydroxycholecalciferol). A B deficient diet may also affect calcium metabolism and thus affects the composition, structure, and strength of bone. Signs of B deficiency in animals vary in nature and severity as the diet varies in its content of calcium, phosphorus, magnesium, potassium, cholecalciferol, aluminum, and methionine. Boron is also thought to have an estrogenic effect. In post-menopausal women consuming a very low B diet, B supplementation reduced the total plasma concentration of calcium and the urinary excretions of calcium and magnesium, and elevated the serum concentrations of 17ß-estradiol, testosterone, and ionized calcium, mimicking the effects of estrogen ingestion in postmenopausal women. In another study of magnesium and B deprivation among 13 healthy postmenopausal women (aged 50-78 years), it was found that marginal magnesium and B deprivation may also affect brain function as measured by EEG. It seems there may be increased CNS activity following boron deprivation. In long term hemodialysis patients serum boron levels may be excessively decreased.

No B requirements have been set as of 1998. Estimates are that between 1-2 mg/day may be required. Average intake in the U.S. has been estimated at between 1.7-4.3 mg/day.

Copper Low

Low urinary copper may or may not correspond to subnormal copper levels in body tissues, and other laboratory tests are more indicative of copper status. Such tests include measurement of: whole blood or blood cell copper, hair copper, erythrocyte superoxide dismutase activity, and serum ceruloplasmin.Because the major route of copper excretion is via bile and feces, urinary levels may fluctuate without reflecting or influencing body stores.

Lower than normal excretion of copper (and other elements) can occur in renal insufficiency; in which case blood levels may be normal or elevated. Inadequate levels of molybdenum or zinc allow increased retention of copper, and transient hypocuprinuria may occur during periods when copper stores are accumulating.

Low urinary copper may also correspond to copper deficiency of nutritional or gastrointestinal origins.The richest dietary sources of copper are: nuts, shellfish, liver, raisins and legumes. Dairy products generally are low in copper content. Gastric hypochlorhydria, sprue, and pancreatic dysfunction may inhibit copper uptake.

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
  - Gluten enteropathy, sprue
  - Immune dysregulation, food reactivities with villous atrophy in the small intestine
  - Intestinal dysbiosis
  - Intestinal fistulas, bypass or resection surgery
  - Radiation enteritis
  - Gastric hypochlorhydria
  - Pancreatic insufficiency
  - 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.

Potassium Low

The level of potassium (K) is lower than expected in this sample. K is an electrolyte and a potentiator of enzyme functions in cells. K can be low in the body as the result of gastrointestinal or renal dysfunction, or as a side effect of some diuretics. In adrenocortical hyperactivity, blood levels of K are depressed, while urinary K is increased. Diabetic acidosis and other medical conditions may result in severe K loss. Symptoms of true K deficiency include: muscle weakness, fatigue, and tachycardia. An electrocardiogram may show abnormalities when K is low in serum/plasma or whole blood.

Appropriate tests to confirm low K in body tissues may include measurements of packed red blood cell K; serum or whole blood K and sodium/K ratio.

Sodium Low

The concentration of sodium in this urine sample is lower than expected and is more than two standard deviations below the mean. Low urine sodium levels are uncommon but may be seen, for example, with severe vomiting and/or diarrhea. Further, a low urine sodium concentration implies that the kidney's capacity to reabsorb sodium must be intact and that some stimulus to conserve sodium is present. Urine sodium can vary from day to day depending on the degree of water reabsorption. To get an accurate assessment of renal clearance of sodium, both urine and serum sodium can be compared - this can be done with the fractional excretion of sodium (FENa) calculation (1).

Most of the sodium in the human body can be found either in blood or lymphatic fluid. Sodium levels are regulated by aldosterone (from the adrenal cortex) which acts on the proximal tubules of the nephron to increase reabsorption of sodium and water and to increase the excretion of potassium. Urine sodium testing has a role in the assessment of sodium concentration in the extracellular fluid (ECF) - urine sodium test results should be correlated clinically with sodium and water intake, observation for clinical signs of ECF volume contraction or expansion, serum sodium levels, estimation of renal function and GFR as well as with urine osmolality.

In a normal individual, urine sodium excretion generally reflects dietary intake - the less one ingests (e.g. low salt diet, etc.) the less one excretes. In dehydration (e.g. vomiting, diarrhea, etc.) sodium may be retained (less sodium output in urine) in efforts to retain water. Decreased urine sodium concentration also may be associated with disease states such as Conn's syndrome (primary hyperaldosteronism due to an aldosterone-producing adenoma), congestive heart failure, liver disease and/or nephrotic syndrome. Low urine sodium has been associated with greater risk of myocardial infarction in males with high blood pressure (2).

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).

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.