Test requisition # Physician:

MOSAIC DIAGNOSTICS
Formerly Great Plains Laboratory

CLIENT #:

ORDER:
TEST:
CLIENT REF:
PATIENT:
ID:
SEX:
AGE: **DOB:**

Toxic Metals; urine

TOXIC METALS		RESULT µg/g Creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	9.6	< 25	
Antimony	(Sb)	0.15	< 0.18	
Arsenic	(As)	390	< 50	
Barium	(Ba)	13	< 5	
Beryllium	(Be)	<dl	< 0.01	
Bismuth	(Bi)	0.32	< 1	
Cadmium	(Cd)	0.45	< 0.9	
Cesium	(Cs)	15	< 10	
Gadolinium	(Gd)	<dl	< 0.8	
Lead	(Pb)	6.3	< 1.2	
Mercury	(Hg)	21	< 1.3	
Nickel	(Ni)	0.7	< 5	
Palladium	(Pd)	<dl	< 0.3	
Platinum	(Pt)	<dl	< 0.1	
Tellurium	(Te)	<dl	< 0.5	
Thallium	(Tl)	0.39	< 0.5	
Thorium	(Th)	<dl	< 0.02	
Tin	(Sn)	0.44	< 5	
Tungsten	(W)	<dl	< 0.4	
Uranium	(U)	<dl	< 0.03	

URINE CREATININE	RESULT	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	71.9	30 – 225	

SPECIMEN DATA

Comments:
Date Collected: 04/19/2023
Date Received: 04/21/2023
Date Reported: 04/24/2023
Provoking Agent: DMSA 500 MG
Provocation: Post Provocative
Collection Period: 6 hours
pH upon receipt: Acceptable
Methodology: ICP-MS QQQ, Creatinine by Jaffe Reaction

< 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)	259	45 – 200	
Potassium	(K)	70.5	20 – 110	
		RESULT µg/mg Creat		
Phosphorus	(P)	907	180 – 1100	
Calcium	(Ca)	308	30 – 350	
Magnesium	(Mg)	156	25 – 230	
Zinc	(Zn)	0.31	0.1 – 1.5	
Copper	(Cu)	0.0321	0.006 – 0.026	
Sulfur	(S)	1430	250 – 1050	
Molybdenum	(Mo)	0.0196	0.013 – 0.13	
Boron	(B)	2.0	0.6 – 4	
Lithium	(Li)	0.414	0.009 – 0.2	
Selenium	(Se)	0.340	0.03 – 0.25	
Strontium	(Sr)	0.321	0.045 – 0.3	

		RESULT µg/g Creat	**REFERENCE INTERVAL**	68th 95th
Cobalt	(Co)	0.55	< 1.7	
Iron	(Fe)	16	< 50	
Manganese	(Mn)	0.29	< 0.6	
Chromium	(Cr)	0.51	< 2	
Vanadium	(V)	0.09	< 0.8	

URINE CREATININE			
	RESULT	**REFERENCE INTERVAL**	-2SD -1SD MEAN +1SD +2SD
Creatinine	71.9	30 – 225	

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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.

Arsenic High

This individual’s urine arsenic (As) is higher than expected. Because urine is the major mode of excretion for arsenic, an elevated level reflects increased assimilation of As. Ingestion of organic species of As in seafood is not uncommon and may be associated with very elevated urine As. Arsenobetaine and arsinocholine, commonly found in shellfish are relatively non-toxic and 90% is excreted in the urine with a half-life of about 48 hours.

Sources of As include: contaminated foods (e.g. chicken), 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 As-containing products include: sodium arsenite, calcium arsenate, lead arsenate and "Paris green" which is cupric acetoarsenite, a wood preservative (pressure treated wood). Undesirable levels of As have been found in many Ayruvedic herbs.

Chronic exposure to or ingestion of inorganic As 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 As exposure include: fatigue, malaise, eczema or allergic-like dermatitis, and garlic-like breath. Increased salivation may occur. Hair element analysis may provide further evidence of As exposure to inorganic As. Blood As levels are not dose related and may or may not reflect As exposure or net retention of As. Levels of As may exceed the expected range after administration of DMPS or DMSA depending upon cumulative exposures. This does not necessarily indicate As excess to the point of toxic effects or physiological impairment.

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 activities add 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, bricks, 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 exposure to Ba.

Calcium High

Urine analysis is not a preferred way to assess body calcium stores. Nutritional sufficiency of calcium should be assessed through dietary analysis. Whole blood calcium level, serum calcium ion level, parathyroid hormone determinations, and bone density measurement are tests that are more indicative of calcium status.

High urinary calcium may be an artifact of diet, or of nutritional supplementation of calcium,or of excessive use of vitamin D or of vitamin A. Very high protein diets may cause increased uptake and excretion of dietary calcium. Cessation of these dietary inputs typically normalizes the urinary calcium level within several days.

High urinary calcium is associated with detoxification therapies in which EDTA is administered. High urine calcium also can be a consequence of immobilization or extended bed rest. Steroid therapy and glucocorticoid excess can raise urinary calcium levels.

Pathological conditions that may feature elevated urinary calcium include: renal acidosis, hyperparathyroidism, hyperthyroidism, diabetes mellitus, ulcerative colitis and some cases of Crohn's disease, sarcoidosis, acromegaly, myeloma, carcinoma of the thyroid or metastatic to bone, and Paget's disease.

Osteoporosis is NOT reliably indicated by urine calcium measurement only because the calcium loss is typically too slow and insidious to significantly raise urinary calcium.

Cesium High

This individuals urine Cesium (Cs) level is higher than expected, reflecting exposure to Cs but symptoms may not be evident. Very high levels of Cs in urine are often associated with the use of cesium chloride as a questionable anti-cancer treatment. Cesium is a naturally-occurring element found in rocks, soil and dust at low concentrations. It is present in the environment only in the stable form of Cs133; the radioactive isotopes 134Cs and 137Cs are not measured or reported by Doctor's Data. Natural deposits of Cs ores occur in Main, South Dakota and Manitoba (Bernic Lake), Canada. Cesium may bio-accumulate in aquatic food chains; higher levels of cesium have been found in Pacific deep-sea fish and local shellfish since the 2011 Fukoshima reactor accident. Cesium may be used in high-density drilling fluids (oil and gas industry) and may contaminate local water and vegetation; Cs has been found in cow's milk. Cesium may occur naturally in mineral waters; one study analyzed the Cs concentration in 163 mineral and thermal waters and found the level ranged from 4.5 to 148 µg per liter.

Cesium can be absorbed after oral ingestion, upon breathing contaminated air and through contact with the skin. Cesium is readily absorbed across the brush border of the intestines in a manner similar to potassium and most is eventually excreted through the urine and feces. The biological half-life of Cs in humans ranges from 15 days in infants to 100-150 days in adults.

The cesium-137 isotope is used in cancer treatments, for ventricular function and pulmonary imaging studies, industrial radiology, and for food and instrument sterilization; Cs137 agents may contain small amounts of Cs133. Non-radioactive cesium chloride may be advertised on the internet as "high pH therapy." Currently there is no support in the scientific literature for that purpose as advertised. Radioactive Cs isotopes may contaminate soil at nuclear waste sites. Cesium may be used in industry for the production of photoelectric cells, vacuum tubes, spectrographic instruments, scintillation counters, DNA biochemistry, in various optical or detecting devices.

Target organs of potential toxic effects of Cs are the liver, intestine, heart, and kidneys. Physiological effects of excessive Cs include ventricular arrhythmias and displacement of potassium from muscle cells and erythrocytes. Cesium can have significant effects on both the central and peripheral nervous systems. Cesium may cause epileptic seizures because it can share the same receptor as the excitatory bioamine glycine. Cesium can interfere with active ion transport by blocking potassium channels and also can interfere with lipid metabolism. Excessive Cs may modify plasma membrane integrity, alter cytoplasmic components and cause cytogenetic damage.

It is unlikely that children or adults would be exposed to enough Cs133 to experience any health effects that could be related to the stable Cs itself. Animals given very large doses of Cs compounds have shown changes in behavior, such as increased activity or decreased activity, but it is unlikely that a human would be exposed to enough stable Cs to cause similar effects.

The isotope Cs137 is used in radiation therapy for certain types of cancer. Other medical uses of Cs are monitoring left ventricular function with Cs137 iodide probes and monitoring pulmonary endothelial permeability with Cs137 iodide crystal mini-detectors. Again, it is emphasized that Cs measured at Doctor's Data is Cs133, not Cs137. Environmental contamination by Cs137 as a result of radioactive fallout could be a concern. Exposure to Cs may be assessed by hair elemental analysis.

Commonly used chelating agents are not effective binders of Cs.

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 slightlyor 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 diagnosticprocedure can yield a 5 - 10X increase in urinary copper levels in normal individuals. Incontrast, Wilson's disease patients may then excrete 50-100 times the normal levels or 1000 to2000 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.

Iron High

High urinary iron may or may not correspond to global iron overload or iron loss from body tissues because the major route for iron uptake, reuptake, and excretion is via the bile, intestinal transport and feces. Urinary iron levels may fluctuate without reflecting or influencing body stores.

Very high urinary iron levels are expected to result from administration of deferoxamine (desferrioxamine, desferal) or of EDTA. For adults, urinary iron normally may vary from about 0.5 to about 2 mg per 24 hours after IM administration of deferoxamine. In cases of iron overload, this level is increased: 2-5 mg/24 hour for early or asymptomatic hemochromatosis; 9-23 mg/24 hr for symptomatic hemochromatosis (Powell and Isselbacher, Chapter 345 in Harrison's Principles of Internal Medicine, 13th Ed., 1994).

Hematuria (isolated), proteinuria with hematuria, and glomerulonephritis feature urinaryloss of iron. These conditions may have infections, toxic insults, malignancies, or physicalinjury as possible origins. Urinary iron may be elevated by contamination with blood if theurine was collected during menstruation.

Biliary obstruction or insufficiency can decrease normal excretion of iron via the bile while increasing urinary levels. Porphyria with urinary loss of porphyrins (before heme can be formed) can feature increased urinary iron. Beta-thalassemia and alcoholic liver are also iron-wasting conditions. Excessive supplementation of iron may also cause iron overload and increased urinary iron.

Iron status is best assessed by measurement of: plasma/serum iron, total iron binding capacity, percent of transferrin that is saturated with iron, serum ferritin level, and a CBC with hemoglobin and cell parameter analysis. The above referenced text by Powell and Isselbacher is an authoritative reference on differential diagnosis of iron overload.

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).

Lithium High

The concentration of lithium (Li) in this urine specimen is unexpectedly high. Li occurs almost universally at low concentrations in water and in plant and animal food products. Li has important functions in the nervous system, and possibly the immune system. Assimilation of Li from food, water and even commonly available organic Li supplements (when taken as directed) would not be expected to be associated with abnormally high levels of Li in urine. In contrast, much higher doses of inorganic Li carbonate, which are often prescribed for specific mood disorders, would be expected to be associated with markedly elevated urine Li if ingestion was recent or chronic.

Occupational/accidental assimilation of excessive amounts of Li could possibly be associated with the manufacture or improper handling of lightweight metal alloys, glass, lubrication greases, and batteries.

Li, when assimilated in excessive quantities, may cause dermatitis, nausea, confusion, course hand tremor, slurred speech, edema, or hypotension. Li toxicity may be more pronounced with low sodium intake. Point-in-time Li doses/exposure are rapidly excreted in urine, and blood analysis may not be indicative of exposure after 5 to 7 days.

Magnesium High

This individual's magnesium level exceeds one standard deviation above the mean of the reference population which means that this individual's urine magnesium level corresponds to the highest 17% (approximately) of that population.

Elevated urine magnesium is an expected finding after administration of EDTA, with levels of 150 to 300 mg/24 hr commonly seen (adults). Elevated urine magnesium is not expected with administration of sulfhydryl agents (DMPS, DMSA, D-penicillamine).

Homeostatic regulation of blood magnesium levels is normally maintained within close limits, and homeostasis closely controls intestinal uptake and renal conservation. There are, however, many possible metabolic, hormonal, drug and (toxic) chemical influences which can increase renal excretion of magnesium, perhaps causing "magnesium wasting". These are listed below.

- Hypermagnesemia, excessive infusion of magnesium
- Hypercalcinuria/hypercalcinemia, excessive supplementation or infusion of calcium
- Hyperphosphaturia/hypophosphatemia
- Hypokalemia with urinary potassium wasting
- Hyperaldosteronism
- Hyperparathyroidism
- Alcoholism
- Hypertaurinuria/hypotaurinemia
- Diuresis: diabetes, use of thiazides, other diuretics
- Acidosis: fasting, diabetic ketoacidosis
- Renal tubular dysfunction/damage, postrenal obstruction, nephritis, Bartter's syndrome
- Nephrotoxic drugs/chemicals: amphotericin, cisplatin, aminoglycides, cyclosporin, theophylline, pentamidine.

Many pesticides, herbicides and fungicides are nephrotoxic, and may cause renal wasting; others may cause renal insufficiency, depending upon dose and time elapsed after exposure (Kuloyanova and El Batawi, Human Toxicology of Pesticides, CRC Press 1991; Sittig, Pesticide Manufacturing and Toxic Materials Control Encyclopedia, Noyes Data Corp., 1980).

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 of intravenous Mg, followed by 24-hour Mg measurement. A deficiency is judged to be present if less than 80% of the Mg challenge is excreted. Ref. Jones, et al. "Magnesium Requirements in Adults", Med Journal Clin Nutr, 20 (1967) p.632-35.

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 increaseas 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 wastingsyndromes, 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.

Mercury High

This individual's urine mercury (Hg) is higher than expected but may not be sufficiently high to be associated with overt pathophysiological effects. Symptomatology depends on many factors: the chemical form of Hg, its accumulation in specific tissues, presence of other toxicants, presence of disease that depletes glutathione or inactivates lymphocytes or is immunosuppressive, and the concentration of protective nutrients, (e.g. zinc, selenium).

Early signs of excessive Hg exposure 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/ dysregulation. Advanced disease processes from excessive Hg assimilation include: tremors and incoordination, anemia, psychoses, manic behaviors, possibly autoimmune disorders and renal dysfunction or failure. Note that in Hg exposure of long duration, renal excretion of Hg (and normal metabolites) may become impaired, and the urine level of Hg might be only mildly elevated or not elevated at all due to renal failure.

Mercury is used in: dental amalgams (50% by weight), explosive detonators; some vaccines, pure liquid form in thermometers, barometers, and laboratory equipment; batteries and electrodes,some medications and Ayurveic herbs, fungicides and pesticides, and in the paper industry. The fungicide/pesticide use of mercury has declined due to environmental concerns, but Hg residues persist in the environment. Emissions from coalfired power plants and hospital/municipal incinerators are significant sources of mercury pollution.

Methylmercury, the most common, organic form of Hg, occurs by methylation of inorganic Hg 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 highest 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. Daily ingestion of fish can result in the assimilation of 1 to 10 micrograms of mercury/day.

Depending upon the extent of cumulative Hg exposure, elevated levels of urine Hg may occur after administration of DMPS, DMSA or D-penicillamine. Blood and especially red blood cell elemental analyses are useful for assessing recent or ongoing exposure to organic (methyl) Hg.

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.

Phosphorus High

The level of phosphorus (P) in this sample is higher than expected. P is a major component of mineralized tissue such as bone and teeth. Phosphates also are present in every cell of the body where they are involved in chemical energy transfer and enzyme regulation. Phosphorylation chemistry is part of carbohydrate, amino acid, and lipid metabolism. Along with calcium, P assimilation is regulated by vitamin D. Serum P levels may be affected by abnormal calcium, P or vitamin D metabolism, and the presence of chronic disease. Hyperphosphatemia is common in kidney disease. Symptoms of P excess will be related to the underlying condition causing the excess. High serum P levels have been associated with increased risk of cardiovascular disease and mortality.

Phosphorus is found in most food sources and is a common ingredient of food additives. Up to 100% of the inorganic phosphorus found in processed foods (processed cheese and some soda (cola) drinks) may be absorbed.

Excess phosphorus may be confirmed by serum, packed blood cell (RBC) element analysis, or whole blood elements. If clinically indicated by patient symptoms or history, vitamin D levels may be assessed.

Selenium High

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. However, selenium excess or overload can feature high urinary levels. Without occupational or environmental exposure, or excessive dietary intake, urinary selenium is expected to be below 100 micrograms per liter.

Selenium can be toxic with long-term intake as low as 750 mcg/day. Essential daily selenium requirements range from 10 micrograms (infants) to 50-70 micrograms (adults). Some manifestations of chronic selenium exposure are: fatigue, jaundice, hyperpigmentation of skin, unstable blood pressure, reddish discoloration and structural degeneration of nails and teeth, and dizziness. A garlic-like breath odor usually occurs and there may be a metallic taste in the mouth. Acute selenium contamination generally occurs from inhalation of selenium fumes which inflame mucous membranes and cause coughing and irritation of eyes and nasal passages.

Packed red blood cell elements analysis is a more definite test for selenium status. Hair analysis may provide confirmation of selenium excess if exogenous sources of contamination(antidandruff shampoos) are eliminated.

Sodium High

The concentration of sodium in this urine sample is higher than expected and is more than two standard deviations above the mean. A high urine sodium concentration can indicate that the kidney's capacity to reabsorb sodium might be impaired and/or that some stimulus to excrete 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 more one ingests (e.g. added dietary salt, drinking and cooking with softened water, salt poisoning, etc.) the more one excretes. High urine sodium may be associated, for example, with diuretic use or conditions such as Addison's disease (primary adrenal insufficiency).

Strontium High

The primary use of Strontium (Sr) has been in the production of glass for color television cathode ray tubes (to block x-ray emissions) and in the production of metal alloys (e.g.aluminum, magnesium). The stable form of Sr is not known to pose any health threat. The prescription drug Strontium Ranelate is used in many countries (but not Canada or the USA) to increase bone density and reduce the occurrence of fractures. The isotope 90Sr (found in nuclear fallout) can lead to bone disorders, including bone cancer. The isotope 89Sr is a beta emitter used for palliation of pain in patients with metastatic bone cancer - after intravenous administration, up to 80% of the isotope is eliminated in urine (1).

Urine Sr levels provides useful information in the biological monitoring of the presence of this element in individuals therapeutically or environmentally exposed to Sr.