MedPrimex
info@medprimex.com
www.MedPrimex.com

LAB #: Sample Report
PATIENT: Sample Patient
ID:
SEX: Female
AGE: 67

CLIENT #: 12345
DOCTOR: Sample Doctor
MedPrimex, S.A. de C.V.
Calle Laplace Num 34 Int. 4-B
Colonia Anzures, Alcaldía Miguel Hidalgo
C.P. 11590. Ciudad de México

Toxic Metals; Urine

TOXIC METALS		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	**32**	< 35	
Antimony	(Sb)	**< dl**	< 0.2	
Arsenic	(As)	**17**	< 80	
Barium	(Ba)	**17**	< 7	
Beryllium	(Be)	**< dl**	< 1	
Bismuth	(Bi)	**< dl**	< 4	
Cadmium	(Cd)	**0.9**	< 1	
Cesium	(Cs)	**24**	< 10	
Gadolinium	(Gd)	**< dl**	< 0.8	
Lead	(Pb)	**37**	< 2	
Mercury	(Hg)	**1.7**	< 4	
Nickel	(Ni)	**16**	< 10	
Palladium	(Pd)	**< dl**	< 0.3	
Platinum	(Pt)	**< dl**	< 0.1	
Tellurium	(Te)	**< dl**	< 0.5	
Thallium	(Tl)	**1.2**	< 0.5	
Thorium	(Th)	**< dl**	< 0.03	
Tin	(Sn)	**8.6**	< 5	
Tungsten	(W)	**< dl**	< 0.4	
Uranium	(U)	**< dl**	< 0.04	

URINE CREATININE	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	**57.4**	30– 225	

SPECIMEN DATA

Comments:

Date Collected: **02/22/2019**
Date Received: **02/25/2019**
Date Completed: **02/27/2019**
Method: **ICP-MS**

pH upon receipt: **> 8.0**
<dl: **less than detection limit**
Provoking Agent: **EDTA 3G**
Creatinine by Jaffe Method

Collection Period: **timed: 5 hours**
Volume:
Provocation: **POST PROVOCATIVE**

Results are creatinine corrected to account for urine dilution variations. **Reference intervals and corresponding graphs are representative of a healthy population under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.

V13

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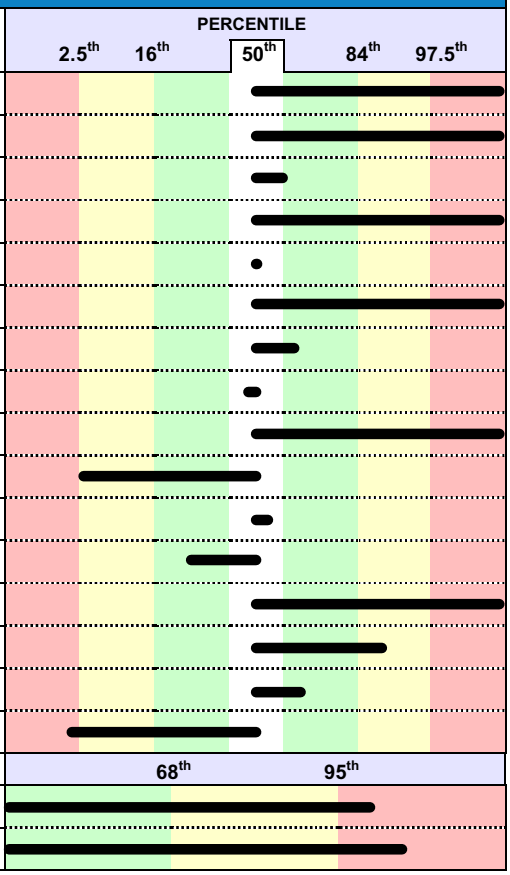
Essential Elements ; Urine

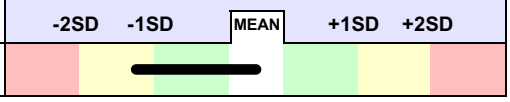
ESSENTIAL AND OTHER ELEMENTS

		RESULT/UNIT per creatinine		REFERENCE INTERVAL
Sodium	(Na)	**260**	mEq/g	45– 200
Potassium	(K)	**150**	mEq/g	20– 110
Phosphorus	(P)	**620**	µg/mg	180– 1100
Calcium	(Ca)	**640**	µg/mg	30– 350
Magnesium	(Mg)	**80**	µg/mg	25– 230
Zinc	(Zn)	**27**	µg/mg	0.1– 1.5
Copper	(Cu)	**0.021**	µg/mg	0.007– 0.06
Sulfur	(S)	**630**	µg/mg	275– 1200
Manganese	(Mn)	**0.04**	µg/mg	0.0004– 0.007
Molybdenum	(Mo)	**0.013**	µg/mg	0.013– 0.15
Boron	(B)	**1.6**	µg/mg	0.5– 4
Chromium	(Cr)	**0.0006**	µg/mg	0.0003–0.0025
Lithium	(Li)	**4.5**	µg/mg	0.009– 0.2
Selenium	(Se)	**0.18**	µg/mg	0.03– 0.25
Strontium	(Sr)	**0.22**	µg/mg	0.045– 0.5
Vanadium	(V)	**< dl**	µg/mg	0.0001–0.0017
Cobalt	(Co)	**0.025**	µg/mg	< 0.008
Iron	(Fe)	**1.4**	µg/mg	< 1



URINE CREATININE

	RESULT mg/dL	REFERENCE INTERVAL
Creatinine	**57.4**	30– 225



SPECIMEN DATA

Comments:

Date Collected: **02/22/2019**
Date Received: **02/25/2019**
Date Completed: **02/27/2019**
pH Upon Receipt: **> 8.0**
<dl: **less than detection limit**
Provoking Agent: **EDTA 3G**
Collection Period: **timed: 5 hours**
Volume:
Provocation: **POST PROVOCATIVE**
Method: **ISE;Na, K Spectrophotometry; P ICP-MS; B, Ca, Cr, Co, Cu, Fe, Mg, Mn, Mo, Se, Sr, S, V, Zn Creatinine by Jaffe method**

Results are creatinine corrected to account for urine dilution variations. **Reference intervals and corresponding graphs are representative of a healthy population under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.

V13

INTRODUCTION

This analysis of urinary elements was performed by ICP-Mass Spectroscopy following acid digestion of the specimen. Urine element analysis is intended primarily for: diagnostic assessment of toxic element status, monitoring detoxification therapy, and identifying or quantifying renal wasting conditions. It is difficult and problematic to use urinary elements analysis to assess nutritional status or adequacy for essential elements. Blood, cell, and other elemental assimilation and retention parameters are better indicators of nutritional status.

1) 24 Hour Collections
"Essential and other" elements are reported as mg/24 h; mg element/urine volume (L) is equivalent to ppm. "Potentially Toxic Elements" are reported as μg/24 h; μg element/urine volume (L) is equivalent to ppb.

2) Timed Samples (< 24 hour collections)
All "Potentially Toxic Elements" are reported as μg/g creatinine; all other elements are reported as μg/mg creatinine. Normalization per creatinine reduces the potentially great margin of error which can be introduced by variation in the sample volume. It should be noted, however, that creatinine excretion can vary significantly within an individual over the course of a day.

If one intends to utilize urinary elements analysis to assess nutritional status or renal wasting of essential elements, it is recommended that unprovoked urine samples be collected for a complete 24 hour period. For provocation (challenge) tests for potentially toxic elements, shorter timed collections can be utilized, based upon the pharmacokinetics of the specific chelating agent. When using EDTA, DMPS or DMSA, urine collections up to 12 hours are sufficient to recover greater than 90% of the mobilized metals. Specifically, we recommend collection times of: 9 - 12 hours post intravenous EDTA, 6 hours post intravenous or oral DMPS and, 6 hours post oral bolus administration of DMSA. What ever collection time is selected by the physician, it is important to maintain consistency for subsequent testing for a given patient.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. Because renal excretion is a minor route of excretion for some elements, (Cu, Fe, Mn Zn), urinary excretion may not influence or reflect body stores. Also, renal excretion for many elements reflects homeostasis and the loss of quantities that may be at higher dietary levels than is needed temporarily. For these reasons, descriptive texts are provided for specific elements when deviations are clinically significant. For potentially toxic elements, a descriptive text is provided whenever levels are measured to be higher than expected. If no descriptive texts follow this introduction, then all essential element levels are within acceptable range and all potentially toxic elements are within expected limits.

Reference intervals and corresponding graphs shown in this report are representative of a healthy population under non-provoked conditions. Descriptive texts appear in this report on the basis of measured results and correspond to non-challenge, non-provoked conditions.

Chelation (provocation) agents can increase urinary excretion of metals/elements. Provoked

reference intervals have not been established therefore non-provoked reference intervals shown are not recommended for comparison purposes with provoked test results. Provoked results can be compared with non-provoked results (not reference intervals) to assess body burden of metals and to distinguish between transient exposure and net retention of metals. Provoked results can also be compared to previous provoked results to monitor therapies implemented by the treating physician. Additionally, Ca-EDTA provoked results can be used to calculate the EDTA/Lead Excretion Ratio (LER) in patients with elevated blood levels.

CAUTION: Even the most sensitive instruments have some detection limit below which a measurement cannot be made reliably. Any value below the method detection limit is simply reported as "< dl." If an individual excretes an abnormally high volume of urine, urinary components are likely to be extremely dilute. It is possible for an individual to excrete a relatively large amount of an element per day that is so diluted by the large urine volume that the value measured is near the dl. This cannot automatically be assumed to be within the reference range.

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 activitiesadd 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, brics, 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 exosure to Ba.

Cesium High

This individuals urine Cesium (Cs) level is higher than expected, reflecting exosure to Cs but symptoms may not be evident. 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 Datais 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.

Resources:

Agency for Toxic Substances & Disease Registry (2015) Toxicological Profile for Cesium. https://www.atsdr.cdc.gov/toxprofiles/TP.asp(c)id=578&tid=107 Accessed 21 February 2017

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Yamagata N, Iwashima K, Nagai T, Watari K, Iinuma T (1966) In Vivo Experiment on the Metabolism of Cesium in Human Blood with Reference to Rubidium and Potassium. Journal of Radiation Research 1966 vol: 7 (1) pp: 29-46

Yorita Christensen KL (2013) Metals in blood and urine, and thyroid function among adults in the United States 2007-2008. International Journal of Hygiene and Environmental Health 2013 vol: 216 (6) pp: 624-632

LEAD HIGH

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

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

Lead accumulates extensively in bone and inhibits formation of heme and hemoglobin in

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

Excessive retention of lead can be assessed by urinalysis after provocation with Ca-EDTA (iv) or oral DMSA. Whole blood analysis can be expected to reflect onlyrecent exposures and does not correlate well with total body burden of lead.

BIBLIOGRAPHY FOR LEAD

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2. Lead Tech ’92, ”Proceedings and Papers from the Lead Tech ’92: Solutions for a Nation at Risk” Conference, Sept 30-Oct 2, 1992. Bethesda, MD, IAQ Publications, 4520 East-West Highway, Ste 610, Bethesda, MD, 20814.

3. ”Preventing Lead Poisoning in Young Children”, US Centers for Disease Control, Atlanta, GA, Oct. 1991 Statement, US Dept. of Health and Human Services.

4. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, Inc., Chelsea, MI, p. 128-135, 1986.

5. Tsalev D.L. et al. Atomic Absorption Spectrometry in Occupational and Environmental Health Practice Vol 1, CRC Press, BocaRaton, FL 1983.

6. Piomelli S. et al. ”Management of Childhood Lead Poisoning”, J. Pediatr 105 (1990) p. 523-32.

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NICKEL HIGH

This individual’s urine nickel (Ni) is elevated which may or may not be of significance. Urinary excretion of nickel bound to cysteine or other thiol compounds (such as glutathione) or to amino acids (histidine, aspartic acid, arginine) is the predominant mode of excretion. With the exception of specific occupational exposures, most absorbed Ni comes from food or drink, and intakes can vary by factors exceeding 100 depending upon geographical location, diet, and water supply. Depending upon chemical form and physiological factors, from 1 to 10% of dietary Ni may be absorbed from the gastrointestinal tract. Urine Ni only reflects recent exposure to Ni and may vary widely from day to day.

Sources of nickel are numerous and include the following.

. Cigarettes (2 to 6 mcg Ni per average cigarette)
. Diesel exhaust (particulates may contain up to 10 mg/gram)
. Foods, especially: cocoa, chocolate, soya products, nuts, hydrogenated oils, and coffee
. Nickel-cadmium batteries (Ni-Cd)
. Nonprecious, semiprecious dental materials
. Nickel-containing prostheses
. Electroplating, metal plated objects, costume jewelry
. Pigments (usually for ceramics or glass)
. Catalyst materials (for hydrogenation processes in the food, petroleum and petrochemical industries)
. Arc welding
. Nickel refining and metallurgical processes

Most clinically relavant Ni exposures are manifested as dermatoses - contact dermatitis and atopic dermatitis. However, Ni hypersensitizes the immune system and may cause hyperallergenic responses to many different substances. Because Ni can displace zinc from binding sites on enzymes it can affect abnormal enzymatic activity. Nickel sensitivity is observed to be three to five times more prevelant in females than in males.

Other laboratory tests or possible clinical findings that may be associated with Ni exposure are; hair elements analysis, presentation of multiple allergic sensitivities, dermatitis, positive patch test for "Ni allergy", proteinuria, hyperaminoaciduria (by 24-hour urine amino acid analysis). Administration of EDTA or sulfhydryl agents (DMPS, DMSA, D-penicillamine) may increase urine Ni levels; such chelator-induced elevations may or may not be associated with adverse health effects.

BIBLIOGRAPHY FOR NICKEL

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2. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, Chelsea MI, pp 162-67, 1986.
3. Nielsen F.H. in Modern Nutrition in Health and Disease ed. by Shils et al, Lea & Febiger, Philadelphia, PA, pp 279-81, 1994.
4. Medical and Biological Effects of Environmental Pollutants: Nickel, Nat. Acad. Sci, Washington DC,1975.
5. Ambient Water Quality Criteria for Nickel, US EPA NTIS, Springfield, VA. Publ No. PB81-117715,1980.

THALLIUM HIGH

This individual's urine thallium (Tl) is markedly higher than expected and is indicative of high level exposure to Tl. Thallium can be an insidious toxic element, sometimes with delayed and diverse manifestations. Multiple organs, tissues and nervous system involvement may occur. Specific symptoms can depend upon: 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 Tl in the body.

Thallium can be assimilated transdermally, by inhalation, or by oral ingestion. Both valence

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

Symptoms associated with excessive exposure to Tl are often delayed for up to 5 days. Early symptoms may include nausea, gastrointestinal distress and diarrhea if a sufficiently large dose of Tl is ingested. Early signs of chronic, low-level exposure and retention may include: mental confusion, fatigue, and peripheral neurological signs: paresthesias, myalgias, tremor and ataxia. After 3 to 4 weeks, diffuse hair loss with sparing of pubic and body hair and a lateral fraction of eyebrows usually occurs. Increased salivation occurs less commonly. Longer term or residual symptoms may include: alopecia, ataxia, tremor, memory loss, weight loss, proteinuria (albuminuria), and possibly psychoses. Ophthalmologic neuritis and strabismus may be presented.

Environmental and occupational sources of Tl include: contaminated drinking water, airborne plumes or waste streams from lead and zinc smeltering, photoelectric, electrochemical and electronic components (photoelectric cells, semiconductors, infrared detectors, switches), pigments and paints, colored glass and synthetic gem manufacture, and industrial catalysts used in some polymer chemistry processes. Thallium is present in some ”weight loss” products (e.g. Active 8) at an undisclosed level (”trade secret”).

Hair (pubic or scalp) element analysis may be used to test for suspected thallium exposure. Although urine is the primary natural route for excretion of Tl, the biliary/fecal route also contributes. Therefore, fecal metals analysis provides a confirmatory test for chronic ongoing exposure to Tl. Other clinical findings that might be associated with excessive Tl include: albuminuria, EEG with diffuse abnormalities, positive fecal blood (occurs with more severe Tl contaminations), hypertension, and elevated urine creatinine phosphokinase (CPK).

BIBLIOGRAPHY FOR THALLIUM

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2. Graef J.W. ”Thallium” in Harrison’s Principles of Internal Medicine, 13th ed., Isselbacher et al eds., McGraw Hill, pp 2465, 1994.
3. Tsalev D.L. and Zaprianov Z.K. Atomic Absorption Spectrometry in Occupational and Environmental Health Practice CRC Press, Boca Raton FL, pp 196-199, 1983.
4. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans Lewis Publishers, Chelsea, MI, pp 243-254, 1987.

TIN HIGH

Tin is elevated in this individual’s urine, and urine accounts for at least 80% of excreted tin that is ingested and absorbed from the gastrointestinal tract. Ingested tin is not significantly absorbed if it is an inorganic form. Oxide coatings readily form on metallic tin, and salts can quickly oxidize making them insoluble. Organic tin, however, is bioavailable and more readily absorbed. Some organic tin compounds such as short-chain alkyltins can be absorbed transdermally and can cause degeneration of myelin.

Food and drink usually provide small daily intakes of (nontoxic) tin, with amounts depending upon type of food, packaging, quality of drinking water and water piping materials. Total daily

intake is expected to vary from about 0.1 to 15 milligrams. Tin is present in many metal alloys and solders; bronze, brass and pewter contain the element. Dyes, pigments and bleaching agents often contain tin. Anticorrosion plating of steel and electrical components may also use tin. "Tin cans" are tin-plated steel with a thin outer oxide layer allowing the surface to be shiny but inert. Modern food-containing cans usually have polymer coatings that prevent food-metal contact. In the past some toothpastes contained stannous fluoride, a soluble fluoride source for strengthening tooth enamel. Currently most brands of fluoridated toothpastes contain sodium fluoride. Organic tins, the usually toxic forms, are: biocides (triphenyltin and alkyltins) used against rodents, fungi, insects and mites; curing agents for rubbers and silicones (dialkyltin); and methyltin formed bacteriologically (similar to methylmercury).

Mildly elevated levels of tin in urine may reflect sporadic dietary intake and excretion; there may be no associated symptoms. A two- or three-fold increase in urine tin levels is not uncommon following administration of EDTA or with sulfhydryl agents (DMSA, D-penicillamine, DMPS). Early signs of chronic organic tin excess can be: reduced sense of smell, headaches, fatigue and muscle aches, ataxia and vertigo. Hyperglycemia and glucosuria are reported. Also, for organic tin exposure, there can be irritation of contacted tissues (eyes, skin, bronchial tubes, or GI tract). Later, immune dysfunction may occur with reduced lymphocytes and leukocytes; mild anemia may occur. A hair element analysis can be used to corroborate tin excess. Tin is commonly elevated in urine from autistic patients following administration of DMSA or DMPS.

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Urine 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).

1. Bazari H. Approach to the patient with renal disease. In: Goldman L, Ausiello D, eds. Cecil Medicine. 23rd ed. Philadelphia, Pa: Saunders Elsevier; 2007:chap 115.

2. National Adrenal Diseases foundation (NADF). What is Addison's Disease. Undated; accessed at http://www.nadf.us/diseases/addisons.htm

Potassium High

The level of potassium (K) is higher than expected in this sample. Symptoms of elevated K may include mental confusion, weakness, numbness, tingling in the extremities, brady-cardia or irregular heart rhythm and ventricular fibrillation.

K is an electrolyte and a potentiator of enzymatic reactions in the body. Elevated K in hair may reflect overall retention of K by the body or maldistribution of this element. In adrenocortical insufficiency, K is increased in blood, while it is decreased in urine; cellular K may or may not be increased.

Appropriate tests to confirm excess K in body tissues may include measurements of packed red blood cell K; serum or whole blood K and sodium/K ratio. An assessment of adrenocortical function may be indicated for symptomatic patients with a confirmed elevation in serum K.

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.

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ZINC HIGH

High urinary zinc may or may not correspond to global zinc excess or to zinc loss from body tissues, because the major route for zinc excretion is via the bile, intestinal transport and feces. Typically, from two to ten percent of total zinc excretion occurs via urine; a similar amount occurs in sweat; the remainder (about 80 to 95%) occurs via biliary secretion to the intestine and is excreted in feces. Urine levels may fluctuate without reflecting or influencing body stores.

Very high urinary zinc levels are expected to result from EDTA detoxification therapy; 3 to 20 mg/L is commonly measured in the 12 hours following intravenous administration of EDTA. Lesser elevations of urine zinc also are expected to result from sulfhydryl agent detoxification therapy (DMPS, DMSA, D-penicillamine). One to five mg/L is commonly found in the 24 hours following administration of these agents. Zinc repletion may be beneficial or required during such therapies.

Breakdown of tissue releases zinc into extracellular fluids and increases urinary zinc levels. This may be observed following or in conjunction with: accidental injury, surgery, catabolism of diseased/disordered tissue, starvation (ketosis) and diabetes. Zinc wasting may occur in alcoholic cirrhosis.

Zinc overload or toxicity can occur from ingestion of zinc contaminated food or drink; galvanized pipes or pails can be sources. Occupational or environmental exposure to zinc fumes may produce an acute contamination or poisoning. Elevated urinary zinc beyond two standard deviations high (without provocation) warrants investigation of possible sources of zinc excess, or of tissue catabolism or injury.

Excessive amounts of zinc in body tissues may displace copper and/or iron from tissue binding sites and may provoke anemia. Symptoms consistent with chronic zinc toxicity include: lethargy, difficulty writing and with fine motor skills, light-headedness, and renal failure. Immediate

symptoms (within 12 hours) of acute zinc excess via ingestion include: nausea, vomiting, diarrhea, exhaustion, headache, dizziness, and myalgia. Other laboratory findings consistent with zinc toxicity would be: elevated leukocyte count, elevated serum amylase and lipase, elevated whole blood zinc concentration, elevated hair zinc level (if the zinc excess is chronic).

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MANGANESE HIGH

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

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

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

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

Relative to other essential trace elements, excessive Mn retention can be neurotoxic. Inhalation, as a result of occupational exposure, is the route of assimilation that is most harmful. Some symptoms and manifestations of excess Mn exposue include:psychiatric disturbances (hyperirritability, hallucinations,

violence), tremor,Parkinson's-like symptoms, anorexia, sexual impotence, and speech disturbance.

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

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MOLYBDENUM LOW

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

Molybdenum is an essential activator of some important enzymes in the body: sulfite oxidase (catalyzes formation of sulfate from sulfite), xanthine oxidase (formation of uric acid and superoxide ion from xanthine), and aldehyde oxidase (processes aldehydes). Over 50% of absorbed Mo is normally excreted in urine; the remainder is excreted via bile to the feces or is excreted in sweat.

The level of molybdenum in urine may be a transient finding and may not reflect body tissue or liver levels. In copper deficiency, retention of molybdenum is increased (tissue levels could be normal or high), while urine levels might be subnormal. In renal insufficiency, molybdenum (and other elements) can be low in urine. Creatinine clearance and blood metabolite levels should be measured if a renal transport disorder is suspected.

Individuals receiving prolonged total parenteral nutrition ("TPN") may have low body tissue and urine levels of molybdenum because it is occasionally omitted from TPN formulations.

Molybdenum in foods is mostly in soluble complexes, and only a small amount is required daily (100 to 200 micrograms, adults). Therefore, frank molybdenum deficiency is uncommon. However, GI dysfunctions, poor-quality diet, and stressors can combine to produce inadequate molybdenum. Tungsten is a powerful antagonist of molybdenum retention, copper less so. Episodic exposures to high levels of either may result in periods of low Mo excretion that follow prior periods of high Mo excretion. Sulfites, aldehydes and high amounts of purines in the diet may increase need for and retention of molybdenum. Prolonged use of dithiol chelators (DMPS, DMSA) or MSM can result in poor molybdenum status as indicated by low levels in red blood cells (DDI observations).

A multielement hair analysis plus analyses for serum and urine uric acid can be used to confirm or rule out molybdenum insufficiency.

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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.

Vanadium Low

A low level of Vanadium (V) was found in this urine sample. Excessively low urinary V excretion may reflect a deficiency state due to poor dietary intake and/or poor absorption (less than 5% of dietary V is absorbed).

Dietary vanadium is found in seafood, eggs, black pepper, mushrooms, dill seed, parsley, soy, corn, olive oil, radishes and other root vegetables, lettuces, nuts, strawberries and gelatin. A balanced diet may provide 10 to 30 mcg of V per day. This trace element is important in cellular metabolism, bone and tooth formation, reproduction and growth. Also, V appears to be involved in glucose metabolism.

There are no known symptoms of V deficiency. Although trace amounts of V may have essential metabolic functions, over-zealous supplementation of V is not warranted. There is no RDA for V

but, if supplementation is warranted, a common daily dose of tetravalent vanadyl sulfate is 20 to 30 mcg per day.

Diabetics should not use supplemental V as the sole intervention in the management of their diabetes and should only use it with the advice of their attending practitioner. People with hypoglycemia should not use supplemental V as it may further lower blood glucose.

A more direct confirmatory test for V deficiency is the Doctor's Data whole blood vanadium test.

COBALT HIGH

The level of cobalt (Co) is higher than expected. Urine Co will be higher than normal if relatively high dose vitamin B-12 is chronically administered; such is not likely of clinical concern because the majority of the Co that was measured is a very likely constitutive of intact excreted B-12. While cobalt is an essential element, cobalt deficiency has not been reported in humans.

Sources of exposure to elemental Co and Co alloys include occupational work with metals (particles, dust, fumes), and colorants in glass, ceramics, and paints, as catalysts, and as paint dryers. Metal-on-metal (M/M) hip prostheses are associated indefinitely with elevated levels of Co in serum and urine for all patients with such prosthetic devices. The elevated Co levels are due to wear of the prostheses. Rates of wear are highly dependent on device design, surgical technique, level of physical activity and other factors that affect the health of involved bone and surrounding soft tissue. Excessive wear may be associated with local tissue damage (bone and soft tissue) that can cause failure of the devices and further exacerbate release of Co debris. Although no safe level of Co in circulation has been determined, guidelines for urine Co have been provided to assist in evaluation of the status of M/M prostheses. It has been proposed that urine Co levels of 2-4 mcg/L are likely to be associated with a prosthetic device in good condition. Urine Co >5 mcg/L is purportedly associated with prosthesis wear, and levels >20 mcg/L in a patient with a Co alloy implant suggest significant prosthesis wear. However, there is limited published data on appropriate reference ranges which raises question regarding the clinical utility of the data. It has been emphasized that elevated serum Co and Cr levels in the absence of corroborating symptoms do not independently predict prosthesis failure.

There are well established adverse systemic health effects associated with excess Co in the body. Elevated levels of Co may be associated with excessive oxidative stress, inflammation, and low levels of glutathione. Further, excessive Co has been shown to compromise hepatic cytochrome P450 activity in laboratory rats (Phase I detoxification). Other well documented effects of excess Co include visual and auditory impairment, tinnitus, vertigo, cardiomyopathy, cognitive dysfunction /dementia, mood disorders, hypothyroidism, peripheral neuropathy and skin rashes.

Urinary Co levels may be increased following intravenous administration of the chelator calcium-disodium-EDTA (Ca-EDTA) or DMPS. Urine Co levels after Ca-EDTA provide an estimate of the net retention of Co.

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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 urinary loss of iron. These conditions may have infections, toxic insults, malignancies, or physical injury as possible origins. Urinary iron may be elevated by contamination with blood if the urine 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.

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