105 - 32 Royal Vista Drive NW
Calgary, AB T3R 0H9
www.rmalab.com

Phone: 403-241-4500
Fax: 403-241-4501

Advancing the Science of Wellness
Rocky Mountain Analytical

Date of Collection: 03-Sep-2013
Sample Received: 04-Sep-2013
Reported Date: 28-Nov-2013
Sample Type: Urine

Urine Element Analysis — Accession Number: 333335

Provider:

Patient:
Jane Smith

Date of Birth : 01-Mar-1969
Age : 44
Gender : F

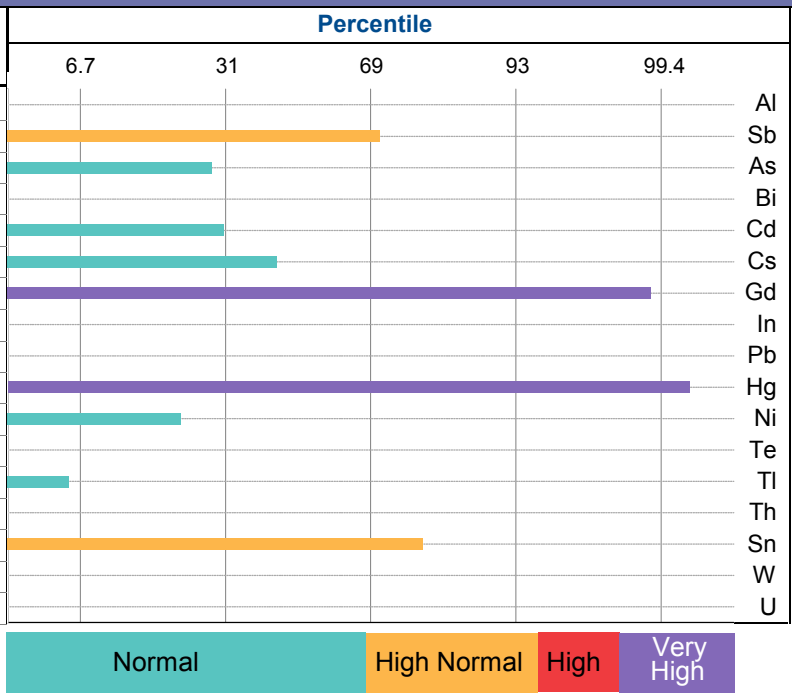
Phone:
Fax:

Collection Period: TIMED 6.00 hrs
Urine Volume: N/A

Provocation: *POST-PROVOCATION
Provoking Agent: DMPS

TOXIC or POTENTIALLY TOXIC ELEMENTS — Accession Number: 333335

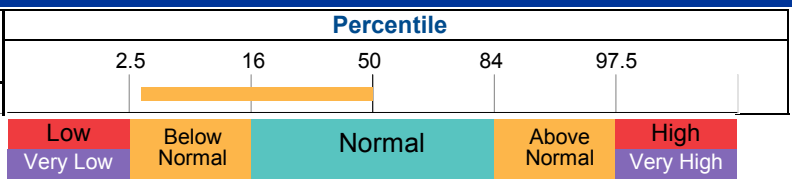
Toxic Elements	Results ug/g Cr	Reference Range ug/g Cr
Aluminum	< dl	<39
Antimony	0.12	<0.35
Arsenic	11	<81
Bismuth	< dl	<0.20
Cadmium	0.29	<1.1
Cesium	3.5	<10
Gadolinium	0.093	<0.040
Indium	< dl	<0.029
Lead	< dl	<3.9
Mercury	4.1	<1.3
Nickel	4.6	<14
Tellurium	< dl	<0.41
Thallium	0.10	<0.40
Thorium	< dl	<0.062
Tin	1.4	<2.6
Tungsten	< dl	<0.29
Uranium	< dl	<0.027



< dl = result is less than detection limit

URINE CREATININE

Analyte	Result g/L	Reference Range g/L
Creatinine	0.28	0.50 - 1.8



*Note: Urine sample was marked post-provocation. Reference ranges are based on measurement of unprovoked specimens and are provided for information purposes only. Graphing of the post-provocation sample using pre-provocation ranges may not accurately reflect the clinical picture. Providers must exercise caution interpreting the meaning of these graphs.

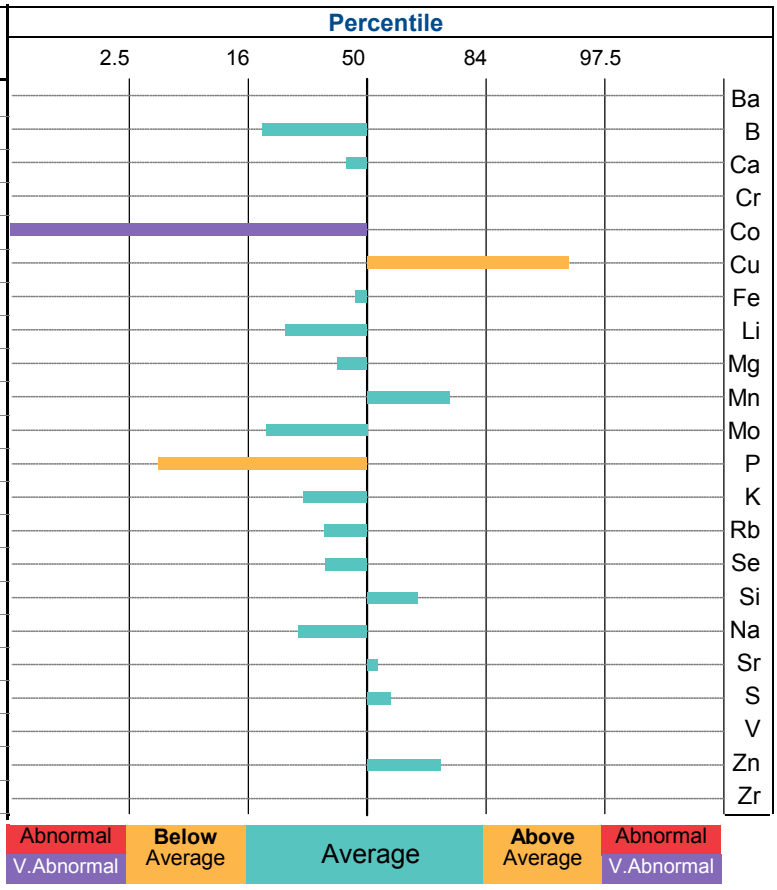
Collection Period: TIMED
Urine Volume: 0 ml

Provocation: *POST-PROVOCATION
Provoking Agent: DMPS

ESSENTIAL ELEMENTS — Accession Number: 333335

Essential Elements	Results ug/mg Cr	Reference Range ug/mg Cr
Barium	< dl	0.0024 - 0.019
Boron	0.99	0.90 - 3.9
Calcium	79	41 - 200
Chromium	< dl	0.000080 - 0.00025
Cobalt	0.000075	0.00028 - 0.0010
Copper	0.11	0.022 - 0.071
Iron	0.0097	0.0051 - 0.022
Lithium	0.019	0.016 - 0.056
Magnesium	73	48 - 150
Manganese	0.0015	0.00043 - 0.0019
Molybdenum	0.027	0.025 - 0.098
Phosphorus	320	480 - 1,300
Potassium	1,000	740 - 2,800
Rubidium	0.79	0.53 - 1.8
Selenium	0.063	0.046 - 0.12
Silicon	16	8.2 - 21
Sodium	1,300	980 - 3,500
Strontium	0.13	0.063 - 0.25
Sulphur	770	420 - 1,200
Vanadium	< dl	0.000038 - 0.00018
Zinc	0.34	0.11 - 0.45
Zirconium	< dl	0.000046 - 0.00028

< dl = result is less than detection limit



Interpretation — Accession Number: 333335

DISCLAIMER FOR TOXIC/POTENTIALLY TOXIC ELEMENTS
This specimen was obtained following administration of a chelating agent, which binds to chemical elements such as lead and mercury, and makes them available for elimination in urine. This is a widely established practice, which has helped many people identify hidden factors affecting their health; however, there is no perfect, "gold standard" chelating agent, so there is always the possibility that the elements appearing in the urine under-represent what is actually present in the various body tissues, depending on the agent used, and the route of administration.

Our reference ranges are derived from normal, healthy, age and gender matched persons who did not receive a chelating agent prior to specimen collection. This begs the question: When healthy, asymptomatic people are given the same chelating agent as this patient, how much of the various toxic elements do they excrete? In other words, are there threshold levels of toxic element excretion that most people can tolerate without incident? The best we can say is that we always have to treat each person as an individual. Even if we could find a "safe threshold" for a given element, there are always going to be people who might still be affected by a body burden below that threshold, for that element.

The fact that a chelating agent pulls a certain amount of a toxic element or elements out of storage and into urine does not always correlate with symptoms/presence of chronic illness. Practitioners need to use their experience and skill, along with the clinical picture, when interpreting these test results.

DMPS DOSE
The recommended oral dose for DMPS is 3 to 5 mg/kg. This patient's dose was 3.3 mg/kg.

ANTIMONY HIGHER THAN AVERAGE WITH PROVOCATION (YELLOW BAR)
Accepting the limitations of the reference ranges, if one hundred healthy, normal people are tested without provocation, about twenty-seven of them would have a result encompassing the range into which this patient's result falls (yellow bar). In an unprovoked sample, this result would be considered higher than average.

Note that DMPS and DMSA are more likely to increase urinary antimony excretion, compared to EDTA. (EDTA has poor affinity for metal ions with valences of +3 or higher.) The literature is not conclusive as to which valence is excreted in urine. Some sources say urine favours the +3 form; others say that both +3 and +5 are both excreted in urine. This is relevant because the +3 valence is less toxic than the +5 form. A urine antimony result should not be casually dismissed based on the assumption that it represents only the less toxic +3 valence.

Most people have some exposure to antimony through the flame-retardant treatments applied to upholstery, carpets, drapes and some clothing (in particular children's pyjamas), as well as contaminated food and water. Interestingly, a 2010 study did not see elevated antimony in firefighters wearing flame-retardant clothing. (de Perio MA et al. A health hazard evaluation of antimony exposure in fire fighters. J Occup Environ Med 2010;52(1):81-84.) Trace amounts of trimethylstibine gas can be produced by the action of the fungus S. brevicaulis and other micro-organisms on inorganic sources of antimony, so there is theoretical concern about toxicity from moulds growing on antimony-containing substrates. Antimony can be found in PETE plastic. Antimony is also found in gunpowder; individuals who frequent firing ranges and load their own ammunition may be exposed to antimony. Cigarette smoke contains some absorbable antimony.

Acute and chronic inhalation of antimony causes upper and lower respiratory tract irritation. Chronic antimony intoxication can lead to nonspecific complaints such as fatigue, achiness, GI complaints and general malaise, as well as muscle weakness. These symptoms may be due to interference with cellular metabolism through binding to sulphydryl sites on various enzymes. Antimony is toxic to the liver and heart, and can cause cardiac arrhythmia and cardiomyopathy.

DMPS/DMSA DO NOT CHELATE CADMIUM WELL
According to Crinnion (Alt Med Rev 2009;14:103-108), cadmium is not mobilized well by DMPS or DMSA. EDTA is the best choice to mobilize cadmium. Cadmium results obtained post-DMPS or post-DMSA challenge may under-reflect tissue stores.

GADOLINIUM VERY HIGH WITH PROVOCATION (PURPLE BAR)
Accepting the limitations of the reference ranges, if one hundred healthy, normal people are tested without provocation, approximately one of them would have a gadolinium result in the same range into which this patient's result falls (purple bar). In an unprovoked sample, this result would be considered very high. A provoked result in this range does not automatically mean that the body burden of gadolinium is also very high.

Note that a right-going red or purple bar for gadolinium might be seen if the creatinine is markedly low. The reported value is the raw result divided by the creatinine; a markedly low creatinine can artificially inflate the normalized result. In the absence of an artifact due to low creatinine, a result in the red or purple range for a toxic/potentially toxic element should, at a minimum, prompt a very thorough review of the clinical picture.

Gadolinium has a few industrial uses. Its alloys are formulated into magnets and electronic components such as video recording heads, compact disks and computer memory. There are no known food sources of gadolinium. The only significant source of exposure in humans comes from its use in contrast agents for magnetic resonance imaging (MRI), where it is present in chelated form (e.g gadopentetate dimeglumine). Unchelated gadolinium is highly toxic. Gadolinium-containing contrast agents have been recently recognized to cause acute nephrogenic systemic fibrosis, as well as acute tubular necrosis when administered to individuals with decreased kidney function. Toxic effects of gadolinium chloride were published more than 15 years ago (Spencer AJ et al. Toxicol Pathol 1997;25:245-255.) and include mineralization in various tissues in the liver, immune cells and cells of reticuloendothelial system. It is unclear whether toxicity of chelated gadolinium arises from the intact molecule, or due to displacement of gadolinium by other metals such as zinc.

MERCURY VERY HIGH WITH PROVOCATION (PURPLE BAR)
Accepting the limitations of the reference ranges, if one hundred healthy, normal people are tested without provocation, approximately one of them would have a mercury result in the same range into which this patient's result falls (purple bar). In an unprovoked sample, this result would be considered very high. A provoked result

in this range does not automatically mean that the body burden of mercury is correspondingly high.

The finding of a purple bar for mercury does not automatically guarantee that mercury is causing a problem for this patient, but it does call for a very careful review of the clinical picture, since mercury toxicity can manifest with a wide variety of symptoms, many of them nonspecific. Note that chronic exposure to mercury can result in kidney damage with decreased excretion of mercury in urine.

Note that a purple bar for mercury might also be seen if the creatinine is markedly low. The reported value is the raw result divided by the creatinine; a markedly low creatinine can artificially inflate the normalized result. In the absence of an artifact due to low creatinine, a result in the red or purple range for a toxic/potentially toxic element should, at a minimum, prompt a very thorough review of the clinical picture.

Individuals can be more or less sensitive to a given level of mercury depending on other factors such as the presence or absence of other toxic elements such as lead, cadmium and arsenic, nutritional status (e.g. antioxidant levels), exposure to other toxins/free radicals, and the overall health status of the individual.

Mercury has the potential to damage/affect any organ system, as it binds to the sulphur-containing portions of proteins found throughout the body. Suppression of the immune system and dysregulation of immunity may occur. General symptoms such as fatigue, headache and loss of appetite are also noted. Neurologic symptoms are prominent and can include numbness, tingling and eventual loss of sensation in the extremities. Alteration in taste (metallic taste), hearing and vision may be seen. Tremor and problems with balance and coordination are common with chronic exposure. Irritability and excitability may also manifest with chronic exposure. Advanced mercury intoxication can result in manic/psychotic behaviour.

Dietary sources of mercury include seafood (especially larger fish toward or at the top of the food chain, e.g. tuna and swordfish) and high fructose corn syrup (HFCS). A serving of larger, predatory fish can contain up to 10 micrograms of methymercury. Children and pregnant women should limit intake of, or avoid eating altogether, these larger fish. The levels of mercury found in HFCS-containing products are substantially lower than those found in seafood, but children consume large (and increasing) amounts of HCFS, and children are more sensitive to the effects of mercury than adults. Non-food sources of mercury include vaccines received prior to around 2002, allergy shots received from the 1960's through 1990's, silvery coloured dental amalgams and some laboratory equipment. Mercury is present in the emissions from coal-fired power plants, and residue from previous use of mercury-based fungicides and pesticides may contaminate some cropland.

Elemental (metallic/liquid) mercury primarily causes health effects when it is breathed as a vapor where it can be absorbed through the lungs. These exposures can occur when elemental mercury is spilled or products that contain elemental mercury break and expose mercury to the air, particularly in warm or poorly-ventilated indoor spaces. Symptoms include: tremors, emotional changes (e.g., mood swings, irritability, excitability, nervousness, excessive shyness), insomnia, neuromuscular changes (such as weakness, muscle atrophy, twitching); headaches; sensorimotor disturbances, diminished performance on tests of cognitive function. At higher exposures there may be kidney effects, respiratory failure and death.

High exposures to soluble mercury salts (inorganic mercury) may result in damage to the gastrointestinal tract, the nervous system, and the kidneys. Symptoms of high exposure to inorganic mercury include: skin rashes and dermatitis, mood swings, memory loss, mental disturbances, and muscle weakness.

The primary health effect of methylmercury on fetuses, infants and children is impaired neurological development. Symptoms of methylmercury intoxication may include: impairment of peripheral vision, peripheral/periorbital sensory disturbances, incoordination, speech impairment, hearing impairment, ataxia; and muscle weakness.

TIN HIGHER THAN AVERAGE WITH PROVOCATION (YELLOW BAR)
Accepting the limitations of the reference ranges, if one hundred healthy, normal people are tested without provocation, about twenty-seven of them would have a tin result in the same range into which this patient's result falls (yellow bar). In an unprovoked sample, this result would be considered higher than average.

Tin exposure consists of inorganic and organic tim compounds. Most inorganic tin (such as tin leaching from food tins) is not absorbed well, but urine does reflect the small fraction that is absorbed after ingestion. (The remaining fraction is excreted in feces.) Depending on the structure, some organotin compounds may be excreted in urine (dimethyltin,trimethyltin). Tributyl tin is excreted in bile. Depending on exposure, urine tin may reflect exposure to toxic or relatively nontoxic forms of tin, but most often will represent absorbed inorganic tin from the diet. Many of the organotin compounds found in seafood, such as tributyltin, are not well represented in

urine.

Soils are widely contaminated with tin, and tin is also naturally present in soil, so tin is present in food. The main sources of tin are canned foods, cereal grains, and toothpaste formulations for sensitive teeth. Leaching of tin from tin cans is much lower in cans that have a protective polymer coating on the inside surface, and in uncoated tins, the levels in food are much higher. Acidic foods and beverages are more likely to leach tin from container walls. Seafood may be contaminated with tin due to bioaccumulation from use of organotin antifouling paint for boat hulls. Water passing through PVC pipes contains organotin as mentioned below. Some multivitamins e.g. Centrum do include a small amount of tin in the formulation and some authorities consider tin to be an essential nutrient in minute amounts.

Tin is used in a wide range of industrial settings: metal food containers, paint, alloys, glassmaking, ceramics, catalysis, electroplating, ink manufacture, as a stabilizer in PVC polymers. Organotin compounds are also used as disinfectants and fungicides used for treating wood.

Tin intoxication presents differently depending on the type of tin. Chronically inhaled low level tetrahydratin exposure manifests with fatigue, depression, headaches, insomnia, adrenal dysfunction, dyspnea and asthma. neurologic symptoms including poor co-ordination, balance problems, neuropathy as well as problems with vision and memory. Other nonspecific complaints such as fatigue, achiness, general malaise and depression can also be noted. Chronic inorganic tin exposure may also be associated with increased risk of heart disease. In general, however, there are no data to indicate any adverse effects in humans associated with chronic exposure to inorganic tin in levels which do not result in acute GI symptoms.

Organotin compounds interfere with heme synthesis and can produce anemia, kidney and hepatocyte damage. They may also stimulate catecholamine production, leading to fluctuating blood glucose and hypertension. In general, organotin compounds are potent neurotoxins because they are quite lipophilic. Acute exposure can lead to encephalopathy and cerebral edema, probably through uncoupling of oxidative phosphorylation. Acute exposure can also result in tremor, seizures, hallucinations and psychosis. Presumably, some or all of these could signs and symptoms might manifest with chronic, low level exposure as well, but this has not been extensively studied. Tributyltin (TBT) has been studied for its role in promoting obesity "TBT and its congeners are chemical stressors or obesogens that activate RXR:PPAR gamma signaling to promote long-term changes in adipocyte number and/or lipid homeostasis after developmental or chronic lifetime exposure". (Grün F et al. Endocrine-disrupting organotin compounds are potent inducers of adipogenesis in vertebrates. Mol Endocrinol 2006;20:2141-2155.) Organotin compounds are highly irritating to skin, and are absorbed through the skin.

URINARY EXCRETION OF URANIUM MAY BE SUPPRESSED BY DMPS

Here the uranium result is below detection limit. There is some literature indicating that DMPS may suppress urinary excretion of uranium. (Br J Radiol. 1978 Aug;51(608):599-607. An evaluation of the use of 99Tcm-dimercaptosuccinic acid (DMSA) as a static renal imaging agent. Bingham JB, Maisey MN.) Urinary excretion of uranium is definitely facilitated by administration of EDTA, according to analysis of our internal database.

DISCLAIMER FOR ESSENTIAL ELEMENTS

A chelating agent was used prior to collection of this specimen. The intended purpose for doing this is to "tease out" toxic elements which might be present in the body, but aren't being released under normal homeostatic conditions. This effect is not restricted to toxic/potentially toxic elements; chelating agents will also pull out excessive amounts of nutritionally important elements such as copper, zinc, manganese and so forth. Therefore, a finding of an elevated level of a nutritionally important element, after chelation, does not mean there is an excess of the element in question. In theory, reference ranges for the essential elements could be developed by chelating normal, healthy people who have no complaints. In practice, this has not been done.

BARIUM BELOW DETECTION LIMIT

The bar corresponding to the result for barium is not shown, because the result is very low, below detection limit. Urine is not the principal route of excretion for barium; probably less than 10% of barium absorbed by mouth is excreted in urine, with most barium excreted in feces. Therefore the clinical relevance of urine barium is not clear. The amount excreted can vary over a wide range, without reflecting a significant change in body burden.

Barium compounds which are water soluble are normally found in small amounts in many different foods, so some barium is expected to be present in the body. Low barium excretion might be seen in the context of renal failure, but otherwise, there is no recognized clinical significance to low barium excretion.

CHROMIUM BELOW DETECTION LIMIT WITH PROVOCATION

There is no bar for chromium because the raw result (not normalized to creatinine) was below the detection limit of the instrument. In general, this means the result is quite low.

A significant amount of chromium is excreted in urine, so urine chromium is reflective of exposure and bodily stores. Diurnal variation in urine chromium secretion may limit the utility of spot urine chromium determination. (A 24-hour collection will be more accurate and should be performed if significant chromium deficiency is suspected.) While low chromium may be associated with high LDL cholesterol and peripheral neuropathy, chromium is particularly important for proper potentiation of insulin signalling and acts via chromodulin or low molecular weight chromium binding substance (LMWCr). LMWCr is an oligopeptide naturally found in the body. (J Am Coll Nutr 1999;18:6-12. Mechanisms of chromium action: low-molecular-weight chromium-binding substance. see also Vincent JB. J Nutr 2000;130:715-718.The biochemistry of chromium. Vincent JB.) Chromium is often supplemented as GTF or glucose tolerance factor, which is a chromium-glutathione-nicotinic acid complex found in yeast.

Refined carbohydrates are low in chromium, so an excess of these in the diet may eventually lead to chromium deficiency. Excessive consumption of refined carbohydrates may also actively lead to the depletion of chromium (Arch Iran Med 2008;11:57-64.The effect of high and low glycemic index diets on urinary chromium in healthy individuals: a cross-over study. Hajifaraji M, Leeds AR). Long term TPN can result in chromium deficiency. Intense endurance exercise may deplete chromium.

Good sources of chromium include romaine lettuce, broccoli, onions, tomatoes, Brewer's yeast, cinnamon, oysters, liver, kidney, whole grains, bran cereals, and potatoes. If chromium is to be supplemented, many practitioners prefer to use GTF chromium as opposed to chromium picolinate.

Low urine chromium excretion following administration of an agent capable of binding chromium may lend support to the notion that there might be decreased chromium available in body tissues.

COBALT VERY LOW WITH PROVOCATION (PURPLE BAR POINTING LEFT)
Accepting the limitations of the reference ranges, if several hundred healthy, normal people are tested without provocation, approximately one of them would have a cobalt result in the same range into which this patient's result falls (purple bar pointing left). In an unprovoked sample, this result would be considered very low.The Handbook on the Toxicology of Metals 3rd ed. unequivocally states that urine is the primary route of excretion for cobalt in humans and is a good marker of intake and exposure to cobalt. The ATSDR Public Health Statement on cobalt concurs.

Although some cobalt is ingested in the form of Vitamin B12 (organic), most of the cobalt consumed in the diet is inorganic cobalt. Therefore, urine cobalt does not reflect Vitamin B12 status, and low urinary excretion of cobalt should not be taken as an indication of Vitamin B12 deficiency. No human deficiency state has been identified for cobalt. There are various reports on a need for cobalt in the diets of ruminants such as sheep and cattle; the cobalt is needed to allow the gut flora to manufacture Vitamin B12, so the growth problems seen in cobalt-deprived animals actually stem from a Vitamin B12 deficiency. Excess iron can inhibit the absorption of cobalt. Low urine cobalt might conceivably be seen in the face of excess iron ingestion, but the clinical implication of this is not known.

Dietary cobalt restriction has been used to address skin rashes, so low urinary cobalt excretion on such a diet would indicate compliance. (Foods high in cobalt include Brazil nuts, flaxseeds and garbanzo beans as well as other dried fruits and nuts, chocolate, oysters and clams, beef liver, beet sugar. Cobalt is also added to food in the form cobalt(II) chloride, as a supplement.) Low urine cobalt might also be seen in situations where digestion and assimilation of nutrients is impaired (impaired gastric acid/pancreatic insufficiency/inflammation/surgery).

A low cobalt result in the face of administration of a chelating agent known to bind cobalt would reinforce the notion that there is not much cobalt available in tissue to be chelated.

COPPER HIGHER THAN AVERAGE WITH PROVOCATION (YELLOW BAR POINTING RIGHT)
Accepting the limitations of the reference ranges, if one hundred healthy, normal people are tested without provocation, about thirteen of them would have a copper result in the same range into which this patient's result falls (yellow bar pointing right). In an unprovoked sample, this result would be considered higher than average. All commonly used chelating agents will cause more copper to appear in the urine than would be the case under homeostatic conditions, elevations up to ten times normal are not uncommon. Urine is normally a minor excretion pathway for copper, with most copper coming out in bile/feces. The finding of high urinary copper after challenge with a chelating agent is not reflective of high bodily stores of copper. Elevated copper in urine after chelation is not automatically indicative of copper overload.

PHOSPHORUS LOWER THAN AVERAGE WITH PROVOCATION (YELLOW BAR POINTING LEFT)
Accepting the limitations of the reference ranges, if one hundred healthy, normal people are tested without

provocation, about thirteen of them would have a phosphorus result in the same range into which this patient's result falls (yellow bar pointing left). In an unprovoked sample, this result would be considered lower than average. The issue of reference ranges is of less concern when the chelating agent does not interact with the element in question, as is the case here.

Urine phosphorus fluctuates throughout the day; a timed 24-hour collection is the preferred specimen to measure phosphorus. Random samples are most often used in children. Urine phosphorus excretion is related to dietary intake. A low phosphorus diet may decrease urine phosphorus. (Dietary sources of phosphorus include milk and other dairy products, eggs, green leafy vegetables; peas and beans, nuts, chocolate, liver, and turkey.) Other causes of low urine phosphorus include high insulin, high growth hormone, elevated thyroid hormone, excess Vitamin D intake, high dietary potassium intake, hypoparathyroidism and decreased intestinal absorption of phosphorous (phosphate-binding antacids, bile acid sequestrants, other malabsorption states).

VANADIUM BELOW DETECTION LIMIT WITH PROVOCATION
There is no bar for vanadium because the raw result (not normalized to creatinine) was below the detection limit of the instrument. In general, this means the result is quite low.

Urine is the primary route of excretion for vanadium, so urinary vanadium is thought to be reflective of exposure to vanadium. Low vanadium probably reflects lower than average vanadium intake. Vanadium is similar to chromium, and will potentiate the post-receptor signalling of insulin. There is debate as to whether vanadium has a unique, essential biochemical function; therefore the clinical implications of of low urinary vanadium are not known. It may play a role in bone formation and joint health, based on research in vanadium-deprived goats. It might be worthwhile assessing for metabolic syndrome and insulin resistance in the face of low vanadium. Foods richer in vanadium include seafood (especially oysters but seafood in general harvested near offshore oilwells), dry mushrooms, dill, parsley, and black pepper.

The daily intake of vandium is estimated to be in the range of 20 to 30 micrograms per day. Vanadium is available as a nutritional supplement, and doses are often in the tens of mg per day. Recognize that doses of vanadium in the mg range are pharmacologic, not physiologic.

Low urine vanadium excretion following administration of an agent capable of binding vanadium may lend support to the notion that there might be decreased vanadium available in body tissues.

ZIRCONIUM BELOW DETECTION LIMIT
There is no bar for zirconium because the raw result (not normalized to creatinine) is below the detection limit of the instrument. In general, this means the zirconium result is quite low. Zirconium is very poorly absorbed by the GI tract, and although it is excreted in urine, levels are low. The clinical significance of urinary zirconium is unclear.

Most adults are routinely exposed to zirconium due to use of antiperspirants. The element is ubiquitous in many kinds of plants, and foods. Higher levels are found in dairy, cereals, nuts oils and fats, relative to meat. (Ghosh et al. Zirconium: An Abnormal Trace Element in Biology. Biological Trace Element Research 1992;35:247-271.) It has a wide range of industrial uses including adhesives, alloys, catalysts, ceramics, drilling mud, gemstones and glass. It is used in the nuclear industry, for construction of reactors. Occupational exposure takes place mostly through inhalation of Zr-containing dust.

Zirconium is thought to be more or less nontoxic, but also nonessential. Zirconium does have some modest immunostimulatory properties and it does accumulate in the brain. Inhalation of zirconium-containing dust has been linked to sarcoidosis and topical application of zirconium can induce a granulomatous reaction in susceptible individuals.

George Gillson, MD PhD
Medical Director

With the exception of toxicity investigations for selected elements, the College of Physicians and Surgeons of Alberta considers urine elemental analysis to be complementary medicine. Analysis of elements in urine as an indication of nutritional status has been used in research but is not yet approved by the College of Physicians and Surgeons. Rocky Mountain Analytical does not diagnose or make treatment recommendations as data is provided for research and educational purposes only.