

# URINE TOXIC METALS



**LAB #:** U000000-0000-0  
**PATIENT:** Sample Patient  
**ID:** PATIENT-S-00014  
**SEX:** Male  
**AGE:** 12

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

## POTENTIALLY TOXIC METALS

METALS	RESULT µg/g creat	REFERENCE RANGE	WITHIN REFERENCE RANGE	ELEVATED	VERY ELEVATED
Aluminum	210	< 60			
Antimony	0.1	< 0.5			
Arsenic	< dl	< 117			
Barium	2.4	< 8			
Beryllium	0.2	< 0.3			
Bismuth	0.05	< 20			
Cadmium	1.1	< 0.5			
Cesium	2.6	< 12			
Gadolinium	0.3	< 0.4			
Lead	63	< 5			
Mercury	1.6	< 5			
Nickel	24	< 15			
Palladium	< dl	< 0.3			
Platinum	0.04	< 1			
Tellurium	< dl	< 0.3			
Thallium	0.1	< 0.8			
Thorium	0.03	< 0.05			
Tin	1	< 15			
Titanium	< dl	< 15			
Tungsten	0.3	< 0.6			
Uranium	0.2	< 0.04			

## URINE CREATININE

	RESULT mg/dL	REFERENCE RANGE	2SD LOW	1SD LOW	MEAN	1SD HIGH	2SD HIGH
Creatinine	35.7	25- 180					

## SPECIMEN DATA

**Comments:**  
 Date Collected: 12/15/2009      pH upon receipt: 7.4      Collection Period: **timed: 6 hours**  
 Date Received: 12/17/2009      <dl: less than detection limit      Volume:  
 Date Completed: 12/22/2009      Provoking Agent: **CAEDTA**      Provocation: **POST PROVOCATIVE**  
 Method: **ICP-MS**

Toxic metals are reported as µg/g creatinine to account for urine dilution variations. **Reference ranges are representative of a healthy population under non-challenge or non-provoked conditions.** No safe reference levels for toxic metals have been established.

V12

# URINE ESSENTIAL ELEMENTS



**LAB #:** U000000-0000-0  
**PATIENT:** Sample Patient  
**ID:** PATIENT-S-00014  
**SEX:** Male  
**AGE:** 12

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

## ESSENTIAL ELEMENTS

ELEMENTS	RESULT mEq/g creat	REFERENCE RANGE	PERCENTILE					
			2.5 <sup>th</sup>	16 <sup>th</sup>	50 <sup>th</sup>	84 <sup>th</sup>	97.5 <sup>th</sup>	
Sodium	340	43.5 - 348						
Potassium	28	26 - 180						
	µg/mg creat							
Phosphorus	490	350 - 1700						
Calcium	820	30 - 250						
Magnesium	83	20 - 300						
Zinc	29	0.15 - 2.5						
Copper	0.024	0.012 - 0.12						
Sulfur	660	308 - 1980						
Manganese	0.11	0.0005 - 0.02						
Molybdenum	0.22	0.02 - 0.25						
Boron	1.4	0.8 - 8.4						
Chromium	0.004	0.0005 - 0.01						
Selenium	0.093	0.04 - 0.35						
Strontium	0.15	0.06 - 0.48						
Vanadium	0.007	0.0002 - 0.004						
					68 <sup>th</sup>		95 <sup>th</sup>	
Cobalt	0.044	< 0.007						
Iron	3.5	< 2						

## URINE CREATININE

	RESULT mg/dL	REFERENCE RANGE					
			2SD LOW	1SD LOW	MEAN	1SD HIGH	2SD HIGH
Creatinine	35.7	25 - 180					

## SPECIMEN DATA

**Comments:**

Date Collected: 12/15/2009      pH Upon Receipt: 7.4      Collection Period: **timed: 6 hours**  
 Date Received: 12/17/2009      <dl: less than detection limit      Volume:  
 Date Completed: 12/22/2009      Provoking Agent: CAEDTA      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

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

V12

## INTRODUCTION

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

### 1) 24 Hour Collections

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

### 2) Timed Samples (< 24 hour collections)

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

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

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

For essential elements, the mean and the reference ranges apply to human urine under non-challenge, non-provocation conditions. Detoxification therapies can cause significant deviations in essential element content of urine. For potentially toxic elements, the expected range also applies to conditions of non-challenge or non-provocation. Diagnostic or therapeutic administration of detoxifying agents frequently raise the urinary levels content of potentially

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toxic elements. Descriptive texts appear in this report on the basis of measured results and correspond to non-challenge, non-provocation conditions.

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

#### ALUMINUM HIGH

This individual's urine aluminum is higher than expected; urine is the primary mode of excretion for absorbed aluminum.

Common sources of bioavailable aluminum include: aluminum cookware, flatware and especially coffee pots; aluminum hydroxide anti-acid formulations; some types of cosmetics, especially deodorants; some colloidal minerals and some herbs or herbal products. Aluminum cookware is particularly of concern if acid foods are cooked such as tomato paste (contains salicylates). In cosmetics and deodorants, aluminum chloride may be present as an astringent. In water purification, alum (sodium aluminum sulfate) may be used to coagulate dispersed solids and improve water clarity. Alumina or  $Al_2O_3$  is very stable chemically and not bioavailable. Silica limits the solubility of aluminum and aluminum silicate is not very bioavailable. Clays, bentonite for example, contain aluminum that has poor bio-availability. Aluminum food containers are manufactured with polymer or plastic coatings that prevent direct food-aluminum contact provided such coatings are not damaged.

In the GI tract, phosphates react with aluminum ions forming insoluble aluminum phosphates. If this phosphate-blocking were 100% efficient, then virtually no aluminum would be absorbed. Evidently, this phosphate-forming process is incomplete because body tissue levels (such as hair) usually contain measurable amounts of aluminum. In the body aluminum follows a path of increasing phosphate concentration: plasma, cytosol, cell nucleus. Once in the nucleus, it adversely affects protein formation. Long-lived cells such as neurons are susceptible to long-term accumulation. Al is considered neurotoxic. Without intervention, aluminum accumulates continually in the body with the highest concentration occurring at old age or death.

A hair element test can be used to corroborate increased body burden of aluminum. An oral provocation with the amino acid glycine, 80 mg/Kg body weight (in divided doses) 24 hours before a diagnostic EDTA chelation with subsequent urine collection can be done to confirm aluminum excess. (Eliminate food/beverage sources of Al during this procedure.)

#### BIBLIOGRAPHY FOR ALUMINUM

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2. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publ, Chelsea MI p 16-20 1986.
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#### CADMIUM HIGH

This individual's urine cadmium level equals or exceeds twice the maximum expected level. This element is insidiously toxic with chronic accumulations affecting renal function, pulmonary and cardiovascular tissues, bone, and the peripheral nervous system. Without intervention, the biological half-life of Cd in humans exceeds 20 years (Harrison's Principles of Internal Medicine, 13th ed, pp 2463-64).

Chronic manifestations associated with this degree of cadmium excess include: hypertension, weight loss, microcytic-hypochromic anemia, lymphocytosis, proteinuria with wasting of beta2 microglobulin, emphysema and pulmonary fibrosis (if inhalation was a route of contamination), atherosclerosis, osteomalacia and lumbar pain, and peripheral neuropathy. Acute inhalation of Cd dusts, fumes or soluble salts may produce cough, pneumonitis and fatigue. Manifestations of Cd toxicity may be lessened or delayed by an individual's protective and detoxication capacities. Zinc and vitamin E are protective; metallothionein and glutathione bind Cd and help detoxify it initially.

Smoking can be a source for as much as 0.1 mcg Cd per cigarette (HEW Pub. No. NIOSH 76-192, US Govt. Printing Ofc., 1976). Some medical authorities consider Cd to be a carcinogen for lung cancer (Harrison's Principles, 13th ed, op. cit. pp 2463). Other occupational or environmental sources include: mining and smelting activities, pigments and paints, electroplating, electroplated parts (e.g., nuts and bolts), batteries (Ni-Cd), plastics and synthetic rubber, photographic and engraving processes, old drums from some copy machines, photoconductors and photovoltaic cells, and some alloys used in soldering and brazing.

Depending upon body burden and upon type, duration and dosage of detoxifying agents, elevated urine cadmium may occur after administration of: EDTA, DMPS, DMSA or D-penicillamine. A confirming test for Cd excess is elemental hair analysis, barring exogenous contamination. Blood Cd measurement may not be indicative (Harrison's Principles of Internal Medicine, 13th ed., pp 2463).

#### BIBLIOGRAPHY FOR CADMIUM

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## LEAD HIGH

This individual's urine lead exceeds three times the upper expected limit per the reference population. Because most of the body burden of lead is excreted in urine, this finding indicates significant burden with attempted detoxication of lead.

Sources of lead include: old lead-pigment paints, batteries, industrial smelting and alloying, some types of solders, 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 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.

Confirming tests for lead excess are: urinary lead following provocation with intravenous EDTA or DMPS, or oral DMSA, and hair element analysis. Whole blood analysis can be expected to reflect only recent exposures and does not correlate well with total body burden of lead (Carson, Ellis and McCann, Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, p. 130, 1987). Increased blood protoporphyrins is a finding consistent with lead excess, but may occur with other toxic exposures as well. Also, zinc protoporphyrin measurement may yield a false negative result in zinc deficiency. Preliminary studies performed at DDI indicate a significant increase in the biliary/fecal excretion of lead following intravenous administration of vitamin C.

### BIBLIOGRAPHY FOR LEAD

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2. "Preventing Lead Poisoning in Young Children", US Centers for Disease Control, Atlanta, GA, Oct. 1991 Statement, US Dept. of Health and Human Services.
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6. Shubert J. et al. "Combined Effects in Toxicology - a Rapid Systematic Testing Procedure: Cadmium, Mercury and Lead" - J. Toxicology and Environmental Health, 4:763-776, 1978.

## NICKEL HIGH

This individual's urine nickel is elevated which may or may not be of significance. Urinary excretion of nickel bound to cysteinyl or 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 nickel comes from food or drink, and intakes can vary by factors exceeding 100 depending upon geographical location, food type, and water supply. Depending upon chemical form and physiological factors, from 1 to 10% of dietary nickel may be absorbed from the gastrointestinal tract into the blood. Urine reflects recent exposure to nickel and may vary widely in nickel content from day to day due to the above factors.

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, and hydrogenated oils
- . Nickel-cadmium batteries
- . Nonprecious, semiprecious dental materials
- . Nickel-containing prostheses
- . Electroplating, 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 observed nickel contaminations are manifested as dermatoses - contact dermatitis and atopic dermatitis. However, Ni hypersensitizes the immune system causing hyperallergenic responses to many different substances. Because nickel can displace zinc from binding sites on enzymes, it can have inhibiting or activating effects on such enzymes. Nickel sensitivity is observed to be three to five times more frequent in women than in men.

Other laboratory tests or clinical findings that would be indicative of nickel excess are; hair element analysis, presentation of multiple allergic sensitivities, dermatitis, positive patch test for "Ni allergy", proteinuria, hyperaminoaciduria (by 24-hour urine amino acid analysis). Detoxification treatments with administration of EDTA or sulfhydryl agents (DMPS, DMSA, D-penicillamine) may increase urine nickel levels depending upon: body burden and mobility in tissues, duration of treatment, dosage and other factors.

### 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.
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4. Medical and Biological Effects of Environmental Pollutants: Nickel, Nat. Acad. Sci, Washington DC, 1975.

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5. Ambient Water Quality Criteria for Nickel, US EPA NTIS, Springfield, VA. Publ No. PB81-117715,1980.

#### URANIUM HIGH

This individual's urine uranium level exceeds three times the upper expected limit. Because renal excretion is the major mode for body excretion of absorbed uranium, this finding is consistent with an excessive exposure to uranium or to an unusually high body burden of this element.

Uranium is a radioactive element having 10 isotopes with half lives that exceed one hour. U238 constitutes about 99% of the naturally-occurring uranium and this is the isotope measured at DDI and reported for this individual. U238 has a half life of  $4.5 \times 10^9$  years. It decays by alpha emission to produce thorium, Th234, the initial step in a decay chain that eventually leads to lead. Alpha, beta and gamma emissions occur during this decay process. Because of the very long half life, the radioactivity danger is only slight. However, exposure to enriched or nuclear fuel grade uranium (high in U235) does pose a health hazard. The measured result (U238) does not reflect or imply exposure to enriched uranium.

The major concern for (natural) uranium excess is toxochemical rather than radiochemical. Uranium is a chemically-reactive element, has four valences (3,4,5 or 6), and may combine with: carbonate, phosphate, citrate, pyruvate, malate, lactate, etc. in body tissues. When not excreted in urine, it may accumulate in the kidneys, spleen, liver, and in bone (substituting for calcium in hydroxyapatite). Uranium is nephrotoxic, causing damage to the glomeruli and proximal tubules. An early sign of uranium excess is general fatigue. Renal damage is reflected by proteinuria, hyperaminoaciduria and glucosuria. Albuminuria and urinary catalase are findings consistent with uranium excess. Elevated hair uranium is a confirmatory finding; whole blood and fecal analyses may corroborate recent or ongoing exposures.

Uranium is more common than mercury, silver or cadmium in the earth's rock strata, and may be present, at low levels, in ground (drinking) water. Most commercial use of uranium is for nuclear fuel, but it may be present in ceramics or colored glass, especially ancient or antique, yellow-colored glass.

#### BIBLIOGRAPHY FOR URANIUM

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#### CALCIUM HIGH

Urine analysis is not a preferred way to assess body calcium stores. Nutritional sufficiency of

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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.

#### BIBLIOGRAPHY FOR 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.

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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).

#### BIBLIOGRAPHY FOR ZINC

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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

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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.

#### BIBLIOGRAPHY FOR IRON

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#### MANGANESE HIGH

High urinary manganese may or may not correspond to global manganese excess or manganese loss from body tissues because the major route for manganese uptake, reuptake, and excretion is via the bile, intestinal transport and feces. Typically, less than one-half 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 manganese may be increased in patients with biliary obstruction or cirrhosis. Urinary manganese levels may fluctuate without reflecting or influencing body stores.

Elevated urinary manganese can occur following intravenous administration of EDTA; levels up to 50 micrograms per 24 hours are commonly seen without evidence of manganese overload (DDI experience). Elevated urinary manganese also occurs following oral administration of D-penicillamine with levels up to 25 micrograms per 24 hours commonly seen. DMSA and DMPS treatments also may result in similar increases in urinary manganese 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. Dopamine, glucagon and cyclic AMP are reported to do this. Environmental or industrial sources of manganese include: mining, metals or ores refining and processing, 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 manganese, 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 contain high concentrations of manganese.

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Relative to other essential trace elements, excessive manganese retention can be quite neurotoxic. Inhalation, as a result of occupational exposure, is the route of assimilation that is most harmful. Some symptoms and manifestations of manganese excess or poisoning are: 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 manganese excess is suspected. These are: whole blood manganese analysis, blood cell elements, and hair elemental analysis.

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#### MOLYBDENUM HIGH

This individual's molybdenum level exceeds one standard deviation above the mean of the reference population which means that this individual's urine molybdenum level corresponds to the highest 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 intestines or is excreted in sweat.

Administration of EDTA is not observed to raise molybdenum levels in the urine. Significant urine Mo levels in molybdenum normal individuals (adults) may occur with D-penicillamine administration and up to 300 micrograms/24 hours is commonly observed (Doctor's Data). Similar increases with DMSA administration would be expected. For DMPS (administered slow-push intravenously) up to 250 micrograms Mo/24 hours is commonly seen, and prolonged use of dithiol chelators can deplete molybdenum stores.

Elevated Mo in urine can occur in renal wasting syndromes, nephritis, and biliary dysfunction or blockage. Other elements would then be relatively more increased (Mn, Fe, Cu). Administration of supplemental copper in high doses can result in elevated urine molybdenum; copper and molybdenum are mutually antagonistic with respect to body retention. Tungsten is a more powerful antagonist. Individuals doing tungsten-inert-gas ("TIG") welding may episodically excrete high amounts of molybdenum (but may actually be subnormal in body tissue levels). Increased dietary sulfate levels reduce intestinal absorption and increase renal excretion of molybdenum (eg. MSM).

Molybdenum is relatively nontoxic. Studies with animals show that huge oral doses are required to produce clinical symptoms which are those of copper deficiency: loss of appetite, anemia, arthritic signs, diminished glucose tolerance, loss of skin pigmentation. Moderately excessive molybdenum uptake can produce gout-like symptoms and elevated blood/urine levels of uric acid.

If molybdenum excess is suspected, the following laboratory tests could be informative: serum and urine uric acid levels, hair multielement analysis including copper and molybdenum, packed blood cell molybdenum and copper levels, erythrocyte SOD activity.

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#### Vanadium High

A high level of Vanadium (V) was found in this urine sample. Increased V, especially in an unprovoked urine sample, reflects recent excessive exposure/intake and absorption.

Vanadium can be highly toxic. Excess levels of V can result from over-zealous V supplementation. It may also result from chronic consumption of fish, shrimp, crabs, and oysters that have been harvested near offshore oil rigs. Industrial/environmental sources of V include: processing of mineral ores, phosphate fertilizers, combustion of oil and coal, production of steel, and chemicals used in the fixation of dyes and print (Metals in Clinical and Analytical Chemistry, 1994). V is used in producing rust-resistant, spring and high speed tool steels. Vanadium pentoxide and other vanadates are used as catalysts in the production of sulfuric acid and formaldehyde. Urban air sheds in industrialized areas may have higher levels of V.

Symptoms of V toxicity vary with chemical form and route of absorption. Inhalation of excess V may produce respiratory irritation and bronchitis. Excess ingestion of V can result in decreased appetite, depressed growth, diarrhea/gastrointestinal disturbances, nephrotoxic and hematotoxic effects. Pallor, diarrhea, and green tongue are early signs of excess V and have been reported in human subjects consuming about 20 mg V/day (Modern Nutrition in Health and Disease, 8th edition, eds. Shils, M., Olson, J., and Mosha, S., 1994).

Confirmatory tests for excess exposure to V are the Doctor's Data red blood cell element analysis or whole blood vanadium. EDTA (but not DMPS or DMSA) is an effective chelator of V. Therefore excessive retention (body burden) of V can be assessed by comparing pre- and post-Ca-Na<sub>2</sub>-EDTA urine V levels.