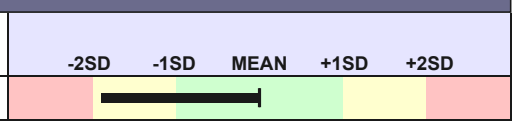
PATIENT: Patient Sample
ID: Rxxx
SEX: Female
AGE: 62 DOB: 09/22/1963

Overland Park, KS 66213 U.S.A.

Toxic Metals; urine

TOXIC METALS		RESULT µg/g Creat	REFERENCE INTERVAL
Aluminum	(Al)	2.3	< 15
Antimony	(Sb)	0.099	< 0.18
Arsenic	(As)	9.2	< 40
Barium	(Ba)	1.2	< 5
Beryllium	(Be)	<dl	< 0.01
Bismuth	(Bi)	<dl	< 0.8
Cadmium	(Cd)	0.20	< 0.6
Cesium	(Cs)	5.9	< 9
Gadolinium	(Gd)	<dl	< 0.5
Lead	(Pb)	0.66	< 1.1
Mercury	(Hg)	0.50	< 0.8
Nickel	(Ni)	1.8	< 4
Palladium	(Pd)	<dl	< 0.3
Platinum	(Pt)	<dl	< 0.1
Tellurium	(Te)	<dl	< 0.5
Thallium	(Tl)	0.22	< 0.4
Thorium	(Th)	<dl	< 0.015
Tin	(Sn)	0.19	< 3
Tungsten	(W)	0.13	< 0.4
Uranium	(U)	0.018	< 0.03

URINE CREATININE		RESULT mg/dL	REFERENCE INTERVAL
Creatinine		40.2	35 – 240



SPECIMEN DATA

Comments:
Date Collected: 12/08/2025
Date Received: 12/11/2025
Date Reported: 12/11/2025
Methodology: ICP-MS QQQ, Creatinine by Jaffe Reaction

Collection Period: Random
Collection Time: 06:26
pH upon receipt: Acceptable

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

PATIENT: Patient Sample
ID: Rxxx
SEX: Female
AGE: 62 DOB: 09/22/1963

Overland Park, KS 66213 U.S.A.

Essential Elements; urine

ESSENTIAL ELEMENTS				
		RESULT mEq/g Creat	REFERENCE INTERVAL	PERCENTILE (2.5th, 16th, 50th, 84th, 97.5th)
Sodium	(Na)	291	40 – 200	
Potassium	(K)	61.0	20 – 90	
		RESULT µg/mg Creat		
Phosphorus	(P)	774	150 – 1000	
Calcium	(Ca)	316	20 – 250	
Magnesium	(Mg)	312	20 – 200	
Zinc	(Zn)	0.57	0.09 – 1.3	
Copper	(Cu)	0.0078	0.003 – 0.022	
Sulfur	(S)	664	250 – 900	
Molybdenum	(Mo)	0.0995	0.01 – 0.11	
Boron	(B)	3.5	0.5 – 3.8	
Lithium	(Li)	3.22	0.008 – 0.18	
Selenium	(Se)	0.235	0.03 – 0.2	
Strontium	(Sr)	0.285	0.035 – 0.26	

		RESULT µg/g Creat	REFERENCE INTERVAL	(68th, 95th)
Cobalt	(Co)	0.40	< 1	
Iron	(Fe)	20	< 50	
Manganese	(Mn)	0.28	< 0.4	
Chromium	(Cr)	1.2	< 1.5	
Vanadium	(V)	0.18	< 0.6	

URINE CREATININE				
		RESULT mg/dL	REFERENCE INTERVAL	-2SD, -1SD, MEAN, +1SD, +2SD
Creatinine		40.2	35 – 240	

SPECIMEN DATA

Comments:
Date Collected: 12/08/2025
Date Received: 12/11/2025
Date Reported: 12/11/2025
Collection Period: Random
Collection Time: 06:26
pH upon receipt: Acceptable
Methodology: ISE, Spectrophotometry, ICP-MS QQQ, Creatinine by Jaffe Reaction

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

Introduction

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

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

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

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

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

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

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

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

If one intends to utilize urinary elements analysis to assess nutritional status or renal wasting of essential elements, it is recommended that unprovoked urine samples be collected for a complete 24 hour period. For 24 hour urine collections essential elements are reported as mg/24 h. For timed or first morning urine collections, elements are normalized per gram creatinine to avoid the potentially great margin of error which can be introduced by variation in the sample volume (concentration). It should be noted that creatinine excretion for an individual may vary to some extent over the course of a day, and from day to day.

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

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

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

If one intends to utilize urinary elements analysis to assess nutritional status or renal wasting of essential elements, it is recommended that unprovoked urine samples be collected for a complete 24 hour period. For 24 hour urine collections essential elements are reported as mg/24 h, and toxic metals are reported as µg/24 h. For timed, random or first morning urine collections, elements and metals are normalized per gram creatinine to avoid the potentially great margin of error that can be introduced by variation in the sample volume (concentration). It should be noted that creatinine excretion for an individual may vary to some extent over the course of a day, and from day to day.

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

Boron High

Boron (B) is introduced to the body mainly through food (noncitrus fruits, leafy vegetables, nuts, legumes, wine, cider, beer) and drinking water but is also found in food preservatives (sodium borate), and insecticides (boric acid). Evidence for biological essentiality in animals (including humans) has been presented. It has been proposed that boron contributes to living systems by acting indirectly as a proton donor and that it exerts a particular influence on cell membrane and structure and function. In humans boron is responsible for the hydroxylation of various substances in the body. It may enhance the production of various hormones such as testosterone, estrogen, DHEA, and 1,25 dihydroxycholecalciferol. Boron is very readily absorbed and excreted in the urine yet its concentration remains quite low in the serum. Based on urinary recovery findings, more than 90% of ingested B is usually absorbed. Boron is distributed throughout the tissues and organs of animals and humans at concentrations mostly between 4.6 and 55.5 nmol (0.05 and 0.6 µg)/g fresh weight. Among the organs that contain the highest amounts of B are bone, spleen, and thyroid. It appears to be most concentrated in the thyroid gland.

Boron has a low order of toxicity even with intakes as high as 40mg/day in some parts of the world. However, high body burden of the element may be harmful, especially to young animals (including human neonates). Reports have shown that when doses equivalent to more than 46 mmol (0.5 g) B daily were consumed, disturbances in appetite, digestion, and health occurred. Acute toxicity signs include nausea, vomiting, diarrhea, dermatitis, and lethargy. High B ingestion also induces riboflavinuria.

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.

Chromium High

The chromium level in this urine sample is high. Chromium (Cr) is essential for proper metabolism of glucose in humans. It potentiates the action of insulin via glucose tolerance factor (GTF) which is Cr+3 bound in a dinicotinic acid-glutathione complex. Other functions of Cr include aiding in lipid metabolism and assisting with HDL/LDL cholesterol balance.

Sources of exposure to hexavalent Cr (Cr+6) include: manufacture and use of ferrochromium and stainless steel, chromium plating (plumbing, electrical appliances, automotive parts), welding, commercial spray painting, wood finishing and leather tanning industries, and handling of cement. Extensive mining of Cr and disposal of spent ore presents a serious environmental problem in certain regions.

The molecular process of reducing Cr+6 to Cr+3 determines the degree of Cr toxicity due to the fact that Cr+6 can react with, for example, reduced glutathione, ascorbic acid, NADH, NADPH, lipids, proteins, and nucleic acids.

Phytates decrease oral assimilation of Cr+3 whereas nicotinic acid and vitamin C increase absorption of Cr+3. Zinc, vanadium and iron compete with Cr for absorption.

Significance of High Chromium: When present in excessive amounts, Cr+6 may be mutagenic and carcinogenic. Elevated Cr levels have been found in patients with cerebral thrombosis and cerebral hemorrhage. Self-supplementation has been reported to be associated with insomnia and increased unpleasant dream activity in some individuals. Exposure to Cr via excessive skin contact with reactive chemical forms can result in allergic dermatitis (sometimes with permanent skin sensitization to chromium) skin ulcers, bronchitis, and lung and nasal carcinomas. Systemic effects of absorbed Cr+6 may feature bronchial asthma (from inhaling Cr dusts) and kidney and/or liver dysfunction.

Other Useful Analyses: Depending upon the route of absorption of chromium, dermatological chromium problems may or may not be reflected by whole blood levels. Measurement of hyaluronidase activity in serum may be helpful as this has been seen to increase in Cr overexposure. Hair element analysis can be used to corroborate suspected recent Cr exposure. Provocative urine testing with EDTA can be used to assess Cr stores. EDTA, but not DMPS or DMSA, is an effective chelator of Cr.

Iron High

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

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

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

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

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

Lithium High

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

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

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

Magnesium High

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

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

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

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

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

Magnesium status can be difficult to assess; whole blood and blood cell levels are more indicative than serum/plasma levels. The magnesium challenge method may be most indicative: baseline 24-hour urine Mg measurement, followed by 0.2 mEq/Kg of intravenous Mg, followed by 24-hour Mg measurement. A deficiency is judged to be present if less than 80% of the Mg challenge is excreted. Ref. Jones, et al. "Magnesium Requirements in Adults", Med Journal Clin Nutr, 20 (1967) p.632-35.

Manganese High

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

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

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

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

Relative to other essential trace elements, excessive Mn retention can be neurotoxic. Inhalation, as a result of occupational exposure, is the route of assimilation that is most harmful. Some symptoms and manifestations of excess Mn exposue include:psychiatric disturbances (hyperirritability, hallucinations, violence), tremor,Parkinson's-like symptoms, anorexia, sexual impotence, and speech disturbance.

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

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 dithiolchelators 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 does 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.

Phosphorus High

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

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

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

Selenium High

Urine accounts for about one-half of the total body excretion of dietary selenium when normal amounts are ingested. Seafood, organ meats, cereal grains, and seleniferous vegetables (garlic, onions) are good dietary sources. Selenium is also excreted in sweat, and lesser amounts are present in fecal matter. Because diets are highly variable in selenium content, urine is not a reliable indicator of selenium adequacy or function. However, selenium excess or overload can feature high urinary levels. Without occupational or environmental exposure, or excessive dietary intake, urinary selenium is expected to be below 100 micrograms per liter.

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

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

Sodium High

The concentration of sodium in this urine sample is higher than expected and is more than two standard deviations above the mean. A high urine sodium concentration can indicate that the kidney's capacity to reabsorb sodium might be impaired and/or that some stimulus to excrete sodium is present. Urine sodium can vary from day to day depending on the degree of water reabsorption. To get an accurate assessment of renal clearance of sodium, both urine and serum sodium can be compared - this can be done with the fractional excretion of sodium (FENa) calculation (1).

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

In a normal individual, urine sodium excretion generally reflects dietary intake - the more one ingests (e.g. added dietary salt, drinking and cooking with softened water, salt poisoning, etc.) the more one excretes. High urine sodium may be associated, for example, with diuretic use or conditions such as Addison's disease (primary adrenal insufficiency).

Strontium High

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

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

Vanadium High

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

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

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

A confirmatory test for excess exposure to V is the Doctor's Data the whole blood vanadium test. EDTA (but not DMPS or DMSA) is an effective chelator of V. Therefore excessive retention (body burden) of V can be assessed by comparing pre- and post-Ca-Na2-EDTA urine V levels.