DOCTOR'S DATA INC.

ORDER: xxx
TEST: xxx
CLIENT REF: xxx
PATIENT: xxx
ID: xxx
SEX: Male
AGE: xxx DOB: xxx

CLIENT #: xxx
DOCTOR:
Biolab Medical Unit
The Stone House 9 Weymouth St
London, W1W 6DB United Kingdom

Toxic Metals; urine

TOXIC METALS				
		RESULT µg/g Creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	1.8	< 15	
Antimony	(Sb)	0.030	< 0.18	
Arsenic	(As)	4.9	< 40	
Barium	(Ba)	1.9	< 5	
Beryllium	(Be)	<dl	< 0.01	
Bismuth	(Bi)	0.005	< 0.8	
Cadmium	(Cd)	0.06	< 0.6	
Cesium	(Cs)	4.7	< 9	
Gadolinium	(Gd)	<dl	< 0.5	
Lead	(Pb)	0.24	< 1.1	
Mercury	(Hg)	0.73	< 0.8	
Nickel	(Ni)	1.4	< 4	
Palladium	(Pd)	<dl	< 0.3	
Platinum	(Pt)	<dl	< 0.1	
Tellurium	(Te)	<dl	< 0.5	
Thallium	(Tl)	0.25	< 0.4	
Thorium	(Th)	<dl	< 0.015	
Tin	(Sn)	0.11	< 3	
Tungsten	(W)	0.074	< 0.4	
Uranium	(U)	<dl	< 0.03	

URINE CREATININE			
	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	99.8	35 – 240	

SPECIMEN DATA

Comments:
Date Collected:
Date Received:
Date Reported:
Methodology: ICP-MS QQQ, Creatinine by Jaffe Reaction

Collection Period: Random
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.

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Essential Elements; urine

ESSENTIAL ELEMENTS				
		RESULT mEq/g Creat	REFERENCE INTERVAL	PERCENTILE 2.5th 16th 50th 84th 97.5th
Sodium	(Na)	41.1	40 – 200	
Potassium	(K)	9.21	20 – 90	
		RESULT µg/mg Creat		
Phosphorus	(P)	763	150 – 1000	
Calcium	(Ca)	255	20 – 250	
Magnesium	(Mg)	119	20 – 200	
Zinc	(Zn)	0.31	0.09 – 1.3	
Copper	(Cu)	0.0054	0.003 – 0.022	
Sulfur	(S)	858	250 – 900	
Molybdenum	(Mo)	0.0196	0.01 – 0.11	
Boron	(B)	1.1	0.5 – 3.8	
Lithium	(Li)	0.0148	0.008 – 0.18	
Selenium	(Se)	0.028	0.03 – 0.2	
Strontium	(Sr)	0.241	0.035 – 0.26	

		RESULT µg/g Creat	REFERENCE INTERVAL	68th 95th
Cobalt	(Co)	1.1	< 1	
Iron	(Fe)	2	< 50	
Manganese	(Mn)	0.45	< 0.4	
Chromium	(Cr)	0.15	< 1.5	
Vanadium	(V)	0.04	< 0.6	

URINE CREATININE				
		RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine		99.8	35 – 240	

SPECIMEN DATA

Comments:
Date Collected:
Date Received:
Date Reported:
Collection Period: Random
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.

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.

Cobalt High

Urinary cobalt (Co) provides an indication of recent or ongoing exposure to the metallic element, and B-12 vitamers. It should be noted that benign high urine Co levels may be associated with recent high B-12 supplementation. That urinary Co is associated with intact B-12, not free Co ions. All forms of B-12 contain Co safely entrapped in the core of the structure. B-12 vitamers are water soluble and are considered safe even at high doses.

Urinary Co is most commonly used as an indicator of occupational exposure to the metal, because urine is the primary elimination route of Co after respiratory exposure. Exposure to Co has been reported for hard metal workers, gas turbine and space propulsion workers, base metal refinery workers, dental technicians, construction workers and workers in the electronics industry.

Individuals bearing metal-on-metal (M-O-M) prosthetic implants (hip or knee replacements) may excrete higher than normal amounts of Co, albeit typically much lower than that associated with occupational exposure. Co is the most abundant metal released by physical wear from the bearing surfaces, but chromium may also be elevated. For patients with M-O-M prosthetics exhibiting abnormal Co excretion, consider the Implant Profile which assesses the blood levels of the six metals that are most commonly associated with orthopedic devices. Signs and symptoms that may be associated with high exposure to metallic Co include visual and auditory impairment, tinnitus, vertigo, impaired immune and renal function, cardiomyopathy, cognitive dysfunction/dementia, mood disorders, hypothyroidism, peripheral neuropathy and skin rashes.

Chelation may acutely increase urinary excretion of free Co ions, but not Co associated with B-12.

Kettelarij J, et al. Neglected exposure route: cobalt on skin and its associations with urinary cobalt levels Occup Environ Med 2018;75:837–842. doi:10.1136/oemed-2018-105099

Copper Low

Low urinary copper may or may not correspond to subnormal copper levels in body tissues, and other laboratory tests are more indicative of copper status. Such tests include measurement of: whole blood or blood cell copper, hair copper, erythrocyte superoxide dismutase activity, and serum ceruloplasmin.Because the major route of copper excretion is via bile and feces, urinary levels may fluctuate without reflecting or influencing body stores.

Lower than normal excretion of copper (and other elements) can occur in renal insufficiency; in which case blood levels may be normal or elevated. Inadequate levels of molybdenum or zinc allow increased retention of copper, and transient hypocuprinuria may occur during periods when copper stores are accumulating.

Low urinary copper may also correspond to copper deficiency of nutritional or gastrointestinal origins.The richest dietary sources of copper are: nuts, shellfish, liver, raisins and legumes. Dairy products generally are low in copper content. Gastric hypochlorhydria, sprue, and pancreatic dysfunction may inhibit copper uptake.

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 Low

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

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

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

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

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

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

Phosphorus High

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

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

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

Potassium Low

The level of potassium (K) is lower than expected in this sample. K is an electrolyte and a potentiator of enzyme functions in cells. K can be low in the body as the result of gastrointestinal or renal dysfunction, or as a side effect of some diuretics. In adrenocortical hyperactivity, blood levels of K are depressed, while urinary K is increased. Diabetic acidosis and other medical conditions may result in severe K loss. Symptoms of true K deficiency include: muscle weakness, fatigue, and tachycardia. An electrocardiogram may show abnormalities when K is low in serum/plasma or whole blood.

Appropriate tests to confirm low K in body tissues may include measurements of packed red blood cell K; serum or whole blood K and sodium/K ratio.

Selenium Low

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.

Low urinary selenium may be a consequence of: junk food diet or highly-processed food diet, gastrointestinal dysfunctions, renal insufficiency (in which case other elements will be subnormal in urine but possibly elevated in blood), and long-term parenteral nutrition or special diets that are low in selenium.

Selenium is a necessary element for proper activity of two enzymes in human metabolism: glutathione peroxidase (GPx) and iodothyronine deiodinase (ITD). Selenium deficiency may cause weakness or rate limitation for one or both of these enzymes. GPx oxidizes glutathione while reducing oxidized lipids. Weak GPx activity may allow excessive inflammation to occur. ITD deiodinates thyroxine prohormone and catalyzes T4 _ T3. Selenium deficiency may be a cause of insufficient T3 and thyroid dysfunction (Berry J.M. Nature 349, 1991 pp.438-40).

Symptoms consistent with selenium deficiency include: myalgia, increased inflammatory responses, hypothyroidism with low T3. Cardiomyopathy and Keshan disease can occur in cases of severe, chronic Se deficiency. Subnormal selenium may accentuate the effects of cadmium, mercury or arsenic overload. Confirmatory tests for selenium status include packed red bloodcell elements, and hair elemental analysis (provided that antidandruff shampoos have not been used.

Sodium Low

The concentration of sodium in this urine sample is lower than expected and is more than two standard deviations below the mean. Low urine sodium levels are uncommon but may be seen, for example, with severe vomiting and/or diarrhea. Further, a low urine sodium concentration implies that the kidney's capacity to reabsorb sodium must be intact and that some stimulus to conserve 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 less one ingests (e.g. low salt diet, etc.) the less one excretes. In dehydration (e.g. vomiting, diarrhea, etc.) sodium may be retained (less sodium output in urine) in efforts to retain water. Decreased urine sodium concentration also may be associated with disease states such as Conn's syndrome (primary hyperaldosteronism due to an aldosterone-producing adenoma), congestive heart failure, liver disease and/or nephrotic syndrome. Low urine sodium has been associated with greater risk of myocardial infarction in males with high blood pressure (2).

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.