



PATIENT: **Sample Report**

TEST REF: **TST-##-####**

TEST NUMBER: #####  
 PATIENT NUMBER: #####  
 GENDER: Female  
 AGE: 53  
 DATE OF BIRTH: dd-mm-yyyy

COLLECTED: dd/mm/yyyy  
 RECEIVED: dd/mm/yyyy  
 TESTED: dd/mm/yyyy

PRACTITIONER: **Nordic Laboratories**  
 ADDRESS:

**TEST NAME: Urine Toxic and Essential Elements (timed or 24 hour) Post**

*Toxic Metals; Urine*

TOXIC METALS					
	RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE	OUTSIDE REFERENCE	
Aluminum (Al)	6.9	< 35			
Antimony (Sb)	0.4	< 0.2			
Arsenic (As)	46	< 80			
Barium (Ba)	2.2	< 7			
Beryllium (Be)	< dl	< 1			
Bismuth (Bi)	< dl	< 4			
Cadmium (Cd)	0.5	< 1			
Cesium (Cs)	13	< 10			
Gadolinium (Gd)	1.9	< 0.8			
Lead (Pb)	4.3	< 2			
Mercury (Hg)	11	< 4			
Nickel (Ni)	5	< 10			
Palladium (Pd)	< dl	< 0.3			
Platinum (Pt)	< dl	< 0.1			
Tellurium (Te)	< dl	< 0.5			
Thallium (Tl)	1.3	< 0.5			
Thorium (Th)	< dl	< 0.03			
Tin (Sn)	0.9	< 5			
Tungsten (W)	< dl	< 0.4			
Uranium (U)	< dl	< 0.04			

URINE CREATININE							
	RESULT mg/dL	REFERENCE INTERVAL	-2SD	-1SD	MEAN	+1SD	+2SD
Creatinine	21.2	30- 225					

SPECIMEN DATA			
Comments:			
Date Collected: mm/dd/yyyy	pH upon receipt: <b>Acceptable</b>	Collection Period: <b>timed: 6 hours</b>	
Date Received: mm/dd/yyyy	<dl: less than detection limit	Volume:	
Date Completed: mm/dd/yyyy	Provoking Agent:	Provocation: <b>POST PROVOCATIVE</b>	
Method: <b>ICP-MS</b>	<b>Creatinine by Jaffe Method</b>		
Results are creatinine corrected to account for urine dilution variations. <b>Reference intervals and corresponding graphs are representative of a healthy population under non-provoked conditions.</b> Chelation (provocation) agents can increase urinary excretion of metals/elements.			
V13			

TEST NUMBER: #####  
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 AGE: 53  
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**Essential Elements; Urine**

ESSENTIAL AND OTHER ELEMENTS								
		RESULT/UNIT per creatinine	REFERENCE INTERVAL	PERCENTILE				
				2.5 <sup>th</sup>	16 <sup>th</sup>	50 <sup>th</sup>	84 <sup>th</sup>	97.5 <sup>th</sup>
Sodium	(Na)	110 mEq/g	45– 200			•		
Potassium	(K)	150 mEq/g	20– 110					
Phosphorus	(P)	230 µg/mg	180– 1100					
Calcium	(Ca)	220 µg/mg	30– 350					
Magnesium	(Mg)	200 µg/mg	25– 230					
Zinc	(Zn)	2.2 µg/mg	0.1– 1.5					
Copper	(Cu)	0.61 µg/mg	0.007– 0.06					
Sulfur	(S)	700 µg/mg	275– 1200					
Manganese	(Mn)	0.005 µg/mg	0.0004– 0.007					
Molybdenum	(Mo)	0.028 µg/mg	0.013– 0.15					
Boron	(B)	4.8 µg/mg	0.5– 4					
Chromium	(Cr)	< dl µg/mg	0.0003–0.0025					
Lithium	(Li)	0.56 µg/mg	0.009– 0.2					
Selenium	(Se)	0.041 µg/mg	0.03– 0.25					
Strontium	(Sr)	0.23 µg/mg	0.045– 0.5					
Vanadium	(V)	< dl µg/mg	0.0001–0.0017					
				68 <sup>th</sup>	95 <sup>th</sup>			
Cobalt	(Co)	< dl µg/mg	< 0.008					
Iron	(Fe)	0.35 µg/mg	< 1					

  

URINE CREATININE							
	RESULT mg/dL	REFERENCE INTERVAL	-2SD	-1SD	MEAN	+1SD	+2SD
Creatinine	21.2	30– 225					

**SPECIMEN DATA**
**Comments:**

 Date Collected: mm/dd/yyyy      pH Upon Receipt: **Acceptable**      Collection Period: **timed: 6 hours**  
 Date Received: mm/dd/yyyy      <dl: less than detection limit      Volume:  
 Date Completed: mm/dd/yyyy      Provoking Agent:      Provocation: **POST PROVOCATIVE**  
 Method: ISE; Na, K Spectrophotometry; P ICP-MS; B, Ca, Cr, Co, Cu, Fe, Mg, Mn, Mo, Se, Sr, S, V, Zn Creatinine by Jaffe method

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

V13

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**TEST NAME: Urine Toxic and Essential Elements (timed or 24 hour) Post**

**INTRODUCTION**

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

**1) 24 Hour Collections**

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

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

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

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

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

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

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

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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.

**ANTIMONY HIGH**

This individual's urine antimony (Sb) is higher than expected, but potential associated symptoms and toxic effects may not be present. This is because antimony has two valences: Sb+3 and Sb+5. Sb+3 is inherently the more toxic but is mostly excreted in feces. Sb+5, less toxic, binds less well to body tissues and is excreted mostly in urine. The current analysis does not differentiate the two forms of Sb.

Antimony can be assimilated by inhalation of Sb salt or oxide dust, ingested with (contaminated) foods or fluids, or absorbed transdermally. Inhalation may occur in industrial areas that involve smelting or alloying is done (usually with copper, silver, lead, tin). Sb is present in tobacco at about 0.01% by weight; about 20% of this is typically inhaled by cigarette smoking (Carson et al., Toxicology and Biological Monitoring of Metals in Humans, Lewis Pub. p. 21, 1987). Antimony compounds are used for fireproofing textiles and plastics, and this element may be found in battery electrodes, ceramics and pigments. Antimony can be absorbed with the handling of gun powder or the frequent use of firearms. Recent studies indicate high levels of antimony in sheepskin bedding produced in New Zealand. Antimony contamination of soft plastic-bottled water is time and temperature dependent.

Symptoms of mild Sb exposure/retention may be insidious and multiple including: fatigue, muscle weakness, myopathy, and metallic taste. Chlorides and oxides of both valences of Sb can be mutagenic and may affect leukocyte function. Sb can bond to sulfhydryl (-SH) sites on enzymes and may interfere with cellular metabolism. Acute symptoms that may be associated with excessive Sb exposure/retention include: respiratory tissue irritation and pneumoconiosis with (chronic) inhalation of Sb dusts, RBC hemolysis with inhalation of stibine (SbH3) vapor, and gastrointestinal distress if orally ingested. Skin exposure can produce "antimony spots" or rashes which resemble chicken pox. Certain molds can produce the highly neurotoxic stibine gas from Sb; stibine inhibits acetylcholinesterase activity.

A hair element analysis may be used to further assess Sb exposure. Antimony may

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be elevated in urine following administration of DMPS or DMSA if exposures to Sb have resulted in net retention; such levels may or may not be associated with overt adverse health effects.

**BIBLIOGRAPHY FOR ANTIMONY**

1. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, Chelsea MI, pp 21-26, 1987.
2. Tsalev D.L. and Z.K. Zaprianov. Atomic Absorption Spectrometry in Occupational and Environmental Health Practice. CRC Press, Boca Raton FL, pp 85-87, 1983.
3. Scriver C.A. et al The Metabolic Basis of Inherited Disease, 6th ed. McGraw-Hill, New York NY, pp 2349-50 on PFK deficiency. 1989.
4. Westerhof P. et al. Antimony leaching from polyethelene trephthalate (PET) plastic used for bottled drinking water. Water Res(2008)42:51-56.
5. Andra, SS et al. Co-leaching of brominated comounds and antimony from bottled

**Cesium High**

This individuals urine Cesium (Cs) level is higher than expected, reflecting exosure to Cs but symptoms may not be evident. Cesium is a naturally-occurring element found in rocks, soil and dust at low concentrations. It is present in the environment only in the stable form of Cs133; the radioactive isotopes 134Cs and 137Cs are not measured or reported by Doctor's Data. Natural deposits of Cs ores occur in Main, South Dakota and Manitoba (Bernic Lake), Canada. Cesium may bio-accumulate in aquatic food chains; higher levels of cesium have been found in Pacific deep-sea fish and local shellfish since the 2011 Fukoshima reactor accident. Cesium may be used in high-density drilling fluids (oil and gas industry) and may contaminate local water and vegetation; Cs has been found in cow's milk. Cesium may occur naturally in mineral waters; one study analyzed the Cs concentration in 163 mineral and thermal waters and found the level ranged from 4.5 to 148 µg per liter.

Cesium can be absorbed after oral ingestion, upon breathing contaminated air and through contact with the skin. Cesium is readily absorbed across the brush border of the intestines in a manner similar to potassium and most is eventually excreted through the urine and feces. The biological half-life of Cs in humans ranges from 15 days in infants to 100-150 days in adults.

The cesium-137 isotope is used in cancer treatments, for ventricular function and pulmonary imaging studies, industrial radiology, and for food and instrument sterilization; Cs137 agents may contain small amounts of Cs133. Non-radioactive cesium chloride may be advertised on the internet as "high pH therapy." Currently there is no support in the scientific literature for that purpose as advertised. Radioactive Cs isotopes may contaminate soil at nuclear waste sites. Cesium may be used in industry for the production of photoelectric cells, vacuum tubes, spectrographic instruments, scintillation counters, DNA biochemistry, in various optical or detecting devices.

Target organs of potential toxic effects of Cs are the liver, intestine, heart, and kidneys. Physiological effects of excessive Cs include ventricular arrhythmias and displacement of potassium from muscle cells and erythrocytes. Cesium can have significant effects on both the central and peripheral nervous systems. Cesium may cause epileptic seizures because it can share the same receptor as the excitatory bioamine glycine. Cesium can interfere with active ion transport by blocking potassium channels and also can interfere with lipid metabolism. Excessive Cs may modify plasma membrane integrity, alter cytoplasmic components and cause cytogetic damage.

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It is unlikely that children or adults would be exposed to enough Cs133 to experience any health effects that could be related to the stable Cs itself. Animals given very large doses of Cs compounds have shown changes in behavior, such as increased activity or decreased activity, but it is unlikely that a human would be exposed to enough stable Cs to cause similar effects.

The isotope Cs137 is used in radiation therapy for certain types of cancer. Other medical uses of Cs are monitoring left ventricular function with Cs137 iodide probes and monitoring pulmonary endothelial permeability with Cs137 iodide crystal mini-detectors. Again, it is emphasized that Cs measured at Doctor's Datas is Cs133, not Cs137. Environmental contamination by Cs137 as a result of radioactive fallout could be a concern. Exposure to Cs may be assessed by hair elemental analysis.

Commonly used chelating agents are not effective binders of Cs.

**Resources:**

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

Bermejo-Barrera P, Beceiro-Gonzalez E, Bermejo-Barrera A, Martinez F (1989) Determination of cesium in mineral and thermal waters by electrothermal atomic absorption spectrophotometry.  
 Microchemical Journal 1989 vol: 40 (1) pp: 103-108

Davis D, Murphy E, London R (1988) Uptake of cesium ions by human erythrocytes and perfused rat heart: a cesium-133 NMR study. Biochemistry 1988 vol: 27 (10) pp: 3547-3551

Ikenoue T, Takata H, Kusakabe M, Kudo N, Hasegawa K, et. al. (2017) Temporal variation of cesium isotope concentrations and atom ratios in zooplankton in the Pacific off the east coast of Japan.  
 Scientific Reports 2017 vol: 7 pp: 39874

Relman A (1956) The physiological behavior of rubidium and cesium in relation to that of potassium.  
 The Yale Journal of Biology And Medicine 1956 vol: 29 (3) pp: 248-62

Samadani U, Marcotte P (2004) Zero Efficacy With Cesium Chloride Self-Treatment for Brain Cancer.  
 Mayo Clinic Proceedings 2004 vol: 79 (12) pp: 1588

United States Geological Service (2006) Cesium.  
<https://minerals.usgs.gov/minerals/pubs/commodity/cesium/cesiummcs06.pdf> Accessed 22 February 2017

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

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

**Gadolinium High**

This individual's urine level of Gadolinium (Gd) is higher than expected. Gadolinium is one of the most abundant "rare-earth" elements but is never found as a free element in nature. Gadolinium has no known biological role in humans.

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Toxicity is rarely associated with Gd due to its poor gastrointestinal absorption (it is suspected that very little Gd is absorbed from the gastrointestinal tract (<0.05%). If exposure to high enough doses and/or if absorption does occur, symptoms of acute toxicity may develop, including abdominal cramps, diarrhea, lethargy, muscular spasms, and even eventual death due to respiratory collapse. Gadolinium salts can cause irritation of the skin and eyes and are suspected to be possible carcinogens. As reported by Perazella (2009) Gadolinium-based contrast (GBC) agents have been linked on occasion with a rare systemic fibrosing condition called nephrogenic systemic fibrosis (NSF) and their use in patients with even mild kidney disease should be avoided (parenteral administration).

Gd is often used in alloys (e.g. chromium, iron). Other technical uses include the phosphors of color television tubes and in making magnets and electronic components such as recording heads for video recorders and in the manufacture of compact disks and computer memory. In medicine Gd, chelated with diethylenetriaminepentaacetic acid (DTPA), is used as a non-radioactive contrasting agent in magnetic resonance imaging and has a half-life in blood of about 90 minutes. However, residual Gd is retained in tissues for quite some time. It is also used in control rods for nuclear reactors and power plants, in making garnets for microwave applications.

EDTA effectively chelates Gd therefore urinary Gd might be higher than average post-Ca-EDTA provocation, particularly in patients who have had Gd-enhanced MRIs.

**References:**

<http://www.lenntech.com/Periodic-chart-elements/Gd-en.htm>

Perazella M. Current Status of Gadolinium Toxicity in Patients with Kidney Disease. Clin J Am Soc Nephrol 4: 461-69, 2009.

**LEAD HIGH**

This individual's urine lead (Pb) is higher than expected which means that Pb exposure is higher than that of the general population. A percentage of assimilated Pb is excreted in urine. Therefore the urine Pb level reflects recent or ongoing exposure to Pb and the degree of excretion or endogenous detoxification processes.

Sources of Pb include: old lead-based paints, batteries, industrial smelting and alloying, some types of solders, Ayurvedic herbs, some toys and products from China and Mexico, glazes on (foreign) ceramics, leaded (anti-knock compound) fuels, bullets and fishing sinkers, artist paints with Pb pigments, and leaded joints in municipal water systems. Most Pb contamination occurs via oral ingestion of contaminated food or water or by children mouthing or eating Pb-containing substances. The degree of absorption of oral Pb depends upon stomach contents (empty stomach increases uptake) and upon the essential element intake and Pb status. Deficiency of zinc, calcium or iron increases Pb uptake. Transdermal exposure is significant for Pb-acetate (hair blackening products). Inhalation has decreased significantly with almost universal use of non-leaded automobile fuel.

Lead accumulates in extensively in bone and can inhibit formation of heme and hemoglobin in erythroid precursor cells. Bone Pb is released to soft tissues with bone remodeling that can be accelerated with growth, menopausal hormonal changes, osteoporosis, or skeletal injury. Low

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levels of Pb may cause impaired vitamin D metabolism, decreased nerve conduction, and developmental problems for children including: decreased IQ, hearing impairment, delayed growth, behavior disorders, and decreased glomerular function. Transplacental transfer of Pb to the fetus can occur at very low Pb concentrations in the body. At relatively low levels, Pb can participate in synergistic toxicity with other toxic elements (e.g. cadmium, mercury).

Excessive Pb exposure can be assessed by comparing urine Pb levels before and after provocation with Ca-EDTA (iv) or oral DMSA. Urine Pb is higher post-provocation to some extent in almost everyone. Whole blood analysis reflects only recent ongoing exposure and does not correlate well with total body retention of Pb. However, elevated blood Pb is the standard of care for diagnosis of Pb poisoning (toxicity).

**BIBLIOGRAPHY FOR LEAD**

1. ATSDR Toxicological Profile for Lead (2007 update) [www.atsdr.cdc.gov/toxprofile](http://www.atsdr.cdc.gov/toxprofile)
2. Centers for Disease Control and Prevention. Third National Report on Human Exposure to Environmental Chemicals. Atlanta, GA: CDC; 2005.
3. Lead Tech '92, "Proceedings and Papers from the Lead Tech '92: Solutions for a Nation at Risk" Conference, Sept 30-Oct 2, 1992. Bethesda, MD, IAQ Publications, 4520 East-West Highway, Ste 610, Bethesda, MD, 20814.
4. "Preventing Lead Poisoning in Young Children", US Centers for Disease Control, Atlanta, GA, Oct. 1991 Statement, US Dept. of Health and Human Services.
5. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, Inc., Chelsea, MI, p. 128-135, 1986.
6. Tsalev D.L. et al. Atomic Absorption Spectrometry in Occupational and Environmental Health Practice Vol 1, CRC Press, Boca Raton, FL 1983.
7. Piomelli S. et al. "Management of Childhood Lead Poisoning", J. Pediatr 105 (1990) p. 523-32.
8. 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.
9. Mayo Clinic. Mayo Medical Laboratories. <http://www.mayomedicallaboratories.com/test-catalog/clinical+and+Interpretive/60246> [Accessed 10/25/2011]
10. Saper RB et al. "Lead, mercury and arsenic in U.S. and Indian manufactured ayurvedic medicines sold via the internet." JAMA (2008) 300(8): 915-23.

**MERCURY HIGH**

This individual's urine mercury (Hg) far exceeds the expected level for the general population under non-provoked conditions. Presentation of symptoms associated with excessive Hg exposure can depend on many factors: the chemical form of Hg its accumulation in specific tissues, presence of other toxicants, presence of disease that depletes glutathione or inactivates lymphocytes or is immunosuppressive, and the concentration of protective nutrients, (e.g. zinc, selenium).

Early signs of excessive Hg exposure include: decreased senses of touch, hearing, vision and taste, metallic taste in mouth, fatigue or lack of physical endurance, and increased salivation. Symptoms may progress with moderate or chronic exposure to include: anorexia, numbness and paresthesias, headaches, hypertension, irritability and excitability and immune suppression/dysregulation. Advanced disease processes from excessive Hg assimilation include: tremors and incoordination, anemia, psychoses, manic behaviors,

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possibly autoimmune disorders and renal dysfunction or failure.

Mercury is commonly used in: dental amalgams (50% by weight), explosive detonators; in pure liquid form for thermometers, barometers, and laboratory equipment; batteries and electrodes, some medications and ayurvedic herbs, and Hg in fungicides and pesticides. The use of Hg in fungicides/pesticides has declined due to environmental concerns, but mercury residues persist from past use.

Methylmercury, the most common, organic form, occurs by methylation of inorganic in aquatic biota or sediments (both freshwater and ocean sediments). Methylmercury accumulates in aquatic animals and fish and is concentrated up the food chain reaching high concentrations in large fish and predatory birds. Except for fish, the human intake of dietary mercury is negligible unless the food is contaminated with one of the previously listed forms/sources. Daily ingestion of fish can result in the assimilation 1 to 10 micrograms of mercury/day.

Depending upon the extent of cumulative Hg exposure, elevated urine mercury may occur after administration of DMPS, DMSA, or D-penicillamine. Blood and especially red blood cell elemental analyses are only useful for diagnosing very recent or ongoing organic (methyl) mercury exposure.

**BIBLIOGRAPHY FOR MERCURY**

- Centers for Disease Control and Prevention. Third National Report on Human Exposure to Environmental Chemicals. Atlanta, GA: CDC; 2005.  
[http://www.cdc.gov/exposure\\_report/report.htm](http://www.cdc.gov/exposure_report/report.htm) [Accessed 2/01/2009]
- Suzuki T. et al eds, *Advances in Mercury Toxicology*, Plenum Press, New York, 1991.
- World Health Organization: "Methylmercury" *Environ. Health Criteria* 101 (1990); "Inorganic Mercury" *Environ. Health Criteria* 118 (1991) WHO, Geneva, Switzerland.
- Tsalev D.L. and Z.K. Zaprianov, *Atomic Absorption Spectrometry in Occupational and Environmental Health Practice*, CRC Press, Boca Raton FL, pp 158-69, 1983.
- Birke G. et al "Studies on Humans Exposed to Methyl Mercury Through Fish Consumption", *Arch Environ Health* 25, 1972 pp 77-91.
- Pelletier L. "Autoreactive T Cells in Mercury-Induced Autoimmunity", *J. Immunology*, 140 no.3 (1988) pp 750-54.
- Werbach M.R. *Nutritional Influences on Illness*, 2nd ed, Third Line Press, Tarzana CA, pp 249, 647, 679, 1993.
- Saper Rb et al. "Lead, mercury and arsenic in U.S. and Indian manufactured ayurvedic medicines sold via the internet." *JAMA* (2008)300(8): 915-23.

**THALLIUM HIGH**

This individual's urine thallium (TI) is markedly higher than expected and is indicative of high level exposure to TI. Thallium can be an insidious toxic element, sometimes with delayed and diverse manifestations. Multiple organs, tissues and nervous system involvement may occur. Specific symptoms can depend upon: chemical form of the thallium and mode of assimilation, severity and duration of exposure, and organ levels of metabolites and nutrients that effect the action of TI in the body.

Thallium can be assimilated transdermally, by inhalation, or by oral ingestion. Both valence states can have harmful effects: TI+1 may displace potassium from binding sites and influence

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GENDER: Female	TESTED: dd/mm/yyyy	
AGE: 53		
DATE OF BIRTH: dd-mm-yyyy		

**TEST NAME: Urine Toxic and Essential Elements (timed or 24 hour) Post**

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

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

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

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

**BIBLIOGRAPHY FOR THALLIUM**

- Centers for Disease Control and Prevention. Third National Report on Human Exposure to Environmental Chemicals. Atlanta, GA: CDC; 2005. <http://www.cdc.gov/exposurereport.htm> [Accessed 2/01/2009]
- Graef J.W. "Thallium" in Harrison's Principles of Internal Medicine, 13th ed., Isselbacher et al eds., McGraw Hill, pp 2465, 1994.
- Tsalev D.L. and Zaprianov Z.K. Atomic Absorption Spectrometry in Occupational and Environmental Health Practice CRC Press, Boca Raton FL, pp 196-199, 1983.
- Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans Lewis Publishers, Chelsea, MI, pp 243-254, 1987.

**Potassium High**

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

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

Appropriate tests to confirm excess K in body tissues may include measurements of packed red blood cell K;

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PATIENT NUMBER: #####	RECEIVED: dd/mm/yyyy	
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serum or whole blood K and sodium/K ratio. An assessment of adrenocortical function may be indicated for symptomatic patients with a confirmed elevation in serum K.

**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, aminoglycosides, 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.

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**TESTED:** dd/mm/yyyy

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**ADDRESS:**
**TEST NAME: Urine Toxic and Essential Elements (timed or 24 hour) Post**
**BIBLIOGRAPHY FOR MAGNESIUM**

1. Knochel J.P. "Disorders of Magnesium Metabolism", Chapt 360 in Harrison's Principles of Internal Medicine, 13th ed., McGraw-Hill pp. 2187-90, 1994.
2. Shils M. "Magnesium", Chapt. 8 in Modern Nutrition in Health and Disease, 8th ed. vol.1, Lea & Febiger, Philadelphia, PA, pp.164-84, 1994.
3. Harper H.A. et al. Review of Physiological Chemistry, 17th ed., Lange Medical Publications, Los Altos, CA, pp. 578-79, 1979.
4. Jones J.E. et al. "Magnesium Requirements in Adults" Med J. Clin. Nutr. 20 pp. 632-35, 1967.
- 5(a) Halpern M.J. and J. Durlach eds., Magnesium Deficiency Karger (Basel and New York), esp. pp. 146-180, 1985.
- 5(b) See also Magnesium and Trace Elements, official journal of the Am. Soc. for Magnesium Research, B.M. Altura (Brooklyn NY), Ed. in- Chief, S. Karger A.G. Postfach CH-4009 Basel, Switzerland.
6. Galland L. "Magnesium and Inflammatory Bowel Disease" Magnesium 7 no. 2, pp. 78-83, 1988.
7. Rea W.J. "Magnesium Deficiency in Patients with Chemical Sensitivity" Clinical Ecology 4 no. 1, pp 17-20, 1986.

**ZINC HIGH**

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

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

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

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

Excessive amounts of zinc in body tissues may displace copper and/or iron from tissue binding sites and may provoke anemia. Symptoms consistent with chronic zinc toxicity include: lethargy, difficulty writing and with fine motor skills, light-headedness, and renal failure. Immediate symptoms (within 12 hours) of acute zinc excess via ingestion include: nausea, vomiting, diarrhea, exhaustion, headache, dizziness, and myalgia. Other laboratory findings consistent with zinc

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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**

1. Lantzsch H.J. and H. Schenkel "Effect of Specific Nutrient Toxicities in Animals in Animals and Man: Zinc in Handbook Series in Nutrition and Food, Sect.E, vol 1, CRC Press, Boca Raton FL, pp 291-307, 1978.
2. King J.C. and C.I. Keen "Zinc" Chapt. 10 in Modern Nutrition in Health and Disease 8th ed. Shils, Olson and Shike eds, Lea & Febiger, Philadelphia PA, pp 214-230, 1994
3. Tsalev D.L. and Z.K. Zaprianov Atomic Absorption Spectrometry in Occupational and Environmental Health Practice vol 1, CRC Press, Boca Raton FL, pp 209-14, 1983.
4. Aggett P.J. "An Overview of the Physiology and Metabolic Role of Zinc" in Zinc in Human Medicine, Inst. Child Health, London, TIL Publications, Toronto, Ont. pp 9-38, 1984.
5. Fell G.S. "Diagnosis of Zinc Deficiency" in Zinc in Human Medicine ibid. pp 39-50.
6. Petering, H.G. "Some Observations on the Interaction of Zinc, Copper and iron Metabolism in Lead and Cadmium Toxicity." Environ. Health Perspect., pp. 141-145, 1978.
7. Lauwerys, R. et al. "The Influence of Orally-Administered Vitamin C or Zinc on the Absorption of and Biological Response to Lead." J. Occup. Health, 25: pp. 668-678, 1983.

**COPPER HIGH**

Significantly elevated copper in urine can be secondary to provocative challenge with sulfhydryl (-SH) bearing agents such as D-penicillamine ("Cuprimine"), DMSA, or DMPS. Large, multi-gram doses of vitamin C (ascorbic acid), administered orally or intravenously, may slightly or moderately increase excretion of copper.

Increased urinary copper can be an artifact of nutritional supplementation with copper or come from drinking water that is high in copper content. Acidic water carried in copper pipes can dissolve some copper which increases the copper intake if used for drinking or cooking. Molybdenum supplementation at high levels or if inappropriate may cause increased copper excretion; molybdenum and copper are mutually antagonistic in terms of body retention.

Bacterial or other infections may cause hypercupremia with attendant or delayed hypercuprinuria. This is transient and follows the inflammatory stage of the disease. Published studies such as Vivoli, Sci Total Environ, 66 p. 55-64, 1987 have correlated increased urinary copper with increased blood pressures in hypertensives. Biliary obstruction or insufficiency can decrease normal excretion of copper via the bile while increasing blood and urinary levels. Proteinuria also may feature increased copper levels.

Hyperaminoacidurias that include histidinuria can result in urinary copper wasting because histidine is a powerful chelator of copper. Hyperaminoacidurias that include histidine can be of many origins including: genetic factors, chemical or elemental toxicities, infectious agents, hyperthyroidism, sugar intolerances, nephrotic syndromes, etc.

In Wilson's disease, urinary copper is generally increased (above 100 micrograms/24 hours) without provocation or chelation. Use of D-penicillamine or DMPS as a provocative diagnostic procedure can yield a 5 - 10X increase in urinary copper levels in normal individuals. In contrast, Wilson's disease patients may then excrete 50-100 times the normal levels or 1000 to 2000 mcg/24 hr. (Walshe, J. Rheumatology (supp/7) 8 p.3-8, 1981).

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Urine analysis (unprovoked) is not an adequate procedure to assess copper stores or copper metabolism. Blood levels, erythrocyte copper content, erythrocyte superoxide dismutase activity, and serum ceruloplasmin are other more indicative measurements for copper status.

**BIBLIOGRAPHY FOR COPPER**

1. Braunwald et al., eds. Harrison's Principles of Internal Medicine, 11th ed., McGraw Hill, Chapter 311, 1987.
2. Johnson M.A. and B.E. Kays, "Copper: Its Role in Human Nutrition", Nutrition Today, Jan/Feb 1990, pp 6-14.
3. Harper et al. Review of Physiological Chemistry, Lange Medical Publications, 17th ed., pp. 588-89, 1979.
4. J. M. Walshe "The Discovery of the Therapeutic use of D-Penicillamine" J. Rheumatology (Supplement 7) 8, pp. 3-8, 1981.
5. Werbach M.R. Nutritional Influences on Illness 2nd ed., Third Line Press, Tarzana, CA, 1993. Multiple source references on copper physiology and pathology.

**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.

**BIBLIOGRAPHY FOR BORON, HIGH**

- Nielsen, F.H., Hunt, C.D., Mullen, L.M., Hunt, J.R. Effect of dietary boron mineral, estrogen, and testosterone metabolism in postmenopausal women. FASEB 1:394-397, 1987.
- Shils, M.E., Olson, J.A., Shike, M.: Modern nutrition in health and disease. Philadelphia, Lea and Febiger, 1994.

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**CHROMIUM LOW**

The chromium level in this urine sample is low. 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.

Significance of Low Chromium: Clinical findings consistent with Cr deficiency are those of GTF insufficiency including diabetes, hyperglycemia, and possibly transient hyper/hypoglycemia. Excessive LDL cholesterol also may be consistent with Cr deficiency. Some investigators have linked Cr deficiency to ischemic heart disease and atherosclerosis.

Other Useful Analyses: Urine Toxic Metals and Essential Elements provocative testing with EDTA can be used to assess Cr stores.

**BIBLIOGRAPHY FOR CHROMIUM LOW**

1. Anderson R.A. Essentiality of Chromium in Humans” Sci Total Environ. 86, 1989, pp 75-81.
2. Anderson R.A. et al, Am. J. Clin. Nutr. 41, 1985 pp 571-77
3. Mertz W., ”Chromium Occurrence and Function in Biological Systems”. Physiological Review 49(2), 1969, pp 163-239.
4. Mertz, W. Clinical, Biochemical and Nutritional Aspects of Trace Elements, Alan R. Liss, Inc., New York, NY, 1982, pp 315-23.
5. Rennert O.M. and W-Y Chan Metabolism of Trace Metals in Man vol II, CRC Press, Boca Raton FL, pp 122-24.
6. Tsalev D.L. and Z.K. Zaprianov, Atomic Absorption Spectrometry in Occupational and Environmental Health Practice, CRC Press, Boca Raton, FL, 1983, pp 113-17.
7. Wallach S.J. Am. Coll. Nutr 4, 1985, pp 107-120

**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



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markedly elevated urine Li if ingestion was recent or chronic.

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

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

**Vanadium Low**

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

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

There are no known symptoms of V deficiency. Although trace amounts of V may have essential metabolic functions, over-zealous supplementation of V is not warranted. There is no RDA for V but, if supplementation is warranted, a common daily dose of tetravalent vanadyl sulfate is 20 to 30 mcg per day.

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

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