

**Toxic Metals; Urine**

TOXIC METALS						
		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE	OUTSIDE REFERENCE	
Aluminum	(Al)	2	< 25			
Antimony	(Sb)	< dl	< 0.2			
Arsenic	(As)	33	< 75			
Barium	(Ba)	0.5	< 7			
Beryllium	(Be)	< dl	< 1			
Bismuth	(Bi)	< dl	< 2			
Cadmium	(Cd)	< dl	< 0.8			
Cesium	(Cs)	6.4	< 9			
Gadolinium	(Gd)	< dl	< 0.5			
Lead	(Pb)	4.8	< 2			
Mercury	(Hg)	9.7	< 3			
Nickel	(Ni)	3.4	< 8			
Palladium	(Pd)	< dl	< 0.3			
Platinum	(Pt)	< dl	< 0.1			
Tellurium	(Te)	< dl	< 0.5			
Thallium	(Tl)	0.3	< 0.5			
Thorium	(Th)	< dl	< 0.03			
Tin	(Sn)	1.4	< 4			
Tungsten	(W)	< dl	< 0.4			
Uranium	(U)	< dl	< 0.03			

URINE CREATININE						
	RESULT mg/dL	REFERENCE INTERVAL	-2SD	-1SD	MEAN	+1SD +2SD
Creatinine	65.3	35- 240				

SPECIMEN DATA			
Comments:			
Date Collected: 09/15/2020	pH upon receipt: <b>Acceptable</b>	Collection Period: <b>timed: 6 hours</b>	
Date Received: 09/15/2020	<dl: less than detection limit	Volume:	
Date Reported: 09/15/2020	Provoking Agent:	Provocation:	
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			

## 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

---

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.

#### 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 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 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.  
[http://www.cdc.gov/exposure\\_report/report.htm](http://www.cdc.gov/exposure_report/report.htm) [Accessed 02/01/2009]
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, 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 extend 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

1. 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]
2. Suzuki T. et al eds, *Advances in Mercury Toxicology*, Plenum Press, New York, 1991.
3. World Health Organization: "Methylmercury" *Environ. Health Criteria* 101 (1990); "Inorganic Mercury" *Environ. Health Criteria* 118 (1991) WHO, Geneva, Switzerland.
4. 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.
5. Birke G. et al "Studies on Humans Exposed to Methyl Mercury Through Fish Consumption", *Arch Environ Health* 25, 1972 pp 77-91.
6. Pelletier L. "Autoreactive T Cells in Mercury-Induced Autoimmunity", *J. Immunology*, 140 no.3 (1988) pp 750-54.
7. Werbach M.R. *Nutritional Influences on Illness*, 2nd ed, Third Line Press, Tarzana CA, pp 249, 647, 679, 1993.
8. 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.