Nordic Laboratories

Test nr.
Patient Name
Patient nr.
Age
Sex
Doctor Name
Practitioner Address

Toxic Metals; Urine

TOXIC METALS		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	< dl	< 35	
Antimony	(Sb)	0.1	< 0.4	
Arsenic	(As)	6	< 117	
Barium	(Ba)	3.9	< 7	
Beryllium	(Be)	< dl	< 1	
Bismuth	(Bi)	< dl	< 15	
Cadmium	(Cd)	0.4	< 1	
Cesium	(Cs)	4.3	< 10	
Gadolinium	(Gd)	< dl	< 0.4	
Lead	(Pb)	34	< 2	
Mercury	(Hg)	9.1	< 4	
Nickel	(Ni)	1.7	< 12	
Palladium	(Pd)	< dl	< 0.3	
Platinum	(Pt)	< dl	< 1	
Tellurium	(Te)	< dl	< 0.8	
Thallium	(Tl)	0.2	< 0.5	
Thorium	(Th)	< dl	< 0.03	
Tin	(Sn)	11	< 10	
Tungsten	(W)	0.1	< 0.4	
Uranium	(U)	< dl	< 0.04	

URINE CREATININE	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	70.1	35- 225	

SPECIMEN DATA

Comments:

Date Collected:
Date Received:
Date Completed:
Method: ICP-MS

pH upon receipt: Acceptable
<dl: less than detection limit
Provoking Agent: Creatinine by Jaffe Method

Collection Period: Random
Volume:
Provocation: POST PROVOCATIVE

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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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 exceeds three times the upper expected limit per the reference population. Because a percentage of absorbed or assimilated lead is excreted in urine, the urine lead level reflects recent or ongoing exposure to lead and the degree of excretion or detoxification.

Sources of lead include: old lead-pigment paints, batteries, industrial smelting and alloying, some types of solders, ayruvedic herbs, some toys and products from China, glazes on (foreign) ceramics, leaded (antiknock compound) fuels, bullets and fishing sinkers, artist paints with lead pigments, and leaded joints in some municipal water systems. Most lead contamination occurs via oral ingestion of contaminated food or water or by children mouthing or eating lead-containing substances. The degree of absorption of oral lead depends upon stomach contents (empty stomach increases uptake) and upon the body's mineral status. Deficiency of zinc, calcium or iron may increase lead uptake. Transdermal exposure is slight. Inhalation has decreased significantly with almost universal use of non-leaded automobile fuel.

Lead accumulates extensively in bone and inhibits formation of heme and hemoglobin in erythroid precursor cells. Bone lead is released to soft tissues with bone remodeling that can be accelerated with growth, menopausal hormonal changes and osteoporosis. Lead has physiological and pathological effects on body tissues that may be manifested from relatively low lead levels up to acutely toxic levels. In children, developmental disorders and behavior problems may occur at relatively low levels: loss of IQ, hearing loss, poor growth. In order of occurrence with increasing lead concentration, the following can occur: impaired vitamin D metabolism, initial effects on erythrocyte and erythroid precursor cell enzymology, increased erythrocyte protoporphyrin, headache, decreased nerve conduction velocity, metallic taste, loss of appetite, constipation, poor hemoglobin synthesis, colic, frank anemia, tremors, nephrotoxic effects with impaired renal excretion of uric acid, neuropathy and encephalopathy. At relatively low levels, lead can participate in synergistic toxicity with other toxic elements (e.g. cadmium, mercury).

Excessive retention of lead can be assessed by urinalysis after provocation with Ca-EDTA (iv)

or oral DMSA. Whole blood analysis can be expected to reflect onlyrecent exposures and does not correlate well with total body burden of lead.

BIBLIOGRAPHY FOR LEAD

1.ATSDR Toxicological Profile for Lead(2007 update) www.atsdr.cdc.gov/toxprofile

2. 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.

3. "Preventing Lead Poisoning in Young Children", US Centers for Disease Control, Atlanta, GA, Oct. 1991 Statement, US Dept. of Health and Human Services.

4. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, Inc., Chelsea, MI, p. 128-135, 1986.

5. Tsalev D.L. et al. Atomic Absorption Spectrometry in Occupational and Environmental Health Practice Vol 1, CRC Press, BocaRaton, FL 1983.

6. Piomelli S. et al. "Management of Childhood Lead Poisoning", J. Pediatr 105 (1990) p. 523-32.

7. 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.

MERCURY HIGH

This individual's urine mercury (Hg) far exceeds the expected level for the general population under non-provoked condititons. 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 assimilaion 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 ayruvedic 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 [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 ayruvedic medicines sold via the internet." JAMA (2008)300(8): 915-23.

TIN HIGH

Tin is elevated in this individual's urine, and urine accounts for at least 80% of excreted tin that is ingested and absorbed from the gastrointestinal tract. Ingested tin is not significantly absorbed if it is an inorganic form. Oxide coatings readily form on metallic tin, and salts can quickly oxidize making them insoluble. Organic tin, however, is bioavailable and more readily absorbed. Some organic tin compounds such as short-chain alkyltins can be absorbed transdermally and can cause degeneration of myelin.

Food and drink usually provide small daily intakes of (nontoxic) tin, with amounts depending upon type of food, packaging, quality of drinking water and water piping materials. Total daily intake is expected to vary from about 0.1 to 15 milligrams. Tin is present in many metal alloys and solders; bronze, brass and pewter contain the element. Dyes, pigments and bleaching agents often contain tin. Anticorrosion plating of steel and electrical components may also use tin. "Tin cans" are tin-plated steel with a thin outer oxide layer allowing the surface to be shiny but inert. Modern food-containing cans usually have polymer coatings that prevent food-metal contact. In the past some toothpastes contained stannous fluoride, a soluble fluoride source for strengthening tooth enamel. Currently most brands of fluoridated toothpastes contain sodium fluoride. Organic tins, the usually toxic forms, are: biocides (triphenyltin and alkyltins) used against rodents, fungi, insects and mites; curing agents for rubbers and silicones (dialkyltin); and methyltin formed bacteriologically (similar to methylmercury).

Mildly elevated levels of tin in urine may reflect sporadic dietary intake and excretion; there may be no associated symptoms. A two- or three-fold increase in urine tin levels is not uncommon following administration of EDTA or with sulfhydryl agents (DMSA, D-penicillamine, DMPS). Early signs of chronic organic tin excess can be: reduced sense of smell, headaches, fatigue and muscle aches, ataxia and vertigo. Hyperglycemia and glucosuria are reported. Also, for organic tin exposure, there can be irritation of contacted tissues (eyes, skin, bronchial tubes, or GI tract). Later, immune dysfunction may occur with reduced lymphocytes and leukocytes; mild anemia may occur. A hair element analysis can be used to corroborate tin excess. Tin is commonly elevated in urine from autistic patients following administration of DMSA or DMPS.

BIBLIOGRAPHY FOR TIN

1. Winship K.A. ”Toxicity of Tin and Its Compounds”, Adverse Drug Reactions and Acute Poisoning Reviews, 7 no.1, pp 19-38, 1988.
2. Gray B.H. et al. ”Inhibition of Tributyltin Mediated Hemolysis by Mercapto Compounds” J. Applied Toxicology 6 no.5 pp 363-70, 1986. Discussed BAL, DMSA, DMPS relative effectiveness in inhibiting a toxic effect of tin.
3. Ganguly B.B. et al ”Cytotoxicity of Tin in Human Peripheral Lymphocytes in Vitro” Mutation Research, 282 no.2, pp 61-67, 1992.
4. Tsalev D.L. and Z.K. Zaprianov, Atomic Absorption Spectrometry in Occupational and Environmental Health Practice, vol. 2, CRC Press, Boca Raton, FL, pp 199-204, 1983.
5. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publ., Chelsea MI, pp 260-63, 1987.