LAB #: Sample Report
PATIENT: Sample Patient
ID:
SEX: Female
DOB: 01/01/1978 AGE: 41

CLIENT #: 12345
DOCTOR: Sample Doctor
Doctor's Data, Inc.
3755 Illinois Ave.
St. Charles, IL 60174 U.S.A.

Essential Elements; Urine

ESSENTIAL AND OTHER ELEMENTS		RESULT/UNIT per creatinine		REFERENCE INTERVAL	PERCENTILE 2.5th 16th 50th 84th 97.5th
Sodium	(Na)	**97**	mEq/g	45- 200	
Potassium	(K)	**60**	mEq/g	20- 110	
Phosphorus	(P)	**460**	µg/mg	180- 1100	
Calcium	(Ca)	**89**	µg/mg	30- 350	
Magnesium	(Mg)	**66**	µg/mg	25- 230	
Zinc	(Zn)	**1.3**	µg/mg	0.1- 1.5	
Copper	(Cu)	**0.015**	µg/mg	0.007- 0.06	
Sulfur	(S)	**870**	µg/mg	275- 1200	
Manganese	(Mn)	**0.002**	µg/mg	0.0004- 0.007	
Molybdenum	(Mo)	**0.11**	µg/mg	0.013- 0.15	
Boron	(B)	**1.9**	µg/mg	0.5- 4	
Chromium	(Cr)	**< dl**	µg/mg	0.0003-0.0025	
Lithium	(Li)	**0.022**	µg/mg	0.009- 0.2	
Selenium	(Se)	**0.057**	µg/mg	0.03- 0.25	
Strontium	(Sr)	**0.09**	µg/mg	0.045- 0.5	
Vanadium	(V)	**< dl**	µg/mg	0.0001-0.0017	
					68th 95th
Cobalt	(Co)	**< dl**	µg/mg	< 0.008	
Iron	(Fe)	**< dl**	µg/mg	< 1	

URINE CREATININE	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	**66.2**	30- 225	

SPECIMEN DATA

Comments:

Date Collected: **03/06/2019**
Date Received: **03/08/2019**
Date Completed: **03/11/2019**
pH Upon Receipt: **Acceptable**
<dl: **less than detection limit**
Provoking Agent:
Collection Period: **Random**
Volume:
Provocation: **PRE 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

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.

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 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 Medi- cine 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.

MOLYBDENUM HIGH

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

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

Administration of EDTA is not observed to raise molybdenum levels in the urine. Significant urine Mo levels in molybdenum normal individuals (adults) may occur with D-penicillamine administration and up to 300 micrograms/24 hours is commonly observed (Doctor’s Data). Similar increases with DMSA administration would be expected. For DMPS (administered slow-push intravenously) up to 250 micrograms Mo/24 hours is commonly seen, and prolonged use of dithiol chelators can deplete molybdenum stores.

Elevated Mo in urine can occur in renal wasting syndromes, nephritis, and biliary dysfunction or blockage. Other elements would then be relatively more increased (Mn, Fe, Cu). Administration of supplemental copper in high doses can result in elevated urine molybdenum; copper and molybdenum are mutually antagonistic with respect to body retention. Tungsten is a more powerful antagonist. Individuals doing tungsten-inert-gas (”TIG”) welding may episodically excrete high amounts of molybdenum (but may actually be subnormal in body tissue levels). Increased dietary sulfate levels reduce intestinal absorption and increase renal excretion of molybdenum (eg. MSM).

Molybdenum is relatively nontoxic. Studies with animals show that huge oral does are required to produce clinical symptoms which are those of copper deficiency: loss of appetite, anemia, arthritic signs, diminished glucose tolerance, loss of skin pigmentation. Moderately excessive molybdenum uptake can produce gout-like symptoms and elevated blood/urine levels of uric acid.

If molybdenum excess is suspected, the following laboratory tests could be informative: serum and urine uric acid levels, hair multielement analysis including copper and molybdenum, packed blood cell molybdenum and copper levels, erythrocyte SOD activity.

BIBLIOGRAPHY FOR MOLYBDENUM

1. Carson B.L. et al., Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, Chelsea, MI, pp 157-61, 1987.
2. Nielson F.H., Chapter 14 in Modern Nutrition in Health and Disease vol 1, 8th ed., Shils, Olson and Shike eds., Lea & Febiger, Phildelphia PA, pp 277-79, 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 169-73, 1983.
4. Smith B.S.W. and H. Wright, "Effect of Dietary Molybdenum on Copper Metabolism" Clin. Chem.Acta. 62, pp 55-63, 1975.
5. Cohen J.J. et al., "Molecular Basis of the Biological Function of Molybdenum" Proc. Nat. Acad. Sci. 70 no. 2 (Part I), pp 3655-59, 1973.

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

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.