Nordic Laboratories

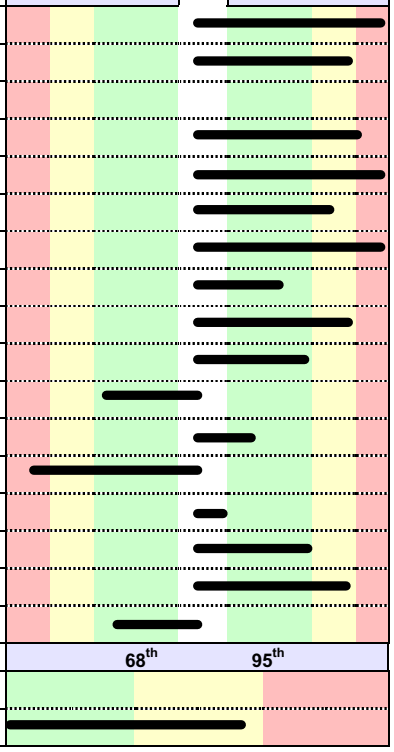
PATIENT:	**Sample Report**			TEST REF:	**TST-##-#####**
TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy	ADDRESS:	
GENDER:	Female	TESTED:	dd/mm/yyyy		
AGE:	54				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Urine Essential Elements

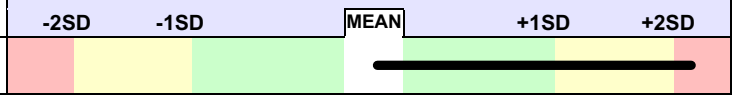
Essential Elements; Urine 24 hour

ESSENTIAL ELEMENTS PER CREATININE	RESULT mEq/g creat	REFERENCE INTERVAL
Sodium (Na)	**180**	45– 200
Potassium (K)	**53**	20– 110
	µg/mg creat	
Phosphorus (P)	**770**	180– 1100
Calcium (Ca)	**280**	30– 350
Magnesium (Mg)	**100**	25– 230
Zinc (Zn)	**1.2**	0.1– 1.5
Copper (Cu)	**0.016**	0.007– 0.06
Sulfur (S)	**820**	275– 1200
Manganese (Mn)	**0.002**	0.0004– 0.007
Molybdenum (Mo)	**0.015**	0.013– 0.15
Boron (B)	**1.6**	0.5– 4
Chromium (Cr)	**< dl**	0.0003–0.0025
Lithium (Li)	**0.027**	0.009– 0.2
Selenium (Se)	**0.064**	0.03– 0.25
Strontium (Sr)	**0.24**	0.045– 0.5
Vanadium (V)	**< dl**	0.0001–0.0017
Cobalt (Co)	**< dl**	< 0.008
Iron (Fe)	**0.42**	< 1

ESSENTIAL ELEMENTS PER 24 HOURS	RESULT mEq/24 HR	REFERENCE INTERVAL
		PERCENTILE 2.5th 16th 50th 84th 97.5th
	390	40– 250
	120	25– 120
	mg/24 HR	
	1690	200– 1600
	610	40– 350
	220	35– 250
	2.6	0.12– 1.5
	0.034	0.008– 0.07
	1800	310– 1800
	0.004	0.0008– 0.008
	0.032	0.015– 0.18
	3.5	1– 6.5
	< dl	0.0003– 0.003
	0.059	0.01– 0.25
	0.14	0.03– 0.27
	0.52	0.05– 0.53
	< dl	0.0001– 0.002
		68th 95th
	< dl	< 0.008
	0.92	< 1



URINE CREATININE	RESULT mg/24 hr	REFERENCE INTERVAL
		-2SD -1SD MEAN +1SD +2SD
Creatinine	**2210**	600– 2100



SPECIMEN DATA			
Comments:			
Date Collected:	01/01/2018	pH Upon Receipt: **Acceptable**	Collection Period: **24 hr**
Date Received:	01/01/2018	<dl: **less than detection limit**	Volume: **3500 ml**
Date Completed:	01/01/2018	Provoking Agent:	Provocation:
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

Nordic Laboratories

PATIENT:	**Sample Report**			TEST REF:	**TST-##-#####**
TEST NUMBER:	##########	COLLECTED:	dd/mm/yyyy	PRACTITIONER:	**Nordic Laboratories**
PATIENT NUMBER:	##########	RECEIVED:	dd/mm/yyyy		
GENDER:	Female	TESTED:	dd/mm/yyyy	ADDRESS:	
AGE:	54				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Urine Essential Elements

INTRODUCTION

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

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.

Urine Sodium High

The concentration of sodium in this urine sample is higher than expected and is more than two standard deviations above the mean. A high urine sodium concentration can indicate that the kidney's capacity to reabsorb sodium might be impaired and/or that some stimulus to excrete sodium is present. Urine sodium can vary from day to day depending on the degree of water reabsorption. To get an accurate assessment of renal clearance of sodium, both urine and serum sodium can be compared - this can be done with the fractional excretion of sodium (FENa) calculation (1).

Most of the sodium in the human body can be found either in blood or lymphatic fluid. Sodium levels are regulated by aldosterone (from the adrenal cortex) which acts on the proximal tubules of the nephron to increase reabsorption of sodium and water and to increase the excretion of potassium. Urine sodium testing has a role in the assessment of sodium concentration in the extracellular fluid (ECF) - urine sodium test results should be correlated clinically with sodium and water intake, observation for clinical signs of ECF volume contraction or expansion, serum sodium levels, estimation of renal function and GFR as well as with urine osmolality.

In a normal individual, urine sodium excretion generally reflects dietary intake - the more one ingests (e.g. added dietary salt, drinking and cooking with softened water, salt poisoning, etc.) the more one excretes. High urine sodium may be associated, for example, with diuretic use or conditions such as Addison's disease (primary adrenal insufficiency).

1. Bazari H. Approach to the patient with renal disease. In: Goldman L, Ausiello D, eds. Cecil Medicine. 23rd ed. Philadelphia, Pa: Saunders Elsevier; 2007:chap 115.

2. National Adrenal Diseases foundation (NADF). What is Addison's Disease. Undated; accessed at http://www.nadf.us/diseases/addisons.htm

TEST NAME: Urine Essential Elements

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; 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.

Phosphorus High

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

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

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

CALCIUM HIGH

Urine analysis is not a preferred way to assess body calcium stores. Nutritional sufficiency of calcium should be assessed through dietary analysis. Whole blood calcium level, serum calcium ion level, parathyroid hormone determinations, and bone density measurement are tests that are more indicative of calcium status.

High urinary calcium may be an artifact of diet, or of nutritional supplementation of calcium, or of excessive use of vitamin D or of vitamin A. Very high protein diets may cause increased uptake and excretion of dietary calcium. Cessation of these dietary inputs typically normalizes the urinary calcium level within several days.

High urinary calcium is associated with detoxification therapies in which EDTA is administered.

High urine calcium also can be a consequence of immobilization or extended bed rest. Steroid therapy and glucocorticoid excess can raise urinary calcium levels.

Pathological conditions that may feature elevated urinary calcium include: renal acidosis, hyperparathyroidism, hyperthyroidism, diabetes mellitus, ulcerative colitis and some cases of Crohn's disease, sarcoidosis, acromegaly, myeloma, carcinoma of the thyroid or metastatic to bone, and Paget's disease.

Osteoporosis is NOT reliably indicated by urine calcium measurement only because the calcium loss is typically too slow and insidious to significantly raise urinary calcium.

BIBLIOGRAPHY FOR CALCIUM
1. D.S. Jacobs et al. Laboratory Test Handbook, 2nd ed., Williams and Wilkins, Baltimore MD, pp 121-122, 1990.
2. D.W. Martin et al. Harper's Review of Biochemistry, 20th ed., Lange Medical Publications, Los Altos, CA, pp 651-52, 1985.
3. H. Bickel and J. Stern Inborn Errors of Calcium and Bone Metabolism, MTP Press, Ltd., St. Leonardsgate, Lancaster, Great Britain, 1976.
4. K. Isselbacher et al. (eds) Harrison's Principles of Internal Medicine, 13th ed. McGraw-Hill, New York,NY, pp 2151-71, 1994.
5. R.K. Murry et al. Harper's Biochemistry, Appleton and Lange, Norwalk, CT 1993, pp 515-22, 1993.
6. R.A. Goyer. "Nutrition and Toxicity" Am.J.Cl.Nutr. 61: pp. 6465-6505, 1995.

MAGNESIUM HIGH

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

Elevated urine magnesium is an expected finding after administration of EDTA, with levels of 150 to 300 mg/24 hr commonly seen (adults). Elevated urine magnesium is not expected with administration of sulfhydryl agents (DMPS, DMSA, D-penicillamine).

Homeostatic regulation of blood magnesium levels is normally maintained within close limits, and homeostasis closely controls intestinal uptake and renal conservation. There are, however, many possible metabolic, hormonal, drug and (toxic) chemical influences which can increase renal excretion of magnesium, perhaps causing "magnesium wasting". These are listed below.

. Hypermagnesemia, excessive infusion of magnesium
. Hypercalcinuria/hypercalcinemia, excessive supplementation or infusion of calcium
. Hyperphosphaturia/hypophosphatemia
. Hypokalemia with urinary potassium wasting
. Hyperaldosteronism
. Hyperparathyroidism
. Alcoholism
. Hypertaurinuria/hypotaurinemia
. Diuresis: diabetes, use of thiazides, other diuretics

. Acidosis: fasting, diabetic ketoacidosis
. Renal tubular dysfunction/damage, postrenal obstruction, nephritis, Bartter's syndrome
. Nephrotoxic drugs/chemicals: amphotericin, cisplatin, aminoglycides, cyclosporin, theophylline, pentamidine.

Many pesticides, herbicides and fungicides are nephrotoxic, and may cause renal wasting; others may cause renal insufficiency, depending upon dose and time elapsed after exposure (Kuloyanova and El Batawi, Human Toxicology of Pesticides, CRC Press 1991; Sittig, Pesticide Manufacturing and Toxic Materials Control Encyclopedia, Noyes Data Corp., 1980).

Magnesium status can be difficult to assess; whole blood and blood cell levels are more indicative than serum/plasma levels. The magnesium challenge method may be most indicative: baseline 24-hour urine Mg measurement, followed by 0.2 mEq/Kg of intravenous Mg, followed by 24-hour Mg measurement. A deficiency is judged to be present if less than 80% of the Mg challenge is excreted. Ref. Jones, et al. "Magnesium Requirements in Adults", Med Journal Clin Nutr, 20 (1967) p.632-35.

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.

TEST NAME: Urine Essential Elements

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.

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.