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:	50				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Urine Toxic Metals (timed or 24 hour) Pre

Toxic Metals; Urine 24 hour

TOXIC METALS PER CREATININE		RESULT µg/g creat	REFERENCE INTERVAL
Aluminum	(Al)	**< dl**	< 35
Antimony	(Sb)	**< dl**	< 0.2
Arsenic	(As)	**130**	< 80
Barium	(Ba)	**1.4**	< 7
Beryllium	(Be)	**< dl**	< 1
Bismuth	(Bi)	**< dl**	< 4
Cadmium	(Cd)	**< dl**	< 1
Cesium	(Cs)	**16**	< 10
Gadolinium	(Gd)	**< dl**	< 0.8
Lead	(Pb)	**0.5**	< 2
Mercury	(Hg)	**< dl**	< 4
Nickel	(Ni)	**9.5**	< 10
Palladium	(Pd)	**< dl**	< 0.3
Platinum	(Pt)	**< dl**	< 0.1
Tellurium	(Te)	**< dl**	< 0.5
Thallium	(Tl)	**0.7**	< 0.5
Thorium	(Th)	**< dl**	< 0.03
Tin	(Sn)	**0.3**	< 5
Tungsten	(W)	**< dl**	< 0.4
Uranium	(U)	**< dl**	< 0.04

TOXIC METALS PER 24 HOURS	RESULT µg/24 HOUR	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
	< dl	< 30	
	< dl	< 0.2	
	200	< 90	
	2.2	< 7	
	< dl	< 1	
	< dl	< 3	
	< dl	< 1.2	
	25	< 10	
	< dl	< 0.6	
	0.7	< 2	
	< dl	< 5	
	15	< 13	
	< dl	< 0.3	
	< dl	< 0.2	
	< dl	< 0.5	
	1.1	< 0.5	
	< dl	< 0.03	
	0.4	< 4	
	< dl	< 0.4	
	< dl	< 0.04	

URINE CREATININE	RESULT mg/24 hr	REFERENCE INTERVAL	- 2SD	-1SD	MEAN	+1SD	+2SD
Creatinine	**1550**	600– 2100					

SPECIMEN DATA		
Comments:		
Date Collected: **dd/mm/yyyy**	pH Upon Receipt: **Acceptable**	Collection Period: **24 hr**
Date Received: **dd/mm/yyyy**	<dl: **less than detection limit**	Volume: **3500 ml**
Date Completed: **dd/mm/yyyy**	Provoking Agent:	Provocation: **PRE PROVOCATIVE**
Method: **ICP-MS**	**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 Aps
Nygade 6, 3.sal • 1164 Copenhagen K • Denmark
Tel: +45 33 75 10 00

UK Office:
11 Old Factory Buildings • Stonegate • E. Sussex TN5 7DU • UK
Tel: +44 (0)1580 201 687

www.nordic-labs.com
info@nordic-labs.com

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:	50				
DATE OF BIRTH:	dd-mm-yyyy				

TEST NAME: Urine Toxic Metals (timed or 24 hour) Pre

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.

ARSENIC HIGH

This inidividual's urine arsenic (As) is higher than expected. Because urine is the major mode of excretion for arsenic, an elevated level reflects increased assimilation of As. Ingestion of organic species of As in seafood is not uncommon and may be associated with very elevated urine As. Arsenobetaine and arsinocholine, commonly found in shellfish are relatively non-toxic and 90% is excreted in the urine with a half-life of about 48 hours.

Sources of As include: contaminated foods (e.g. chicken), water or medications. Industrial sources are: ore smelting/refining/processing plants, galvanizing, etching plating processes. Tailing from or river bottoms near gold mining areas (past or present) may contain arsenic. Insecticides, rodenticides and fungicides (Na-, K- arsenites, arsenates, also oxides are commercially available). Commercial As-containing products include: sodium arsenite, calcium arsenate, lead arsenate and "Paris green" which is cupric acetoarsenite, a wood preservative (pressure treated wood). Undesirable levels of As have been found in many Ayruvedic herbs.

Chronic exposure to or ingestion of inorganic As causes tissue levels to gradually increase as the element binds to sulfur, phosphorus and selenium. An important detrimental effect is inactivation of lipoic acid,a vitamin cofactor needed for metabolism of pyruvate and alpha-ketoglutarate.

Symptoms consistent with mild or moderate As exposure include: fatigue, malaise, eczema or allergic-like dermatitis, and garlic-like breath. Increased salivation may occur. Hair element analysis may provide further evidence of As exposure to inorganic As. Blood As levels are not dose related and may or may not reflect As exposure or net retention of As. Levels of As may exceed the expected range after administration of DMPS or DMSA depending upon cumulative exposures. This does not necessarily indicate As excess to the point of toxic effects or physiological impairment.

BIBLIOGRAPHY FOR ARSENIC

1. Centers for Disease Control and Prevention. Third National Report on Human Exposure to Environmental Chemicals. Atlanta, GA; CDC: 2005. http://www.cdc.gov/exposurereport/report.htm [Accessed 2/01/2009]
2. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers,

Chelsea, MI, pp 27-33, 1987.
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. 87-93, 1983.
4. Clarkson T.W. et al. eds. Biological Monitoring of Toxic Metals, Plenum Press, NY, NY, pp 309-15, 1988.
5. Harrison's Principles of Internal Medicine, 13th ed., McGraw Hill, New York, NY, pp 2461-62, 1994.
6. Heyman A. et al. "Peripheral Neuropathy Caused by Arsenical Intoxication" New Eng. J. Med., 254 no.9, pp 401-9 1956.
7. 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.

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 cytogenetic damage.

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 Datais 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 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/cesiumcs06.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

NICKEL HIGH

This individual's urine nickel (Ni) is elevated which may or may not be of significance. Urinary excretion of nickel bound to cysteine or other thiol compounds (such as glutathione) or to amino acids (histidine, aspartic acid, arginine) is the predominant mode of excretion. With the exception of specific occupational exposures, most absorbed Ni comes from food or drink, and intakes can vary by factors exceeding 100 depending upon geographical location, diet, and water supply. Depending upon chemical form and physiological factors, from 1 to 10% of dietary Ni may be absorbed from the gastrointestinal tract. Urine Ni only reflects recent exposure to Ni and may vary

widely from day to day.

Sources of nickel are numerous and include the following.

. Cigarettes (2 to 6 mcg Ni per average cigarette)
. Diesel exhaust (particulates may contain up to 10 mg/gram)
. Foods, especially: cocoa, chocolate, soya products, nuts, hydrogenated oils, and coffee
. Nickel-cadmium batteries (Ni-Cd)
. Nonprecious, semiprecious dental materials
. Nickel-containing prostheses
. Electroplating, metal plated objects, costume jewelry
. Pigments (usually for ceramics or glass)
. Catalyst materials (for hydrogenation processes in the food, petroleum and petrochemical industries)
. Arc welding
. Nickel refining and metallurgical processes

Most clinically relavant Ni exposures are manifested as dermatoses - contact dermatitis and atopic dermatitis. However, Ni hypersensitizes the immune system and may cause hyperallergenic responses to many different substances. Because Ni can displace zinc from binding sites on enzymes it can affect abnormal enzymatic activity. Nickel sensitivity is observed to be three to five times more prevelant in females than in males.

Other laboratory tests or possible clinical findings that may be associated with Ni exposure are; hair elements analysis, presentation of multiple allergic sensitivities, dermatitis, positive patch test for "Ni allergy", proteinuria, hyperaminoaciduria (by 24-hour urine amino acid analysis). Administration of EDTA or sulfhydryl agents (DMPS, DMSA, D-penicillamine) may increase urine Ni levels; such chelator-induced elevations may or may not be associated with adverse health effects.

BIBLIOGRAPHY FOR NICKEL

1. Tsalev D.L. and Z.K. Zaprianov Atomic Absorption Spectrometry in Occupational and Environmental Health Practice, CRC Press, Boca Raton FL, pp 173-78, 1983.
2. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, Chelsea MI, pp 162-67, 1986.
3. Nielsen F.H. in Modern Nutrition in Health and Disease ed. by Shils et al, Lea & Febiger, Philadelphia, PA, pp 279-81, 1994.
4. Medical and Biological Effects of Environmental Pollutants: Nickel, Nat. Acad. Sci, Washington DC,1975.
5. Ambient Water Quality Criteria for Nickel, US EPA NTIS, Springfield, VA. Publ No. PB81-117715,1980.

THALLIUM HIGH

This individual's urine thallium (Tl) is markedly higher than expected and is indicative of high level exposure to Tl. 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 Tl in the body.

TEST NAME: Urine Toxic Metals (timed or 24 hour) Pre

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

1. Centers for Disease Control and Prevenstion. Third National Report on Human Exposure to Enviornmental Chemicals. Atlanta, GA: CDC; 2005. http://www.cdc.gov/exposurereport.htm [Accessed 2/01/2009]
2. Graef J.W. ”Thallium” in Harrison’s Principles of Internal Medicine, 13th ed., Isselbacher et al eds., McGraw Hill, pp 2465, 1994.
3. 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.
4. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans Lewis Publishers, Chelsea, MI, pp 243-254, 1987.