Micro Trace Minerals Laboratory

environmental & clinical laboratory

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MINERAL ANALYSIS — DMPS Urine

		Lab Number		1UP120000	
Doctor	-			Test Date	2/16/2012
Patient Name	-	Sex	w	D.O.B.	1/1/1959
Clinical Information	DMPS IV 1h				
Creatinine (g/l) *	0.30			Page	1/7

	Baseline URINE Norm	Chelator-specific orientation range	Test Value		
Essential Trace Elements (mcg/g Creatinine)					
Chromium	< 3.50		3.10		
Cobalt	< 5.00		0.14		
Copper	< 60.00	700.00	333.14		
Iron	2.00 --- 95.00		25.47		
Manganese	< 4.50	10.00	n.n.	↓	
Molybdenum	9.70 --- 100.00		10.78		
Selenium	12.00 --- 90.00		7.23	↓	
Vanadium	< 1.40		0.05		
Essential Macro- & TraceElements (mg/g creatinine)					
Calcium	55.00 --- 245.00		120.19		
Magnesium	12.00 --- 150.00		38.93		
Zinc	0.07 --- 7.00	10.00	1.11	↓	
Trace Elements in mcg/g Creatinine					
Germanium	< 1.50		0.00		
Lithium	< 175.00		19.58		
Strontium	< 570.00		96.19		
Potentially Toxic Elements in mcg/g Creatinine					
Aluminum	< 125.00		30.28		
Antimony	< 1.00		0.36		
Arsenic-total	< 50.00	100.00	11.50		
Barium	< 8.22		1.61		

n.n. = not detected
These 95percentile Reference Ranges listed above are representative for a healthy population. All elements are tested quantitatively.
Accreditation: DIN EN ISO 17025; Quality control: Dipl. Ing. Friedle, Ing. J. Merz, Dr. Rauland; Validation: Dr. E.Blaurock-Busch PhD, Laboratory physician: Dr. med. A. Schönberger

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MINERAL ANALYSIS — DMPS Urine

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	Baseline URINE Norm	Chelator-specific orientation range	Test Value		
Potentially Toxic Elements in mcg/g Creatinine					
Beryllium	< 1.20		0.06		
Bismuth	< 0.15		0.05		
Cadmium	< 0.80	1.50	0.28		
Lead	< 5.00	12.00	4.17		
Mercury	< 1.00	18.00	3.60		
Nickel	< 3.00	7.00	11.40	↑	
Platinum	< 0.60		n.n.		
Silver	< 1.40		0.03		
Thallium	< 0.60		0.09		
Tin	< 5.00	15.00	16.73	↑	

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URINE ANALYSIS AND CHELATION INFORMATION:
Urine analysis is an indispensable tool for assessing the renal ability to excrete toxic metals, especially before and after provocation.

Results are reported in mg/g creatinine for the macro elements and mcg/g creatinine for the trace elements and heavy metals. Normalization per mg or mcg creatinine reduces the potentially great margin of error which otherwise can result from sample collection and variation in sample volume given. A creatinine value of <0,3g/L is the borderline level for the conversion of test values to mg/g and mcg/g creatinine. When lower creatinine levels are measured (usually due to a high fluid intake during urine collection time), the borderline value of 0,3g/l is used for the conversion.

Chelation treatment or provocation with complexing agents increase metal binding and urinary excretion. DMPS stimulates, even forces the binding and excretion of metals such as copper, arsenic and mercury. Clinical and laboratory evaluation indicate that a copper value of 700mcg/g creatinine is to be expected after the administration of 1 ampule DMPS IV or the oral provision of 300mg.

This report provides DMPS-specific orientation values, which were obtained following statistical observations.

Test values are compared to urine baseline reference ranges (UB RR) and DMPS specific Orientation Ranges. When challenged with 1 ampule (5ml) DMPS, 65% of testpersons showed values equal to or lower than the DMPS-specific Orientation Range (OR)

A test value higher than the UB RR and lower than the OR may be viewed as a marginal to moderate exposure on the test value.

A test value higher than the UB RR that also exceeds the OR represents a moderate to high exposure, depending on the test value.

The toxicological of effect of one minor burdens may be significant, depending on the patient's condition; two or more minor burdens may affect health significantly more.

The type of exposure must be medically evaluated. Patient history and symptoms must be taken into consideration.

RELATED INFORMATION: The data of this report is based on ICP-MS Spectroscopy utilizing cell technique. Strict quality control measurements and licensing requirements are followed, including round robin blind testing by licensing authorities. For more information: www.microtrace.de

The information contained in this report is designed as an interpretive adjunct to normally conducted diagnostic procedure. The findings are best viewed in the context of a medical examination and history.

LITERATUR:

Berlin M. et al. Handbook on the Toxicology of Metals, 3rd Edition. Academic Press nc. 675-729, 2007
Blaurock-Busch, Antidota- Handbook of Chelation Therapy, MTM 2010
Thomas L. Labor & Diagnose, 4. Auflage Med. Verlag Marburg 1992
VanderSchaar, IBCMT Textbook of Clinical Metal Toxicology 2009

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COPPER (Cu) HIGH:
This trace element is an important metallo-enzyme, essential in hemoglobin synthesis. Adults absorb approximately 56% of dietary Cu, with < 50mcg/day excreted in the urine under normal and unprovoked conditions. The adult body contains approximately 80mg copper, one third in muscle and the reminder in other tissue and body fluids. In the divalent state copper has the capacity to readily complex with many amino acids and proteins, such as metallothionin , which facilitate Cu-absorption from the stomach and the duodenum. In a large number of cuproproteins, Cu is a fixed proportion of the molecular structure, and these metalloproteins form an important group of oxidase enzymes, including ceruloplasmin (ferroxidase), SOD (superoxide dismutase), cytochrome oxidase, lysyl oxidase, dopamine beta-hydroxylase, tyrosinase, uricase, spermine oxidase, benzylamin oxidase, diamine oxidase, and tryptophan-2,3 dioxygenase (tryptophan pyrrolase).

LABORATORY INFORMATION:
DMPS stimulates copper binding. A urinary copper level of 700mcg/g creatinine can be expected. A level higher than that may mask the mercury burden i.e. block mercury excretion. Therefore, the German toxicologist Dr. Daunderer recommended a follow-up DMPS challenge test after 3-4 weeks. After copper levels dropped, an increase in mercury levels may be seen.

Continued DMPS treatment may affect the copper and zinc metabolism and reduce tissue storage. Biomonitoring of Blood and hair analysis may be useful.

LITERATURE:

Daunderer, M. Handbuch der Umweltgifte. www.amazon.de
Kaplan LA; Pesce AJ. Clin Chem. Theory, analysis,correlation. 2nd ed. Mosby 1989, p535-536

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MERCURY (Hg):
Mercury compounds are readily bound by DMPS and an excretion level greater than the baseline urine reference range is to be expected. Such a level may be viewed as a reflection of a moderate burden and can be seen in nonsymptomatic and symptomatic patients. A level even greater than the orientation range should be viewed in context with other minor or moderate burdens, especially when treatment schedules are developed. When a urinary copper level greater than 700mcg/g creatinine is present, follow-up DMPS treatment may result in a rise of mercury excretion after copper levels decrease.

Toxicity Signs and Symptoms:
Researchers at the University of Calgary demonstrated that even minute levels of mercury are potent neurotoxins, causing neuronal death, meaning no level of mercury may be considered safe.

The most frequent causes of exposure to toxic levels of mercury are those related to acute accidental or chronic industrial exposure. Mercury vapour exposure (including from dentistry) produces toxic effects due to the accumulation in the brain. Neurological signs may include increased excitability, severe behavioural and personality changes, insomnia and loss of memory. With continued or acute exposure, gastrointestinal disturbance and renal damage are likely to occur.

•Early Symptoms of Chronic overexposure
Insomnia, dizziness, fatigue, drowsiness, weakness, depression, tremors loss of appetite, loss of memory, nervousness, headache, dermatitis, numbness, and tingling of lips and feet, emotional instability and kidney damage.

Sources:
Overexposure may stem from paints, bleaches, explosives, electrical apparatus, batteries, mercurial diurectics, fungicides, fluorescent lamps, cosmetics, hair dyes, amalgams in dentistry, contaminated seafood, and petroleum products. Vaccines such as tetanus toxoid contain thiomersal which is a mercury compound. Improper disposal of broken mercury thermometers and other apparatuses that use mercury including button cells and tube lights are additional sources of mercury exposure.

Literature:

Berlin M: Mercury, In Friberg L. Nordberg GF and Vouk, VB, editors: Handbook on the toxicology of metals. Amsterdam, 1979, Elsevier/No Holland Biomed Press
Clarkson TW. Mercury poisoning. In Brown SS, editor: Clinical chemistry and chemical toxicology of metals. Amsterdam, 1977. Elsevier/No Holland Biomed Press.
Daunderer M. Handbuch der Umweltgifte. www.amazon.de
Kaplan LA, Pesce AJ. Clinical Chemistry, Theory, analysis, and correlation. 2nd ed. Mosby UK 1989, p 541
Thomas L. Labor und Diagnose.4th ed. Med.Verlag Marburg 1992, p436

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NICKEL (Ni) HIGH:
Smoke, cigarette smoking and food are major sources of nickel exposure, hence a level greater than the reference range and lower than the DMPS-Orientation range may be due to dietary or environmental exposure. If the urinary excretion level is greater than the orientation range, the source of exposure should be evaluated. Treatment schedules should consider the presence (or absence) of other metal burdens and carefully weigh patient lifestyle, history and symptoms.

Environmental/Occupational Sources
Ni is found in ambient air at very low levels, as a result of releases from manufacturing facilities, oil and coal combustion, sewage sludge incineration, and other sources.
Exposure may be through contact with everyday items such as nickel-containing jewelry, cooking utensils, stainless steel kitchens, and clothing fasteners.

Toxicity and Symptoms:
Nickel carbonyl is the most acutely toxic nickel compound, also found in cigarette smoke. Symptoms include headache, vertigo, nausea, vomiting, insomnia, and irritability, followed by chest pains, dry coughing, cyanosis, gastrointestinal symptoms, sweating, visual disturbances, and severe weakness.Lung and kidney appear to be the target organs for acute nickel carbonyl toxicity in humans and animals, with pulmonary fibrosis and renal edema reported.

EPA's Office of Air Quality Planning and Standards, for a hazard ranking under Section 112(g) of the Clean Air Act Amendments, considers nickel carbonyl to be a "high concern" pollutant based on severe acute toxicity.

Chronic Effects (Noncancer):
Contact dermatitis is the most common effect in humans from nickel exposure, and have been reported following occupational and non-occupational exposure, with symptoms of itching of the fingers, wrists, and forearms. Chronic exposure to nickel in humans also results in respiratory effects, including asthma due to primary irritation or an allergic response, and an increased risk of chronic respiratory tract infections.

Cancer Risk:
Human studies have reported an increased risk of lung and nasal cancers among nickel refinery workers exposed to nickel refinery dust. Nickel refinery dust is a mixture of many nickel compounds, including nickel subsulfide. EPA has classified nickel refinery dust and nickel subsulfide as carcinogens. Nickel carbonyl has been reported to produce lung tumors in rats exposed via inhalation.

References:

U.S. Environmental Protection Agency (EPA). Integrated Risk Information System (IRIS) on Nickel. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH. 1993.
Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Nickel (Draft). U.S. Public Health Service, U.S. Department of Health and Human Services, Altanta, GA. 1993.
U.S. EPA. Technical Background Document to Support Rulemaking Pursuant to the Clean Air ActCSection 112(g). Ranking of Pollutants with Respect to Hazard to Human Health. EPAB450/3-92-010. Emissions Standards Division, Office of Air Quality Planning and Standards, Research Triangle Park, NC. 1994

SELENIUM (Se): Low urinary levels may reflect the dietary intake. The biochemical role of this essential trace element is to serve as an essential constituent of the enzyme gluthathione peroxidase. Se is linked to cysteine residues in the protein as selenocysteine, which is found in the cytoplasm and mitochondria of liver, erythrocytes, platelets and other tissues. The antioxidant role of Se parallels that of vitamin E, and Se-deficiency responds to Vit. E supplementation. Chronic selenium deficiency has been epidemiologically associated with certain cancers, cardiovascular disease, cardiomyopathy and immune dysfunctions. Causes of deficiency include inadequate selenium or cysteine intake, exposure to toxins incl. mercury, arsenic, cadmium, PCB, etc. SOURCES: wheat bran and germ, Brewer's yeast, garlic, whole grains, liver, kidney, fish. The RDA is 10-200mcg/day. THERAPEUTIC CONSIDERATION: increase intake of selenium, cysteine and vitamin E.

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TIN (Sn)
A test value greater than the Baseline Reference Range and lower than the Orientation Range is considered inconspicuous, and may be due to oral intake. Large amounts of tin can accumulate in foods that are in contact with tin plate or are absorbed as tin fluoride from toothpaste.

A test value greater than the orientation value should be carefully evaluated, especially when tin toxicity symptoms are present. A controlled fecal metal test may be considered, especially when industrial exposure is suspected.

Tin is poorly absorbed and retained by humans and is excreted mainly in the feces. Once tin is absorbed, both the bile and urine are routes of excretion and the level of accumulation seems related to the intake.

Tin has a low toxicity, but tin salts are gastric irritants causing nausea, vomiting, and diarrhea. High tin levels influence the metabolism of several minerals, including calcium, zinc and alkaline phosphates activities in liver and femur. Tin is a potent inducer of heme oxygenase and thus affects heme-dependent functions.

TOXICITY SYMPTOMS: vomiting, diarrhea, abdominal cramps, loss of appetite, tightness of chest, metallic taste in mouth, dry throat. Excessive inhalation of tin oxide can cause Stannosis (pneumoconiosis).