Micro Trace Minerals Laboratory

environmental & clinical laboratory

Röhrenstrasse 20, 91217 Hersbruck, Germany
P.O.Box 4613; Boulder, CO 80306-4613, USA

MINERAL ANALYSIS — DMSA Urine

		Lab Number		1UA180436	
Doctor	Dr.			Test Date	5/09/2016
Patient Name	John Hargrave	Sex	m	D.O.B.	31/10/1973
Clinical Information	DMSA oral 1.5g + Glycine 3g + Biosil 40 drops 12h				
Creatinine (g/l) *	0.540			Page	1/6

	Baseline URINE Norm	Chelator-specific orientation range	Test Value		
Essential Trace Elements (mcg/g Creatinine)					
Chromium	0.550 --- 4.830		1.412		
Cobalt	< 5.000		0.157		
Copper	1.450 --- 60.000		11.974		
Iron	2.200 --- 45.000		120.728	↑	
Manganese	< 4.500		4.260		
Molybdenum	9.700 --- 100.000		7.893	↓	
Selenium	12.000 --- 90.000		17.641		
Vanadium	< 1.000		n.n.		
Essential Macro- & TraceElements (mg/g Creatinine)					
Calcium	55.000 --- 245.000		30.695	↓	
Magnesium	12.000 --- 150.000		47.470		
Zinc	0.060 --- 0.780		0.250		
Trace Elements (mcg/g Creatinine)					
Germanium	< 1.500		0.579		
Lithium	< 175.000		14.920		
Strontium	< 200.000		56.739		
Tungsten	< 0.790		< DL		
Potentially Toxic Elements (mcg/g Creatinine)					
Aluminum	< 40.000		6.056		
Antimony	< 1.000		< DL		
Arsenic-total	< 15.000		22.837	↑	

n.n. = not detected, < DL = below Detection Limit
Accreditation: DIN EN ISO 17025; Quality control: Dipl. Ing. Friedle, Ing. J. Merz, Dr. Rauland; Validation: Dr. E. Blaurock-Busch PhD

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	Baseline URINE Norm	Chelator-specific orientation range	Test Value	
Potentially Toxic Elements (mcg/g Creatinine)				
Barium	< 5.700		1.180	
Beryllium	< 1.200		< DL	
Bismuth	< 0.150		< DL	
Cadmium	< 0.800		< DL	
Cesium	< 11.000		7.700	
Gallium	< 7.760		n.n.	
Lead	< 5.000	10.000	2.892	
Mercury	< 1.000	2.800	0.688	
Nickel	< 3.000	5.000	3.383	
Palladium	< 1.400		< DL	
Platinum	< 0.600		n.n.	
Silver	< 1.400		< DL	
Thallium	< 0.600		0.475	
Tin	< 2.000		0.389	
Titanium	< 13.000		< DL	
Uranium	< 0.060		n.n.	
Zirconium	< 2.500		< DL	

n.n. = not detected, < DL = below Detection Limit
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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 chelation.

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 < 0.3 g/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 0.3 g/l is used for the conversion.

Chelation treatment or provocation with complexing agents increase metal binding and urinary excretion. DMSA stimulates, even forces the binding and excretion of metals such as lead, arsenic and mercury.

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

Test values are compared to urine baseline reference ranges (UB RR) and DMSA-SPECIFIC ORIENTATION RANGES. When provoked with 500mg DMSA (oral), 65% of the test persons showed values equal to or lower than the DMSA-specific Orientation Range.

A test value higher than the URINE BASELINE REFERENCE RANGE (UB RR) and lower than the ORIENTATION RANGE may be viewed as a marginal to moderate exposure, depending on the test value.

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

The toxicological of effect of one minor burden may be significant, depending on the patient's condition; two or more minor burden 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.microtraceminerals.com

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.

LITERATURE:

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.

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ARSENIC (As):
Environmental sources of arsenic exposure include food, water, soil, and air. Arsenic is ubiquitous in the environment. Natural sources are arsenic-containing mineral ores and groundwater (especially near geothermal activity). In industry, arsenic is a by-product of the smelting process for many metal ores such as lead, gold, zinc, cobalt, and nickel. Other potential sources of arsenic exposure are:
• Commercial products: Wood preservatives, insecticides, herbicides (weed killers and defoliants), fungicides, cotton desiccants, cattle and sheep dips, paints and pigments, antifouling paints, leaded gasoline, and fire salts (multicolored flame).
• Food: Wine (grapes sprayed with arsenic-containing pesticides), and seafood (especially bivalves, certain cold water and bottom-feeding finfish, and seaweed).
• Smokers may also inhale small amounts of arsenic as a result of pesticide residue on tobacco leaves.
• Industrial processes: purifying industrial gases (removal of sulfur), burning fossil fuels, burning wood treated with arsenic preservatives, electronics manufacturing (microwave devices, lasers, light-emitting diodes, photoelectric cells, and semiconductor devices), hardening metal alloys, preserving animal hides, bronze plating, and clarifying glass and ceramics.
• Medicinals: Fowler's solution (potassium arsenite), antiparasitic drugs (carbazone), Donovan's solution, folk remedies ("Asiatic pill," kushtay, yellow root), kelp-containing health foods, some naturopathic remedies.

Laboratory Information:
The given reference range applies only if 48hrs prior to the urine collection no fish, or algae products were consumed. Mineral waters high in arsenic may also raise urinary excretion levels. Consumption of any of these sources raises urine levels considerably, at least 2-3 times above the given range.
Smoking may also raise urinary excretion levels or arsenic.

Health Effects:
• Acute arsenic toxicity may be associated with hepatic necrosis and elevated levels of liver enzymes.
• Gastrointestinal effects are seen primarily after arsenic ingestion, and less often after inhalation or dermal absorption.
• Arsenic is capable of causing acute renal failure, as well as chronic renal insufficiency.
• Long-term ingestion of arsenic in drinking water has resulted in pronounced peripheral vascular changes.
• Acute arsenic poisoning may cause both diffuse capillary leak and cardiomyopathy, resulting in shock.
• Arsenic-exposed patients develop destruction of axonal cylinders, leading to peripheral neuropathy.
• Pigment changes and palmoplantar hyperkeratosis are characteristic of chronic arsenic exposure.
• Benign arsenical keratosis may progress to malignancy.
• Inhalation of high concentrations of arsenic compounds produces irritation of the respiratory mucosa.
• Increased frequency of spontaneous abortions and congenital malformations has been linked to arsenic exposure.
• The carcinogenicity of arsenic in humans has been established, but no animal model has been developed.
• Latency for skin cancer associated with ingestion of arsenic may be 3 to 4 decades, whereas the noncarcinogenic skin effects typically develop several years after exposure.
• In arsenic-exposed workers, there is a systematic gradient in lung cancer mortality rates, depending on duration and intensity of exposure.

Source: Agency for Toxic Substances and Disease Registry. 2006.

CALCIUM (Ca):
Urine is not the specimen of choice to detect nutritional deficiencies; however, when urine levels are low, it can be safely assumed that the calcium availability was low. This may be a reflection of an adequate dietary intake.
Calcium essential for bone and teeth growth, muscular and neuronal functions; it influences hormonal secretion and is involved in immune/oxidant responses. Deficiency symptoms are muscle cramps, musculoskeletal pain, menstrual cramps, periodontal disease, and osteoporosis. The RDA is 800-1800 mg/day, depending on age and condition. The ability of the body to absorb calcium decreases with age, due to hormonal changes, reduced gastric ability and decreased activity levels.

SOURCES: Dairy products, green leafy vegetables, citrus fruits, molasses and fish with edible bones.

THERAPEUTIC CONSIDERATION: Vitamin D, the amino acid lysine and digestive enzymes, containing hydrochloric acid and pepsin assist calcium absorption. Lactobacillus acidophilus assists intestinal absorption.

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IRON (Fe) HIGH:
The physiological distribution of iron in urine is low, and the excretion of iron in unprovoked urine is unusual. High urinary iron may or may not correspond with the total iron status or body stores, because the main route for iron uptake, re-uptake and excretion is via the bile, intestinal transport and feces. Urine levels may fluctuate without reflecting or influencing body stores.

Chelation Information:
• For adults, iron levels of unprovoked iron are relatively low (<40mcg/g creatinine). Urine collected during menstruation may significantly elevate urinary iron levels without reflecting on the iron body status or chelation.
• Chelating agents such as IV EDTA, the DTPAs or deferoxamine have a significant ability to bind free iron, and elevated post urine values are to be expected.
• DMSA and DMPS increase the binding of free iron to a much lesser degree above levels of unprovoked urines.

Pathophysiology:
In cases of iron overload or hemochromatosis, urinary iron levels may increase significantly. Typically, hematuria (isolated), proteinuria with hematuria, and glomerulonephritis increase the urinary iron loss. Infections, malignancy or physical injury may be the cause. Biliary obstruction or insufficiency can decrease normal iron excretion while increasing urinary iron levels. Porphyria with urinary loss of porphyrins (before heme can be formed) can result in an increase in urinary iron.

Nutritional Information:
Excessive iron supplementation may result in iron overload and increased urine iron.

Laboratory Information:
The best tests for assessing the iron status are total iron binding capacity, transferrin levels and serum ferritin levels.

Literature: Thomas L. Labor & Diagnose, Med. Verlagsges. Marburg, 1992, p 394

MOLYBDENUM (Mo) serves as a co-factor for xanthine and aldehyde oxidases. Dietary molybdenum is readily absorbed by the intestine and is excreted in the urine and bile. SOURCES: whole grains, legumes, leafy vegetables and organ meats. The RDA is 0.15-0.5 mg/day, depending on age and status. Acute deficiency symptoms are unknown in humans. THERAPEUTIC CONSIDERATION: increase molybdenum intake and support intestinal function.

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NICKEL (Ni) HIGH:
Smoke, cigarette smoking and food are major sources of nickel exposure. A equal or above the Reference Range indicates a mild exposure; a value between the Baseline Reference Range and the Orientation Range represents a moderate exposure. When the urine concentration levels is higher than the Orientation Range, a chronic or acute case of intoxication might be present. A physician experienced in metal toxicology should be consulted.

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 (Non-cancer):
• 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:

1. 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.
2. 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.
3. 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.