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MINERAL ANALYSIS — DMSA Urine

		Lab Number		5UA150001	
Doctor	Sample Doctor			Test Date	9/25/2014
Patient Name	Sample Patient	Sex	w	Age	34
Clinical Information	DMSA oral 500mg				
Creatinine (g/l) *	0.30			Page	1/9

	Baseline URINE Norm	Chelator-specific orientation range	Test Value		
Essential Trace Elements (mcg/g Creatinine)					
Chromium	0.55 --- 4.83		4.53		
Cobalt	< 5.00		1.13		
Copper	1.45 --- 60.00		73.18	↑	
Iron	12.10 --- 131.00		20.01		
Manganese	< 4.50		6.93	↑	
Molybdenum	9.70 --- 100.00		29.00		
Selenium	12.00 --- 90.00		61.40		
Vanadium	< 1.40		n.n.		
Essential Macro- & TraceElements (mg/g Creatinine)					
Calcium	55.00 --- 245.00		33.95	↓	
Magnesium	12.00 --- 150.00		34.01		
Zinc	0.07 --- 7.00		0.41		
Trace Elements in mcg/g Creatinine					
Germanium	< 1.50		0.65		
Lithium	< 175.00		24.13		
Strontium	< 570.00		46.28		
Potentially Toxic Elements in mcg/g Creatinine					
Aluminum	< 40.00		15.48		
Antimony	< 1.00		0.10		
Arsenic-total	< 15.00		3.58		
Barium	< 8.22		3.89		

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, Laboratory physician: Dr. med. A. Schönberger

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	Baseline URINE Norm	Chelator-specific orientation range	Test Value		
Potentially Toxic Elements in mcg/g Creatinine					
Beryllium	< 1.20		< DL		
Bismuth	< 0.15		n.n.		
Cadmium	< 0.80		0.29		
Lead	< 5.00	10.00	6.41		
Mercury	< 1.00	2.80	1.36		
Nickel	< 3.00	5.00	20.86	↑	
Platinum	< 0.60		n.n.		
Silver	< 1.40		< DL		
Thallium	< 0.60		0.23		
Tin	< 5.00		0.60		

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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 <0,3g/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.

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 testpersons 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 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: http://www.tracemin.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.

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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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 800-1800mg/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 acidophius assists intestinal absorption.

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 AND TESTING INFORMATION:
Urine analysis of unprovoked urine is not an adequate measure to assess copper stores or copper metabolism. Blood copper levels, SOD levels and serum ceruloplasmin are other, often more indicative measurements for copper status. Increased urinary copper levels can be caused by nutritional supplementation with copper or be the result of a high dietary intake due to copper-containing drinking water. Supplements containing high molybdenum may stimulate an increase in copper excretion, because Cu and Mo are mutually antagonistic in terms of body retention.

CLINICAL SIGNS AND SYMPTOMS:
•Bacterial or other infection may cause hypercupremia, and published studies such as Vivoli, Sci Total Eniviron, 66p 55-64, 1987 have correlated increased urinary copper levels with increased blood pressure in hypertensives. Biliary obstruction or insufficiency can decrease normal excretion of copper via the bile while increasing blood and urine levels.
•Hyperaminoacidurias, including histidinuria can result in copper wasting since histidine is a powerful chelator of copper. Hyperaminoacidurias can be of various origins including genetic factors, chemical or elemental toxicities (high urinary copper is often seen with high mercury levels) infectious agents, hyperthyroidism, sugar intolerances, nephrotic symptoms, etc.
•In Wilson's disease, urinary copper levels may increase to above 100mcg/24hrs without provocation or chelation.

LITERATURE:

Kaplan LA; Pesce AJ. Clin Chem. Theory, analysis,correlation. 2nd ed. Mosby 1989, p535-536

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MERCURY (Hg):
Mercury compounds readily react covalently with sulfhydryl groups in proteins, resulting in inhibition of functional activity. Both organic and inorganic mercury are potent toxic compounds.
TOXICITY:
•Excretion levels of 100mcg/g creatine in random urine prior to chelation are representative of acute exposure, reflecting toxicity. Values equal to the Hg-Orientation Range indicate a mild exposure, values above that range and below the excretion level of 100mcg/g crea are representive of a past or present intoxication. Early Symptoms of Chronic overexposure may occur at much lower levels including 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.
TREATMENT: Consider chelation treatment with DMPS or DMSA.
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
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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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MANGANESE (Mn) HIGH:
High urinary concentration may correspond with an excessive manganese intake. Studies by Micro Trace Minerals indicate that drinking black or green tea during chelation and prior to urine collection can increase urinary manganese excretion.

Biochemical basis:
The major route for manganese uptake, re-uptake, and excretion is via bile, intestinal transport and feces. Typically, less than one-half of one percent of total manganese excretion occurs via urine, 3-5% in sweat; the reminder of approx. 95% occurs via bile and feces. Hence, urinary manganese may be increased in patients with biliary obstruction or cirrhosis.

Urine levels may fluctuate without reflecting or influencing body stores. In cases of manganese overexposure, intravenous EDTA chelation therapy may be the method of choice. DMPS and DMSA have a lesser binding capacity.

Pathophysiology:
Manganese excess in urine without provocative challenge are seen in renal wasting syndromes, nephritis, biliary insufficiency or obstruction, and dietary overload or excessive supplementation. Some hormones and drugs inhibit biliary excretion of manganese, resulting in increased urinary excretion. Dopamin, glucagon and cyclic AMP are reported to do this.

Environmental or industrial sources of manganese include: mining, refining and processing of metals and ores, welding, glazes, and pigments, petrochemicals, plastics and synthetic rubbers in industry, some type of batteries, and certain gasoline additives. Ground water used as drinking water may contain manganese, and EPA water surveys indicate that the manganese content of city drinking water fluctuates between <5 to 350mcg/L. Manganese-rich water promotes bacterial growth.

Neurotoxicity of Manganese
Manganese can be neurotoxic. Symptoms of intoxication (esp. after inhaling Mn) include hyperirritability, hallucinations, violence, tremor, Parkinson-like symptoms, anorexia, sexual impotence, and speech disturbance. Excess manganese can interfere with the absorption of iron, and if excess continues, it may result in iron-deficiency anemia.

Nutritional and Laboratory Information
A high manganese exposure increases the need for vitamin C. If manganese overexposure or toxicity is suspected, additional laboratory measurements such as blood or hair manganese levels are recommended.

Literature:

Paige DM, editor; Manual of clinical nutrition. Nutrition Publ. Pleasantville, NJ 1983

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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 intoxicaton 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 (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:

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.

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LEAD (Pb) HIGH:
Occupational and environmental exposure are common causes of high urinary concentrations. A lead concentration above the baseline reference range and below the Orientation Range reflects a mild exposure; a concentration equal to or higher than the Orientation Range may reflect a chronic or acute exposure in need of detoxification treatment. Consult a physician knowledgeable in clinical metal toxicology.

Common Sources:
leaded gasoline, canned goods, lead paint, newsprint, tobacco smoke, air pollution, and contaminated water.

Biochemical basis of toxicity
Only 8 to 12% of the orally ingested lead is absorbed by the small intestine, but toxic effects are severe. Lead can react with sulfhydryl groups in enzymes, thereby inactivating important enzymes such as the aminolevulinic acid dehydratase (ALA) and ALA synthetase, leading to haematological manifestations. Lead reduces the body's ability to utilize calcium, magnesium, zinc, iron and other important nutrients. Lead is easily absorbed by children.

Clinical Signs:
The pathological effects of Pb have been recognized for centuries. Lead affects all physiological systems including renal, nervous, reproductive, endocrine, immune, and hemopoietic. Exposure to lead, either chronic or acute, presents a variety of signs, symptoms, and chemical evidence. The exposed person may be asymptomatic or symptomatic.
•Mild Symptoms include tiredness, lack of energy, constipation, slight abdominal pain and discomfort, anorexia, altered sleep, irritability, anemia, hair loss, pallor, and less frequently diarrhea and nausea. Formation and precipitation of lead sulphide may be evidenced as a blue-black ‘lead line’ near the gingival margin of the teeth.
•Severe symptoms include colic, reduction of muscle power, muscle tenderness, parethesia, and symptoms of neuropathy and encephalopathy.
•Frequent Symptoms in children as reported by the Center for Disease control are irritability, vomiting, abdominal pain, ataxia, anorexia, behavioural changes, speech disturbances, seizures, intercurrent fever, and dehydration. Other symptoms reported are ataxia and stupor.

Treatment:
Chelating agents such as EDTA, Dimercaprol (BAL) and penicillamine have been medically recommended. The use of oral DMSA was approved by the FDA for the detoxification of children. Micro Trace Minerals data evaluation indicates that DMPS, ZnDTPA and CaDTPA are highly effective chelating agents with a binding capacity that equals or is greater than BAL and penicillamine. This data also indicates that the combination treatment of EDTA + DMSA has a great lead binding capacity and significantly increases the lead excretion. Vitamin C and cysteine have a good lead binding capacity and may be used as supportive measures during chelation therapy.

Laboratory Analysis:
Two classes of persons must be considered:
1. the occupationally exposed
•A case of occupational lead overexposure is defined as an adult (15 years of age or older) with a BLL greater than or equal to 25 mcg/dL or 250mcg/L.
•Childhood lead poisoning is defined by the Centers for Disease Control and Prevention as a blood lead level of 10 ug/dL or above. Long-term effects may include slow development, reduced Intelligence Quotient (IQ), learning disabilities, hearing loss, reduced height and hyperactivity. Most lead intoxicated children do not have any symptoms. Appearing symptoms are often confused with other childhood illnesses. Very severe lead exposure (levels greater than 80 ug/dL) can cause coma, convulsions and even death.
2.low level chronic exposure
•blood levels may or may not be significant, depending on the immediate exposure.
•longterm exposed individuals typically show elevated hair lead.

Blood lead and urine concentrations have been reported to be about 10 to 20% higher in males than females, both in children and adults.

Literature:

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Lead-Based Paint Hazard Reduction and Financing Task Force. Putting the Pieces Together: Controlling Lead Hazards in the Nation's Housing. Washington, DC: US Dept of Housing and Urban Development; 1995
National Research Council. Measuring Lead Exposure in Infants, Children and Other Sensitive Populations. Washington, DC: National Academy Press; 1993
Med 1996; 150:609-614
Centers for Disease Control and Prevention. Blood Lead Proficiency Testing. Atlanta, GA: US Dept of Health and Human Services, Public Health Service; 1994

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