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491 College Street, Boulder, CO 80302-8713, USA
P.O.Box 4613, Boulder, CO 80306-4613, USA

Phone: +1 (720) 325-2652
Facsimile: +1 (720) 325-2653
http://www.tracemin.com
service@tracemin.com

MINERAL ANALYSIS — EDTA Urine

		Lab Number		5UE150000	
Doctor	Sample Doctor			Test Date	7/31/2014
Patient Name	Sample Patient	Sex	w	Age	52
Clinical Information	NaMgEDTA iv 3g				
Creatinine (g/l) *	0.31			Page	1/9

	Baseline URINE Norm	Chelator-specific orientation range	Test Value		
Essential Trace Elements (mcg/g Creatinine)					
Chromium	0.55 --- 4.83		3.12		
Cobalt	< 5.00		42.24	↑	
Copper	< 60.00		322.56	↑	
Iodine	< 719.00		249.33		
Manganese	< 4.50	35.00	9.13		
Molybdenum	9.70 --- 100.00		27.78		
Selenium	12.00 --- 90.00		78.30		
Vanadium	< 1.40		0.22		
Essential Macro- & TraceElements (mg/g Creatinine)					
Zinc	0.07 --- 7.00	19.50	5.66	↓	
Trace Elements in mcg/g Creatinine					
Boron	< 3,770.00		1,037.09		
Strontium	< 570.00		158.99		
Potentially Toxic Elements in mcg/g Creatinine					
Aluminum	< 40.00		48.92	↑	
Arsenic-total	< 15.00		261.22	↑	
Barium	< 8.22		1.36		
Beryllium	< 1.20		< DL		
Cadmium	< 0.80	1.30	0.43		
Cerium	< 2.70		0.02		
Cesium	< 11.00		5.33		

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					
Gallium	< 7.76		0.08		
Iridium	< 0.20		n.n.		
Lead	< 5.00	22.00	7.53		
Mercury	< 1.00	3.50	9.07	↑	
Nickel	< 3.00	14.80	7.01		
Palladium	< 1.40	1.80	< DL		
Platinum	< 0.60		< DL		
Rhodium	< 0.06		n.n.		
Silver	< 1.40		< DL		
Tantalum	< 0.60		n.n.		
Thallium	< 0.60		0.28		
Tin	< 5.00		2.98		
Titanium	< 13.00		1.86		
Uranium	< 0.06		< DL		
Zirconium	< 2.50		< DL		

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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.

Chelation treatment or provocation with complexing agents increase metal binding and urinary excretion. EDTA stimulates, even forces the binding and excretion of metals such as copper, lead and zinc. This report provides EDTA-specific orientation values, which were obtained following statistical observations.

Test values are compared to urine baseline reference ranges and EDTA-SPECIFIC ORIENTATION RANGES. When provoked with 2gr EDTA (CaEDTA or NaEDTA), 65% of the testpersons showed values equal to or lower than the EDTA-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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ALUMINIUM (Al) HIGH
This naturally-occurring element is commonly ingested with food, medicine and water. Since aluminum was considered virtually non-absorbable, it has been, and still is, freely used in a variety of food additives and over-the-counter drugs such as antacids, anti-diarrhea medication, and cosmetics. Aluminum finds its way into food through cooking acidic foods in aluminum ware, or storing it in aluminium foil and Alu-containers. Ayurvedic medicines and certain covering called Waraq, another source of silver in India also contain aluminum.

Biochemistry and Pathophysiology:
More than 98% of the oral aluminium passes through the gastrointestinal tract, some is absorbed into plasma where it is bound primarily with transferrin. Some of the plasma aluminium equilibrates with tissues and it deposited primarily in bone and nerve (esp. brain) tissue. Al can bind to DNA, resulting in abnormal neurofibrillary tangles in the brain. Al inhibits the enzyme, hexokinase. It is excreted almost exclusively in the urine.

Toxicity
In persons with abnormal kidney function, the urinary excretion ability is reduced and aluminum is deposited in bones and nerve tissue. Therefore, Al-toxicity is a major concern in dialysis patients with end-stage renal disease, and those who are treated chronically with aluminium-contaminated parenteral nutrition fluid. New research suggests that Al overexposure can cause neurological changes such as seen in Alzheimer's and Parkinson's disease, and dialysis dementia.

Chelation Information:
•Toxicity in dialysis patients.
Once the presence of Al-toxicity has been established, the patient may be treated with desferrioxamine or other chelating agents. In the case of dialysis, Desferrioxamine binds Al from tissue deposits and removes the bound metal by dialysis. After successful chelation treatment, serum Al-levels fall, usually well below the pre treatment levels. Reversal of symptoms has been noted in patients after treatment. Such therapy may need to be continued for years.
•High Al-excretion in patients with normal renal function
Chelation with desferrioxamine and other chelating agents (Studies Micro Trace Minerals 2004) increases urinary elimination. Compare pre and post urine level. If elimination in the pre urine is excessive, check blood levels to confirm, or rule out immediate and acute exposure. Hair tissue levels confirm or rule out longterm exposure and tissue storage.

Therapeutic Consideration:
•A high intake of Aluminum-containing food or drink results in increased urinary elimination, but is rarely indicative of toxicity.
•Vitamin B6 intake supports renal function.
•Inadequate Calcium intake or Ca-deficiency may accompany high aluminum exposure.

Literature:

Bertolf RL. Roman JM, Brown S. et al. Aluminum hydroxide-induced osteomalacia, encephalopathy and hyperaluminemia in CAPD: treatment with desferoxamine. Peritoneal Dial Bull 4:30-32, 1984
Wills MR and Savory J. Aluminum poisoning, dialysis encephalopathy, osteomalacia, and anaemia. Lancet 1:29:34, 1983

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ARSENIC (As) –
Environmental sources of arsenic exposure include food, water, soil, and air, esp. around arsenic-containing mineral ores. In industry, arsenic is a by-product of the smelting process for many metal ores such as lead, gold, zinc, cobalt, and nickel. Arsenic is used for purifying industrial gases (removal of sulfur), burning fossil fuels, 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.

Other potential sources of arsenic exposure are 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). Wine (grapes sprayed with arsenic-containing pesticides), or seafood (especially certain cold water and bottom-feeding finfish) and seaweed can be cause of dietary exposure.
• Smokers inhale small amounts of arsenic as a result of pesticide residue on tobacco leaves.
• Medicinals: Fowler's solution (potassium arsenite), antiparasitic drugs (carbasone), Donovan's solution, folk remedies ("Asiatic pill," kushtay, yellow root), kelp-containing health foods, some naturopathic remedies.

CHELATION INFORMATION:
If fish, seaweed or other potentially arsenic-rich food has been consumed within 3 days prior to chelation, and if the patient smoked prior to treatment, elevated arsenic levels may result.

Reference:
Agency for Toxic Substances and Disease Registry. 2006
Blaurock-Busch E. Antidote- Handbook of Chelation Therapy. MTM 2010
BUA (Bundesumweltamt) 2009

COBALT (Co) is part of the Vitamin B12 molecule. High levels may due to Vit.12 therapy. Excess cobalt can increase the toxic effect of selenium and suppress iron absorption. Cobalt is stored in the liver and the main excretion is via bile. SOURCES: Excessive environmental exposure as found near smelter emissions and inhalation of industrial cobalt dust causes contact dermatitis, cardiomyopathy, liver and kidney damage. Thyroid hypertrophy (goiter) has been linked to cobalt poisoning. THERAPEUTIC CONSIDERATION: a high protein or amino acid intake protects against industrial poisoning. Fatty acids stimulate the excretion via bile.

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:
EDTA stimulates some copper binding. 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):
EDTA has a low mercury binding ability. A level exceeding the baseline reference range and below the orientation range signals a mild exposure. A level greater than the Orientation Range should be viewed as a reflection of a burden that can be seen in nonsymptomatic and symptomatic patients. Consider a DMPS or DMSA challenge to evaluate the patient's mercury exposure.

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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MANGANESE (Mn):
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.

CHELATION INFORMATION:
A urine value higher than the Urine Baseline Reference Range and lower than the Orientation Range indicates a mild exposure. Urine levels exceeding the Orientation Range are indicative of a chronic or acute manganese exposure, and should be viewed in context with patient symptoms, history and lifestyle.

A baseline urine or blood test should be considered as urinary manganese may be increased in patients with biliary obstruction or cirrhosis.

SOURCES:
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 chronic manganese overexposure is suspected, additional laboratory measurements such as 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, hence a level greater than the reference range and lower than the 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

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LEAD (Pb) HIGH:
EDTA is a good lead chelator, and levels exceeding the baseline reference range is expected. If the urine concentration is greater than the orientation range, accupational and environmental exposure is considered the expected cause. Consult your physician for further treatment.

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

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.

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American Academy of Pediatrics, Committee on Environmental Health Lead poisoning: from screening to primary prevention. Pediatrics 1993; 92:176-183
Casey R, Wiley C, Rutstein R, Pinto-Martin J Prevalence of lead poisoning in an urban cohort of infants with high socioeconomic status. Clin Pediatr 1994; 33:480-484
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