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MINERAL ANALYSIS — DMSA Urine

		Lab Number		5UA180387	
Doctor	Wendy Myers FDN CHHC			Test Date	9/5/2016
Patient Name	Ben Greenfield	Sex	m	D.O.B.	12/20/1991
Clinical Information	DMSA oral 1625mg + Glycine 3.5g + Biosil 40 drops 24h				
Creatinine (g/l) *	0.420			Page	1/10

	Baseline URINE Norm	Chelator-specific orientation range	Test Value		
Essential Trace Elements (mcg/g Creatinine)					
Chromium	0.550 --- 4.830		3.894		
Cobalt	< 5.000		1.374		
Copper	1.450 --- 60.000		2.969		
Iron	2.200 --- 45.000		188.632	↑	
Manganese	< 4.500		24.963	↑	
Molybdenum	9.700 --- 100.000		2.681	↓	
Selenium	12.000 --- 90.000		24.889		
Vanadium	< 1.000		0.252		
Essential Macro- & TraceElements (mg/g Creatinine)					
Calcium	55.000 --- 245.000		98.852		
Magnesium	12.000 --- 150.000		112.067		
Zinc	0.060 --- 0.780		< DL		
Trace Elements (mcg/g Creatinine)					
Germanium	< 1.500		1.903	↑	
Lithium	< 175.000		315.561	↑	
Strontium	< 200.000		254.482	↑	
Tungsten	< 0.790		< DL		
Potentially Toxic Elements (mcg/g Creatinine)					
Aluminum	< 40.000		65.150	↑	
Antimony	< 1.000		0.401		
Arsenic-total	< 15.000		69.511	↑	

n.n. = not detected, < DL = below Detection Limit
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	Baseline URINE Norm	Chelator-specific orientation range	Test Value		
Potentially Toxic Elements (mcg/g Creatinine)					
Barium	< 5.700		19.029	↑	
Beryllium	< 1.200		< DL		
Bismuth	< 0.150		< DL		
Cadmium	< 0.800		< DL		
Cesium	< 11.000		7.426		
Gallium	< 7.760		0.817		
Lead	< 5.000	10.000	< DL		
Mercury	< 1.000	2.800	2.436		
Nickel	< 3.000	5.000	7.570	↑	
Palladium	< 1.400		0.976		
Platinum	< 0.600		n.n.		
Silver	< 1.400		< DL		
Thallium	< 0.600		0.055		
Tin	< 2.000		1.805		
Titanium	< 13.000		< DL		
Uranium	< 0.060		0.041		
Zirconium	< 2.500		< 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 < 0.3 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: 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.

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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ALUMINUM (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 aluminum foil and aluminum 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 aluminum passes through the gastrointestinal tract, some is absorbed into plasma where it is bound primarily with transferrin. Some of the plasma aluminum equilibrates with tissues and is 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 aluminum-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.

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. 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.

BARIUM (Ba):
Barium is not readily absorbed; however intestinal dysfunctions support the uptake. Barium is then distributed in very low concentration in soft tissues. It appears to inhibit the calcium absorption and has properties that are similar to lead and cadmium. Barium X-ray techniques can increase tissue levels.
SOURCES: Drinking water. The EPA allows a maximum level of 1PPM.
THERAPEUTIC CONSIDERATION: Digestive support to reduce intestinal uptake. Zinc and antioxidants, including selenium are recommeded to normalize barium levels.

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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

GERMANIUM (Ge) is not routinely found in urine, and the clinical significance of high levels has not been established. Some forms of germanium may accumulate in the body, causing mild skin irritation, digestive disturbance and nephropathy. There is no evidence that germanium is essential in mammalian nutrition, but excess germanium intake has caused kidney failure in humans and 24-hr urinary levels may be most reflective of a toxic overload. SOURCE: found in minute amounts in most foods, esp garlic, camphor, aloe vera and in water, however concentrations vary greatly due to location. THERAPEUTIC CONSIDERATION: support kidney function (vitamin B6 etc)

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 100 mcg/g creatinine 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 creatinine 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 diuretics, fungicides, fluorescent lamps, cosmetics, hair dyes, amalgams in dentistry, contaminated seafood, and petroleum products. Vaccines such as tetanus toxoid contain thimerosal, 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.
Clarkson TW. Mercury poisoning. In Brown SS, editor: Clinical chemistry and chemical toxicology of metals. Amsterdam, 1977. Elsevier/No Holland Biomed Press.
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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LITHIUM (Li) is found in variable amounts in foods; esp grains and vegetables. Drinking water can provide significant amounts of the element but the content varies significantly due to geography. Traces of lithium were detected in human organs and fetal tissues already in the late 19th century, leading to early suggestions as to possible specific functions in the organism. Other sources are batteries or inhaling fumes from printing and copying machine.
The biochemical mechanisms of action of lithium appear to be multifactorial and are intercorrelated with the functions of several enzymes, hormones and vitamins, as well as with growth and transforming factors

MEDICAL USES Lithium is commonly used in the treatment of depressive and bipolar affective disorders. Because it has a comparatively narrow therapeutic index, lithium intoxication is a frequent complication of chronic lithium therapy.Lithium is available only for oral administration. It is almost completely absorbed from the GI tract. Peak levels occur 2-4 hours postingestion, although absorption can be much slower in massive overdose or with ingestion of sustained-release preparations. Hypokalemia is a potential adverse effect of lithium therapy.

EXCRETION Lithium clearance is predominantly through the kidneys and a mild increase in urine baseline levels most likely reflects nutrititional exposuse. Because it is minimally protein bound, lithium is freely filtered at a rate that is dependent upon the glomerular filtration rate (GFR). Consequently, dosing must be adjusted based on renal function. Individuals with chronic renal insufficiency must be closely monitored if placed on lithium therapy.

TOXICITY symptoms include tremor, nausea and vomiting, weakness and seizure. Check Serum levels.

LITERATURE.

James G Linakis, PhD, MD, Associate Professor of Emergency Medicine and Pediatrics, Brown Medical School; Associate Director, Department of Pediatric Emergency Medicine, Rhode Island Hospital, Hasbro Children's Hospital; Toxicity, Lithium: Treatment & Medication. E-medicine Jan2007 Schrauzer G.N. Lithium: Occurrence, Dietary Intakes, Nutritional Essentiality. Journal of the American College of Nutrition, Vol. 21, No. 1, 14-21 (2002)

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MANGANESE (Mn) HIGH:
High urinary concentration may correspond with an excessive manganese intake. Studies by Micro Trace Minerals / Trace Minerals International 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. Dopamine, 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 350 mcg/L. Manganese-rich water promotes bacterial growth.

Neurotoxicity of Manganese:
Manganese can be neurotoxic. Symptoms of intoxication (especially 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.

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.

STRONTIUM (Sr) possesses physiological and chemical properties similar to calcium. Strontium is poorly absorbed by humans, and the intestinal uptake lies between 5-25%. Of that, about 99% is found in bone and teeth. People living in areas where high levels are found in the water supply, show higher levels. The daily intake varies considerably from 1 mg/day to 4.7 mg/day, according to geography. Strontium can interfere with the calcium metabolism, leading to bone disorders, incl. rickets.

THERAPEUTIC CONSIDERATION: Strontium may compete with the calcium absorption and storage in bone and teeth. Algae and fibrous cellulose reduce strontium uptake.

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ZINC (Zn):
Zinc is a cofactor for many metalloenzymes, incl. those involving RNA and DNA synthesis. Low urinary levels reflect the dietary intake and absorption ability. To confirm zinc deficiency, evaluate blood and hair tissue. Zinc is necessary for growth, healthy cell division and insulin production. Pregnant women, cancer and burn patients are at high risk for zinc deficiency, causing fatigue, poor growth, menstrual problem and delayed sexual maturity. Deficiency causes are malnutrition and malabsorption. The zinc absorption occurs mainly in the small intestine. The RDA is 3-10 mg/day, depending on age and physiology. In severe zinc deficiency states, a much higher intake is warranted with proper supervision.
SOURCE: Yeast, meat, fish, legumes, and eggs. The zinc in whole grains has a low bio-availability. Phytates block zinc absorption and a high intake of uncooked grains or unleavened bread can cause zinc deficiency.
THERAPEUTIC CONSIDERATION: A high exposure to toxic metals interferes with zinc absorption and increases the need for zinc and vitamin B6. Before chelation, evaluate and optimize the zinc status.

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