URINE TOXIC METALS

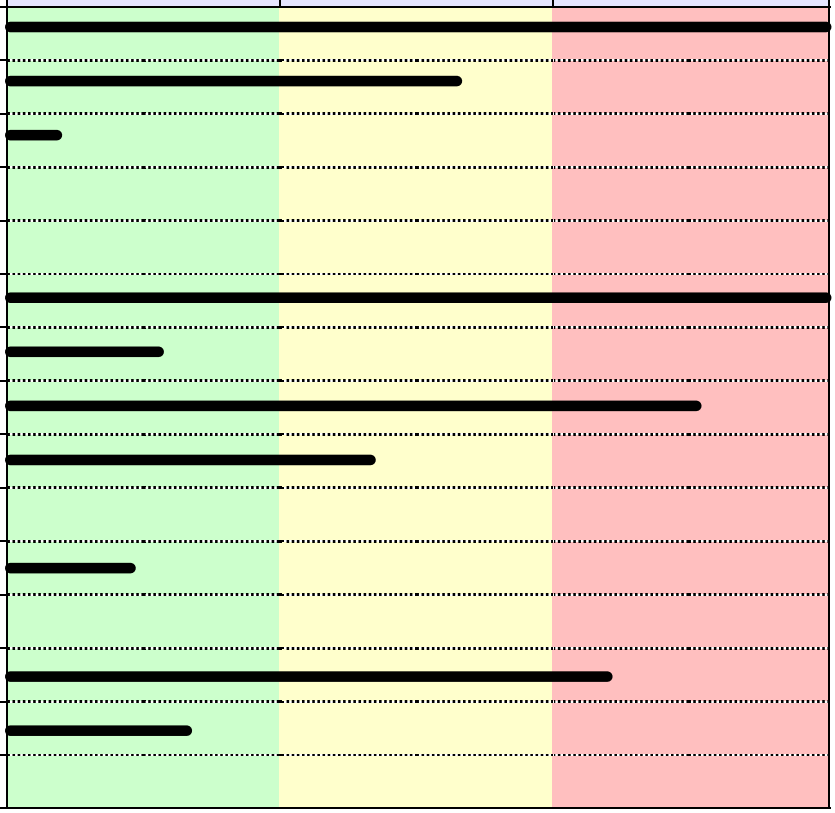
DOCTOR'S DATA INC

LAB#: U081212-0468-1
PATIENT: Jacqueline Heckenlivey
SEX: Female
AGE: 10

CLIENT#: 27080
DOCTOR: Amy Yasko, ND
Holistic Health
279 Walkers Mills Rd
Bethel, ME 04217

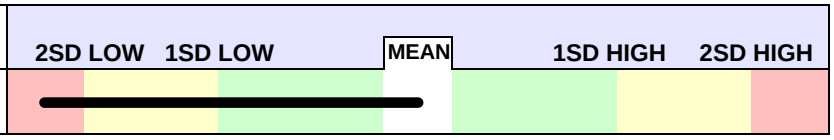
POTENTIALLY TOXIC METALS

METALS	RESULT µg/g CREAT	REFERENCE RANGE
Aluminum	**920**	< 60
Antimony	**3.5**	< 1.5
Arsenic	**23**	< 130
Beryllium	**< dl**	< 0.6
Bismuth	**< dl**	< 20
Cadmium	**6.3**	< 2
Lead	**2.8**	< 5
Mercury	**23**	< 5
Nickel	**36**	< 15
Platinum	**< dl**	< 1
Thallium	**0.5**	< 1.1
Thorium	**< dl**	< 0.5
Tin	**91**	< 15
Tungsten	**1**	< 1.5
Uranium	**< dl**	< 0.2



CREATININE

	RESULT mg/dL	REFERENCE RANGE
Creatinine	**10**	25- 180



SPECIMEN DATA

Comments: **1023 Results checked.**
Date Collected: **12/9/2008** Method: **ICP-MS** Collection Period: **Random**
Date Received: **12/12/2008** <dl: **less than detection limit** Volume:
Date Completed: **12/17/2008** Provoking Agent: Provocation:

Toxic metals are reported as µg/g creatinine to account for urine dilution variations. **Reference ranges are representative of a healthy population under non-challenge or non-provoked conditions.** No safe reference levels for toxic metals have been established.

V10.00

INTRODUCTION

This analysis of urinary elements was performed by ICP-Mass Spectroscopy following acid digestion of the specimen. Urine element analysis is intended primarily for: diagnostic assessment of toxic element status, monitoring detoxification therapy, and identifying or quantifying renal wasting conditions. It is difficult and problematic to use urinary elements analysis to assess nutritional status or adequacy for essential elements. Blood, cell, and other elemental assimilation and retention parameters are better indicators of nutritional status.

1) 24 Hour Collections
"Essential and other" elements are reported as mg/24 h; mg element/urine volume (L) is equivalent to ppm. "Potentially Toxic Elements" are reported as µg/24 h; µg element/urine volume (L) is equivalent to ppb.

2) Timed Samples (< 24 hour collections)
All "Potentially Toxic Elements" are reported as µg/g creatinine; all other elements are reported as µg/mg creatinine. Normalization per creatinine reduces the potentially great margin of error which can be introduced by variation in the sample volume. It should be noted, however, that creatinine excretion can vary significantly within an individual over the course of a day.

If one intends to utilize urinary elements analysis to assess nutritional status or renal wasting of essential elements, it is recommended that unprovoked urine samples be collected for a complete 24 hour period. For provocation (challenge) tests for potentially toxic elements, shorter timed collections can be utilized, based upon the pharmacokinetics of the specific chelating agent. When using EDTA, DMPS or DMSA, urine collections up to 12 hours are sufficient to recover greater than 90% of the mobilized metals. Specifically, we recommend collection times of: 9 - 12 hours post intravenous EDTA, 6 hours post intravenous or oral DMPS and, 6 hours post oral bolus administration of DMSA. What ever collection time is selected by the physician, it is important to maintain consistency for subsequent testing for a given patient.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. Because renal excretion is a minor route of excretion for some elements, (Cu, Fe, Mn Zn), urinary excretion may not influence or reflect body stores. Also, renal excretion for many elements reflects homeostasis and the loss of quantities that may be at higher dietary levels than is needed temporarily. For these reasons, descriptive texts are provided for specific elements when deviations are clinically significant. For potentially toxic elements, a descriptive text is provided whenever levels are measured to be higher than expected. If no descriptive texts follow this introduction, then all essential element levels are within acceptable range and all potentially toxic elements are within expected limits.

For essential elements, the mean and the reference ranges apply to human urine under non-challenge, non-provocation conditions. Detoxification therapies can cause significant deviations in essential element content of urine. For potentially toxic elements, the expected range also applies to conditions of non-challenge or non-provocation. Diagnostic or therapeutic administration of detoxifying agents frequently raise the urinary levels content of potentially

toxic elements. Descriptive texts appear in this report on the basis of measured results and correspond to non-challenge, non-provocation conditions.

CAUTION: Even the most sensitive instruments have some detection limit below which a measurement cannot be made reliably. Any value below the method detection limit is simply reported as "< dl." If an individual excretes an abnormally high volume of urine, urinary components are likely to be extremely dilute. It is possible for an individual to excrete a relatively large amount of an element per day that is so diluted by the large urine volume that the value measured is near the dl. This cannot automatically be assumed to be within the reference range.

ALUMINUM HIGH

This individual's urine aluminum exceeds 5X the expected level. Urine is the primary mode of excretion for absorbed aluminum, and this high urine level indicates ingestion or absorption of relatively high amounts and perhaps an excessive body burden.

Common sources of bioavailable aluminum include: aluminum cookware, flatware and especially coffee pots; aluminum hydroxide anti-acid formulations; some types of cosmetics, especially deodorants; and some herbs or herbal products. Aluminum cookware is particularly of concern if acid foods are cooked such as tomato paste (contains salicylates). In cosmetics and deodorants, aluminum chloride may be present as an astringent. In water purification, alum (sodium aluminum sulfate) may be used to coagulate dispersed solids and improve water clarity. Alumina or Al2O3 is very stable chemically and not bioavailable. Silica limits the solubility of aluminum and aluminum silicate is not very bioavailable. Clays, bentonite for example, contain aluminum that has poor bio-availability. Aluminum food containers are manufactured with polymer or plastic coatings that prevent direct food-aluminum contact provided such coatings are not damaged.

In the GI tract, phosphates react with aluminum ions forming insoluble aluminum phosphates. If this phosphate-blocking were 100% efficient, then virtually no aluminum would be absorbed. Evidently, this phosphate-forming process is incomplete because body tissue levels (such as hair) usually contain measurable amounts of aluminum. In the body aluminum follows a path of increasing phosphate concentration: plasma, cytosol, cell nucleus. Once in the nucleus, it adversely affects protein formation.Long-lived cells such as neurons are susceptible to long-term accumulation. Al is considered neurotoxic and is implicated as a stabilizing agent (via aluminum phosphate bonds) in neurofibrillary tangles in Alzheimer's disease (Science, 267, pp 793-4, 1995). In cells, Al inhibits the citric acid cycle enzyme isocitrate dehydrogenase which catalyzes formation of alpha-ketoglutaric acid. An effect of this inhibition could be hyperammonemia. Al also inhibits hexokinase, a magnesiumdependent phosphorylating enzyme. Without intervention, aluminum accumulates continually in the body with the highest concentration occurring at old age or death.

Fatigue, hypophosphatemia, increased prothrombin time, and porphyria are consistent with aluminum excess. A hair element test can be used to corroborate increased body burden of aluminum. An oral provocation with the amino acid glycine, 80 mg/Kg body weight (in divided doses) 24 hours before a diagnostic EDTA chelation with subsequent urine collection can be done to confirm aluminum excess.(Eliminate food/beverage sources of Al during this procedure.)

BIBLIOGRAPHY FOR ALUMINUM
1. Ganrot P.O. ”Metabolism and Possible Health Effects of Aluminum”, Environ. Health Perspectives, 65, pp. 363-441 1986.
2. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publ, Chelsea MI pp 16-20 1986.
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4. Fulton B. and E.H. Jeffery, ”Absorption and Retention from Drinking Water”, Fund. & Appl. Toxicology 14 pp 788-96 1980.
5. Tsalev D.L. et al. Atomic Absorption Spectrometry in Occupational and Environmental Health Practice vol 1, CRC Press, Boca Raton FL, pp 81-84, 1983.

ANTIMONY HIGH

This individual’s urine antimony is higher than expected, but associated symptoms and toxic effects may not be presented. This is because antimony (chemical symbol Sb) has two valences: Sb+3 and Sb+5. Sb+3 is the more toxic but is mostly excreted in feces. Sb+5, less toxic, binds less well to body tissues and is excreted mostly in urine.

Antimony can be assimilated by inhalation of Sb salt or oxide dust, ingested with (contaminated) foods or fluids, or absorbed transdermally. Inhalation may occur in industrial areas where smelting or alloying is done (usually with copper, silver, lead, tin). Sb is present in tobacco at about 0.01% by weight; about 20% of this is typically inhaled by cigarette smoking (Carson et al., Toxicology and Biological Monitoring of Metals in Humans, Lewis Pub. p. 21, 1987). Antimony compounds are used for fireproofing textiles and plastics, and this element may be found in battery electrodes, ceramics and pigments. Antimony can be absorbed with the handling of gun powder or the frequent use of firearms. Recent studies indicate high levels of antimony in sheepskin bedding produced in New Zealand.

Symptoms of mild Sb contamination may be insidious and multi- ple including: fatigue, muscle weakness, myopathy, and metallic taste. Chlorides and oxides of both valences of Sb can be mutagenic and may affect leukocyte function. Sb can bond to sulfhydryl (-SH) sites on enzymes and interfere with cellular metabolism. Acute symptoms of Sb contamination include: respiratory tissue irritation and pneumoconiosis with (chronic) inhalation of Sb dusts, RBC hemolysis with inhalation of stibine (SbH3) vapor, and GI distress if orally ingested. Skin exposure can produce ”antimony spots” or rashes which resemble chicken pox. Certain molds can produce the highlyneurotoxic stibine gas from antimony; stibine inhibits acetylcholinestelase activity.

A hair element analysis may be used as a corroborative test for increased body burden of antimony. Fecal metal analysis can be used to confirm exposure/retention of toxic Sb+3.Antimony may be elevated in urine following administration of DMPS or DMSA.

BIBLIOGRAPHY FOR ANTIMONY

1. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, Chelsea MI, pp 21-26, 1987.
2. Tsalev D.L. and Z.K. Zaprianov. Atomic Absorption Spectrometry in Occupational and Environmental Health Practice. CRC Press, Boca Raton FL, pp 85-87, 1983.
3. Scriver C.A. et al The Metabolic Basis of Inherited Disease, 6th ed. McGraw-Hill, New York NY, pp 2349-50 on PFK deficiency. 1989.

CADMIUM HIGH

This individual's urine cadmium level equals or exceeds twice the maximum expected level. This element is insidiously toxic with chronic accumulations affecting renal function, pulmonary and cardiovascular tissues, bone, and the peripheral nervous system. Without intervention, the biological half-life of Cd in humans exceeds 20 years (Harrison's Principles of Internal Medicine, 13th ed, pp 2463-64).

Chronic manifestations associated with this degree of cadmium excess include: hypertension, weight loss, microcytic-hypochromic anemia, lymphocytosis, proteinuria with wasting of beta2 microglobulin,emphysema and pulmonary fibrosis (if inhalation was a route of contamination), atherosclerosis,steomalacia and lumbar pain, and peripheral neuropathy. Acute inhalation of Cd dusts, fumes or soluble salts may produce cough, pneumonitis and fatigue. Manifestations of Cd toxicity may be lessened or delayed by an individual's protective and detoxication capacities. Zinc and vitamin E are protective; metallothionein and glutathione bind Cd and help detoxify it initially.

Smoking can be a source for as much as 0.1 mcg Cd per cigarette (HEW Pub. No. NIOSH 76-192, US Govt. Printing Ofc., 1976). Some medical authorities consider Cd to be a carcinogen for lung cancer (Harrison's Principles, 13th ed, op. cit. pp 2463). Other occupational or environmental sources include:mining and smelting activities, pigments and paints, electroplating, electroplated parts (e.g., nuts and bolts), batteries (Ni-Cd), plastics and synthetic rubber, photographic and engraving processes, old drums from some copy machines, photoconductors and photovoltaic cells, and some alloys used in soldering and brazing.

Depending upon body burden and upon type, duration and dosage of detoxifying agents, elevated urine cadmium may occur after administration of: EDTA, DMPS, DMSA or D-penicillamine. A confirming test for Cd excess is elemental hair analysis, barring exogenous contamination. Blood Cd measurement may not be indicative (Harrison's Principles of Internal Medicine, 13th ed., pp 2463).

BIBLIOGRAPHY FOR CADMIUM

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2. Nat. Inst. Occup. Safety and Health (NIOSH), "Criteria for a Recommended Standard... Occupational Exposure to Cadmium", H.E.W. Publication No. (NIOSH) 76-192, 1976.
3. Carson, B.L. et al, Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers,Chelsea MI, pp 51-58, 1987.
4. Werbach M.R. Nutritional Influences on Illness 2nd ed., Third Line Press, Tarzana, CA, pp 102, 348-49, 643, 679, 1993.
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Clinical Chem. 40 no 7, pp 1391-94, 1994.
6. Whittemere A.S. et al. ”Urinary Cadmium and Blood Pressure: Results from the NHANES II Survey” Environ. Health Persp. 91, pp 133-40, 1991.

MERCURY HIGH

This individual’s urine mercury equals or exceeds twice the maximum expected level. Presentation of symptoms associated with excessive mercury can depend on many factors: the chemical form of absorbed Hg and its transport in body tissues, presence of other synergistic toxics (Pb, Cd have such effects),presence of disease that depletes or inactivates lymphocytes or is immunosuppressive, organ levels of xenobiotic chemicals and sulfhydryl-bearing metabolites (e.g. glutathione), and the concentration of protective nutrients, (e.g. zinc, selenium, vitamin E).

Early signs of mercury contamination include: decreased senses of touch, hearing, vision and taste, metallic taste in mouth, fatigue or lack of physical endurance, and increased salivation. Symptoms may progress with moderate or chronic exposure to include: anorexia, numbness and paresthesias, headaches, hypertension, irritability and excitability, and immune suppression, possibly immune dysregulation. Advanced disease processes from mercury toxicity include: tremors and incoordination, anemia, psychoses, manic behaviors, possibly autoimmune disorders, renal dysfunction or failure.

Mercury is commonly used in: dental amalgams, explosive detonators; in pure liquid form for thermometers, barometers, and laboratory equipment; batteries and electrodes (”calomel”); and in fungicides and pesticides. The fungicide/pesticide use of mercury has declined due to environmental concerns, but mercury residues persist from past use.

Methylmercury, the common, poisonous form, occurs by methylation in aquatic biota or sediments (both freshwater and ocean sediments). Methylmercury accumulates in aquatic animals and fish and is concentrated up the food chain reaching high concentrations in large fish and predatory birds. Except for fish, the human intake of dietary mercury is negligible unless the food is contaminated with one of the previously listed forms/sources. A daily diet of fish can cause 1 to 10 micrograms of mercury/day to be ingested, with about three-quarters of this (typically) as methylmercury.

Depending upon body burden and upon type, duration and dosage of detoxifying agents, elevated urine mercury may occur after administration of: DMPS, DMSA, D-penicillamine, or EDTA. Blood and especially blood cell analyses are only useful for diagnosing very recent or ongoing organic (methyl) mercury exposure.

BIBLIOGRAPHY FOR MERCURY

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2. World Health Organization: ”Methylmercury” Environ. Health Criteria 101 (1990); ”Inorganic Mercury” Environ. Health Criteria 118 (1991) WHO, Geneva,

Switzerland.
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NICKEL HIGH

This individual's urine nickel is elevated which may or may not be of significance. Urinary excretion of nickel bound to cysteinyl or thiol compounds (such as glutathione) or to amino acids (histidine, aspartic acid, arginine) is the predominant mode of excretion. With the exception of specific occupational exposures, most absorbed nickel comes from food or drink, and intakes can vary by factors exceeding 100 depending upon geographical location, food type, and water supply. Depending upon chemical form and physiological factors, from 1 to 10% of dietary nickel may be absorbed from the gastrointestinal tract into the blood. Urine reflects recent exposure to nickel and may vary widely in nickel content from day to day due to the above factors.

Sources of nickel are numerous and include the following.

- Cigarettes (2 to 6 mcg Ni per average cigarette)
- Diesel exhaust (particulates may contain up to 10 mg/gram)
- Foods, especially: cocoa, chocolate, soya products, nuts, and hydrogenated oils
- Nickel-cadmium batteries
- Nonprecious, semiprecious dental materials
- Nickel-containing prostheses
- Electroplating, plated objects, costume jewelry
- Pigments (usually for ceramics or glass)
- Catalyst materials (for hydrogenation processes in the food, petroleum and petrochemical industries)
- Arc welding
- Nickel refining and metallurgical processes

Most clinically observed nickel contaminations are manifested as dermatoses - contact dermatitis and atopic dermatitis. However, Ni hypersensitizes the immune system causing hyperallergenic responses to many different substances. Because nickel can displace zinc from binding sites on enzymes, it can have inhibiting or activating effects on such enzymes. Nickel sensitivity is observed to be three to five times more frequent in women than in men.

Other laboratory tests or clinical findings that would be indicative of nickel excess are; hair element analysis, presentation of multiple allergic sensitivities, dermatitis, positive patch test for "Ni allergy", proteinuria, hyperaminoaciduria (by 24-hour urine amino acid analysis). Detoxification treatments with administration of EDTA or sulfhydryl agents (DMPS, DMSA, D-penicillamine) may increase urine nickel levels depending upon: body burden and mobility in tissues, duration of

treatment, dosage and other factors.

BIBLIOGRAPHY FOR NICKEL
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2. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Publishers, Chelsea MI, pp 162-67, 1986.
3. Nielsen F.H. in Modern Nutrition in Health and Disease ed. by Shils et al, Lea & Febiger, Philadelphia, PA, pp 279-81, 1994.
4. Medical and Biological Effects of Environmental Pollutants: Nickel, Nat. Acad. Sci, Washington DC,1975.
5. Ambient Water Quality Criteria for Nickel, US EPA NTIS, Springfield, VA. Publ No. PB81-117715,1980.

TIN HIGH

The tin level in this individual's urine exceeds 5X the maximum expected level. Because urine accounts for at least 80% of ingested and absorbed tin, this is a significant finding. Although most forms of tin are considered nontoxic or only mildly toxic, a few forms, the organic ones, can be quite toxic and cause degeneration of meylin. Therefore, it is advisable to investigate the source of the tin and prevent further exposure for this individual.

Food and drink usually provide small daily intakes of (non- toxic) tin, with amounts depending upon type of food, packaging, quality of drinking water and water piping materials. Total daily intake is expected to vary from about 0.1 to 15 milligrams. Tin is present in many metal alloys and solders; bronze, brass and pewter contain the element. Dyes, pigments and bleaching agents often contain tin. Anticorrosion plating of steel and electrical components may also use tin. "Tin cans" are tin-plated steel with a thin outer oxide layer allowing the surface to be shiny but inert. Modern food-containing cans usually have polymer coatings that prevent food-metal contact. Some toothpastes contain stannous fluoride, a soluble fluoride source for strengthening tooth enamel. Organic tins, the usually toxic forms, are: biocides (triphenyltin and alkyltins) used against rodents, fungi, insects and mites; curing agents for rubbers and silicones (dialkyltin); and methyltin formed bacteriologically (similar to methylmercury).

Early signs of chronic organic tin excess can be: reduced sense of smell, headaches, fatigue and muscle aches, ataxia and vertigo. Hyperglycemia and glucosuria are reported. Also for organic tin exposures there can be irritation of contacted tissues (eyes, skin, bronchial tubes, or GI tract). Later, immune dysfunction may occur with lowered lymphocytes and leukocytes. Erythrocyte hemolysis and mild anemia can occur. Other tin-related lesions may occur in bile ducts (inflammation, congestion), kidneys (dystrophic changes of tubular epithelium in renal cortex, nephrosis), testes and ovaries (histological tissue changes).

Inorganic tin excess may provoke ataxia and muscle weakness and may also feature irritation of contacted tissues (skin, respiratory tract, GI tract) and may affect liver and kidney functions. Tin can impair liver function by reducing the P-450 mixedfunction oxidase enzyme system thereby hampering enzymatic detoxication.

A hair element analysis may be used to corroborate tin excess. Administration of EDTA or with dithiol chelators (DMSA, DMPS) are expected to cause some increase in urinary tin depending upon body burden and treatment parameters.

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1. Winship K.A. ”Toxicity of Tin and Its Compounds”, Adverse Drug Reactions and Acute Poisoning Reviews, 7 no.1, pp 19-38, 1988.
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