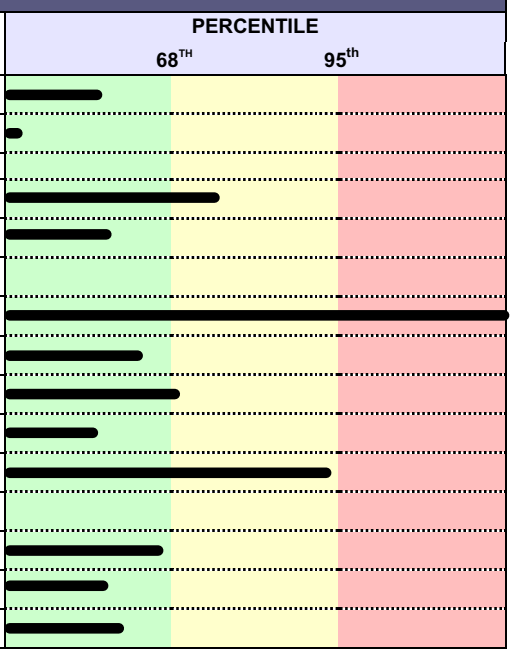
LAB #: F000000-0000-0
PATIENT: Sample Patient
ID: PATIENT-S-00003
SEX: Male
AGE: 7

CLIENT #: 12345
DOCTOR:
Doctor's Data, Inc.
3755 Illinois Ave.
St. Charles, IL 60174

Toxic Metals; Feces

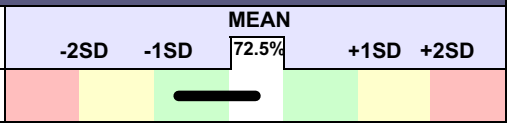
TOXIC METALS

		RESULT mg/kg Dry Wt	REFERENCE INTERVAL
Mercury	(Hg)	**0.031**	**<.05** w/o amalgams*
Mercury	(Hg)	**0.031**	**<0.5** with amalgams*
Antimony	(Sb)	**0.100**	< 0.080
Arsenic	(As)	**0.20**	< 0.30
Beryllium	(Be)	**< dl**	< 0.009
Bismuth	(Bi)	**229.8**	< 0.050
Cadmium	(Cd)	**0.41**	< 0.50
Copper	(Cu)	**63**	< 60
Lead	(Pb)	**0.27**	< 0.50
Nickel	(Ni)	**11.8**	< 8.0
Platinum	(Pt)	**< dl**	< 0.003
Thallium	(Tl)	**0.019**	< 0.020
Tungsten	(W)	**0.054**	< 0.090
Uranium	(U)	**0.085**	< 0.120



WATER CONTENT

	RESULT % H_2O	REFERENCE INTERVAL
% Water Content	**67.6**	60 - 85%



INFORMATION

Analysis of elements in feces provides a comprehensive evaluation of environmental exposure, accumulation and endogenous detoxification of potentially toxic metals. For several toxic elements such as mercury, cadmium, lead, antimony and uranium, biliary excretion of metals into feces is the primary natural route of elimination from the body. Studies performed at DDI demonstrate that the fecal mercury content and number of amalgam surfaces are highly correlated, as is the case for post-DMPS urine mercury levels and amalgam surface area.

Results are reported as mg/kg dry weight of feces to eliminate the influence of variability in water content of fecal specimens. The reference values that appear in this report have been derived from both published data and in-house studies at DDI. *Due to exposure to mercury in the oral cavity, people with dental amalgams typically have a considerably higher level of mercury in the feces than individuals without dental amalgams; therefore, two reference ranges have been established for mercury.

To provide guidance in interpretation of results, patient values are plotted graphically with respect to percentile distribution of the population base. Since this test reflects both biliary excretion and exposure (metals to which the patient is exposed may not be absorbed), it may not correlate with overt clinical effects. Further testing can assist in determining whether the metals are from endogenous (biliary excretion) or exogenous (oral exposure) sources.

1. Bjorkman, L, Sandborgh-Englund, G, and Ekstand, J,. Mercury in Saliva and Feces after Removal of Amalgam Fillings. Toxicology & Applied Pharmacology 144: 156-162 (1997)
2. Zalups, R, Progressive Losses of Renal Mass and the Renal and Hepatic Disposition of Administered Inorganic Mercury. Toxicology & Applied Pharmacology 130: 121-131 (1995)
3. Adamsson, E., Piscator, M., and Nogawa, K., Pulmonary and Gastrointestinal Exposure to Cadmium Oxide Dust in a Battery Factory. Environmental Health Perspectives, 28: 219-222 (1979)
4. Smith,J., et al., The Kinetics of Intravenously Administered Methyl Mercury in Man. Toxicology & Applied Pharmacology 128:251-256 (1994)
5. Bass, D., et al., "Measurement of Mercury in Feces", Poster presentation 1999 AACC

SPECIMEN DATA

Comments:

Date Collected: 5/16/2014
Date Received: 5/17/2014
Date Completed: 5/19/2014

Provocation:
Detoxification Agent:
Dosage:

Dental Amalgams: not indicated
Quantity:
Methodology: ICP-MS

ARSENIC HIGH

Fecal Arsenic (As) can be used as an estimate of exposure to the element. Inorganic As accumulates in hair, nails, skin, thyroid gland, bone and the gastrointestinal tract. Organic As is rapidly and mainly excreted in the urine and a smaller percentage in the feces.

As can cause malaise, muscle weakness, vomiting, diarrhea, dermatitis, and skin cancer. Long-term exposure may affect the peripheral nervous, cardiovascular and hematopoietic systems. As is a major biological antagonist to selenium. Arsenic, along with antimony is often found in fecal specimens from autistic children.

Common sources of As are insecticides (calcium and lead arsenate), fungicides (orchards, vineyards), well water, smog, shellfish (arsenobetaine), and industrial exposure, particularly in the manufacture of electronic components (gallium arsenide), wood preservatives (pressure treated wood/sawdust).

As burden can be confirmed by urine elements analysis. Comparison of urine As levels pre and post provocation (DMPS, DMSA, D-penicillamine) permit differentiation between recent uptake and retention in the body.

BERYLLIUM HIGH

Fecal Beryllium (Be) is an excellent measure of oral exposure to the element. Be is poorly absorbed in the gastrointestinal tract because of the alkalinity (forms precipitate) but is readily absorbed by the skin and lungs. Inhalation is the primary route of exposure to Be and chronic uptake results in dyspnea, cough and pulmonary distress (Am. Rev. Respir. Dis.;173:464-473, 1988). Be can be toxic to humans and animals. Be is a biological antagonist of magnesium. Be has a long-term effect of inducing abnormal activity in T lymphocytes, causing immune dysregulation and hypersensitivity reactions. In animals, Be has been shown to induce rickets and to damage liver, kidney, lungs, and skin.

Possible sources of Be are: electronic components, metal alloys used in aircraft and aerospace applications (especially aluminum-copper-beryllium alloys), bearing sleeves, optical lens coatings, and some phosphors in fluorescent lights. Tobacco contains Be, and smoking immediately increases the Be levels in the blood and urine.

Be is slowly excreted in urine and may be found elevated many months after high level exposure. To date, there are no known effective chelators for Be. A confirmatory test for Be exposure is the Be lymphocyte proliferation assay. As listing of laboratories that perform the test can be obtained form the Medical Department at Brush Wellman, Inc., 17876 St. Clair Ave., Cleveland, OH 44110-2697.

Beryllium has been detected in hair, but documentation correlating exposure, tissue levels and hair levels is lacking.

BISMUTH HIGH

Bi is a non-essential element of relatively low toxicity. However, excessive intake of insoluble, inorganic Bi containing compounds can cause nephrotoxicity and encephalopathy. Absorption is dependent upon solubility of the Bi compound, with insoluble Bi excreted in the feces while soluble forms are excreted in the urine. Sources of Bi include: cosmetics (lipstick), Bi containing medications such as rantidine Bi-citrate, antacids (Pepto Bismol), pigments used in colored glass and ceramics, dental cement, and dry cell battery electrodes. Several organometallic Bi compounds are used for bactericidal and fungicidal applications.

Symptoms of moderate Bi toxicity include: constipation or bowel irregularity, foul breath, blue/black gum line, and malaise. High levels of Bi accumulation can result in nephrotoxicity (nephrosis, proteinurea) and neurotoxicity (tremor, memory loss, monoclonic jerks, dysarthria, dementia).

Urine elements analysis can be used to corroborate Bi absorption for a period of days or a few weeks after the exposure. Dithiol chelating/complexing agents (DMPS, DMSA) markedly reduced Bi levels in liver and kidneys, and increased Bi in urine in animal studies (J. Lab. Clin. Med.; 119:529-537,1992).

CADMIUM HIGH

Approximately 90% of ingested (Cadmium) Cd is excreted through the feces and therefore fecal measurements of Cd can be used to estimate Cd ingestion and uptake.

Cd adversely affects the kidneys, lungs, testes, arterial walls, and bones and interferes with many enzymatic reactions. Chronic Cd excess (T ½ ˜ 30 years) can lead to microcytic, hypochromic anemia and proteinuria with loss of beta-2-microglobin, and functional zinc deficiency. A recently published epidemiological study found a significant correlation between elevated hair Cd and distractibility in young children (Le Clair, J.A. and D.W. Quig. Mineral Status, Toxic Metal Exposure and Children's Behavior. J. Orthomol. Med. (2001) 16: 13-32. Cd excess is also commonly associated with fatigue, weight loss, osteomalacia, and lumbar pain.

Cd absorption is reduced by zinc, calcium, and selenium. Cd is found in varying amounts in foods, from .04 ppm for some fruits to 3-5 ppm in some oysters and anchovies. Cigarette smoking significantly increases Cd intake. Refined carbohydrates have very little zinc in relation to the Cd. Other sources of Cd include human biosolids, pigments and paints, batteries (Ni-Cd), plastics and synthetic rubber (tires).

A confirming test for elevated body burden of Cd is urine analysis following administration of appropriate chelating agents: EDTA, sulfhydryl agents (DMSA,D-penicillamine, DMPS). Urinary beta-2-microglobulin is considered to be the best test for assessment of Cd-associated functional damage to the proximal tubules of the kidneys.

COPPER HIGH

The biliary or fecal route is the main route of excretion for Copper (Cu). About half of daily ingested Cu (~10 mg/d) is excreted in the feces (1 – 5 mg). Estimates of daily absorbed Cu are also in the range of 1 – 5 mg/d.

Sources of excessive Cu include contaminated food or drinking water, excessive Cu supplementation, wood preservatives (pressure treated wood/sawdust) and occupational or environmental exposures. Insufficient intake of competitively absorbed elements such as zinc or molybdenum can lead to, or worsen Cu excess.

Medical conditions that may be associated with excess Cu include: biliary obstruction (reduced ability to excrete Cu), liver disease (hepatitis or cirrhosis), and renal dysfunction. Symptoms associated with excess Cu accumulation are muscle and joint pain, depression, irritability, tremor, hemolytic anemia, learning disabilities, and behavioral disorders.

Confirmatory tests for Cu excess are a comparison of Cu in pre vs. post provocation (D-penicillamine, DMPS) urine elements tests, and a whole blood elements analysis.

MERCURY HIGH

Mercury (Hg) is an extremely toxic element. Fecal Hg is an excellent measure of exposure and possible accumulation of the element. Both fecal and urinary excretion are the main elimination routes for inorganic and methyl mercury.

It is quite clear that sensitivity to Hg varies greatly among individuals; some individuals exhibit extreme symptoms with levels of Hg which are without obvious effects in others. The symptomatology of Hg excess can depend on many factors: the chemical form of absorbed Hg and its transport in body tissues, presence of other synergistic toxics (Pb and 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 the mouth, fatigue or lack of physical endurance, and increased salivation. Symptoms may progress with moderate or chronic exposure to include: anxiety, depression, 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 (50% by weight), explosive detonators, in elemental or liquid form for thermometers, barometers, and laboratory equipment; batteries and electrodes, some vaccines and in fungicides and pesticides. The fungicide and pesticide use of mercury (including that in paints) has declined due to environmental concerns, but mercury residues persist from past use. Methylmercury, the common, most 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 food is contaminated with one of the previously listed forms/sources.

Data collected at DDI indicate positive correlations between fecal Hg levels and the number of amalgams, and the amount of fish consumed.

Hg burden can be confirmed by urine elements analysis. Comparison of urine Hg levels pre and post provocation (DMPS, DMSA, D-penicillamine) permit differentiation between recent uptake and retention in the body.

NICKEL

From 1 to 10% of dietary nickel may be absorbed from the gastrointestinal tract into the blood. Nonabsorbed Ni is eliminated in the feces. Therefore, fecal Ni levels are primarily reflective of oral exposure.

There is substantial evidence that Ni is an essential element which is required in extremely low amounts. However, excess Ni has been well established to be nephrotoxic, and carcinogenic. 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.

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, soy products, nuts, and hydrogenated oils (margarine)
Nickel-cadmium batteries
Non-precious, 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
Prostheses or surgical implants

Symptoms of Ni toxicity are dermatitis (hand eczema) and pulmonary inflammation (following exposure to Ni dust, smoke). Long term or chronic Ni toxicity may lead to liver necrosis and carcinoma.

A confirmatory test for elevated assimilation of Ni is the measurement of urine Ni before and after administration of chelating agents that mobilize Ni e.g., EDTA, DMPS, D-penicillamine, DMSA.

LEAD HIGH

Absorbed lead (Pb) is excreted primarily in urine and gastrointestinal secretions (urine 76% and feces 16%). Fecal Pb provides an excellent indication of oral exposure and an approximation of assimilation/excretion.

Lead (Pb) has pathological, neurotoxic and nephrotoxic effects in humans that may be manifested with relatively low Pb levels up to acutely toxic levels. Pb may also affect the body's ability to utilize the essential elements calcium, magnesium, and zinc. At moderate levels of body burden, Pb may have adverse effects on memory, cognitive function, nerve conduction, and metabolism of vitamin D. In children, developmental disorders and behavior problems may occur at relatively low levels: loss of IQ, hearing loss, poor growth. In order of occurrence with increasing lead concentration, the following can occur: impaired vitamin D metabolism, initial effects on erythrocyte and erythroid precursor cell enzymology, increased erythrocyte protoporphyrin, headache, and decreased nerve conduction velocity. Further effects of Pb excess include: metallic taste, loss of appetite, constipation, poor hemoglobin synthesis, colic, frank anemia, tremors, nephrotoxic effects with impaired renal excretion of uric acid, neuropathy and encephalopathy.

Sources of lead include: old lead-pigment paints, batteries, industrial smelting and alloying, some types of solders, glazes on (foreign) ceramics, leaded (antiknock compound) fuels, bullets and fishing sinkers, artist paints with lead pigments, and leaded joints in some municipal water systems. Most lead contamination occurs via oral ingestion of contaminated food or water or by children mouthing or eating lead-containing substances. The degree of absorption of oral lead depends upon stomach contents (empty stomach increases uptake) and upon the body's mineral status. Deficiency of zinc, calcium or iron may increase lead uptake. Transdermal exposure is slight. Inhalation of lead has decreased significantly with almost universal use of non-leaded automobile fuel.

Pb burden can be confirmed by urine elements analysis. Comparison of urine Pb levels pre and post provocation (EDTA, DMSA, DMPS) permit differentiation between recent uptake and retention in the body. Increased erythrocyte zinc protoporphyrin is a finding consistent with lead excess.

PLATINUM HIGH

Platinum (Pt) is a nonessential element that is sometimes detected in feces. However, the significance of higher Pt levels in feces is not well studied.

Pt is poorly absorbed in the gut but may be absorbed via inhalation. Since it is a relatively rare element, most Pt exposures are of occupational origin. In recent years, there may have been a slight increase in environmental Pt due to the use of Pt as a catalyst in automobile exhaust converters. Pt is a byproduct of copper refining and used as an alloy in dental and orthopedic materials. Symptoms excess exposure to Pt include: dermatitis, irritation of mucus membranes, dyspnea and

wheezing (for inhaled Pt dusts or salts), development of chronic allergic reactions ("platinosis"), nephrosis, and immunosuppression (from Pt diamine salts).

Pt containing drugs, such as cisplatin and carboplatin, are used as chemotherpeutic agents. Such drugs are extremely toxic and cause nephrotoxicity with associated magnesium wasting and hypomagnesemia, myelosupression, ototoxicity, and neurotoxicity. Hair elements analysis may be utilized to assess excessive exposure and assimilation of Pt.

ANTIMONY HIGH

The measurement of Antimony (Sb) in feces can be used as an estimate of exposure to and biliary excretion of the element.

Sb is a nonessential element that is chemically similar to but less toxic than some forms of arsenic. Food and smoking are the usual sources of Sb. Gunpowder (ammunition) often contains Sb. Other possible sources are textile industry, metal alloys, and some antihelminthic and antoprotozoic drugs. Sb is also used in the manufacture of paints, glass, ceramics, solder, batteries, bearing metals and semiconductors. Confirming a report from New Zealand, analysis performed at DDI revealed high levels of Sb and arsenic in sheepskin bedding designed for an infants crib. Sb along with arsenic is also commonly found in fecal specimens from autistic children.

Like arsenic, Sb has a high affinity for sulfhydryl groups on many enzymes. Sb is conjugated with glutathione and excreted in urine and feces. Therefore, excessive exposure to Sb has the potential to deplete intracellular glutathione pools.

Early signs of Sb excess include: fatigue, muscle weakness, myopathy, nausea, low back pain, headache, and metallic taste. Later symptoms include hemolytic anemia, myoglobinuria, hematuria and renal failure. Transdermal absorption can lead to "antimony spots" which resemble chicken pox. Respiratory tissue irritation may result from inhalation of Sb particles or dust.

Comparison of pre and post provocation (DMPS, DMSA) urine Sb levels can be used to further evaluate potential accumulation of Sb in the body.

THALLIUM HIGH

Thallium (Tl) is a highly toxic element which is generally tasteless and odorless. Like lead and mercury, Tl accumulates in many body tissues. Although the kidneys are the major route of elimination for Tl, the biliary fecal route also contributes.

Common sources of Tl are: foods (marine organisms concentrate Tl up to 700 times), tobacco, contaminated water, electronics components, fly ash, cement dust, and some fertilizers, pesticides and rodenticides. Tl is rapidly and completely absorbed when ingested, inhaled or brought into contact with skin. Consumption of Tl containing rodenticides is the primary means of acute Tl toxicity.

Symptoms of chronic Tl excess include: sleep disturbances, cardiac, optical, dermatatological, liver, GI, and kidney dysfunctions. Albuminuria and alopecia are consistent with Tl excess. Potassium, selenium and sulfhydryl compounds (e.g. glutathione) diminish Tl retention and toxicity. Tl toxicity can have a long latency period before clinical symptoms become apparent. In contrast, acute Tl poisoning is associated with extreme abdominal and retrosternal pain, respiratory distress and excessive thirst.

Hair elemental analysis can be utilized to assess chronic, low-level exposure to Tl.

URANIUM HIGH

The levels of Uranium (U) in feces have been used to arrive at the total daily intake of U while urinary U represents a smaller fraction of the total daily elimination of U. Most U passes through the intestine unabsorbed. Some excretion occurs from blood into the intestine, via bile (endogenous excretion).

U is a nonessential element that is very abundant in rock, particularly granite. U is present at widely varying levels in ground (drinking) water, root vegetables, and present in high phosphate fertilizers. Other sources of U include: ceramics, some colored glass, many household products (uranyl acetate) and tailings from U mines.

Uranyl cations bind tenaciously to protein, nucleotides, and bone, where it substitutes for Ca. Published data are sparse, but there appears to be a correlation between U exposure, nephrotoxicity and all forms of cancer. Kidney and bone are the primary sites of U accumulation.

All isotopes of U are radioactive; U-238 is the most abundant and lowest energy emitter. It is important to note that the measured result, which is U-238, does NOT indicate or imply exposure to highly enriched U-235, which is used in nuclear power and weaponry.

Hair elements analysis can be performed to assess chronic exposure to U. Uranium is infrequently found to be elevated in unprovoked urine and only reflects ongoing exposure. To date, there are no available chelating agents that can be definitively used to assess U accumulation in the body.

TUNGSTEN HIGH

After exposure and absorption via inhalation, ingestion, or injection, Tungsten (W) is rapidly eliminated via urine and feces. W has no known biological role. Long-term pulmonary exposures have been associated with lung disease (pneumoconiosis or “hard metal lung disease”) and lung cancer. Skin contact with W may produce contact eczema, pruritis, folliculitis, and neurodermatitis. Tungsten has a potent antagonistic relationship to Mo decreasing hepatic Mo concentration and reducing the activities of sulfite and xanthine oxidases.

Tungsten is a silvery-white lustrous element usually obtained as a grey powder and is mainly utilized as tungsten carbide in metal-working, mining and petroleum industries. Calcium and magnesium tungstates are widely used in as filaments for electric lamps, electron tubes and

television tubes. Since Tungsten has the highest melting point of all metals it is used for high-speed and hot-worked steels. Other sources of W include catalysts and reagents in biological analysis, fire and waterproof materials, industrial lubrifications, and ash from incineration of sewage sludge.

Intestinal absorption of tungsten is rapid and significant. W is rapidly transported to the blood and then to the kidneys for filtration and eventual excretion from the body. Pulmonary absorption of W-tungstic oxide has been studied in dogs. 60% of W is rapidly deposited in the respiratory tract and 33% of that fraction reaches systemic circulation. Tungsten is also easily transferred from mother to fetus, usually later in gestation.

Comparison of pre and post provocation (DMPS, DMSA) urine W levels can be used to further evaluate potential accumulation of W in the body.