FECAL METALS

LAB#: F000000-0000-0
PATIENT: Sample Patient
SEX: Female
AGE: 73

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

POTENTIALLY TOXIC METALS

METALS	RESULT mg/kg	REFERENCE RANGE	PERCENTILE (68th / 95th)
Mercury	0.246	<.05 w/o amalgams*	
Mercury	0.246	< .5 with amalgams*	
Antimony	0.054	< 0.08	
Arsenic	0.27	< 0.3	
Beryllium	0.014	< 0.009	
Bismuth	0.032	< 0.05	
Cadmium	0.38	< 0.5	
Copper	30	< 60	
Lead	9.13	< 0.5	
Nickel	5.4	< 8.00	
Platinum	0.010	< 0.003	
Thallium	0.010	< 0.02	
Tungsten	0.124	< 0.09	
Uranium	0.062	< 0.12	

% WATER CONTENT

	RESULT % H_2O	EXPECTED RANGE	MEAN (2SD LOW / 1SD LOW / 72.5% / 1SD HIGH / 2SD HIGH)
% WATER CONTENT	75.4	60-85%	

DISCUSSION

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: **6/3/2002**
Date Received: **6/7/2002**
Date Completed: **6/15/2002**

Detoxification Agent:
Methodology: **ICP-MS**

Dental Amalgams: **Yes**
Quantity:

V02.00

MERCURY HIGH

FecalHG

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.

LEAD HIGH

FecalPB

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.