LAB #: Sample Report
PATIENT: Sample Patient
ID:
SEX: Female
AGE: 46

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

Toxic Metals; Feces

TOXIC METALS		RESULT mg/kg Dry Wt	REFERENCE INTERVAL	PERCENTILE (68TH / 95th)
Mercury	(Hg)	**0.215**	**<.05** w/o amalgams*	
Mercury	(Hg)	**0.215**	**<0.5** with amalgams*	
Antimony	(Sb)	**0.042**	< 0.080	
Arsenic	(As)	**0.20**	< 0.30	
Beryllium	(Be)	**0.009**	< 0.009	
Bismuth	(Bi)	**67.50**	< 0.050	
Cadmium	(Cd)	**0.51**	< 0.50	
Copper	(Cu)	**47**	< 60	
Lead	(Pb)	**0.29**	< 0.50	
Nickel	(Ni)	**6.3**	< 8.0	
Platinum	(Pt)	**< dl**	< 0.003	
Thallium	(Tl)	**0.047**	< 0.020	
Tungsten	(W)	**0.028**	< 0.090	
Uranium	(U)	**0.073**	< 0.120	

WATER CONTENT	RESULT % H_2O	REFERENCE INTERVAL	MEAN (-2SD / -1SD / 72.5% / +1SD / +2SD)
% Water Content	**69.8**	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: **02/19/2019**
Date Received: **02/20/2019**
Date Completed: **02/25/2019**

Provocation: **Pre Provocative**
Detoxification Agent:
Dosage:

Dental Amalgams: **not indicated**
Quantity: **0**
Methodology: **ICP-MS**

V08.10

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.

BISMUTH HIGH

FecalBI

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).

THALLIUM HIGH

FecalTL

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.