LAB #: F140326-2612-1
PATIENT: Matthew Webber
ID: WEBBER-M-00067
SEX: Male
AGE: 23

CLIENT #: 39238
DOCTOR: , MD
Neurological Research Institute, Llc
279 Walkers Mills Rd
Bethel, ME 04217 USA

Toxic Metals; Feces

TOXIC METALS		RESULT mg/kg Dry Wt	REFERENCE INTERVAL	PERCENTILE (68TH / 95th)
Mercury	(Hg)	**0.024**	**<.05** w/o amalgams*	
Mercury	(Hg)	**0.024**	**<0.5** with amalgams*	
Antimony	(Sb)	**0.076**	< 0.080	
Arsenic	(As)	**0.12**	< 0.30	
Beryllium	(Be)	**0.004**	< 0.009	
Bismuth	(Bi)	**120.4**	< 0.050	
Cadmium	(Cd)	**0.14**	< 0.50	
Copper	(Cu)	**31**	< 60	
Lead	(Pb)	**0.20**	< 0.50	
Nickel	(Ni)	**3.5**	< 8.0	
Platinum	(Pt)	**< dl**	< 0.003	
Thallium	(Tl)	**0.008**	< 0.020	
Tungsten	(W)	**0.221**	< 0.090	
Uranium	(U)	**0.643**	< 0.120	

WATER CONTENT	RESULT % H_2O	REFERENCE INTERVAL	MEAN (-2SD / -1SD / 72.5% / +1SD / +2SD)
% Water Content	**74.8**	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: **03/23/2014**
Date Received: **03/26/2014**
Date Completed: **04/01/2014**

Provocation:
Detoxification Agent:
Dosage:

Dental Amalgams: **No**
Quantity:
Methodology: **ICP-MS**

V08.10

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).

URANIUM HIGH

FecalU

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.