### Toxic Elements

<table>
<thead>
<tr>
<th>Element</th>
<th>Reference Range</th>
<th>TMPL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lead</td>
<td>0.4</td>
<td>&lt;= 1.4</td>
</tr>
<tr>
<td>Mercury</td>
<td>6.46</td>
<td>&lt;= 2.19</td>
</tr>
<tr>
<td>Aluminum</td>
<td>25.7</td>
<td>&lt;= 22.3</td>
</tr>
<tr>
<td>Antimony</td>
<td>&lt;dl</td>
<td>&lt;= 0.149</td>
</tr>
<tr>
<td>Arsenic</td>
<td>23</td>
<td>&lt;= 50</td>
</tr>
<tr>
<td>Barium</td>
<td>4.0</td>
<td>&lt;= 6.7</td>
</tr>
<tr>
<td>Bismuth</td>
<td>&lt;dl</td>
<td>&lt;= 2.28</td>
</tr>
<tr>
<td>Cadmium</td>
<td>0.17</td>
<td>&lt;= 0.64</td>
</tr>
<tr>
<td>Cesium</td>
<td>12.8</td>
<td>&lt;= 10.5</td>
</tr>
<tr>
<td>Gadolinium</td>
<td>&lt;dl</td>
<td>&lt;= 0.019</td>
</tr>
<tr>
<td>Gallium</td>
<td>&lt;dl</td>
<td>&lt;= 0.028</td>
</tr>
<tr>
<td>Nickel</td>
<td>2.06</td>
<td>&lt;= 3.88</td>
</tr>
<tr>
<td>Niobium</td>
<td>&lt;dl</td>
<td>&lt;= 0.084</td>
</tr>
<tr>
<td>Platinum</td>
<td>0.084</td>
<td>&lt;= 0.033</td>
</tr>
<tr>
<td>Rubidium</td>
<td>2.421</td>
<td>&lt;= 2.263</td>
</tr>
<tr>
<td>Thallium</td>
<td>0.167</td>
<td>&lt;= 0.298</td>
</tr>
<tr>
<td>Thorium</td>
<td>&lt;dl</td>
<td>&lt;= 4.189</td>
</tr>
<tr>
<td>Tin</td>
<td>0.81</td>
<td>&lt;= 2.04</td>
</tr>
<tr>
<td>Tungsten</td>
<td>0.167</td>
<td>&lt;= 0.211</td>
</tr>
<tr>
<td>Uranium</td>
<td>&lt;dl</td>
<td>&lt;= 0.026</td>
</tr>
</tbody>
</table>

The performance characteristics of all assays have been verified by Genova Diagnostics, Inc. Unless otherwise noted with *, as cleared by the U.S. Food and Drug Administration, assays are For Research Use Only.

### Sulfur

<table>
<thead>
<tr>
<th>Element</th>
<th>Reference Range</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur*</td>
<td>660</td>
<td>367-1,328</td>
</tr>
</tbody>
</table>

* Elevated sulfur may indicate the presence of a chelating agent.

### Creatinine Concentration

- Urine Creatinine: 17.96 mg/dL (23.00-205.00 mg/dL)

### Collection Information

- Urine Total Volume (in milliliters): Not given
- Length of Collection: (in hours) 16.0
- Provocation Comment: Post-provocation agent: Advanced Cellular Zeolite (ACZ) nano

### Tentative Maximum Permissible Limit (TMPL)

- Tentative Maximum Permissible Limit (TMPL) - Element excretion is significantly elevated, consistent with increased body burden. Increased element concentrations can have a negative impact on overall health and well-being. These values are derived from Casaret and Doull's *Toxicology: The Basic Science of Poisons*, 5th Ed. 1996 McGraw Hill NY, NY pp 997-998. Units have been standardized.

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Lab Comments

January 24, 2006
Please note that the reference ranges for Bismuth and Thorium have been updated. This report includes the new reference ranges. A statistical analysis was performed on the data, in compliance with NCCLS guidelines and recommendations for reference ranges.

<dl = Unable to determine results due to less than detectable levels of analyte.

Commentary is provided to the practitioner for educational purposes, and should not be interpreted as diagnostic or treatment recommendations. Diagnosis and treatment decisions are the responsibility of the practitioner.

Reference Range Information:
Elemental reference ranges were developed from a healthy population under non-provoked/non-challenged conditions. Provocation with challenge substances is expected to raise the urine level of some elements to varying degrees, often into the cautionary or TMPL range. The degree of elevation is dependent upon the element level present in the individual and the binding affinities of the challenge substance.

Urine creatinine concentration is below the reference range. This may be due to increased fluid intake, a low protein diet, low body weight, or low levels of physical activity. Conditions such as diuretic use, dietary deficiencies of precursor amino acids (arginine, glycine, or methionine), malnutrition, or hypothyroidism may also lower creatinine levels. Measurement of serum creatinine or a creatinine clearance test can help determine if there are changes in renal function.

Mercury is measured to be elevated. In body tissues, mercury behaves differently in different tissues, depending on its chemical form, and interchange between forms can occur in vivo. For elemental and inorganic mercury, biliary excretion predominates with low-level toxicity, but urinary excretion increases and is favored as the degree of exposure and burden increases. For organic mercury (methyl, ethyl, alkyl), bile accounts for about 90% of excretion and urine accounts for about 10%. Significant day-to-day and diurnal variations are typically observed. Urinary excretion of mercury is notably increased following administration of chelating or detoxifying agents (DMSA, DMPS); intravenous administration of EDTA results in relatively minor urinary increases. The GSDL laboratory procedure measures total urine mercury, regardless of chemical form, and the procedure is not hindered by tightly-bound sulphydryl-mercury that might be unavailable (and unmeasured) by the old standard procedure ("cold-vapor atomic absorption").

There is great variability in individual tolerances to mercury. In some individuals, relatively low levels can cause immune dysregulation. Lymphocyte inhibition and dysfunction is reported, immunosuppression can occur, and autoimmune conditions are documented in animals. At the cellular level, mercury can induce cytotoxicity, oxidative stress (via loss of glutathione function), and increased secretion of beta-amyloid in neuronal cells, linking it to Alzheimer's disease. Outside cells, mercury can bind to and strongly inhibit a cell surface-bound protein called dipeptidylpeptidase IV, CD26, and adenosine deaminase binding protein. This one protein is responsible for digestion of proline-containing dietary peptides, T-cell activation, and the metabolism of adenosine. Inside cells, mercury binds to lipoic acid, glutathione, coenzyme A and cysteinyl sites, and it can impair pyruvate metabolism and citric acid (Kreb's) cycle function, leading to impaired energy production. Chronic mercury exposure may produce
increased excitability and tremor, memory loss, insomnia, lassitude, anorexia and weight loss, gingivitis and stomatitis. Young children may exhibit “pink disease” (acrodynia), commonly featuring rash, photophobia, increased perspiration and salivation. Acute mercury vapor exposure may inflame the bronchial tubes and cause pneumonitis. Irreversible neurologic damage is reported in acute mercury toxicity. Inorganic mercury concentrates mostly in kidneys, while organic (methyl) mercury has high affinity for the posterior cortex of the brain.

Mercury sources have increased in the environment, resulting in increased amounts in soils, sediments and bodies of water. Coal-fired power plants emit over 30% of environmentally released mercury. Other industrial sources are chlorine or “chlor-alkali” plants, cement plants, pulp and paper mills, municipal waste incinerators (19% of total release), and hazardous/medical waste incinerators. As of 2001, over 100 tons of mercury are “missing” from the EPA-surveyed inventory of chlor-alkali plants which admit to releasing the element to air and landfills. These sources, along with increased farming, forest fires, mining and excavations, and volcanoes, have served to increase surface deposition (micrograms per square meter) of mercury in surveyed areas by over 300% since 1850. This mercury can be biologically changed into organic forms and made bioavailable. Fish, shellfish and edible seaweed then become dietary sources of this element. Other sources include: old latex paint (manufactured before 1990), antifungal and antifouling (marine) paints, some fluorescent light tubes and vapor lamps, medicinal products such as those containing "Thimerosal" (sodium ethyl mercurithiosalicylate or mercurothiolate--often contained in routine vaccines), explosives and detonators, batteries and "calomel" electrodes, electrical switches, thermostats and relays, and scientific or laboratory equipment (thermometers, barometers). Dental amalgams are primarily a source of elemental or amalgamated mercury that is typically found in feces for several days following dental procedures; very little of this dental-procedure mercury appears in urine. However, mercury vapor from in-place amalgam fillings can be absorbed, biotransformed and excreted in urine, but its level is typically much less than that which is attributable to food sources, especially seafood.

Aluminum is above the reference range. Most ingested aluminum comes from food and drink, while additional amounts may come from pharmaceuticals. Absorption from the GI tract is normally minimal, typically decreased by the presence of dietary phosphates, but increased by the presence of citric or malic acids (carboxylic acids). Excretion of aluminum from blood plasma is primarily by urine, while RBC-bound aluminum is mostly excreted via the bile. Urinary excretion predominates overall. Administration of the chelating agent EDTA can release aluminum from the bloodstream and increase its level in urine.

Once in the body, aluminum follows increasing concentrations of phosphate. However, it may also bind to transferrin in the blood and to citric or malic acids (carboxylic acids). Binding and increased transport also occurs with the amino acid, glycine. Aluminum may bind to DNA, ATP, NADP, NADPH or phosphorylated proteins, once inside a cell. Excessive aluminum in neuronal tissue is implicated as a contributory agent in senile dementia and Alzheimer's disease. In neuronal and other tissues, aluminum may become a permanent resident with accumulations continuing throughout life.

Common sources of aluminum include cookware (coffee pots, pizza pans, utensils), tomato sauce cooked in aluminum pots or pans, deodorants and cosmetics, aluminum hydroxide antacids (when G.I. phosphate is inadequate), aluminum baking powder, drinking water clarified by "alum", and drinking from surface-damaged or very old aluminum soft drink cans. Fluoride-treated water increases aluminum bioavailability and uptake.
Cesium is above the reference range. This element is known for its radioactive isotopes, which are uranium fission products and arise from nuclear power plant fuel and from atomic bomb testing. It is also known for its medical and research uses (Cs-137, "Radiogardase-Cs"). Measured and reported here is the stable (nonradioactive), natural isotope of cesium, Cs-133; natural cesium is 100% Cs-133. Elevation of Cs-133 does not imply, and has no relationship to, the presence (or absence) of radioactive isotopes of cesium. When ingested or otherwise assimilated, cesium behaves like, and transports like, potassium in body tissues; in blood it binds inside erythrocytes. Much of the body's long-term burden is in muscle tissue where there is higher affinity for cesium than for potassium. Tests with injected Cs-137 show that 70% is excreted within seven days, mostly via urine. Dietary cesium forms may have a longer biologic half-time in humans; 50 to 60 days is reported. Increased dietary intake of potassium can increase urinary excretion of cesium. Cesium (like thallium) can be chelated and removed with "Prussian Blue" (potassium ferrihexacyanoferrate II).

Cesium has multiple toxic effects. In erythrocytes, cesium obstructs the release of oxygen from hemoglobin, thereby decreasing the RBC's ability to oxygenate body tissues. Additionally, exposure during pregnancy can result in organ abnormalities, impaired liver function (weak aldehyde and alcohol dehydrogenase), and reduction of CNS tissue mass in offspring.

Significant exposures are industrial or occupational. The element is used in the petrochemical industry as a catalyst for hydrogenation and polymerization, and it is used to manufacture some types of photoelectric cells. Halogenated cesium (chloride, bromide or iodide) is used in x-ray fluorescent screens. It may also be used in the manufacture of glass, lenses and prisms.

Platinum is above the reference range. Excessive platinum in food or drink is rare, and most exposures are industrial via inhalation, or as a result of the administration of "cis-platin", a chemotherapeutic agent for cancer. Elemental platinum has very low toxicity except for those who have dermal sensitivity. Most inhaled, ingested or injected platinum is excreted via urine. This excretion is biphasic with most being eliminated within several hours while the remainder may require 12 days or more for excretion. Elevated urine platinum may be observed after administration of sulfhydryl-bearing detoxification agents (DMSA or DMPS).

Platinum as a complex can upregulate heme oxygenase activity (>10x), thereby disordering heme synthesis in the liver. Platinum inhibits DNA synthesis in the same manner that it exerts its antitumor activity. Binding to and blocking sulfhydryl sites and inhibition of dehydrogenase enzymes are other modes of toxicity. Platinum deposits in liver and in the kidney, where chronic deposition can damage proximal tubules and cause renal insufficiency. "Platinosis", caused by chronic exposure, features rhinorrhea, coughing and sneezing, eczematous dermatitis, and a lung syndrome with dyspnea, wheezing and an asthma-like condition.

Besides cis-platin, sources of platinum include: catalytic converters on gasoline engines (cars, trucks), electroplating operations, catalyst production and catalytic equipment in the chemical process industries and petroleum refineries, precious dental materials, jewelry, smelting and refining of nickel and copper, purification of gold ores, and electronic parts such as thermocouples, resistance wires and contacts.

Rubidium is above the reference range. Natural rubidium is present in virtually all soils, plants and vegetables, and animal products. Foods with highest rubidium content are soybeans, tomatoes and beef. Rubidium has very low
toxicity, and health problems arise primarily from industrial or occupational exposures. Ingested rubidium is almost completely absorbed into portal blood, carried temporarily by erythrocytes, and deposited in muscle, liver, spleen, kidney, heart, lung and brain. The biologic half time in humans is 50 to 60 days with about 85% excreted via urine and about 15% excreted via bile/feces. Increased potassium intake can cause increased excretion of accumulated rubidium.

Rubidium can dysregulate potassium-dependent transport processes and metabolism and is believed to increase the release of norepinephrine. When tissue levels are excessive, excitability and aggressive behaviors occur. Human occupational exposures to high levels of rubidium have produced headaches, lassitude, irritability, disturbed sleep, cardiac arrhythmia, peripheral neuropathy, inflammation of the respiratory tract, and kidney damage with proteinuria.

Some photoelectric cells, specialized glasses, lenses and prisms, and vacuum tubes contain this element. Rubidium is encountered wherever alkali elements are processed, often by electrolysis. Potassium mineral deposits and natural brines usually have high rubidium content.