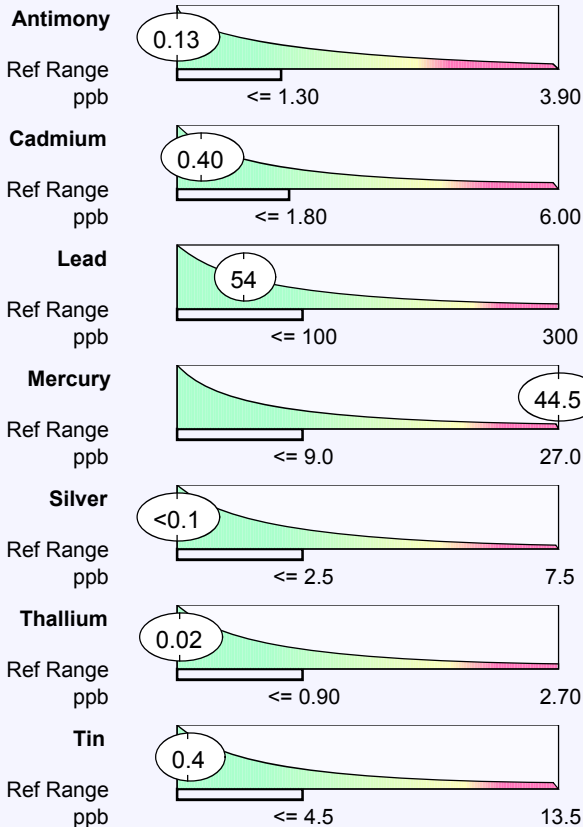


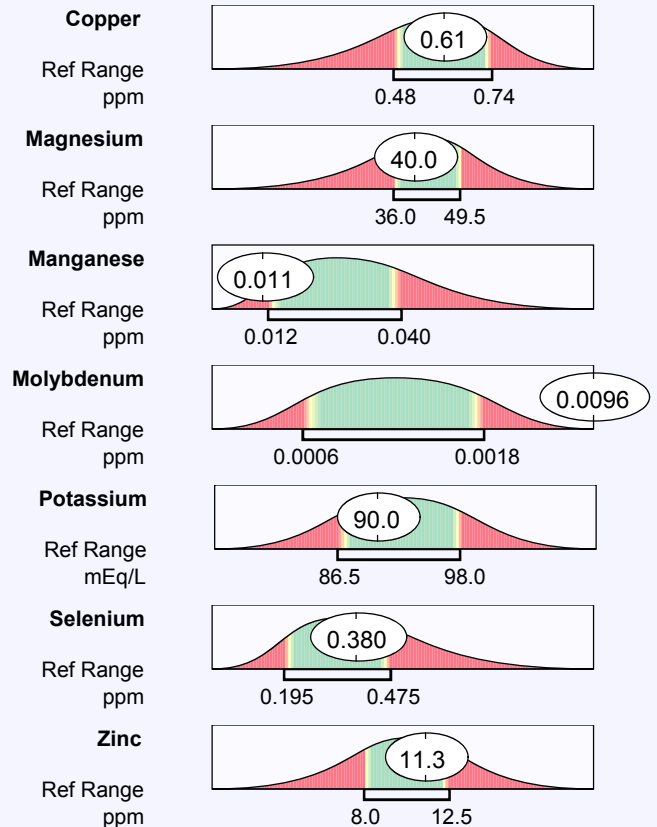


Patient:	<b>Order Number:</b>	INSTITUUT VOOR FUNCTIONELE GENEESKUNDE B
Age:	Completed:	Mathaak 3
Sex:	Received:	Malden, 6581VK
MRN:	Collected:	THE NETHERLANDS

### Toxic Elements



### Essential Elements



Histograms on this report are not based on data from reference populations and should be used for illustrative purposes only. This test has been developed and its performance characteristics determined by GSDL, Inc. It has not been cleared or approved by the U.S. Food and Drug Administration.

### Commentary

#### Lab Comments

**Elevated results verified through a repeat analysis. rbw 10/15/02**

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.

**Molybdenum (Mo)** is measured to be elevated in erythrocytes. Most blood Mo is tightly bound to enzyme proteins

## Commentary

and globulin inside the erythrocyte; only about one-third is in plasma bound to globulins. Significantly elevated Mo is unusual, and some toxic effects are due to displacement of copper or inactivation of copper enzymes. Copper deficiency predisposes to Mo excess. Normally, Mo excretion is by both urine and bile. Increased erythrocyte Mo may occur in biliary dysfunction. Copper supplementation enhances Mo excretion. For mildly elevated Mo there is typically no clinical significance.

Sources of Mo include: metal alloying, metal machining, catalysts in the chemical and petrochemical industries, pigments, lubricants, and coatings and agents for anticorrosive treatment of metals. Molybdenum exposure also occurs in mining, ore refining and smelting operations. Exhaust plumes and ash from fossil fuel combustion, sewage sludge, and oil refinery waste streams may contain Mo.

Symptoms consistent with significant Mo excess include: anemia, loss of skin pigmentation, increased inflammatory responses, sexual dysfunction, loss of calcium from bone and osteoporosis, and weakened tendons and cartilage. Increased uric acid formation and gout also are reported.

**Mercury** is elevated. Possible sources of this toxic element include -

- Contaminated shellfish or seafood
- Contaminated water supplies
- Dental amalgams - recent dental work
- Electrical switches and relays, explosive detonators
- Batteries and electrodes (calomel electrodes)
- Laboratory equipment, barometers, thermometers
- Some specially-formulated fungicides
- Old paint containing Hg fungicide
- Chemical process industry, "chloralkali electrolysis"
- Mining and smelting operations

At least 95% of blood organic mercury rapidly distributes to erythrocytes, and at least 60% of elemental mercury may reside transiently in erythrocytes. Most inorganic mercury does not enter the erythrocyte. Mercury has strong affinity for sulfhydryl (-SH) sites on proteins and enzymes throughout the body and deposits in many tissues and organs. The kidneys eventually carry much of the body burden regardless of route of exposure or chemical form of the Hg. Elemental and inorganic Hg eventually distribute predominately to liver and kidney. Excretion is slow - kidney Hg via urine and liver Hg via feces. Elemental Hg vapor may be dissolved in blood, may enter erythrocytes, and can deposit in brain tissue. Organic Hg (methyl, ethyl) binds to enzymes, proteins and glutathione in blood and various tissues, circulates rather freely, and has a long retention half-time in the body (approximately two months). Hg interferes with catalase, monoamine oxidase, mixed-function oxidases and P-450 in liver tissue, and stimulates thionein formation and is distributed there partly as mercury-metallothionein. In cell mitochondria, organic Hg, especially methylmercury, disrupts respiration, decreases synthesis of RNA and can be mutagenic by altering chromosome structure.

Symptoms consistent with Hg contamination are variable and may include: metallic taste, increased salivation, paresthesias with decreased senses of hearing touch and vision, hypertension, headaches, fatigue, insomnia, and fine muscle tremor possibly displayed as poor handwriting. A hallmark symptom is emotional disturbance, sometimes a bipolar depression but often a form of excitability and lack of ability for mental concentration.

**Manganese** is low. Manganese activates the enzyme glycosyltransferase which is essential for formation of mucopolysaccharides and the ground or packing substance of collagen, bone, and joint tissue. Other enzyme activator roles include: arginase in the urea cycle, some digestive peptidases secreted by the pancreas or intestinal

### *Commentary*

mucosa, Mn-superoxide dismutase in cell mitochondria, and pyruvate carboxylase which forms an important citric acid cycle component, oxaloacetic acid. Manganese also assists isocitrate dehydrogenase which forms alpha-ketoglutaric acid in the citric acid cycle. Alpha-ketoglutaric acid is required for nitrogen balance in body tissues. In erythrocytes, manganese is bound to porphyrin and is a general measure of manganese status. Erythrocytes concentrate manganese, and usually contain about 20X more than does blood serum. However, none of the above-cited manganese functions occur with erythrocyte-porphyrin Mn.

Low or deficient manganese can result from a poor quality "junk-food" diet, maldigestion or malabsorption. Profuse sweating or diarrhea can reduce body retention of manganese; relatively little manganese is excreted via urine. Symptoms consistent with manganese insufficiency may include: increased allergic or inflammatory responses, fatigue, abnormal blood glucose levels, impaired growth of bone, nails or hair, weight loss, poor blood clotting (resistant to vitamin K), hyperaminoacidemia/hyperaminoaciduria with nitrogen excess, possibly hyperammonemia (nausea, headache), and arthritic joint symptoms.