LAB #: 999999-9999
PATIENT: Sample Patient
ID: 999999
SEX: Male
DOB:
AGE: 48

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

Toxic & Essential Elements; Packed Red Blood Cells

ESSENTIAL AND OTHER ELEMENTS		RESULT / UNIT	REFERENCE INTERVAL	PERCENTILE 2.5th 16th 50th 84th 97.5th
Calcium	(Ca)	**10** µg/g	8-25	
Magnesium	(Mg)	**47** µg/g	39-59	
Potassium	(K)	**90** mEq/L	78-97	
Phosphorus	(P)	**582** µg/g	520-670	
Copper	(Cu)	**0.514** µg/g	0.52-0.8	
Zinc	(Zn)	**11.3** µg/g	8-14	
Iron	(Fe)	**947** µg/g	810-1020	
Manganese	(Mn)	**0.011** µg/g	0.008-0.029	
Selenium	(Se)	**0.292** µg/g	0.16-0.49	
Boron	(B)	**0.117** µg/g	0.01-0.11	
Molybdenum	(Mo)	**0.0002** µg/g	0.0002-0.001	

TOXIC METALS		RESULT / UNIT	REFERENCE INTERVAL	PERCENTILE 95th 99th
Arsenic	(As)	**0.0091** µg/g	< 0.008	
Cadmium	(Cd)	**< 0.0005** µg/g	< 0.002	
Cesium	(Cs)	**0.0091** µg/g	< 0.015	
Chromium	(Cr)	**0.0004** µg/g	< 0.0005	
Lead	(Pb)	**0.015** µg/g	< 0.06	
Mercury	(Hg)	**0.0353** µg/g	< 0.01	
Thallium	(Tl)	**< 0.00004** µg/g	< 0.00005	

SPECIMEN DATA

Comments:

Date Collected: **03/14/2023**
Date Received: **03/16/2023**
Date Reported: **03/20/2023**

Methodology: **ICP-MS**

PACKED BLOOD CELL ELEMENTS REPORT

INTRODUCTION

This analysis of elements in packed blood cells was performed by ICP-Mass Spectroscopy following acid digestion of the specimen in a closed microwave system. For a given element, these procedures measure the sum of the amounts of surface-adhering and intracellular content, regardless of chemical form. For units of measurement, mg/l is approximately equivalent to ppm, and mcg/l is approximately equivalent to ppb.

The packed cells are not washed, and therefore, a very small amount of residual plasma remains as part of the specimen. Washing would eliminate some important plasma membranebound elements. Because the cells are not washed, the DDI reference range may vary from published ranges for intracellular content of washed erythrocytes. Blood cell specimens that are not adequately centrifuged, per the kit instructions, may yield distorted or invalid results because of excess plasma content.

Packed blood cell analysis is intended to be a diagnostic method of assessing insufficiency or excess of elements that have important functions inside blood cells or on blood cell membranes. Additional testing of whole blood or serum/plasma or other body tissues may be necessary for differential diagnosis of imbalances. Additional testing also may be necessary to assess specific dysfunctions of assimilation, transport, retention, or excretion of elements. Packed blood cell element analysis is additionally intended to determine elevated or excessive levels of five potentially toxic elements that can accumulate in erythrocytes: thallium, arsenic, cadmium, lead, and mercury.

If an element is sufficiently abnormal per the blood cell measurement, a descriptive text is included with the report. For elements with essential or beneficial functions, a text will print if the measured result is below -1.5 standard deviations from the mean of the reference population, or if the result is above +1.5 standard deviations from the mean of the reference population. For potentially toxic elements, a text prints whenever the measured result exceeds the expected range. If no descriptive element texts follow this introductory discussion, then all essential cell elements were measured to be within +1.5 SD, and all measured potentially toxic elements were within expected ranges.

Doctor’s Data states the reference range as +1 SD from the mean of the reference population. This is considered to be the nutritionally and physiologically optimal range for elements with essential or beneficial functions. Physiological imbalance corresponds to levels beyond +1 SD but pathological consequences are not expected until the blood level is beyond +2 SD. Element levels beyond +2 SD may only be temporary nutritional problems or they may reflect a failure of homeostasis to control blood quantities. Pathological consequences depend upon cell and tissue functions which are disrupted by such levels.

COPPER LOW

Copper (Cu) is an important mineral activator of certain enzymes in humans. As the activator of dopamine beta-hydroxylase, Cu is essential for balanced metabolism of adrenal catecholamines. Copper also activates: tyrosinase (phenolase) which catalyzes formation of quinones from phenols and is needed for melanin forma-tion, cytochrome c oxidase which is necessary for cell respiration and energetics, and lysyl oxidase, which assists proper formation of elastin in collagen tissue. The partitioning of copper in blood normally approximates 60% in serum, 40% in cells. In packed red blood cells, over 80% of copper is bound to cytosolic superoxide dismutase (”SOD”) which helps to control oxidant-antioxidant processes.

If erythrocyte SOD has subnormal activity (which can result from subnormal erythrocyte Cu), membrane damage, cell death and inflammatory conditions can result. Subnormal CuZn-SOD is implicated in hereditary and acquired amyotrophic lateral sclerosis. Wilson's disease can also feature low copper in blood cells, while elevated or toxic levels accumulate in the intestinal mucosa, kidney, and liver. Serum ceruloplasmin should be measured to rule out ceruloplasmin deficiency which is an expected finding in Wilson's disease. Subnormal packed cell Cu may or may not correspond to overall copper deficit or deficit in other specific tissues. Conditions associated with various copper deficits include: CNS disorder with myelination deficit, depression and adrenal medullary disorder with deficient norepinephrine; cardiovascular and lung disorders due to defective elastin in connective tissue; skin pigmentation abnormalities and kinky hair (Menkes' disease);anemia; and hypercholesterolemia, especially if zinc excess is coincident with the Cu deficit.

An indicative test for cell SOD status is measurement of erythrocyte SOD activity. Other tests for copper status include measurement of serum copper, serum ceruloplasmin, whole blood copper and zinc, hair copper (barring exogenous contamination), and platelet cytochrome c oxidase activity.

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BORON HIGH

Boron (B) is introduced to the body mainly through food (noncitrus fruits, leafy vegetables, nuts, legumes, wine, cider, beer) and drinking water but is also found in food preservatives (sodium borate), and insecticides (boric acid). Evidence for biological essentiality in animals (including humans) has been presented. It has been proposed that boron contributes to living systems by acting indirectly as a proton donor and that it exerts a particular influence on cell membrane and structure and function. In humans boron is responsible for the hydroxylation

of various substances in the body. It may enhance the production of various hormones such as testosterone, estrogen, DHEA, and 1,25 dihydroxycholecalciferol. Boron is very readily absorbed and excreted in the urine yet its concentration remains quite low in the serum. Based on urinary recovery findings, more than 90% of ingested B is usually absorbed. Boron is distributed throughout the tissues and organs of animals and humans at concentrations mostly between 4.6 and 55.5 nmol (0.05 and 0.6 µg)/g fresh weight. Among the organs that contain the highest amounts of B are bone, spleen, and thyroid. It appears to be most concentrated in the thyroid gland.

Boron has a low order of toxicity even with intakes as high as 40mg/day in some parts of the world. However, high body burden of the element may be harmful, especially to young animals (including human neonates). Reports have shown that when doses equivalent to more than 46 mmol (0.5 g) B daily were consumed, disturbances in appetite, digestion, and health occurred. Acute toxicity signs include nausea, vomiting, diarrhea, dermatitis, and lethargy. High B ingestion also induces riboflavinuria.

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Nielsen, F.H., Hunt, C.D., Mullen, L.M., Hunt, J.R. Effect of dietary boron mineral, estrogen, and testosterone metabolism in postmenopausal women. FASEB 1:394-397, 1987.

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MOLYBDENUM LOW

Molybdenum (Mo) is an essential nutrient that functions as an obligatory cofactor for the iron- and flavin-containing enzymes aldehyde oxidase, xanthine oxidase, and sulfite oxidase. Aldehyde oxidase oxidizes and detoxifies the pyrimadines, purines, and pteridines. Xanthine oxidase/ dehydrogenase catalyzes the formation of uric acid from hypoxanthine and sulfite oxidase catalyzes the transformation of sulfite to sulfate. Insufficient sulfite oxidase activity can result in deranged cystiene metabolism.

Mo is readily absorbed (40 - 80%) and transported as a complex with protein in blood. Blood levels of Mo are regulated primarily by urinary excretion. Recent surveys indicate that many diets do not provide the recommended safe and adequate intake of 50 - 350 mcg Mo/day. (1) Good sources of Mo include milk products, whole grains, dried legumes, and organ meats.

Symptoms of overt Mo deficiency have only been described for a patient on long-term total parenteral nutrition. However, prolonged exposure to tungsten (T.I.G. welding) or dietary sulfates, aldehydes, and large amounts of purines diet might possibly result in an acquired Mo deficiency. A possible link between Mo deficiency and increased risk for esophageal cancer has been reported. (2)

Mo deficiency would be expected to be associated with abnormally low levels of uric acid in blood and sulfate in urine.

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ARSENIC HIGH

Blood cell arsenic (As) exceeds the expected level for this individual. Usually, arsenic clears the blood rapidly after a point-in-time exposure. The finding of elevated blood cell As suggests: (1) recent exposure, (2) chronic or on-going exposure, (3) decreased metabolic capacity to clear As. Arsenic has two oxidation states or valences, As+3 and As+5. As+3 is more reactive and toxic. Both forms of As accumulate primarily in skin and skeletal tissue; also in liver, kidney and spleen. Over one-half of ingested or absorbed As is normally excreted via urine and feces in 2 to 8 days.

In blood cells, As binds primarily to globulin, but generally As seeks out thiols and sulfhydryl binding sites. The vitamin cofactor, lipoic acid, is particularly affected, and this may be the reason for inhibition of alpha-ketoacid oxidation. Much of the enzymatic inhibition caused by As occurs in cells with mitochondrial structures (not erythrocytes). Arsine gas, AsH3, does react rapidly with erythrocytes, combining with hemoglobin and causing hemolysis, hemoglobinuria and hematuria.

An important detoxication pathway for As involves methylation with methyl groups donated by S-adenosylmethionine; methylated arsenic can produce a garlic-like breath odor.

Early symptoms of arsenic excess include: fatigue, malaise, eczema or allergic-like dermatitis, and increased salivation. Increased body burden of arsenic can lead to further manifestations: skin hypopigmentation, white striae on fingernails, hair loss, stomatitis, peripheral neuropathy, myocardial damage, hemolysis, and anemia (aplastic with leukopenia).

Sources of arsenic include: contaminated foods (especially seafood), water or medications. Industrial sources are: ore smelting/refining/processing plants, galvanizing, etching and plating processes. Tailing from or river bottoms near gold mining areas (past or present) may contain arsenic. Insecticides, rodenticides and fungicides (Na-,K-arsenites, arsenates, also oxides are commercially available). Commercial arsenic products include: sodium arsenite, calcium arsenate, lead arsenate and ”Paris green” which is cupric acetoarsenite, a wood preservative. Elevated blood As of undetermined source is reported in hemodialysis patients.

Hair element analysis can be done for corroborative evidence of arsenic excess. Blood arsenic levels are not dose-related and may not accurately reflect As body burden. Urine analysis following provocation with D-penicillamine or DMSA can corroborate excess, but sequestered As may not show in early trials.

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MERCURY HIGH

Packed cell mercury (Hg) is measured to exceed the expected range. In whole blood, mercury eventually partitions between plasma and cells in various proportions depending upon its chemical form. For inorganic Hg (salts), the RBC/plasma ratio is about 1.0 or less which means that inorganic Hg is equally distributed, or there is somewhat more inorganic Hg in the plasma. For elemental Hg the RBC/plasma ratio is about 2. For organic mercury, the RBC/plasma ratio exceeds 10, which means that at least 90% of organic mercury, such as methylmercury, accumulates in the erythrocytes. However, blood cells may not be indicative of past exposures if the Hg has cleared the blood and deposited in other tissues. This can take up to 2 months after a point-in-time exposure to organic mercury.

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, Cd, organic xenobiotics), presence of disease and status of immune function, and the availability 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: 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, explosive detonators, in elemental or liquid form for thermometers, barometers, and laboratory equipment; batteries and electrodes ("calomel"); 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 occurs in aquatic biota in 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.

Corroborating diagnostic tests for assessment of mercury burden are hair element analysis (for past or chronic exposures),and urine analysis following administration of sulfhydryl agents(DMPS, DMSA, D-penicillamine).

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