RED BLOOD CELL ELEMENTS

LAB#: B000000-0000-0
PATIENT: Sample Patient
SEX: Male
AGE: 5

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

NUTRIENT ELEMENTS

ELEMENTS	RESULT µg/g	REFERENCE RANGE	PERCENTILE 2.5th 16th 50th 84th 97.5th
Calcium	24	8- 31	
Magnesium	41	36- 64	
Potassium mEq/g	67	65- 95	
Phosphorus	577	480- 745	
Copper	0.78	0.52- 0.89	
Zinc	8.3	8- 14.5	
Iron	780	745- 1050	
Manganese	0.011	0.007- 0.030	
Chromium	0.0010	0.0003-0.0060	
Selenium	0.27	0.19- 0.38	
Boron	0.016	0.01- 0.110	
Vanadium	0.0002	0.0001-0.0020	
Molybdenum	0.0007	0.0005-0.0020	

POTENTIALLY TOXIC ELEMENTS

TOXIC ELEMENTS	RESULT µg/g	REFERENCE RANGE	PERCENTILE 95th 99th
Arsenic	0.003	< 0.010	
Cadmium	< 0.0008	< 0.005	
Lead	0.011	< 0.090	
Mercury	< 0.001	< 0.010	
Thallium	< 0.0001	< 0.0005	

SPECIMEN DATA

Comments:
Date Collected: **10/12/2006**
Date Received: **10/13/2006**
Date Completed: **10/14/2006**

Methodology: **ICP-MS**
µg/g = ppm

V10.01

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 membrane-bound 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: antimony, 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.

POTASSIUM LOW

As an electrolyte mineral, potassium (K) is the principal cation in intracellular fluid, and, at lower concentrations, it also is a component of extracellular fluid. It influences the excitability of muscle cells, particularly cardiac muscle. Potassium functions intracellularly (as sodium does

extracellularly) to maintain osmotic pressure and acid-base equilibrium. Some enzymes utilize the K+ ion as a promoter of catalytic activity; the glycolytic enzyme pyruvate kinase is such an enzyme.

Of various blood fractions, erythrocyte K is most indicative of status or function, but there usually is rapid correspondence between serum and erythrocyte levels. An exception is acidosis or alkalosis which shift K out of or into cells from serum. Occasionally, serum potassium may remain normal during conditions of moderate overall potassium insufficiency. Serum K normally ranges between 3.5 to 5.5 mEq/l while erythrocyte K ranges from 80 to over100 Meq/l.

Causative factors for and conditions consistent with subnormal potassium are :

. Excessive perspiration usually associated with extended physical exertion;
. Excessive use of diuretics or laxatives;
. Vomiting or diarrhea;
. Adrenocortical excess, hyperaldosteronism, Cushing's syndrome;
. Bartter's syndrome (producing hypokalemic alkalosis);
. Chronic alcoholism;
. Folic acid deficiency;
. Unbalanced hyperalimentation or parenteral procedures that provide insufficient K;
. Gastrointestinal fistula.

In general, acidemia (low blood pH) shifts K out of erythrocytes (and other cells) and into serum; metabolic and organic acidoses have more pronounced effects on lowering cell K than does respiratory acidosis.

Expected symptoms of subnormal potassium include: muscle weakness, fatigue, abnormal blood pressure, tachycardia, and symptoms consistent with the above-listed conditions.

Diagnostic tests for further characterization of K status include: serum K measurement, tests of endocrine function and steroid levels, urine element analysis, blood folic acid analysis, blood and urine pH determination, and review of the individual's diet.

BIBLIOGRAPHY FOR BLOOD CELL POTASSIUM, LOW

1. Jacobs D.S. et al Laboratory Test Handbook, Williams & Wilkins, Baltimore MD, 1990 pp 302-04.

2. Oh, M.S., Chapt. 6 in Modern Nutrition in Health and Disease 8th ed, Lea & Febiger, Philadelphia PA, 1994 pp 127-28, 131-34.

3. Harper H.A. et al Review of Physiological Chemistry, 17th ed, Lange Med Pub, Los Altos CA, 1979 pp 581-83.

4. Levinsky N.G. "Potassium" in Harrison's Principles of Internal Medicine, 11th ed, McGraw-Hill, New York NY, 1987 pp 204-08.

ZINC LOW

Zinc (Zn) is an activator or cofactor for many enzymatic steps in human metabolism. Many digestive peptidase enzymes contain Zn; an important enzyme controlling chemical energy conversion (lactate dehydrogenase) requires Zn, as does alcohol dehydrogenase. A form of the oxidant-response mediating enzyme, superoxide dismutase ("SOD"), is activated by zinc and copper. Absorption of Zn occurs mainly in the small intestine, and Zn uptake can be competitive with that of iron. Zinc is distributed throughout body tissue; about one-fifth of total body stores of Zn are in skin. Plasma or serum Zn concentration normally varies from about 0.6 to 1.3 mg/dl; RBC Zn normally varies from 0.9 to 1.6 mg/dl.

Zinc inside erythrocytes is bound to Cu,Zn-SOD, carbonic anhydrase, and other proteins. Individuals who have Zn deficiency have low erythrocyte carbonic anhydrase activity (Martin D.W. et al, Harper's Review of Biochemistry, 20th ed, Lange Med. Publ.,1984 p. 659). The role of low Zn and erythrocyte SOD activity in inflammatory diseases, e.g. arthritis, has been studied with inconsistent and controversial conclusions. Hemolytic anemias, especially thalassemia, can feature deficient Zn in erythrocytes.

Conditions associated with zinc deficiency include: incomplete digestive proteolysis and malabsorption, chronic diarrhea, overuse of diuretics, alcoholism, hepatic cirrhosis, renal tubular disease and nephrotic syndrome, diabetes mellitus. Excess dietary phosphates, phytates, fiber, calcium and copper can impair uptake of Zn. Excess copper levels also can interfere with zinc retention bycompetition for albumin binding sites in blood serum. Zinc binds to cysteine and histidine. Cystinuria or histidinuria enhances urinary zinc excretion; usually lowering serum zinc. Packed cell Zn may or may not be affected. Therapeutic detoxification procedures, e.g. EDTA chelation and D-penicillamine therapy, deplete body stores of Zn.

Conditions seen in Zn deficiency are: altered taste, impaired dark adaptation by the eyes, partial (usually) alopecia, poor wound healing, sexual impotency, acral dermatitis, delayed growth in children, dwarfism, and immune dysfunction with impaired T-lymphocyte activity. Elevated lactic acid in blood (lactic acidosis) may occur in Zn deficiency.

Other laboratory tests that may be diagnostic for suspected Zn deficiency are: serum Zn measurement, periodic urine element analysis during detoxification therapy, hair element analysis (low zinc corroborates deficiency, high zinc usually indicates maldistribution and zinc dysfunction), serum lactic acid (mildly elevated in Zn deficiency), erythrocyte SOD activity determination (subnormal in either Zn or Cu deficiency), and erythrocyte carbonic anhydrase activity (subnormal in Zn deficiency).

BIBLIOGRAPHY FOR BLOOD CELL ZINC, LOW

1. Falchuk K.H., Chapt 28 in Harrison's Principles of Internal Medicine, 13th ed, McGraw-Hill, New York, NY, 1994 pp 481-82.

2. Zinc in Human Medicine, Proceedings of a Symposium on the Role of Zinc in Health and Disease, Inst. Child Health (London), TIL Pub Ltd, Toronto, Canada, 1984.

3. Cunnane S.C. Zinc: Clinical and Biochemical Significance, CRC Press, Boca Raton FL, 1988.

4. Prasad A.S. Ed, Clinical, Biochemical and Nutritional Aspects of Trace Elements, Alan Liss, New York, NY, 1988.

5. Dogru U. et al ”Zinc Levels of Plasma, Erythrocyte, Hair and Urine in Homozygote Beta-Thalassemia” Acta. Haematology 62, 1979 pp41- 44.

6. Dore-Duffy P. et al, ”Zinc Profiles in Rheumatoid Arthritis” Clin. Exp. Rheumatology 8,1990 pp 541-46.

7. Milanino R. et al ”Copper and Zinc Status in Rheumatoid Arthritis ...” Clin. Exp. Rheumatology 11, 1993 pp 271-83.

IRON LOW

Nominally, about 97% of erythrocyte iron (Fe) is ferrous iron bound as heme in hemoglobin; only about 3% is nonheme iron. Thus, the packed blood cell Fe measurement is essentially a measurement of the heme iron content of erythrocytes. The fraction of whole blood volume that constitutes erythrocytes, or the packed cell volume (the ”hematocrit”), does not directly influence the packed cell iron result.

Diagnostic testing to assess whether there is anemia requires measurement of red blood cell structure and quantity relative to the whole blood volume. The iron content of erythrocytes would then indicate if the condition is hypochromic (low heme-iron), normochromic (normal heme-iron) or hyperchromic (high heme-iron).

A low packed cell Fe result does not necessarily mean anemia, and diagnostic hematology procedures are suggested when this result is found. Possible reasons for low iron or low hemoglobin in erythrocytes are those of iron deficiency, but not necessarily those of low RBCs or low hematocrit. Sickle cell anemia, thalassemia and disorders of hemoglobin metabolism can feature low packed cell iron.

The suggested tests to assess iron assimilation are those for: serum iron level, serum ferritin, total iron binding capacity, percent saturation of transferr in, investigations of blood loss, dietary iron intake, dietary interferences (phosphates, phytates, oxalates, excess coffee or tea), GI function (especially sufficiency of gastric function), and whole blood or serum copper level.

BIBLIOGRAPHY ON BLOOD CELL IRON, LOW

1. Martin D.W. et al, Harper’s Review of Biochemistry, 20th ed, Lange Med. Pub., Los Altos CA, 1985, pp 41-51.

2. Jacobs D.S. et al, Laboratory Test Handbook, 2nd ed, Williams & Wilkins, Baltimore MD, 1970, pp 188-89; 233-36.

3. Fairbanks V.F. ”Iron in Medicine and Nutrition”, chapt 9 in Modern Nutrition in Health and Disease, 8th ed, Lea & Febiger, Philadelphia PA, 1994, pp 191-202.

4. Harris E.D. ”Iron-Copper Interactions: Some New Revelations” Nutr. Reviews 52(9), 1994, pp

311-19.

5. Bunn H.F. "Disorders of Hemoglobin", Chapt 306 in Harrison's Principles of Internal Medicine, 13th ed, McGraw-Hill, New York, NY1994, pp 1734-43.

BORON LOW

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). Although there is an absolute requirement for B in vascular plants, evidence for biological essentiality in animals (including man) is limited. 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. Boron is rapidly absorbed and excreted largely in the urine. 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.

Boron influences macromineral metabolism and steroid hormone metabolism (testosterone, estrogen, DHEA, and 1,25 dihydroxycholecalciferol). A B deficient diet may also affect calcium metabolism and thus affects the composition, structure, and strength of bone. Signs of B deficiency in animals vary in nature and severity as the diet varies in its content of calcium, phosphorus, magnesium, potassium, cholecalciferol, aluminum, and methionine. Boron is also thought to have an estrogenic effect. In post-menopausal women consuming a very low B diet, B supplementation reduced the total plasma concentration of calcium and the urinary excretions of calcium and magnesium, and elevated the serum concentrations of 17á-estradiol, testosterone, and ionized calcium, mimicking the effects of estrogen ingestion in postmenopausal women. In another study of magnesium and B deprivation among 13 healthy postmenopausal women (aged 50-78 years), it was found that marginal magnesium and B deprivation may also affect brain function as measured by EEG (Penland JG, Magnes Res. 8(4), 1995 pp 341-58). It seems there may be increased CNS activity following boron deprivation. In long term hemodialysis patients serum boron levels may be excessively decreased (Usuda K, Sci Total Environ. 191(3), 1996 pp 283-90).

No B requirements have been set as of 1998. Estimates are that between 1-2 mg/day may be required. Average intake in the U.S. has been estimated at between 1.7-4.3 mg/day.

BIBLIOGRAPHY FOR BORON, LOW

Barr, R.D., Barton, S.A., Schull, W.J. Boron levels in man: preliminary evidence of genetic regulation and some implications for human biology. Med Hypotheses 46(3):286-9, 1996.

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

Shils, M.E., Olson, J.A., Shike, M.: Modern nutrition in health and disease. Philadelphia, Lea and Febiger, 1994.