LAB #: B000000-0000-0
PATIENT: Sample Patient
ID: PATIENT-S-00000
SEX: Female
DOB: AGE: 46

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

Toxic & Essential Elements; Whole Blood

ESSENTIAL AND OTHER ELEMENTS		RESULT / UNIT	REFERENCE INTERVAL	PERCENTILE (2.5th, 16th, 50th, 84th, 97.5th)
Calcium	(Ca)	**6.0 mg/dL**	4.8- 7.1	
Magnesium	(Mg)	**3.2 mg/dL**	3- 4.2	
Copper	(Cu)	**90 µg/dL**	65- 130	
Zinc	(Zn)	**599 µg/dL**	480- 780	
Manganese	(Mn)	**7 µg/L**	4- 22	
Chromium	(Cr)	**0.20 µg/L**	0.2- 0.80	
Lithium	(Li)	**0.3 µg/L**	0.4- 20	
Selenium	(Se)	**192 µg/L**	140- 350	
Strontium	(Sr)	**43 µg/L**	10- 45	
Molybdenum	(Mo)	**0.8 µg/L**	0.3- 2.5	
Vanadium	(V)	**< 0.04 µg/L**	0.04- 0.30	

TOXIC METALS		RESULT / UNIT	REFERENCE INTERVAL	PERCENTILE (95th, 99th)
Arsenic	(As)	**2.9 µg/L**	< 9.0	
Barium	(Ba)	**11.8 µg/L**	< 4.0	
Cadmium	(Cd)	**0.2 µg/L**	< 1.0	
Cobalt	(Co)	**0.1 µg/L**	< 0.8	
Lead	(Pb)	**0.5 µg/dL**	< 3.0	
Mercury	(Hg)	**5.0 µg/L**	< 4.5	
Nickel	(Ni)	**< 1.5 µg/L**	< 3.0	
Platinum	(Pt)	**< 0.05 µg/L**	< 0.10	
Thallium	(Tl)	**0.07 µg/L**	< 0.50	
Tungsten	(W)	**< 0.03 µg/L**	< 0.10	
Uranium	(U)	**< 0.02 µg/L**	< 0.10	

SPECIMEN DATA

Comments:

Date Collected: **04/04/2017**
Date Received: **04/05/2017**
Date Reported: **04/07/2017**

Time Collected: **10:03 AM**
Fasting: **Yes**

Methodology: **ICP-MS**

Blood lead levels in the range of 5-9 µg/dL have been associated with adverse health effects in children aged 6 years and younger.

WHOLE BLOOD ELEMENT REPORT

INTRODUCTION

This analysis of elements in whole blood was performed by ICP Mass Spectroscopy following specimen digestion with nitric acid in a closed container microwave oven system. This procedure measures the total concentration of an element in whole blood, regardless of biochemical form and regardless of partitioning of the element in blood fractions. For units of measurement, mg/L is approximately equivalent to ppm, and mcg/L is approximately equivalent to ppb.

Whole blood element analysis is intended to be a diagnostic method that assists in determining imbalance, insufficiency, or excess of certain elements that have essential or beneficial functions. Additional testing of blood fractions 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. Whole blood element analysis is additionally intended to determine elevated or excessive levels of eleven potentially toxic elements.

If an element is sufficiently abnormal per the whole blood 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.

Doctor's Data states the reference range as + 1SD 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 + 1SD but pathological consequences are not expected until the blood level is beyond + 2SD. Element levels beyond + 2SD 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.

Chromium Low

The level of chromium is lower than expected in this sample. Whole blood chromium represents the levels of trivalent chromium (Cr III) in plasma and hexavalent chromium (Cr VI) in the red blood cells. Recent evidence indicates that red blood cell chromium levels primarily reflect exposure to toxic hexavalent chromium (Cr VI), as the nutrient chromium III is not directly taken up appreciably by erythrocytes. RBC Cr VI levels directly correlated with Cr VI exposures (in vitro, Devoy et al. 2016). The low level of chromium in this patient sample indicates minimal exposure to toxic chromium VI (Cr VI) over the last 60-120 days, and / or low CrIII intake /absorption.

Chromium is an essential micronutrient. Low Cr III levels may exacerbate conditions such as glucose intolerance, type II diabetes, cardiovascular disease or gestational diabetes. Cr III is primarily absorbed by the jejunum of the small intestine.

Cr III is bound by the protein chromodulin, and the Cr-chromodulin complex binds with cellular insulin receptors to amplify insulin signaling. Less than 3% of dietary chromium is absorbed in the gut. However,

vitamin C, niacin, NSAIDs, aspirin, beta-blockers, corticosteroids, or insulin may increase absorption (animal studies). If Cr III supplementation is considered, one should consider that metabolism of chromium picolinate to free Cr III ions may generate oxidative stress. The Institute of Medicine currently recommends a daily intake level of 20û45 µg Cr III for adolescents and adults.

Consider Evaluation of type II diabetes risk (Metabolomic Profile, serum)

Resources:

Agency for Toxic Substances & Disease Registry (2015) Toxicological Profile for Chromium. https://www.atsdr.cdc.gov/toxprofiles/tp.asp(c)id=62&tid=17 Accessed 23 February 2017

Linus Pauling Institute (2014) Chromium. http://lpi.oregonstate.edu/mic/minerals/chromium Accessed 10 February 2017.

National Institutes of Health (2013) Chromium Dietary Supplement Fact Sheet. https://ods.od.nih.gov/factsheets/Chromium-HealthProfessional/ Accessed 10 February 2017.

O'Flaherty, Ellen J.; Kerger, Brent D.; Hays, Sean M.; Paustenbach, Dennis J. (2001) A Physiologically Based Model for the Ingestion of Chromium(III) and Chromium(VI) by Humans. Toxicological Sciences vol. 60 (2) p. 196-213.

Vincent, John B. (2000) The Biochemistry of Chromium. J. Nutr. vol. 130 (4) p. 715-718.

Vincent, John B. (2013) Chromium and Glucose Tolerance Factor. Encyclopedia of Metalloproteins pp 603-607. Springer New York.

LITHIUM LOW

The concentration of lithium (Li) in this blood specimen is lower than expected. Li occurs almost universally in water and in the diet, and Li has essential functions in the body.

Intracellularly, Li inhibits the conversion of phosphorylated inositol to free inositol. In the nervous system this moderates neuronal excitability. Li also influences monamine neurotransmitter concentrations at the synapse (this function is increased when Li is used therapeutically for mania or bipolar illness). Recent studies suggest that long-term, low dose Li supplementation is neuroprotective and may help preserve integrity of the central nervous system with aging.

Bibliography for Lithium, low

1. Williams RSB , Harwood AJ. " Lithium therapy and signal transduction". Trends in Pharmacol. Sci. (2000)21:61-64.

2. Moore Gj et al. "Lithium-induced increase in human grey matter". Lancet (2000)356:1241-1242.

3. Chuang DM. ”Lithium exerts robust neuroprotective effects in vitro and in the CNS in vivo : Therapeutic implications”. Neuropsychopharmacol. (2000)23:S39.

4. Chuang DM et al. ”Beta-amyloid peptide-induced death of PC 12 cells and cerebellar granule neurons is inhibited by long-term lithium treatment”. Eur. J. Pharmacol. (2000)392:117-23.

STRONTIUM HIGH

The concentration of strontium (Sr) is higher than expected in this blood specimen. The element Sr occurs in nature as a mixture of four stable (non-radioactive) isotopes: Sr 84 (0.5%), Sr 86 (9.9%), Sr 87 (7.1%) and Sr 88 (82.5%). The Sr value reported in this test pertains specifically to the concentration of Sr 88. Strontium has a bad reputation because of its radioactive isotopes: Sr 89 (52 day half-life), Sr 90 (28 year half-life), Sr 91 (10 hour half-life), etc. These radioactive isotopes arise from nuclear fission of uranium and plutonium. In past years, atmospheric testing of nuclear devices resulted in contamination of grazing lands and the food chain with radioactive strontium. It is emphasized that the Sr level reported here does not reflect exposure to radioactive Sr.

Sr is chemically similar to calcium and is assimilated by plants and animals along with calcium. Studies with chicks show that vitamin D controls Sr assimilation just as it does for calcium. It is very probable that Sr assists in bone and calciferous tissue formation in humans, and clinical studies have been reported about treatment of osteoporosis with low doses of stable strontium. Controlled studies with animals have shown stimulation of bone formation with low levels of Sr in drinking water.

Elevated strontium in whole blood may reflect problems with calcium assimilation, parathyroid function, or vitamin D activity. Calcium and parathyroid hormone levels and vitamin D activity should be checked to directly assess such problems. Blood Sr levels may rise in osteoporosis (due to bone loss); calcium levels may also rise but may not be as pronounced.

A common reason for elevated whole blood Sr (with normal Ca, parathyroid, and vitamin D) is a high concentration of Sr in drinking water. This varies geographically; two areas having high Sr water are northwest Florida and southeast Wisconsin. Other environmental and occupational sources of Sr are: fireworks and road flares, a dental desensitizer ”Elecol”, diagnostic barium preparations used as radiopaque ”cocktails”, depilatories and luminous paints. Stable strontium has very low toxicity as an element, although there are toxic or corrosive Sr compounds such as Sr(OH)2.

BIBLIOGRAPHY FOR STRONTIUM, HIGH

1. Marie P.J. et al ”Histomorphometry of Bone Changes in Stable Strontium Therapy” in Trace Substances in Environmental Health XIX, Proceedings of the U. of Missouri 19th Annual Conference on Trace Substances in Environmental Health, ed. by D.D. Hemphill, U. of Missouri, Columbia MO, June 1985.

2. Blumsohn A. et al. ”Stable Strontium Absorption as a Measure of Intestinal Calcium Absorption...” Clin. Sci (Colch), 87 no.3, Sept.1994, pp 363-68.

3. Pielte M. et al. ”Determination of Strontium in Human Whole Blood by ICP-AES” Sci. Total Environ. 141 (1-3), Jan. 1994, pp 269-73.

4. El Solh N. and F. Rousselet ”Effects of Stable Strontium on Calcium Metabolism with Reference to Low Calcium Diet”, in Handbook of Stable Strontium, Plenum Pres, New York NY, 1981, pp 515-44.

5. Carson B.L. et al. Toxicology and Biological Monitoring of Metals in Humans, Lewis Pub. Chelsea MI, 1986, pp 231-33.

6. Lederer, C.M. Table of Isotopes, John Wiley & Sons, New York NY,1967.

VANADIUM LOW

The level of vanadium (V) is lower than expected in this patient. Vanadium is not currently considered an essential nutrient cofactor for any human enzyme, although it is essential for some rodents (rats). Vanadium may improve insulin responses and blood sugar control; Wang et al. (2014) found an inverse correlation in new type II Chinese diabetics and plasma V levels. The subjects with the lowest levels of V had the highest risk of diabetes. Accumulating evidence suggests that the presence or absence of V may alter immune responses, particularly humoral immunity (B cell responses). A study of i ndustrial workers indicates that low levels of V may be associated with increased risk for atherosclerosis (higher cholesterol, lower HDL-C, poor apoB / apoA1 ratios); this association was strongest in males.

Very little V is found in red blood cells. The oxidation state of V may determine whether it is found in the cell or the plasma. Vanadium (IV) diffuses through cell membranes, while V enters cells via anionic channels. Intracellular V is found primarily in the kidneys, spleen, bones and liver, and is typically found as vanadium (IV) or V. In the plasma, V is primarily bound to transferrin, albumin or serum lipids. Normal serum concentrations in unexposed human populations average less than 1û2 µg V/L.

Consider:
-Evaluate digestion and absorption (Comprehensive Stool Analysis)
-Evaluate diet. Food sources of V include: mushrooms, shellfish, black pepper, parsley, dill weed, grain and grain products.

Resources:

Agency for Toxic Substances & Disease Registry (2015) Toxicological Profile for Vanadium. https://www.atsdr.cdc.gov/toxprofiles/tp.asp(c)id=276&tid=50 Accessed 23 February 2017

Boyle, Karleen (1996) Evaluating Particulate Emissions from Jet Engines: Analysis of Chemical and

Physical Characteristics and Potential Impacts on Coastal Environments and Human Health. Transportation Research Record 1517 University of California, Los Angeles, 405 Hilgard Avenue, Los Angeles, Calif. 90024-1606.

Korbecki, Jan; Baranowska-Bosiacka, Irena; Gutowska, Izabela and Chlubek, Dariusz (2012) Biochemical and medical importance of vanadium compounds. Acta Biochimica Polonica Vol. 59, No 2/2012 195û200

National Institute of Environmental Health Sciences (2008) Chemical Information Review Document for Oral Exposure to Tetravalent and Pentavalent Vanadium Compounds. https://ntp.niehs.nih.gov/ntp/htdocs/chem_background/exsumpdf/niehs_vanadium_compounds_508.pdf Accessed 22 February 2017

Seargeant L, Stinson R (1979) Inhibition of human alkaline phosphatases by vanadate. The Biochemical Journal 1979 vol: 181 (1) pp: 247-50

Shi Z, Liu H, Yang X (2011) Vanadium stimulates mitochondrial ROS production in different ways. Journal of Chinese Pharmaceutical Sciences 2011 vol: 20 (5)

Tsave O, Petanidis S, Kioseoglou E, Yavropoulou M, Yovos J, et. al. (2016) Role of Vanadium in Cellular and Molecular Immunology: Association with Immune-Related Inflammation and Pharmacotoxicology Mechanisms. Oxidative Medicine and Cellular Longevity 2016 vol: 2016 pp: 1-10

BARIUM HIGH

An elevated level of barium (Ba)was detected in this blood specimen. The half-life of Ba in the blood is less than 1 day, so elevated results indicate an acute or ongoing exposure. A slight trend toward increased serum calcium has been noted with Ba exposure, but may not be clinically significant.

The most significant route of exposure to barium is through drinking water and food. Barium sulfite is commonly used as a radiocontrast agent. However, it is very poorly absorbed from the gastrointestinal tract. Other Ba compounds (i.e. barium carbonate, barium chloride) are more likely to cause symptoms of toxicity, including gastroenteritis, hypokalemia, hypertension, cardiac arrhythmias, and flaccid paralysis.

MERCURY HIGH

The concentration of mercury (Hg) is abnormally high in this blood specimen. Elevated blood Hg indicates higher than average exposure to the metal, but does not provide information about net bodily retention of Hg. Whole blood constitutes both organic (RBC) and inorganic (serum) Hg. However, blood Hg is not indicative of past exposures if the Hg has cleared the blood and deposited in other tissues. The biological half-lives of inorganic and organic Hg in blood are about 3 days and 60 days, respectively. Blood Hg levels are typically higher than average in ”high-end” fish consumers.

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 and Cd have such effects), presence of disease that depletes or inactivates lymphocytes or is immunosuppressive, organ levels of xenobiotic chemicals and sulfhydryl-bearing metabolites (e.g. glutathione), and the concentration of protective nutrients, (e.g. zinc, selenium, vitamin E). Early signs of excessive Hg exposure 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 and retention include: anorexia, numbness and paresthesias, headaches, hypertension, irritability and excitability, and immune suppression, possibly immune dysregulation. Advanced disease processes from Hg 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; and in fungicides and pesticides. The use of Hg as a funcicial/pesticide (including that in paints) has declined somewhat due to environmental concerns, but mercury residues persist from past use. Methylmercury, the common, most neurotoxic form, results from methylation of hg in aquatic biota or sediments, 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 intake of dietary mercury is negligible unless food is contaminated with one of the previously listed forms/sources.

Measurement of fecal Hg provides an indication of exposure to inorganic mercury from dental amalgams. Net retention of Hg in the body can be assessed by comparison of pre- and post-provocation urinalysis using DMPS, DMSA or D-penicillamine.

BIBLIOGRAPHY FOR MERCURY

1. Suzuki T. et al eds, Advances in Mercury Toxicology, Plenum Press, New York, NY, 1991.

2. World Health Organization: ”Methylmercury”, Environ. Health Criteria 101 (1990); ”Inorganic Mercury”, Environ. Health Criteria118 (1991), WHO, Geneva, Switzerland.

3. Tsalev D.L. and Z.K. Zaprianov, Atomic Absorption Spectrometryin Occupational and Environmental Health Practice, CRC Press, Boca Raton, FL, 1983, pp 158-69.

4. Birke G. et al ”Studies on Humans Exposed to Methyl Mercury Through Fish Consumption”, Arch Environ Health 25, 1972, pp 77-91.

5. Ishihara N. et al ”Inorganic and Organic Mercury in Blood, Urineand Hair in Low Level Mercury Vapor Exposure” Int. Arch. Occup. Environ. Health 40, 1978, pp 249-53.

6. Werbach M.R. Nutritional Influences on Illness, 2nd ed, Third Line Press, Tarzana CA, 1993, pp 349, 647, 679.